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Pasteurella anatipestifer as a Cause of Mortality In Captive Wild Waterfowl

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Abstract

An outbreak of *Pasteurella anatipestifer* infection in young wild waterfowl at the Niska Waterfowl Research Center resulted in losses of approximately 100 Blue and Snow Geese, one White-fronted Goose, five Mandarin Ducks, one Black Duck and one Wood Duck. Clinical signs included diarrhea, paralysis and tremors. Gross lesions were fibrin deposits on serosal surfaces, hemorrhages on the epicardium, consolidation of the lungs, cloudy or flaky deposits on the air sacs, and dark, swollen spleens. Microscopic lesions included fibrinous meningitis, pneumonitis, air saculitis and serositis. Swollen leg and foot joints were seen in some cases. Chloramphenicol treatment seemed to be of benefit in controlling the outbreak.

Two hundred and seventy-five eggs of Snow and Blue Geese, collected from nests in the Canadian Arctic, were incubated artificially at the Niska Waterfowl Research Center, Guelph, in June, 1969. Approximately 200 goslings hatched. They were kept in brooders until they were 1-2 weeks of age, when, in groups of 15-20, they were put into outdoor pens with pairs of adult Snow or Blue Geese, which had been resident at Niska. This procedure was followed to see if imprinting would occur and if so, what influence imprinting to blue or white adults might have on later color selection of mates.*

Death losses occurred suddenly, beginning on July 14, 1-2 days after the goslings were put into outdoor pens. Attendants believed the first losses were related to chilling. On July 17, two live goslings were submitted for necropsy. They had diarrhea, were partly paralysed, unable to stand, trembled, and made constant biting movements with their beaks. Their mouths were filled with clear frothy saliva. One gosling had persistent lateral deflection of the neck. The tentative diagnosis was encephalitis. Both birds were killed by decapitation. Necropsy revealed few gross lesions: slight fibrinous epicarditis and cloudy air sacs in one; and white fibrinous exudate on the peritoneum and a 3 mm hemorrhage on the epicardium of the right ventricle in the other.

No bacteria were cultured on blood agar and MacConkey agar inoculated from brains and serosal exudates.

* This genetic and behavioral color type study was part of a research program by Dr. F. Cooke, Queen's University, Kingston, Ontario.

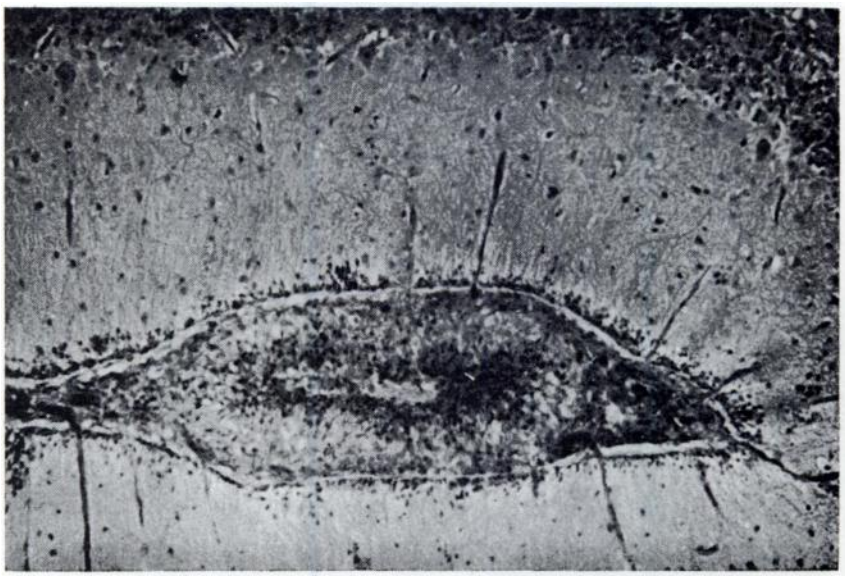

FIGURE 1. *Cerebellum. Note extreme proliferation of vesicular epithelioid cells about a meningeal blood vessel. All figures, 1-5, are representative of hematoxylin and eosin stained sections from Snow Geese infected with* Pasteurella anatipestifer.

