

Causes of mortality in Trumpeter Swans *Cygnus buccinator* in Minnesota 1986-1989

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Between January 1986 and June 1989, 116 Trumpeter Swans were presented to The Raptor Center at the University of Minnesota U.S.A. for post mortem examination to determine the cause of death. In all cases a thorough gross post mortem examination was conducted with histopathology and microbiology included in selected cases. Liver lead analysis was conducted in nearly every case, even when the cause of death was known. Lead poisoning as a result of the ingestion of spent lead shotgun pellets was the major cause of death (53 birds, 45.7%). Aspergillosis, a fungal respiratory disease, accounted for 18 deaths (15.5%). Traumatic injuries such as shooting or power lines resulted in 13 deaths (11.2%). A variety of miscellaneous diseases caused another 13 deaths (11.2%). Ten birds (8.6%) died of, or were euthanized for developmental or congenital diseases. The cause of death could not be determined for nine birds (7.8%).

Between January 1986 and June 1989, 116 Trumpeter Swans, *Cygnus buccinator*, were presented to the Raptor Center at the University of Minnesota, St. Paul, Minnesota U.S.A. for post mortem examination to determine the cause of death. The Raptor Center has been conducting all Trumpeter Swan necropsies since 1986 in an effort to gain a better understanding of the problems faced by these birds in Minnesota, and to make recommendations for changes in their management.

Trumpeter Swan restoration efforts began in the late 1960s with the first releases occurring in 1979. The fall 1988 state-wide population was estimated to be approximately 240 swans, half of which were free-flying and the remainder in captive programs. Many of the captive birds were wing-clipped cygnets and subadults being held for release as two-year-olds. Mortality data reported in this paper include swans from Hennepin Parks, Minnesota Department of Natural Resources, Wisconsin Bureau of Endangered Resources (cygnets housed in Minnesota refuges) plus several small private propagation facilities in the state.

Methods

During the study period of January 1986 - June 1989 almost all Trumpeter Swans that were found dead in the state of Minnesota (Fig. 1 &

2) were sent to The Raptor Center for examination. All swans presented were examined by post-mortem except where scavenging or advanced decomposition precluded complete necropsies. The Department of Diagnostic Investigations at the College of Veterinary Medicine, University of Minnesota conducted histopathologic studies in selected cases to determine or confirm the cause of death. Additional information was obtained from microbiological cultures, parasitology (fecals), and toxicology (lead analysis, using atomic absorption spectrophotometry).

Results and Discussion

The causes of mortality during this three and one-half year period are outlined in Table 1. The main cause of death was lead poisoning (45.7%) and the majority of these birds (40) died during the winter of 1988-89. Drought conditions of 1988 caused water levels to fall, allowing increased access to spent lead shotgun pellets by swans. A statewide ban on lead shot for waterfowl hunting went into effect in 1987, however some of the swans came from a refuge that had not been hunted for more than 20 years. Prior to this study period, there were few documented cases of lead poisoning in the Minnesota population of Trumpeter Swans (Gillette 1988, Degernes & Redig 1990a). Mortality records

