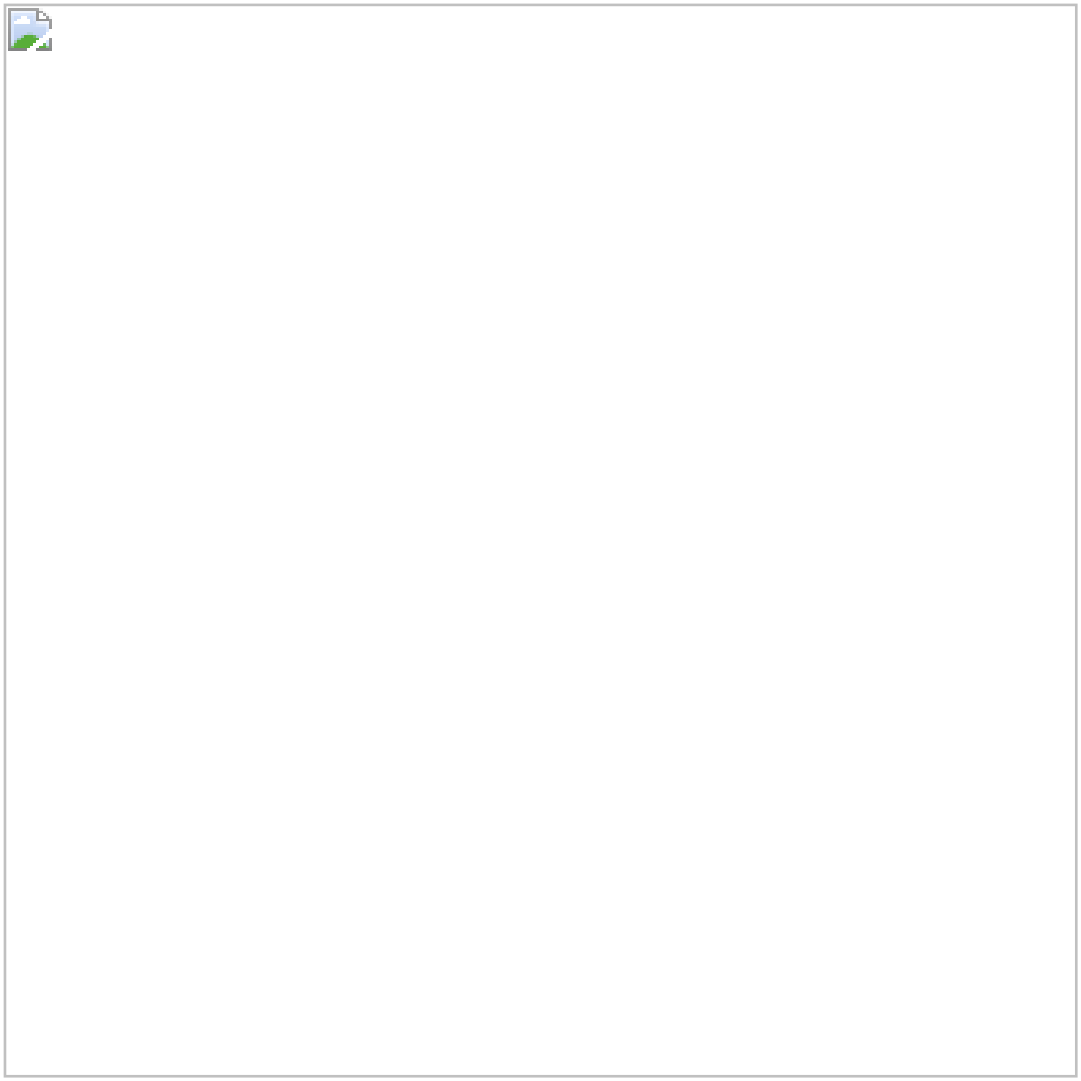

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Source: Journal of Wildlife Diseases, 10(4) : 466-470

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-10.4.466

Pasteurella anatipestifer INFECTION IN MIGRATING WHISTLING SWANS

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Abstract: Pasteurella anatipestifer was isolated from five of seven juvenile whistling swans found sick or dead on two lakes in Saskatchewan, during the 1973 autumn migration. More than 100 sick or dead birds had been reported on one of these lakes. Pathologic lesions were similar to those reported in *Pasteurella anatipestifer* infection in other species of waterfowl. The possible epizootiology of the disease is discussed.

