Full Paper

Successful Treatment of a Canada Goose (*Branta Canadensis)* with Presumed Capture Myopathy

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Abstract: An adult Canada goose *(Branta canadensis)* was contaminated by a release of non-polychlorinated biphenyl (PCB) dielectric oil and was one of 47 birds captured during the wildlife response to the incident. The bird was examined and treated in accordance with established protocols for oiled wildlife rehabilitation. The animal met medical criteria for cleaning, and it was cleaned one day post-capture. Immediately after cleaning it began to exhibit symptoms of capture myopathy. The bird was aggressively treated with intravenous fluids, vitamin E, methocarbamol, gavage nutrition and physical therapy. Significant progressive improvement occurred over the next five days, and seven days post-capture the bird showed no signs of exercise intolerance. The bird was released after conditioning and veterinary examination fourteen days post-capture. The case suggests that aggressive treatment at the first signs of capture myopathy can result in full recovery in wild-caught Canada geese.

Introduction

Capture myopathy (exertional myopathy) presents a risk during any activity that involves capture and/or restraint of wildlife, including oiled wildlife response. Initial capture and cleaning both have the potential to induce capture myopathy in susceptible animals. The condition has been extensively documented in ungulates and birds, and it has also been reported in cetaceans, pinnipeds, ursids and other vertebrates. (Cattet et al 2008, Hartup et al 1999, Herráez et al 2007, Williams and Thorne 1996, Wobeser 1987, Wobeser 1981) Development of capture myopathy is presumed to be related to hyperthermia and increased stress hormones during capture and restraint, leading to a buildup of lactate in the muscle. (Williams and Thorne 1996, Carpenter et al 1991) Clinical signs may vary between species, but often include depression, ataxia, muscle stiffness and recumbency. (Williams and Thorne 1996, Spraker 1993) Clinical pathology may include elevated creatinine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST). (Williams and Thorne 1996, Abbott et al 2005, Dabbert and Powell 1993) Onset of symptoms may begin several hours to several days after capture. Clinical signs may progress for several weeks, often leading to death. (Williams and Thorne 1996, Spraker 1993)

Several treatments for animals with symptoms of capture myopathy have been described, with varying outcomes. (Hartup et al 1999, Carpenter et al 1991, Businga et al 2007, Smith et al 2005) Successful treatment of individuals from several avian orders have been

detailed, but successful treatment of Anseriformes (waterfowl) exhibiting symptoms of capture myopathy has not yet been reported.

Clinical Report

On June 19, 2008 several thousand gallons of non-PCB dielectric oil were released from an underground pipe into the Muddy River in Brookline, MA. Forty-seven contaminated Canada geese (*Branta canadensis*) and mallards (*Anas platyrhyncos*) were captured with hand nets or sheets, occasionally in conjunction with baiting. Each bird was transported individually in a well-ventilated box which was driven to the temporary oiled wildlife rehabilitation center in a cool, quiet vehicle. All animals captured for rehabilitation were treated in accordance with established protocols for oiled wildlife response. (Berg 2003)

On intake all birds were examined by a veterinary team; the physical examination included weight, basic blood values (packed cell volume [PCV], total solids [TS], buffy coat [BC]) and evaluation of other parameters (e.g. neurologic function, musculoskeletal abnormalities). [Table 1] Initial treatment included approximately 2-3% of body weight by volume of intravenous 2.5% dextrose in lactated Ringer's solution (LRS), oral Pedialyte® (Abbot Laboratories, Abbot Park, IL) and oral Pepto-bismol™ (Proctor & Gamble, Cincinnati, OH).

Maintenance before cleaning included housing in 2.4 m x 4.8 m (8 ft x 16 ft)pens, free access to drinking water and gavage feeding of vanilla Ensure® (Abbott Laboratories, Abbott Park, IL) three times daily. The first birds were cleaned on June 24, and cleaning continued through June 27, until each bird had been washed one time. Oil was removed from the feathers by washing in multiple tubs of 1-3% Dawn® (Proctor & Gamble, Cincinnati, OH) detergent in 40ºC (104ºF) water; soap was removed in a meticulous rinse process. Following the cleaning process, the birds were housed in a covered 2.4 m x 2.4 m (8 ft x 8 ft) pen with pet dryers and free access to water. Each bird was gavaged with oral Pedialyte® one hour after it had completed the cleaning process.

