



AN OUTBREAK OF SCHISTOSOMIASIS IN ATLANTIC BRANT GEESE, BRANTA BERNICLA HROTA

Authors: Wojcinski, Zbigniew W., Barker, Ian K., Hunter, D. Bruce, and Lumsden, Harry

Source: Journal of Wildlife Diseases, 23(2) : 248-255

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-23.2.248>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

AN OUTBREAK OF SCHISTOSOMIASIS IN ATLANTIC BRANT GEESE, *BRANTA BERNICLA HROTA*

Zbigniew W. Wojcinski,¹ Ian K. Barker,¹ D. Bruce Hunter,² and Harry Lumsden³

¹ Department of Pathology, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1, Canada

² Department of Clinical Studies, Ontario Veterinary College, University of Guelph,
Guelph, Ontario N1G 2W1, Canada

³ Ontario Ministry of Natural Resources, Wildlife Research Section, Maple, Ontario L0J 1E0, Canada

ABSTRACT: A heavy infection with schistosomes of the genera *Trichobilharzia* and *Dendritobilharzia* was considered the cause of 90% mortality in a group of 40 wild-caught Atlantic brant geese (*Branta bernicla hrota*) that were maintained in captivity on a fresh-water pond in Aurora, Ontario. Numerous adult worms and eggs were disseminated in many organs throughout the body of all birds examined. The main pathological findings, attributed to both eggs and adults, included emaciation, thrombosis of the caudal mesenteric vein and its branches, fibrinohemorrhagic colitis, and in some birds, hepatomegaly. Translocation of brant geese from their natural marine environment to a fresh-water pond may have caused them to be exposed to parasites which they would not normally encounter.

Key words: *Branta bernicla hrota*, Atlantic brant, *Trichobilharzia* sp., *Dendritobilharzia* sp., schistosomiasis, pathology.

INTRODUCTION

Schistosomes commonly occur in waterfowl (McDonald, 1969; Farley, 1971), but they are rarely reported as a cause of mortality (Farr and Blankemeyer, 1956; Levine et al., 1956; Pence and Rhodes, 1982; Wilson et al., 1982). Schistosomes are often overlooked at gross necropsy because of their small size, occurrence within blood vessels and the low degree of pathology associated with infection. This study describes an outbreak of clinical schistosomiasis in a group of translocated captive Atlantic brant geese (*Branta bernicla hrota*).

CASE HISTORY

During the week of 6–12 March 1981, 40 Atlantic brant geese were caught on a golf course on Long Island, New York. They were shipped by air on 13 March to Toronto, then transported 70 km north to Aurora, Ontario, where they were wing-clipped and immediately released in an enclosure.

The birds had access to two spring-fed ponds with muddy or silty bottoms and sandy shores. Aquatic vegetation and algal

growth were plentiful, and combined with slow water flow through the ponds, provided an ideal environment for invertebrates, especially molluscs. Numerous other captive and wild waterfowl (Canada geese, mallards, etc.) also shared these ponds.

On 20 July 1981 a single brant goose was found dead. The first bird showing signs of lameness was recorded on 27 July. The number of brant geese limping increased to 18 by 30 August. However, by 12 September, most birds had recovered and only eight were still lame. During this period (27 July–12 September), three brant geese died and one was killed. However, a diagnosis was not made at this time.

On 9 November, one brant was killed by an owl, and two more died January 1982. Thus, by the spring of 1982, 32 of the original 40 brant geese remained alive. On 2 May, another individual was found dead. In late June, some birds started to limp and by 3 July, eight birds showed marked lameness. Another bird was found dead on 14 July. During the period between 3 August and 27 September 1982, the greatest mortality occurred; 25 birds died (one of which was killed). Finally,

following the death of a single bird 11 October, only four of the original 40 remained alive by the spring of 1983.

The clinical signs were nonspecific ill-thrift, weight loss, and lameness. Often, the birds were merely found dead. The dead birds weighed 800–900 g, which is approximately one-half their normal weight (Bellrose, 1980). Other species of waterfowl inhabiting these ponds were apparently unaffected.