INTRODUCTION

Pasteurella anatipestifer causes septicemic disease in domestic ducklings,[3,9,10] and has also been reported to cause disease in domestic geese,[1] turkeys[11] and pheasants.[1] Outbreaks of the disease have been reported in semi-domestic black ducks (*Anas rubripes*)[4] and in several species of captive wild waterfowl.[6] Donahue and Olson[2] isolated the organism from nasal swabs of clinically normal wild Canada geese (*Branta canadensis*) in Missouri. The only published reports of this disease in free flying wild birds describe an outbreak in black swans (*Cygnus atratus*) in Tasmania.[7,8] The present report describes the disease in whistling swans (*Olor columbianus*) congregated on lakes near Saskatoon, Saskatchewan during the 1973 autumn migration.

CASE HISTORIES AND METHODS

On October 28, 1973 the senior author found a recently dead, juvenile whistling swan (Bird 1) at the edge of a small, shallow unnamed lake located approximately 35 km southeast of Saskatoon, Saskatchewan. At the time approximately 75 whistling swans; 1,000 ducks, primarily mallards (*Anas platyrhinchos*); and 25 Canada geese were present on the lake. No other dead birds were noted; however, the entire shoreline was not searched. By November 2, the date of the next visit, the ground was snow covered, and the lake was almost totally frozen. About 50 swans, 100 ducks and 1 Canada goose were present on the ice, but no dead birds were found.

On November 7, three dead juvenile whistling swans (Birds 2-4) were submitted to the diagnostic laboratory. The consignor had visited Goose Lake, (a shallow intermittent lake, measuring approximately 5 x 10 km and located about 75 km southwest of Saskatoon), on November 3, at which time approximately 2,000 swans were present. On November 4, only about 500 swans remained around the small amount of open water available. Of the 500 birds, at least 100 were exhibiting varying degrees of weakness, and at least 15 dead swans were observed. The consignor stated, that with the possible exception of one bird, all of the sick and dead birds were juveniles. Sixteen of the sick swans were collected by the consignor and taken to a site for care. The three birds submitted November 7 had died shortly after collection and had been frozen until submission. On November 14, 3 of the remaining 13 birds were submitted (Birds 5-7). Two

of these birds had died within 3 days of capture, while the third had survived for an unspecified length of time. No further specimens were submitted and the fate of the remaining 10 birds was not determined.

Complete necropsies were performed on all birds. Tissues collected for histopathologic examination were fixed in 10% neutral buffered formalin, processed routinely, sectioned at 6 μm and stained with hematoxylin-eosin. A variety of tissues from each bird were cultured for the presence of bacteria. Swabs taken aseptically from the meninges were cultured in six of the seven cases. Routine cultures were performed using blood agar and MacConkey agar incubated aerobically at 37C.

PATHOLOGY

The seven swans examined were all juveniles in good body condition with large amounts of subcutaneous and intra-abdominal fat. The sex, and weight of the birds, together with gross lesions observed, are summarized in Table 1. The most consistent gross lesion was a swollen liver which was an unusual brick-red color. Fibrinous exudate was prominent in several sites in most birds (Table 1). The entire alimentary tract was empty of ingesta in all birds. A thick collection of fibrinous debris was present in the trachea of two birds, but the lungs appeared normal in all. The spleen was of normal size and pale. The histopathologic lesions observed are summarized in Table 2. The lesions were similar in all birds, but varied in extent and in type of inflammatory cells present. Heterophils were the most numerous cell type in periportal cellular infiltrates in the liver and in the meningeal exudate of Bird 1. In Birds 5 and 7 the inflammatory response was primarily mononuclear, while in the remaining birds a combination of heterophils and mononuclear cells were present. Vascular thrombosis was a common finding in many organs, as was endothelial proliferation and reticulo-endothelial (R.E.) cell activation as described by Karstad *et al.*[6] Septic thrombi were found in the glomeruli of two birds, and the small foci of myocarditis present in some birds were assocated with septic thrombosis of small vessels.

TABLE 1. Sex, weight and gross lesions observed in juvenile whistling swans with **Pasteurella anatipestifer** infection.

	Bird No.						
	1	2	3	4	5	6	7
Sex	F	F	M	M	F	M	M
Weight (kg)	5.4	6.3	6.3	6.3	5.3	5.9	5.5
Lesions							
Liver swollen, brick red	+*	+	+	+	+	—	+
Fibrinous perihepatitis	—**	+	+	—	+	+	—
Fibrinous pericarditis	—	+	—	—	+	+	+
Fibrinous airsacculitis	—	+	—	—	—	—	—
Fibrinous tracheitis	—	—	—	—	+	—	+

*+ lesion present

**— lesion not present

TABLE 2. Occurrence of histopathologic lesions in juvenile whistling swans with **Pasteurella anatipestifer** infection.