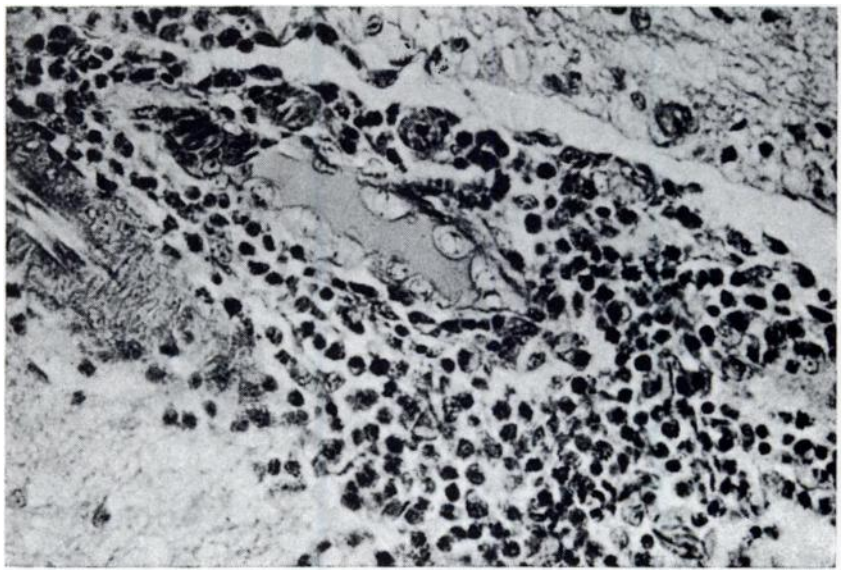

FIGURE 2. *Cerebellum with meningeal exudate composed of mononuclear leukocytes and a venule with swollen endothelial cells.*

Histopathological examination of these two goslings revealed meningitis, fibrinous epicarditis and slight to moderate fibrinous pneumonitis. Meningitis was most pronounced over the cerebellum. Meningeal blood vessels were greatly thickened with swollen endothelial cells and large vesicular epithelioid cells in the adventitia (Figures 1, 2). Meningeal exudates contained fibrin and numerous mononuclear leukocytes.

Losses continued in the young Snow and Blue Geese and by July 22 about 60 birds had died out of the original 200. Some of the young geese were put back into brooders but they continued to die. On July 22 the remaining geese were treated with chloramphenicol in their drinking water, at approximately 1.25 mg/oz. This treatment seemed to have beneficial effects; losses were markedly reduced after three days.

Necropsy examinations revealed similar lesions in 36 waterfowl: 28 young Snow and Blue Geese; one white-fronted Goose hatched and penned with the Snow and Blue Geese; one young Black Duck; one young Wood Duck; and five young Mandarin Ducks. The ducks were from a pen adjacent to the brooder house. Total losses in the young Snow and Blue Geese in the 20 day outbreak finally reached approximately 50% of the original 200 hatched.

Gross lesions were similar in all cases but variable in extent. The most consistently found lesion was a firmly adherent, dull white, fibrinous exudate on the ser-

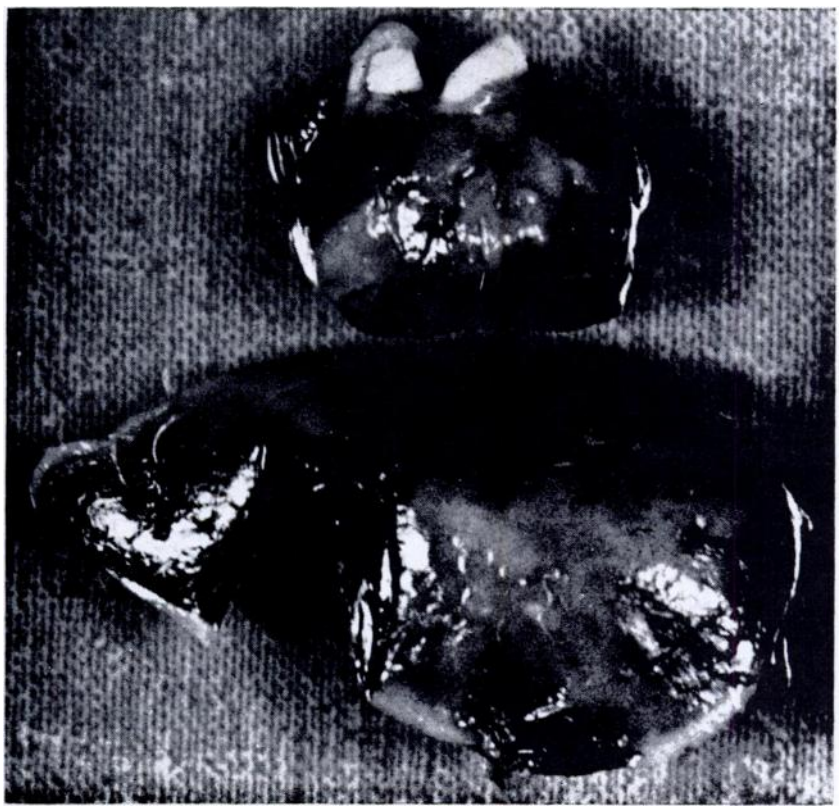

FIGURE 3. *Heart and liver with white exudate on serosal surfaces. Sections had been removed for histology.*