MATERIALS AND METHODS

Sixteen frozen carcasses and one freshly killed bird were necropsied and gross lesions described. Major blood vessels were examined under a dissecting microscope for schistosomes. Mature specimens were teased from thrombi, fixed in 10% neutral buffered formalin, stained in Semichon's acetic carmine (Noble and Noble, 1962) and mounted in permount. Mucosal scrapings and feces were collected from the colon of frozen carcasses and a sucrose centrifugation-flotation technique was used to isolate schistosome eggs. Eggs were examined microscopically and measured with a calibrated ocular micrometer. Representative tissues (listed in Table 2) were collected from the carcasses, fixed in 10% neutral buffered formalin, processed routinely, sectioned at 6–8 μ m and stained with hematoxylin and eosin (H&E) for light microscopic examination. The number of cross-sections of worms in tissue was determined in each of several randomly selected low power fields (10 \times objective) per tissue. The number of eggs was similarly determined in five randomly selected low power fields per tissue.

Snails were collected for identification by random spot dredging of the ponds. These were examined as squash preparations by light microscopy.

RESULTS

Gross findings

All birds examined were markedly emaciated and dehydrated with reduced pectoral muscle mass and prominent keels. Gallbladders were distended with bile and the gastrointestinal tracts were devoid of ingesta. Occasionally, incidental cestodes and nematodes were present in the intestinal tract. The most consistent findings included thrombosis of the caudal mes-

enteric vein and its branches. The distal two-thirds of the colon was thickened and congested, containing a granular fibrino-hemorrhagic exudate adherent to the mucosal surface. Pulmonary congestion and hepatomegaly were variable findings. Freezing artifacts often obscured further interpretation of gross lesions.

Dissection of the thrombosed vessels revealed numerous intravascular parasites of two distinct morphological types. Long white thread-like trematodes were found within many thrombosed veins throughout the body, including the caudal mesenteric and hepatic portal veins (Fig. 1). A second, more robust and flattened parasite was found in the branches of the abdominal aorta.

Parasite identification

Identification of these vascular parasites was based on their morphology and location within the host. The white thread-like specimens were present in greatest numbers and consistently located in the venous system. Measurement of the parasites was difficult owing to their fragility and entanglement with each other and in thrombi. Parasite fragments often exceeded 10 mm in length. These worms were identified as belonging to the genus *Trichobilharzia* (McDonald, 1981), probably *T. brantae* (D. B. Pence, pers. comm.). Representative specimens are deposited in the National Museum of Canada Invertebrate Collection, Ottawa, Ontario (Accession No. NMCP 1985-0063, -0064).

The robust flat parasites (measuring 12.0 \times 1.5 mm) were found only within the arterial system and occurred in lower numbers. These were identified as belonging to the genus *Dendritobilharzia* (McDonald, 1981), probably *D. pulverulenta* (D. B. Pence, pers. comm.). Representative specimens are deposited in the National Museum of Canada Invertebrate Collection, Ottawa, Ontario (Accession No. NMCP 1985-0065, -0066, -0067).

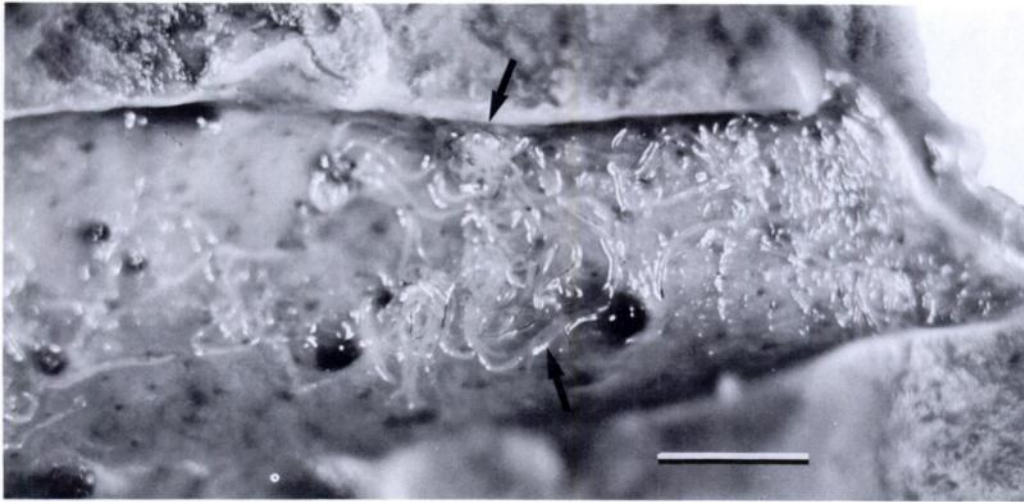


FIGURE 1. Numerous *Trichobilharzia* sp. (arrows) within an opened hepatic vein. H&E. (Bar = 13.3 mm.)