Organ	Lesion	Bird No. 1	2	3	4	5	6	7
Liver	Fibrinous perihepatitis	—*	+	+	—	+	+	—
	Periportal cellular infiltration	+**	+	+	+	+	—	+
	Vacuolar degeneration	+	+	+	+	+	+	+
	Focal necrosis	—	—	+	—	—	—	—
Heart	Fibrinous epicarditis	—	+	—	—	+	+	+
	Subepicardial edema	—	+	—	—	—	—	+
	Myocarditis	—	+	—	—	+	+	+
Kidney	Embolic glomerulitis	—	—	—	+	—	—	+
Spleen	Fibrinous perisplenitis	—	+	—	—	—	—	+
	Lymphoid depletion	+	+	+	+	+	+	+
	R.E. cell hyperplasia	+	+	+	+	+	+	+
	Focal necrosis	+	+	—	+	+	—	+
Lung	Focal pneumonia	—	—	—	—	+	—	—
	Fibrinous airsacculitis	—	+	—	—	—	—	—
Trachea	Fibrinous tracheitis	—	—	—	—	+	—	+
Brain	Fibrinous meningitis	+	—+	+	+	+	—	+

*— lesion not present

**+ lesion present

BACTERIOLOGY

Organisms which resembled *P. anatipestifer* were isolated from five of the seven birds examined (Table 3).

On blood agar, the organism produced small transparent, non-hemolytic colonies which were composed of small gram-negative coccobacilli. The organism was cytochrome oxidase positive, catalase positive, indol negative, did not grow on MacConkey agar, and did not ferment common carbohydrate media. Subcultures of four of the isolates (Birds 1, 2, 5, 7) were sent to Dr. Jessie I. Price, Duck Research Laboratory, Cornell University, Eastport, New York. Dr. Price confirmed the identification of the isolates as *P. anatipestifer*, but found that the isolates failed to agglutinate with any sera available. She reported that the isolate from Bird 2 was tested for pathogenicity in domestic ducklings, and produced lesions in 14 of 19 ducklings inoculated.

TABLE 3. Results of bacteriologic culture for **Pasteurella anatipestifer** from seven juvenile whistling swans.

	Bird No.						
	1	2	3	4	5	6	7
Liver	—*	+**	—	—	—	—	+
Spleen	NC***	+	—	NC	—	—	+
Kidney	+	+	—	—	—	—	+
Meninges	NC	+	—	+	+	—	+
Intestine	—	—	—	—	—	—	—
Lung	—	+	—	—	—	—	+
Trachea	NC	NC	NC	NC	—	—	NC
Thoracic airsac	NC	+	NC	NC	NC	NC	—
Pericardium	NC	NC	NC	NC	NC	NC	—
Nasal cavity	NC	NC	NC	—	NC	NC	—
Abdominal cavity	NC	NC	NC	—	NC	NC	—

*— *P. anatipestifer* not isolated

**+ *P. anatipestifer* isolated

***NC Not cultured

DISCUSSION

There are many similarities between the present cases and the outbreak of *P. anatipestifer* infection in black swans.[7,8] Both occurred in the autumn of the year among birds concentrated during migration, and almost exclusively young birds were involved in both instances. Munday[7] speculated that the causative organism, (at that time unidentified), might be enzootic in swans, only producing disease among birds in overcrowded situations. Under crowded condition, large numbers of organisms might be present in the environment and disease in juvenile birds might be the result of an overwhelming initial infection. Prior to the present outbreak, an unusually large number of swans (over 19,000) were present on Goose Lake.[1] Similarly, Munday[7] reported overcrowding of black swan prior to the outbreak in Tasmania.

The isolation of *P. anatipestifer* from normal Canada Geese[2] suggests that the organism may be present in waterfowl in a sub-clinical form. Recently we have isolated *P. anatipestifer* from the nasal cavity of a 4-year-old captive whistling swan, from a local waterfowl collection, which had died of amyloidosis. This isolate was sent to Dr. Price and was found to agglutinate with sera to an avirulent *P. anatipestifer* strain isolated from domestic ducklings in New York. This supports the occurrence of inapparent infections in waterfowl and also suggests that

[1] Gollop, J. B. 1973. Personal communication. Prairie Migratory Bird Research Centre, Canadian Wildlife Service, Environment Canada, Saskatoon, Saskatchewan.

strains of varying pathogenicity may be present. The pathology of the disease in whistling swans is very similar to that reported in other species[5,6,9]; however, isolation of the organism is necessary for diagnosis of the disease. *P. anatipestifer* is often difficult to isolate from infected birds,[6,9,10] perhaps explaining the failure to find the organism in Birds 3 and 6.

The organism was recovered more frequently from the meninges than from other organs; however, the results from the present cases indicate the importance of culturing from several organs.

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Received for publication 17 July 1974