

CAPTURE MYOPATHY IN A CAPTIVE BLACK-BELLIED WHISTLING DUCK

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A female captive-raised, Black-bellied Whistling Duck exhibited signs of capture myopathy within 24 hours after a strenuous capture. Serum creatine kinase activity 48 hrs after capture was 23,680 IU/L, and aspartate aminotransferase activity was 898 IU/L. Postmortem lesions included large, pale areas of muscle tissue in the hind limbs and heart. Histologic lesions of skeletal muscle included severe myofiber degeneration and necrosis with mineralization, interstitial hypercellularity, and occasional areas of dense fibrous connective tissue scarring.

Key words: Aspartate Aminotransferase, Black-bellied Whistling Duck, Capture Myopathy, Creatine Kinase, *Dendrocygna autumnalis*

A serious outcome occasionally observed while handling wild-caught birds is capture myopathy (CM), which results in damaged striated muscle and kidneys. Excessive production of lactic acid in muscle tissue because of anaerobic glycolysis during intense activity, increased plasma potassium levels, and perhaps the effects of catecholamine release lead to reduced cardiac output, poor tissue perfusion, and severe muscle hypoxia and necrosis resulting in lesions and clinical signs associated with CM. Capture myopathy is also associated with exertion and fear occurring during capture and confinement (Wobeser 1981). Clinical signs of CM may range from acute to chronic. In the periacute form, metabolic acidosis and hyperkalemia cause cardiac fibrillation, circulatory collapse, and death within minutes of capture. The acute form is recognized by muscular stiffness, depression, and rapid heart and respiration rates, with death in about 12 hours resulting from pulmonary edema. The subacute form is characterized by pronounced damage to skeletal muscle and internal organs, and animals are paretic or paralyzed; death may result from acute renal failure. The chronic form is usually associated with fibrotic lesions in skeletal and cardiac muscle (Wobeser 1981).

Gross postmortem lesions are bilateral, but

not symmetrical, areas of skeletal muscle necrosis, swollen kidneys, subcutaneous hemorrhage, and hemorrhagic and congested adrenal glands. Histopathic lesions include tubular necrosis of the kidneys, and focal areas of necrosis and congestion of the adrenal cortex (Spraker 1978). These lesions have been reported in a Greater Sandhill Crane *Grus canadensis tabida* (Windingstad et al. 1983), and Wild Turkeys *Meleagris gallopavo* (Spraker et al. 1987) with clinical signs of CM.

Certain serum or plasma enzymes may be useful in diagnosing CM. Increased cell permeability caused by low tissue pH causes release of the intercellular enzymes creatine kinase (CK), and aspartate aminotransferase (AST) into the blood. Elevated concentrations of these enzymes reflect damage to skeletal and cardiac muscle (Bollinger et al. 1989).

Methods

We suspected a case of CM in a captive Black-bellied Whistling Duck. One pair of pinioned, adult, Black-bellied Whistling Ducks were purchased from a private breeder in Mamou, Louisiana, USA. The ducks were already captured and placed in a cardboard box when the new owner picked them up, and the

Table 1. Mean (with standard deviation in brackets) serum creatine kinase and aspartate aminotransferase activities reported for various waterfowl species.

Species Reference ¹ <i>Anas platyrhynchos</i>	Creatine Kinase (CK) IU/L	Aspartate Aminotransferase (AST) IU/L
Minimal handling (1)	225(52)	19.0(7.0)
Rocket net (1)	12,035(8,125)	330.0(171.0)
Bait trap (2)	-	43.0(33.0)
Rocket net (2)	-	61.0(46.0)
Young (3)	-	88.5(54.1)
Adult (3)	-	17.4(5.7)
Adult (4)	-	13.5(4.6)
Bait trap (5)	-	88.0(2.0)
<i>Anas rubripes</i> (4)	-	18.6(8.2)
<i>Anas superciliosa</i> Cotton net capture (6)	-	61.0(24)
<i>Aythya marila</i> and <i>Aythya affinis</i> Newly caught (7)	4,397	-
Moribund (7)	27,180	1,772
<i>Aythya valisineria</i> (8)	-	15.0(8.1)
<i>Aythya americana</i> (8)	-	23.0(12.0)

¹References:(1) Dabbert & Powell (1993); (2) Bollinger *et al.* (1989); (3) Fairbrother *et al.* (1990); (4) Franson (1982); (5) Driver (1981); (6) Mulley (1979); (7) Harvey-Clark (1991); (8) Franson *et al.*(1985).

breeder reported that the capture had been strenuous. When the ducks were released from the box at their new location after two hours of travel, the female could not stand.

On physical examination, the female appeared bright and alert, and had no obvious central or peripheral neurological deficiencies. There were no palpable fractures or luxations of the pelvic limbs or other visible signs of trauma. While not bearing weight, the legs appeared to move normally, but when forced to bear weight, the extensor muscles were weak and the bird was unable to stand or walk.

Forty-eight hours after capture, initial blood samples were taken from both the female and an apparently normal male for comparison. Blood samples were collected from two other pairs from the same breeder to determine CK and AST levels. Serum chemistries were performed on an Olympus AU 5000 automated blood chemistry analyzer at a private laboratory. Serum levels of creatine kinase (CK) and aspartate aminotransferase (AST) were measured.