Eggs recovered from mucosal scrapings of colon were oval and measured $83 \times 125 \mu\text{m}$. A delicate spine was present at one end and often a miracidium was evident within the egg. While lacking the typical spindle shaped appearance of most schistosome eggs, these eggs are similar in size and shape to at least two described *Tri-*

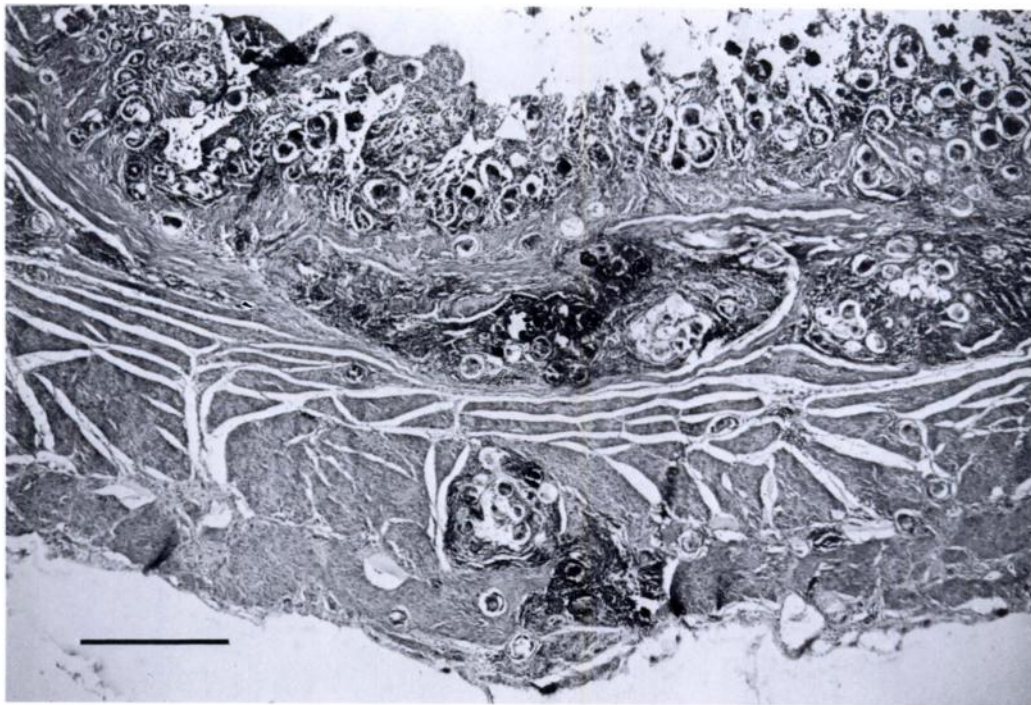


FIGURE 2. Transmural section of colon demonstrating the large numbers of viable, degenerate and/or mineralized eggs inciting a granulomatous reaction in the mucosa and multi-focally in the submucosa and muscularis layers. H&E. (Bar = $800 \mu\text{m}$.)