Cleaned birds were housed in 2.4 m x 7.3 m (8 ft x 24 ft)pens with free access to shallow pools, food and drinking water. Initial pre-release examinations took place on June 30. Release criteria include complete waterproofing, normal blood values (PCV, TS, BC) and appropriate weight. Forty-five birds were released to their capture locations on July 1, 3 and 7, 2008.

Canada goose 038 was captured on June 23 with its family group, consisting of a second adult and two downy goslings; the family group was housed together throughout rehabilitation. The bird was cleaned on June 24 in 22 minutes, below the average cleaning time for an adult Canada goose during this response (avg = 33min, n = 32). Following the cleaning the bird was noted to be lethargic and not standing; it was gavaged orally with Pedialyte® (per standard procedure) and also treated with 200 IU oral vitamin E one hour post cleaning and 60 ml LRS intravenously three hours later. Post-cleaning blood values for goose 038 were PCV = 50%, TS = 3.6g/dL.

On June 25 the bird was not standing. It was given a thorough physical examination, and blood was drawn for lead analysis (LeadCare Analyser, ESA Biosciences, Chelmsford, MA). Physical examination revealed an elevated body temperature (41.3ºC [106.3ºF]), but did not reveal spinal trauma or other musculoskeletal changes, and blood lead level was within normal range for the species (15.8 µg/dl; normal= <40 µg/dl)[2]. The bird had an elevated body temperature at this time (41.3ºC [106.3ºF]).

On June 25 and 26 the bird was treated with 200 IU oral vitamin E once daily, 50 mg oral methocarbamol once daily and 60 ml intravenous LRS twice daily. Enteral feedings of vanilla Ensure® continued throughout this time; the bird was also given free access to food and water. Gentle physical therapy was performed twice daily. Physical therapy consisted of up to five minutes of muscle massage and passively extending and flexing the legs. Although the bird did not stand during these two days, it remained bright, alert and responsive during treatments.

On June 27 the bird was standing prior to handling; in response to this positive progression, treatment was scaled back. Vitamin E was discontinued; treatment included 25 mg oral methocarbamol once, 35 ml intravenous LRS twice and physical therapy. The bird remained down following handling.

On June 28 the bird was observed standing throughout the day as well as self-feeding. Methocarbamol was discontinued; treatment was limited to 60 ml intravenous LRS once and physical therapy once.

Starting on June 29 the bird exhibited normal behavior including standing, walking and preening, and all treatment in excess of normal conditioning and rehabilitation was discontinued.

Canada goose 038 was examined for release on July 4 and noted to tolerate handling without any change in gait. All parameters for release were normal [Table 2], and the bird was banded with a band issued by the US Bird Banding Laboratory in Laurel, MD, and released with its family group on July 7, 2008.

Discussion

Canada goose 038 showed symptoms comparable to previously described incidences of capture myopathy in birds. The differential diagnoses included traumatic (hip or spinal) injury, lead poisoning, botulism, tetanus, West Nile virus, other central nervous system infections (viral, bacterial, fungal or parasitic), renal disease (resulting in pressure on the sciatic nerve) and neoplasm. Radiographs were not taken, but thorough physical examination ruled out traumatic injury, and blood analysis ruled out lead poisoning. The working diagnosis of capture myopathy was based on history of physical capture and transport on a warm summer day, clinical signs, normal blood lead values and eventual response to therapy. Serum biochemical analysis and histopathology may have provided a more definitive diagnosis, but limited resources for treatment of individual animals during oil spill response must be balanced with treatment of the flock (herd health management

approach applies), and resources were not immediately available as the response was being conducted in a vacant warehouse.

Treatment and rehabilitation of wildlife impacted by an oil spill includes at least two events that could lead to capture myopathy: the initial capture event and cleaning. The information taken on initial veterinary examination of all oiled animals indicates whether an animal is medically stable enough to be cleaned. The parameters of goose 038 measured on admission were within the averages for other adult Canada geese treated during this incident. [see Table 1] Oiled wildlife responders took measures to prevent capture myopathy: capture and transport were conducted by experienced personnel, stress reduction methods were implemented in captivity and cleaning was accomplished as quickly as possible by an experienced team. Out of 47 birds rehabilitated during this incident, only one developed persistent signs of capture myopathy. Three other birds were given vitamin E post-cleaning for similar symptoms; all three recovered normally within eight hours, and none developed additional symptoms nor required additional treatment. A study of capture myopathy in mallards suggested that propensity for muscle damage may vary greatly between individuals, a finding that is supported in this case. (Dabbert et al 1993)