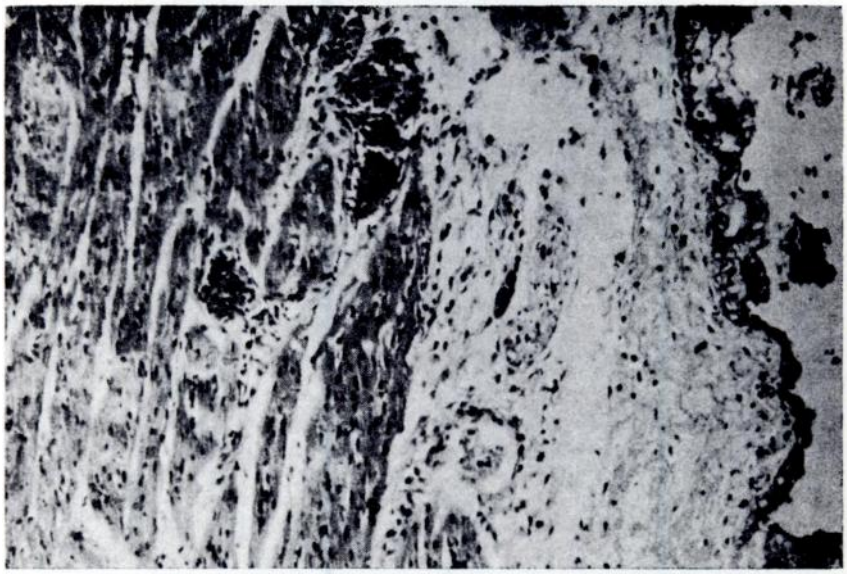

FIGURE 4. *Myocardium. Note the heavy deposit of fibrinous exudate on the epicardium.*

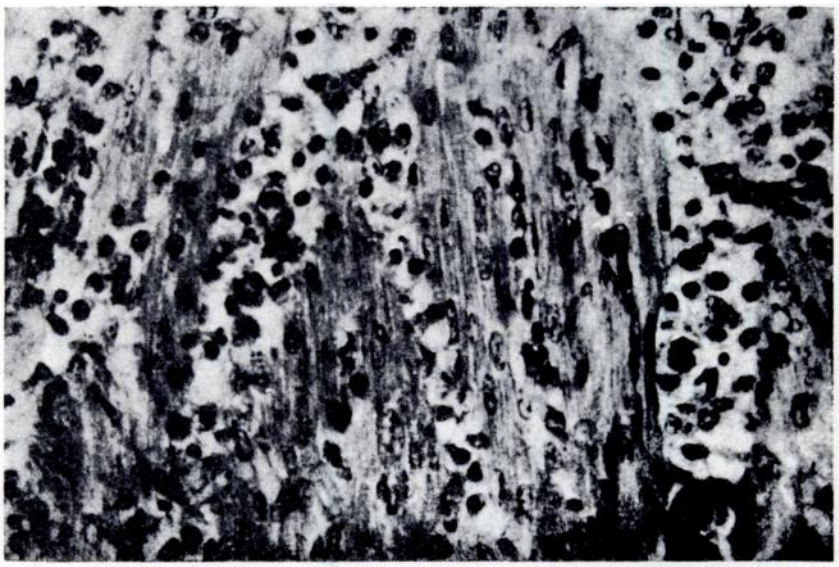

FIGURE 5. *Myocardium. Myofibres are separated by an infiltrate composed mainly of mononuclear leukocytes.*

osa of thoracic and abdominal organs, particularly the liver and pericardium (Figure 3). Lungs were congested and in some cases, consolidated. The spleen was enlarged, up to ten times normal size, dark and pulpy. Meninges were congested. Some of the goslings had edematous swellings of the subcutaneous tissues around the joints of the legs and feet.

Histologic examination confirmed the gross findings. The inflammatory changes consisted of copious fibrinous exudate (Figure 4) containing leukocytes that were mostly large mononuclear phagocytes. Kupfer cells of the liver and reticular phagocytes of the spleen were markedly enlarged. Lung changes varied from acute disseminated vascular thrombosis to acute fibrinous pneumonitis. In some cases the heart lesions were confined mainly to the pericardium and epicardium but more often the myocardium also had variable degrees of congestion, edema and leukocyte infiltration (Figure 5). Liver lesions varied from congestion to acute hepatitis, with periportal leukocytic infiltration and widespread fibrin thrombosis of sinusoids.