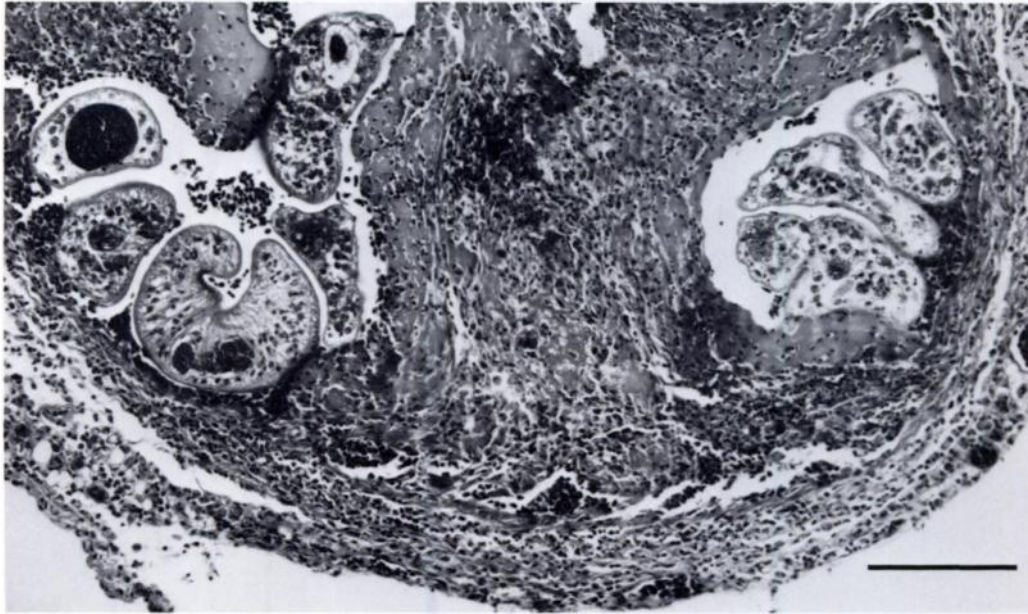


FIGURE 3. Cross-sections of adult *Trichobilharzia* sp. within a thrombosed mesenteric vein. Severe vasculitis and perivascular inflammation is evident around the affected vessel. H&E. (Bar = 200 μ m.)

chobilharzia spp., *T. brantae* and *T. filliformis* (Farr and Blankemeyer, 1956).

Snail identification

Snails collected from the ponds were identified as *Physa gyrina* and *Helosoma trivolvis* (Goodrich, 1932). The *Physa gyrina* were most prevalent. Squash preparations of the snails revealed numerous empty sporocysts, but cercariae were not recovered.

Microscopic findings

The most dramatic microscopic lesions were in the terminal colon. Many viable, degenerate and/or mineralized eggs were present transmurally in the colon causing a severe granulomatous reaction characterized by a pronounced mixed inflammatory cell infiltrate consisting of histiocytes, fibroblasts, plasma cells and lymphocytes, with fewer heterophils and giant cells (Fig. 2). Mucosal destruction was marked in acute lesions, while in more chronic areas fibrosis and thickening of the colon wall were the prominent features. Extensive ex-

udation of fibrin, cellular debris and eggs was a consistent finding in the colonic lumen.

The eggs had a thick eosinophilic shell that was often convoluted as a result of artifactual contraction. The degree of mucosal destruction correlated positively with the number of eggs present. Viable eggs were present free in the lamina propria, while degenerate or mineralized eggs were most often associated with granulomas.

Mature schistosomes were found in the serosal and mesenteric blood vessels. Adults of the genus *Trichobilharzia* were identified in section, distinguished by their long filamentous appearance. Furthermore, similar to previous studies (McMullen and Beaver, 1945; Pence and Rhodes, 1982), the majority appeared to be males based on the presence of sperm packets in the coelomic cavity. Females containing eggs were not observed microscopically. However, adults were usually associated with thrombi and eggs were often observed in close proximity. Extensive hemorrhage and perivascular mononuclear cell infiltration

TABLE 1. Distribution and numbers of adult *Trichobilharzia* sp. and *Dendritobilharzia* sp. in various tissues of captive brant geese.