Successful capture myopathy treatments reported in greater sandhill cranes (*Grus canadensis tabida*) and a rhea (*Rhea americana*) both included fluid therapy. (Businga et al 2007, Smith et al 2005) Aggressive intravenous fluid therapy may reverse metabolic acidosis and help to lower circulating CK, LDH and AST. (Williams and Thorne 1996) In addition, the early treatment with large volumes of fluids was intended to reduce secondary complications of capture myopathy, including circulatory shock and renal damage. (Businga et al 2007)

A study of mallards suggested that some wild waterfowl may have nutritional selenium and/or vitamin E deficiencies that predispose them to capture myopathy. (Dabbert and Powell 1993) In addition, selenium and vitamin E are known to have antioxidant activity that may reduce CK and LDH levels. Goose 038 was treated with vitamin E for this reason, and to address the possibility of an underlying vitamin E deficit predisposing this animal to capture myopathy.

Methocarbamol was used in successful treatment of capture myopathy in a rhea by aiding in muscle fiber relaxation and healing, and in a demoiselle crane (*Anthropoides virgo*) with severe ataxia. (Smith et al 2005, Done et al 1993) This muscle relaxant has also been used in mammals to treat exertional myopathy as well as other paresias such as tetanus. (Cunningham et al 1992, Lee and Jones 1996)

Physical therapy has been an important part of all successful outcomes to capture myopathy in avian patients. Previous therapy has included raising birds in slings, assisted walking and massaging the leg muscles. (Businga et al 2007, Smith et al 2005, Rogers et al 2004) In the authors experience physical therapy has contributed to successful outcomes with paresis of other origins including trauma and West Nile virus, and we believe it aided

the recovery of this bird. (Hanrahan 2005) In order to reduce handling, physical therapy was performed only in conjunction with other treatments.

Appropriate housing and nutritional support contributed to the treatment of goose 038. Studies of captive wildlife have shown that environmental stressors can impact daily time budgets. (Shepherdson et al 2004) Stress reduction measures such as diminishing unexpected aural and visual stimuli should always be considered for captive wildlife and are especially important to resolution of a stress-induced syndrome such as capture myopathy. In order to reduce stress, goose 038 was housed with its family group throughout care. During the initial days of treatment the bird was confined in a child's playpen within a larger pen. The tarp walls of the larger pen created a visual barrier between the family group and other activity in the wildlife center. Goose 038 had full view of its family group from the playpen, but the pen restricted its activity to movements of only a few feet. Extra foam padding on the floor of the playpen prevented development of pressure sores on the keel or hocks while the goose was immobile. In addition, all handling of the bird and its family group was minimized and restricted to a few staff members.

Adequate nutritional support was an important component to successful treatments of capture myopathy in greater sandhill cranes and a rhea. (Businga et al 2007, Smith et al 2005) Goose 038 was gavage-fed during the first few days of treatment in order to minimize weight loss during recovery. Vanilla Ensure® was used because it is readily obtained in a spill response, easily absorbed by the gastrointestinal tract, is high in Kcals, has a suitable protein, carbohydrate and fat ratio and has been used successfully in waterfowl recovering from oil spill contamination for over 15 years. As the bird recovered it ate duck pellets and chopped romaine lettuce readily from bowls and had free access to water; the bird gained 245g in the nine days between post-cleaning and release.

This case suggests that aggressive treatment at the first signs of capture myopathy in wild-caught Canada geese can result in full recovery. We expect that similar protocols could be effective in the treatment of this condition in other species of waterfowl. The authors welcome information on documented or anecdotal cases of treatment of capture myopathy in wild birds.

Literature cited

Abbott, C.W., C.B. Dabbert, D.R. Lucia and R.B. Mitchell. 2005. "Does muscular damage during capture and handling handicap radiomarked northern bobwhites?" *Journal of Wildlife Management* 69(2): 664-670.

Berg, C., ed. *Best practices for migratory bird care during oil spill response.* (Washington DC: US Fish and Wildlife Service, 2003).

Businga, N.K., J. Langenberg and L. Carlson. 2007. "Successful treatment of capture myopathy in three wild greater sandhill cranes (*Grus canadensis tabida*)." *Journal of Avian Medicine and Surgery* 21(4): 294-298.

Carpenter, J.W., N. Thomas and S. Reeves. 1991 "Capture myopathy in an endangered sandhill crane (*Grus canadensis pulla*)." *Journal of Zoo and Wildlife Medicine* 22(4): 488-493.

Cattet, M., G. Stenhouse and T. Bollinger. 2008. "Exertional myopathy in a grizzly bear (*Ursus arctos*) captured by leghold snare." *Journal of Wildlife Diseases* 44(4): 973-978.