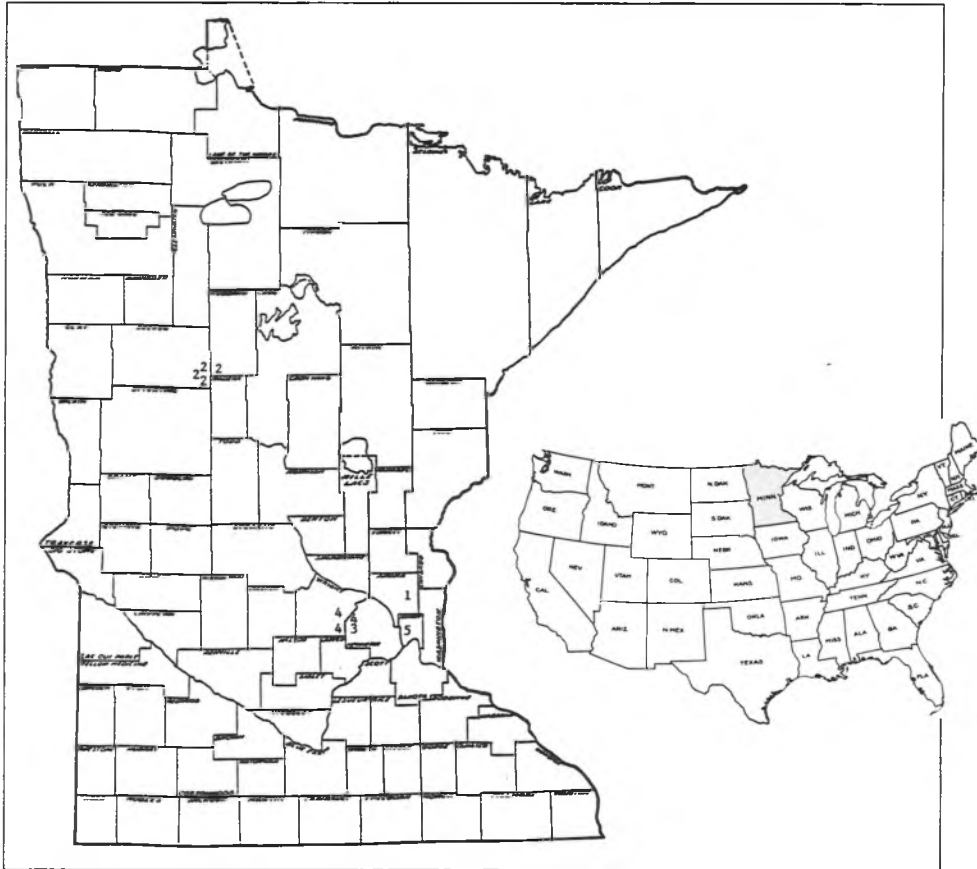


Fig. 1 United States of America - the state of Minnesota (MINN.) is denoted by an asterisk.
 Fig. 2 State of Minnesota - Trumpeter Swan captive and free-flying populations.
 1. Minnesota Department of Natural Resources (DNR) captive rearing facilities. 2. Minnesota DNR free-flying populations. 3. Hennepin Parks captive rearing facilities. 4. Hennepin Parks free-flying population. 5. The Raptor Center at the University of Minnesota, St. Paul.

for the past decade in other Trumpeter Swan populations in the western states have shown no lead-related mortality in Alaska, Oregon, and Wyoming, 20% of the total mortality in Idaho and Montana and as high as 61.5% of the mortalities in Washington state (Blus *et al.* 1989).

Table 1. Causes of mortality: January 1986 - June 1989.

	Total	Per cent of total
1. Lead poisoning	53	45.7%
2. Aspergillosis	18	15.5%
3. Trauma	13	11.2%
4. Miscellaneous diseases	13	11.2%
5. Developmental/congenital diseases	10	8.6%
6. No diagnosis determined	9	7.8%
	116	100.0%

Diagnosis of lead poisoning was based upon gross and histologic observations and liver lead analysis. Gross pathologic observations included weight loss, impacted proventriculus and green livers, all of which have been commonly reported in other lead poisoned swans (Trainer & Hunt 1965, Irwin 1975, Kendall & Driver 1982, Blus *et al.* 1989). Histopathologic changes were detected in multiple systems including gastrointestinal, renal and cardiac systems. Acute necrohemorrhagic enteritis of the proximal duodenum was observed in four swans that died during treatment for lead toxicity. Grossly, the proximal 10 to 20 cm of the duodenum was distended with dark red clotted blood and *Clostridium perfringens* in mixed or pure culture was isolated. Histologically, fibrinoid vascular necrosis was observed. Fibrinoid vascular necrosis was also confirmed in the thyroid gland,

proventriculus, pancreas and/or spleen and has been documented in other lead poisoned avian species (Cook & Trainer 1966, Karstad 1971). Renal lesions consisted of degeneration, necrosis and sloughing of the tubular epithelium with secondary visceral gout in some cases. Multifocal myocardial degeneration and necrosis was observed in one third of the swans examined, varying from very mild to moderate. Secondary problems included bacterial septicemia, aspergillosis and schistosomiasis, and may have been related to immunosuppression. Neurologic lesions were not observed histologically.

Liver lead levels > 6.0 parts per million (ppm or µg/g, wet weight) were generally considered diagnostic but in context with other significant pathologic changes, lower levels may be significant as well (Cook & Trainer 1966, Birkhead 1982). The mean liver lead level in swans that had not been treated was 17.6 ppm (range 2.6 - 34.8 ppm).

The second leading cause of death (15.5%) was aspergillosis, an often fatal respiratory disease caused by *Aspergillus fumigatus*, or less commonly *A. flavus* (Degernes & Redig 1990b). Inhalation of fungal spores from moldy or damp food or bedding is the most likely source of the disease. The majority of the cases (12) involved the generalized form of aspergillosis in which the disease is widespread throughout the lungs and air sac system. However, six birds died of tracheal aspergillosis in which the distal loop of the trachea within the keel section was partially to totally obstructed, leading to asphyxiation. Surprisingly, gross evidence of aspergillosis was not present elsewhere in the lungs or air sacs in these birds. It is speculated that Trumpeter Swans are predisposed to tracheal aspergillosis due to the elongated looped trachea within the keel, resulting in fungal spores settling out in the curved sections. An over-

whelming dose of fungal spores or a localized immune system failure may allow the fungal organisms to grow and occlude the airway.

Various traumatic injuries accounted for 11.2% of the total deaths. Shooting accidents and power lines were the two most common causes of fatality. O'Halloran *et al.* (1989) reported an increased incidence of powerline fatalities in lead poisoned Mute Swans *Cygnus olor*, however, this relationship was not observed in Minnesota Trumpeter Swans in this study period. Other miscellaneous traumatic deaths were caused by automobiles, predators (mink), head-injuries, etc. Shooting-related mortality has decreased significantly since the early 1980s (Gillette 1988), presumably due to increased hunter education and public awareness in the state.

Other miscellaneous diseases comprised 11.2% of the total, and included bacterial septicemia, amyloidosis, visceral gout, neoplasia (ganglioneuroma), ruptured aneurysm of the right atrium and botulism caused by *Clostridium botulinum*.

Developmental diseases included those problems that occur during the rapid growth phase and are most likely the result of management (e.g. nutrition) problems. Severe lameness problems resulting from a medially "slipped" gastrocnemius tendon caused the death or euthanasia of seven cygnets (four other cases were successfully treated with surgery). Severe distal wing deformities ("flip wing") resulted in the euthanasia of one cygnet. Congenital problems observed in two newly hatched cygnets included severely twisted and deformed legs in one and an imperforate vent (no cloacal opening) in another.

Lastly, the cause of death could not be determined in approximately 8% of the cases due to scavenging, decomposition or lack of gross and histologic abnormalities.

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