Location	Mean score ^a	Range of score	Number affected/number examined
<i>Trichobilharzia</i> sp.			
Caudal renal vein	2.88	0-4	6/8
Branches of gonadal veins	2.25	1-4	8/8
Hepatic portal vein	2.00	0-4	7/8
Mesenteric vein	1.75	0-4	5/8
Branches of gastric veins	0.75	0-2	4/8
Pulmonary vein	0.62	0-2	3/8
Esophageal vein	0.50	0-1	2/4
Jugular vein	0.50	0-2	2/8
Femoral vein	0.25	0-2	1/8
Thyroidal vein	0.67	0-2	1/3
<i>Dendritobilharzia</i> sp.			
Caudal mesenteric artery	1.25	0-1	2/8
Cranial mesenteric artery	0.12	0-1	1/8
Renal artery	0.12	0-1	1/8
Meningeal artery	0.33	0-1	1/3

^a 0, no worms observed; 1, 1 worm per low power field; 2, 2-5 worms per low power field; 3, 6-10 worms per low power field; 4, >10 worms per low power field.

were present around most vessels (Fig. 3). Vasculitis was less common. Adults of the genus *Dendritobilharzia* were rarely identified in cross-sections of arteries.

The mucosa of the small intestine was intact and only rarely were degenerating eggs observed in the lamina propria. Arteries and veins in other organs (Tables 1, 2) in the body often contained eggs and mature schistosomes (Fig. 4) resulting in thrombosis and perivascularitis. Eggs were found in a variety of tissues inciting lesions varying from none to marked granulomatous reactions (Fig. 5).

DISCUSSION

The mortality in this group of brant geese was attributed to the effects of infection with schistosomes. The clinical signs of ill-thrift, weight loss and emaciation probably

TABLE 2. Distribution and density of schistosome eggs in tissues from captive brant geese.

Location	Mean number of eggs per LPF ^a	Number of birds affected
Lung	3.23	7/7
Liver	1.75	8/8
Kidney	45.31	7/7
Spleen	0.27	1/8
Colon	76.00	8/8
Small intestine	0.15	1/8
Adrenal/gonads	3.15	6/8
Brain	1.50	2/2
Gizzard	1.32	2/8
Proventriculus	0.05	2/8
Esophagus	0.05	1/8
Heart	0	0/5
Pancreas	0	0/5
Skeletal muscle	0.40	1/4
Foot web	0.80	1/1

^a LPF, low power field 10× objective (5 fields examined/tissue/bird).

resulted from both protein-loss from the severe colitis and inanition due to the systematic illness associated with the heavy parasite infection. Lameness may have been a non-specific sign, often seen in sick waterfowl without any attributable lesions. A possible explanation was thrombosis of the femoral vessels, although this was an inconsistent finding at necropsy.

The lesions associated with avian schistosomiasis vary with schistosome species, the species of host and with individuals in the same host population. Minor lesions are usually egg-induced and not the result of adult or immature schistosome activity (Pence and Rhodes, 1982) reflecting the well-adapted host-parasite relationship in their natural environment. However, there are occasional reports of severe lesions and mortality in waterfowl caused by immature or adult schistosomes. Pence and Rhodes (1982) described an outbreak of mortality in endemic wild ducks in Texas, caused by *Trichobilharzia physellae*. The lesions were attributed to mature schistosomes and were confined to the liver. Adult schistosomes were also observed in the mesenteric veins, but there was no evi-



FIGURE 4. Numerous filamentous *Trichobilharzia* sp. worms and several eggs (arrows) in a thrombosed renal vein. H&E. (Bar = 600 μ m.)

dence of a tissue response in this site. Schistosome eggs associated with granulomas were occasionally observed only in the mucosa and submucosa of the intestine. McMullen and Beaver (1945) reported lung and liver lesions in ducklings, pigeons and canaries which they attributed to the migration of schistosomes in experimental infections. McLeod and Little (1942) described an enlarged liver with abscessation due to migration of immature schistosomes in a pigeon exposed to *Cercariae elvae*.

Other reports of schistosome-associated mortality in waterfowl are related to the aberrant location of adults and eggs within the host. Levine et al. (1956) described encephalitis in a swan (*Cygnus cygnus*) attributed to schistosomes of the genus *Dendritobilharzia*. Adult schistosomes were observed in meningeal arteries, but the lesions were attributed to the inflammatory reaction towards the eggs. Eggs were also found in the villi of the small intestine and in the liver, pancreas, and gizzard mucosa.

Farr and Blankemeyer (1956) attributed mortality in Canada geese to schistosomes identified as *Trichobilharzia brantae* re-

covered from mesenteric and renal veins. The pathological findings, however, were not described.