Results

The female showing signs of capture myopathy had a serum CK value of 23,680 IU/L at the initial sample 48 hours after capture, and 18,020 IU/L in samples collected 96 hours after capture. The female died four days later. Similar samples collected from the male had levels of 862 IU/L and 813 IU/L at 48 and 96 hours after capture, respectively. The samples from the two additional pairs had much lower values for CK (\bar{x} =227 IU/L; range 93-525 IU/L), and AST (\bar{x} =52; range 26-80 IU/L). One bird, which struggled vigorously during the sampling, had a CK value of 525 IU/L, the highest of the four birds sampled.

The affected bird was frozen after death because necropsy could not be performed immediately. The intact carcass had a mass of 652 g after thawing, and appeared to be in good condition. The superficial pectorals appeared normal. The deep pectoral muscles had faint white streaking near their insertion. The pelvic limb muscles were affected to a greater extent. White tissue was present in approximately 75%

of the *gastrocnemius pars lateralis* muscle, and to a lesser and variable extent in the *iliotibialis cranialis*, *iliotibialis lateralis*, *flexor cruris lateralis*, *femorotibialis externus et medius*, *gastrocnemius pars medialis*, *tibialis cranialis*, and *flexor perforatus digiti IV* muscles (as described by Raikow 1985). Pale streaking was grossly evident in the heart muscle. No other lesions were noted.

Gastrocnemius and heart muscle tissues were submitted for histopathic examination. The skeletal muscle sections contained severe myofibre degeneration and necrosis with mineralisation and interstitial hypercellularity (fibroplasia and inflammatory cells-macrophages, few multinucleated giant cells). Occasional areas of dense fibrous connective tissue scarring were noted. The heart contained separation and fragmentation of myofibres believed by the pathologist to be an artifact of freezing. There were rare areas of sarcoplasmic fragmentation, however, which have may represented a real lesion.

Discussion

Creatine kinase is an intracellular enzyme that functions in skeletal muscle and heart muscle and brain tissue making adenosine triphosphate (ATP) available for contraction by the phosphorylation of adenosine diphosphate (ADP) from creatine phosphate (Hochleithner 1994). Elevations are primarily due to muscle damage (Franson *et al.* 1985), leakage into the blood occurs when heart, muscle or nerve tissue is damaged (Hochleithner 1994). Creatine kinase concentrations reported for waterfowl with myopathy are >1,000 IU/L (Wobeser 1981). Bollinger *et al.* (1989) reported CK levels of 4,317 to 6,506 IU/L for birds exhibiting abnormal flight after capture during trapping efforts. Harvey-Clark (1991) reported higher CK levels in moribund birds than in newly caught birds, mostly Greater *Aythya marila* and Lesser Scaup *A. affinis*, which were oiled as a result of a marine fuel spill (Table 1). Dabbert & Powell (1993) found lower CK and AST activity in Mallard *Anas platyrhynchos* captured with minimal handling and holding time than for birds captured by rocket net (Table 1). It was not reported

whether birds captured by rocket net were able to walk or fly normally because they were euthanized after blood samples were taken.

Aspartate aminotransferase activity is found in the liver, skeletal muscle, heart, brain and kidney cells. AST catalyzes the interconversion of amino acids and oxoacids by the transfer of amino groups. AST levels vary by species (Table 1), but variance of 'normal' birds is substantial less than for the Black-bellied Whistling Duck reported here. Elevations are usually associated with liver or muscle damage (Hochleithner 1994), which may appear during strenuous capture (Driver 1981, Bollinger *et al.* 1989). Fairbrother *et al.* (1990) found AST activity increased in young Mallard, and decreased with age until reaching average adult, postreproductive values at 58 days of age. Driver (1981) found AST activities of 28 ± 2.2 to 49 ± 3.1 IU/L in captive Mallard during varying moult stages, which was less than captured birds (Table 1).

A condition which produces lesions similar to CM is white muscle disease, which is believed to be a result of a dietary selenium deficiency (Smith *et al.* 1972). The diet of the ducks in this report was not quantified as to selenium levels, but they were reared in an area in which selenium concentration in plants is considered to be adequate (89% of forage and grain contain >0.1 ppm selenium; Robbins 1983). In addition, evidence is beginning to accumulate to indicate that animals that succumb to CM may have normal selenium-vitamin E levels (Spraker 1978). Considering the history, clinical signs, high CK levels, and muscle lesions seen in this case, it is reasonable to conclude that capture myopathy, rather than white muscle disease was the cause of this bird's lameness, even though there is not enough data to determine what normal CK and AST values are for whistling ducks after capture and handling.

Although the lesions seen in this case are often associated with CM (Spraker 1978, Windingstad *et al.* 1983), it is difficult microscopically to rule out other causes, such as vitamin E/selenium imbalance, since marginal deficiencies can precipitate exertional myopathy (Spraker 1978). It is possible that the female was suffering from a subclinical disease which predisposed the female to CM.

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