All 36 carcasses necropsied were cultured for bacteria on blood agar and MacConkey agar. Inocula consisted of wire loop scrapings from organs with gross lesions after they had been seared with a hot spatula. From 10 of the 36 carcasses, very small dull white, smooth colonies of bacteria were isolated on 5% bovine (BBA) plates incubated for 24 hours at 37°C. These colonies were composed of small gram-negative coccobacilli which stained bipolar with methylene blue. Such organisms were found in direct smears from most of the carcasses, even from those from which bacteria were not cultured. On primary isolation under aerobic conditions, these organisms grew poorly on BBA and were non-hemolytic. When transferred to cystine heart agar, supplemented with 10% bovine blood they grew readily, yielding gray mucoid colonies of medium size in 24 hours. On this medium the organism looked similar to *Pasteurella multocida*, a common cause of fatal septicemia infections in waterfowl. However, it lacked the characteristic odour of *Pasteurella multocida*, was indol negative, unreactive in a number of the common carbohydrates, and was not pathogenic for mice when inoculated intraperitoneally. It was cytochrome oxidase positive and a later examination of a stored isolate showed it to be catalase positive.

Assistance with identification was sought and a representative isolate was sent to the Public Health Laboratories, Ontario Department of Health, Toronto, for further study. Identified there as a "Pasteurella-like organism" it was referred for study to the National Communicable Disease Center, Atlanta, Georgia. NCDC Laboratories identified it as a "Pasteurella - like organism related to *Pasteurella anatipestifer*". Confirmation of its identity as *P. anatipestifer*, serologically identical to the Long Island strain, was provided by Dr. Jessie I. Price of the Cornell University Duck Research Laboratory, Eastport, Long Island, New York.

Discussion

Pasteurella anatipestifer is the cause of an economically important disease of domestic ducks, variously referred to as "new duck disease", duck septicemia, and infectious serositis.

We have been unable to find reports in the literature of *P. anatipestifer* as a cause of disease in free-flying wild waterfowl. Hilbert and Witter,[3] however, mentioned an outbreak of *P. anatipestifer* infection in a flock of semi-domestic Black Ducks and Donahue and Olson[1] isolated four strains of *P. anatipestifer* from naso-pharyngeal swabs taken from 50 Canada Geese trapped at Swan Lake, Missouri, in 1967. One of nine mallard ducklings which Donahue and Olson inoculated with *P. anatipestifer* died 3 days later with gross lesions in the spleen and air sacs. *P. anatipestifer* was recovered on culture. Dr. Jessie I. Price[5] reported isolations of *P. anatipestifer* from four Black Ducks and two Mallards submitted to the Long Island Duck Research Laboratory during the years 1959 to 1964.

The source of the infection in the present outbreak is unknown. In view of the isolation of *P. anatipestifer* from the nasopharynges of 4 of 50 trapped Canada Geese, it seems that a possible source was the adult Blue and Snow Geese which were used as subjects for imprinting the goslings. These adult geese may have been latently or subclinically infected. It is of interest that all of the geese and ducks which died were juveniles, most of them less than a month old. The infected ducks, five Mandarin Ducks, one Black Duck and one Wood Duck were all kept in outdoor pens beside the brooder house. They were infected late in the outbreak, after infection occurred in the brooder house when the geese were returned to the brooders. They had no direct contact with the geese but may have been exposed to an infected aerosol, since the pens were immediately below the open windows of the brooder house. The total waterfowl population at Niska at the time of the outbreak was some 2000 birds, including 60 species.

Chloramphenicol treatment seemed to be of benefit in controlling the outbreak. When tested in vitro, the strain of *P. anatipestifer* isolated from the White-Fronted Goose was found to be sensitive to all of the following antibiotics: penicillin, streptomycin, tetracycline, neomycin, chloramphenicol, ampicillin and nitrofurazone.

Difficulty in isolating *P. anatipestifer* has been reported also by other workers.[3,4] If the present outbreak had been limited to just a few birds, it is possible that a correct diagnosis would not have been made. None of us who observed the lesions and the causative agent had had any prior experience with *P. anatipestifer* or the disease it produced. Plans have been made to make a survey for *P. anatipestifer* carriers among adults of the many species of ducks, geese and swans at Niska. Some of these resident waterfowl are free-flying; thus there is ample opportunity for exchange of pathogens between resident birds and wild transient waterfowl on the Niska ponds and on the adjacent Speed River.

Acknowledgments

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Note added in proof: B. L. Munday has drawn our attention to the occurrence of *P. anatipestifer* infection in free-living Black Swans *(Cygnus atratus)* in Australia. The original description of the outbreak was published in 1966 (Munday, B. L. "Diseases of Tasmania's Free-Living Animals", Agricultural Research Bulletin, Tasmania, No. 5: page 29). A description of the strain of *P. anatipestifer* isolated from the swans is in a paper by Munday, et al., 1970, Aust. Vet. J. 46: 322-325.