Wilson et al. (1982) described mortality in a group of mute swans (*Cygnus olor*) associated with infection by *Dendritobilharzia* sp. Gross lesions were not seen in the birds, but microscopic examination of the brain revealed disseminated granulomatous lesions containing schistosome eggs in the cerebrum and cerebellum. Similar granulomas were also observed in the intestines, lungs and liver. Mature schistosomes were found in the mesenteric artery. It is a characteristic of members of the genus *Dendritobilharzia* to live in arteries rather than veins, the typical location of avian blood flukes (Vande Vusse, 1980).

In our study, the extent of lesions and distribution of the two types of schistosomes were of significance. Worms of the genus *Trichobilharzia* were present in the greatest numbers and probably produced the majority of eggs. Lesions in most organs appeared to be the result of the aberrant distribution of immature and mature schistosomes and eggs of this genus. The

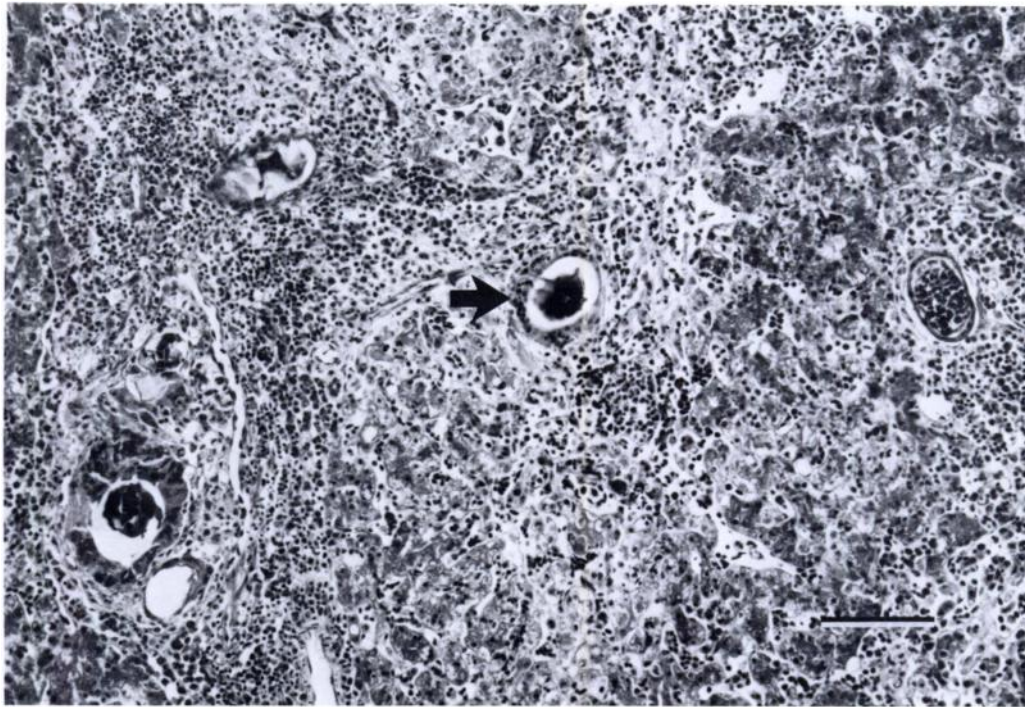


FIGURE 5. Degenerate schistosome eggs randomly distributed in the hepatic parenchyma often inciting a granulomatous reaction (arrow). H&E. (Bar = 150 μ m.)

brain lesions were more likely due to the *Dendritobilharzia* sp. based on the presence of adults in the meningeal arteries, which is in agreement with previous reports (Levine et al., 1956; Wilson et al., 1982). The natural hosts for *Dendritobilharzia* sp. are diving ducks, in which adult worms live in the caudal mesenteric artery, producing eggs which are passed in the feces of the bird (Vande Vusse, 1980). In dabbling ducks, geese and swans the trematodes are found in the aorta and its cranial branches and other abnormal sites, and infections are nonpatent due to marked tissue destruction associated with embolization of the eggs in various organs (Vande Vusse, 1979).