Cunningham, F.E., J.H. Fisher, C. Bevelle, M.J. Cwik and R.C. Jensen. 1992. "The pharmacokinetics of methocarbamol in the thoroughbred race horse." *Journal of Veterinary Pharmacological Therapy* 15: 96-100.

Dabbert, C.B. and K.C. Powell. 1993. "Serum enzymes as indicators of capture myopathy in mallards (*Anas platyrhynchos*)." *Journal of Wildlife Diseases* 29(2): 304-309.

Done, L.B., D.M. Ialeggio and M. Cranfield. "Therapeutic use of methocarbamol in a demoiselle crane (*Anthropoides virgo*) with severe ataxia and lateroflexion of the neck." In *Proceedings of the Annual Conference of the American Association of Zoo Veterinarians* 137-138. Yulee, FL: 1993.

Forbes, N.A. "Nervous Diseases." In *Manual of Raptors, Pigeons and Waterfowl*, ed. P.H. Beynon, N.A. Forbes, and N.H. Harcourt-Brown. (Ames, IA: Iowa State University Press, 1996), 317-321.

Hanrahan, E. "Physical Therapy for Songbirds." *Wildlife Rehabilitation* 22: 35-44.

Hartup B.K., G.V. Kollias, M.C. Jacobsen, B.A. Valentine and K.R. Kimber. 1999. "Exertional myopathy in translocated river otters from New York." *Journal of Wildlife Diseases* 35(3): 542-547.

Herráez, P., E. Sierra, M. Arbelo, J.R. Jaber, A. Espinosa de los Monteros and A. Fernández. 2007. "Rhabdomyolysis and myoglobinuric nephrosis (capture myopathy) in a striped dolphin." *Journal of Wildlife Diseases* 43(4): 770-774.

Lee, E.A. and B.R. Jones. 1996. "Localised tetanus in two cats after ovariohysterectomy." *New Zealand Veterinary Journal* 44(3): 105-108.

Rogers, D.I., P.F. Battley, J. Sparrow, A. Koolhaas and C.J. Hassell. 2004. "Treatment of capture myopathy in shorebirds: a successful trial in northwestern Australia." *Journal of Field Ornithology* 75(2): 157-164.

Shepherdson, D.J., K.C. Carlstead and N. Wielebnowski. 2004. "Cross-institutionals assessment of stress responses in zoo animals using longitudinal monitoring of faecal corticoids and behavior." *Animal Welfare* 13: S105-S113.

Smith, K.M., S. Murray, and C. Sanchez. 2005. "Successful treatment of suspected exertional myopathy in a rhea (*Rhea americana*)." *Journal of Zoo and Wildlife Medicine* 36(2): 316-320.

Spraker, T.R. "Stress and capture myopathy in artiodactylids." In *Zoo and Wild Animal Medicine and Current Therapy Vol. 3,* ed. M.E. Fowler, 481-488. Philadlelphia, PA: WB Saunders, 1993.

Williams, E.S. and E.T. Thorne. "Exertional myopathy (capture myopathy)." In *Noninfectious Diseases of Wildlife, 2nd Edition,* ed. A. Fairbrother, L.N. Locke and G.L. Hoff, 181-193. London: Manson Publishing/Veterinary Press, 1996.

Wobeser, G.A. 1987. "Mortality of waterfowl on a hypersaline wetland as a result of salt encrustation." *Journal of Wildlife Diseases* 23(1): 127-134.

Wobeser, G.A. "Capture Myopathy," in *Diseases of Wild Waterfowl*, 208-210. New York, NY: Plenum Press, 1981.

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	CAGO 038	**mean (range)**
body weight (g)	4262	4168 (3384 - 5230) n=32
body temperature	40.8 ºC [105.5ºF]	41 ºC [105.8 ºF] (40.2 - 42.9 ºC 104.6 - 109.3 ºF n=31
packed cell volume (%)	46	41 (34 - 50) n=32
total solids (g/dL)	4.6	3.7 (2.9 - 5.0) n=24

Table 1. Incoming parameters of Canada goose 038 as compared with all after hatch year Canada geese treated during this incident.

	CAGO 038	**mean (range) n=32**
body weight (g)	4420	4280 (2980 – 5545)
packed cell volume (%)	40	41 (35 – 45)
total solids (g/dL)	5.2	4.8 (3.8 – 6.0)

Table 2. Pre-release parameters of Canada goose 038 as compared with all after hatch year Canada geese treated during this incident.