Snails were the likely source of infection even though we were unable to positively identify cercariae in snails obtained from the ponds on which these geese were maintained. During the summer months when cercariae are most abundant and active in

fresh-water lakes and ponds of the Great Lakes region (Jarcho and Burkalow, 1952), brant geese are normally found in an arctic marine environment (Bellrose, 1980), possibly well out of the range of the parasite. *Trichobilharzia brantae* and *D. pulverulenta*, to our knowledge, have not been reported previously to infect *B. bernicla*. We suspect that translocating and maintaining brant geese in captivity on fresh-water ponds during the summer exposed them to parasites that they would not normally encounter. The endemic waterfowl that shared this pond showed no signs of illness or mortality from these parasites and likely have a well-established host-parasite relationship.

ACKNOWLEDGMENTS

This study was supported by the Ontario Ministry of Natural Resources. The authors wish to thank Drs. J. P. Lautenslager and B. M. McCraw, and Mrs. J. Bagg for their technical as-

sistance. Dr. Danny B. Pence kindly identified the species of schistosomes involved.

LITERATURE CITED

- BELLROSE, R. C. 1980. Ducks, geese and swans of North America, 3rd ed. Stackpole Books, Harrisburg, Pennsylvania, pp. 166–171.
- FARLEY, J. 1971. A review of the family Schistosomatidae: Excluding the genus *Schistosoma* from mammals. *Journal of Helminthology* 45: 289–320.
- FARR, M. M., AND V. G. BLANKEMEYER. 1956. *Trichobilharzia brantae* n. sp. (Trematoda: Schistosomatidae) from the Canada goose (*Branta canadensis* L.). *Journal of Parasitology* 42: 320–325.
- GOODRICH, C. 1932. The Mollusca of Michigan. Michigan Handbook Series No. 5, The University of Michigan Press, Ann Arbor, Michigan, 120 pp. and 7 plates.
- JARCHO, S., AND A. U. BURKALOW. 1952. A geographical study of swimmer's itch in the United States and Canada. *Geographical Review* 42: 212–226.
- LEVINE, N. D., D. T. CLARK, AND L. E. HANSON. 1956. Encephalitis in a swan due to *Dendritobilharzia* sp. (Trematoda: Schistosomatidae). *Journal of Parasitology* 42: 496–500.
- MCDONALD, M. E. 1969. Catalogue of helminths of waterfowl (Anatidae). Bureau of Sport Fisheries and Wildlife Special Scientific Report—Wildlife No. 126, Washington, D.C., 692 pp.
- . 1981. Key to trematodes reported in waterfowl. U.S. Department of the Interior, Fish and Wildlife Service, Resource Publication 142, Washington, D.C., 156 pp.
- MCLEOD, J. A., AND G. E. LITTLE. 1942. Continued studies on cercarial dermatitis and the trematode family Schistosomatidae in Manitoba, Part I. *Canadian Journal of Research* 20: 170–181.
- McMULLEN, D. B., AND P. C. BEAVER. 1945. Studies on schistosome dermatitis. IX. The life cycles of three dermatitis-producing schistosomes from birds and a discussion of the subfamily Bilharziellinae (Trematoda: Schistosomatidae). *American Journal of Hygiene* 42: 128–155.
- NOBLE, E. R., AND G. A. NOBLE. 1962. Animal parasitology laboratory manual. Lea and Febiger, Philadelphia, Pennsylvania, 120 pp.
- PENCE, D. B., AND M. J. RHODES. 1982. *Trichobilharzia physellae* (Digenea: Schistosomatidae) from endemic waterfowl on the High Plains of Texas. *Journal of Wildlife Diseases* 18: 69–74.
- VANDE VUSSE, F. J. 1979. Host-parasite relations of *Dendritobilharzia pulverulenta* (Trematoda: Schistosomatidae) and anatids. *Journal of Parasitology* 65: 894–897.
- . 1980. A review of the genus *Dendritobilharzia* Skrjabin and Zakharow 1920 (Trematoda: Schistosomatidae). *Journal of Parasitology* 66: 814–822.
- WILSON, R. B., J. C. NEW, AND R. G. SCHOLTENS. 1982. Granulomatous encephalitis caused by schistomiasis in swans. *Journal of the American Veterinary Medical Association* 181: 1386–1387.

Received for publication 9 September 1985.