Chapter 30

Tracheal Worms

Synonyms

Gape worm, syngamiasis, gapes

Cause

Infection by tracheal worms often results in respiratory distress due to their location in the trachea or bronchi and their obstruction of the air passage. Infections by these parasitic nematodes or roundworms in waterbirds, primarily ducks, geese, and swans, are usually due to *Cyathostoma bronchialis* and infection of land birds are usually due to *Syngamus trachea*. However, both genera infect a variety of species, including both land and waterbirds. Infections with *S. trachea* have been more extensively studied than infections with *Cyathostoma* sp. because of its previous importance as a disease-causing parasite of poultry in many parts of the world. Changes in husbandry practices to modern intensive methods for poultry production have essentially eliminated *S. trachea* as an agent of disease in chickens, but it is an occasional cause of disease in turkeys raised on range.

Life Cycle

Tracheal worms have an indirect life cycle (Fig. 30.1) that requires a paratenic or transport host which transmits the infectious larvae to the definitive host bird, where they reach adulthood and reproduce. Adult *S. trachea* reside within the trachea. The female releases fertilized eggs, which are swallowed by the bird and voided with the feces into the soil. Eggs may also be directly expelled onto the ground from the trachea. After embryonation (1–2 weeks), infective larvae develop within the egg. Birds can become infected by eating invertebrate paratenic hosts such as earthworms, snails, slugs, or fly larvae that have consumed the eggs. Infective larvae are released from the egg and become encysted within the bodies of these invertebrates and can remain infective for up to three and one-half years. Upon ingestion by birds, the larvae are believed to penetrate the intestinal wall. Some larvae enter the abdominal cavity but most enter the bloodstream, where they are carried to the lungs. After further development in the lungs, the young worms migrate up the bronchi to the trachea. Larvae can reach the lungs within 6 hours after ingestion and eggs are produced by worms in the trachea about 2 weeks after ingestion of those larvae. *C. bronchialis* is very similar in that earthworms transmit the infective stage to the bird. Infection of birds with *C. bronchialis* by direct consumption of fully embryonated eggs has been documented experimentally; however, worm burdens were extremely low.

Species Affected

Disease caused by tracheal worms is not commonly reported for free-ranging birds within the United States and Canada, but it is common within the United Kingdom and in some other countries. High infection rates within wild birds in England attest to the potential for this parasite to be a serious pathogen. More than 50 percent of nestling and fledgling starlings, more than 85 percent of jackdaws, and 100 percent of young rooks were found to be infected in one study. Infection rates in adult birds were considerably lower, but they still exceeded 30 percent for starlings and rooks. Within the United States, *S. trachea* infections have been reported from wild turkeys, other gamebirds, a variety of passerines, (songbirds), and occasionally from other bird species. Large-scale mortalities have occurred among pheasants and other gamebirds being propagated for sporting purposes. Findings from captive bird collections have led to the conclusion that almost any species of cage or aviary bird is susceptible to infection.

S. trachea has been reported infrequently in waterfowl, but members of the genus *Cyathostoma* sp. are "characteristic" or common parasites of waterfowl. Mortality has been reported for several species of young geese, leading some investigators to suggest that *C. bronchialis* are potentially important pathogens for geese. Juvenile free-ranging sandhill cranes have also been reported to have died from *Cyathostoma* sp. infection.

Distribution

S. trachea and *Cyathostoma* sp. are found worldwide.

Seasonality

Infected birds can be found yearround. Young birds are most commonly affected and, therefore, disease is associated with breeding cycles in the spring to summer months for free-ranging birds.

Field Signs

Most birds that are infected show no signs of disease. In general, the severity of disease is dependent upon the degree of infection and the size of the bird. Small birds are more severely affected than larger birds because their narrower tracheal openings result in greater obstruction by the worms. Respiratory distress is the primary clinical sign of disease. Birds with severe infections open their mouth widely and at the same time stretch out their necks, assuming a "gaping" posture. The adult worms that are attached to the lining of

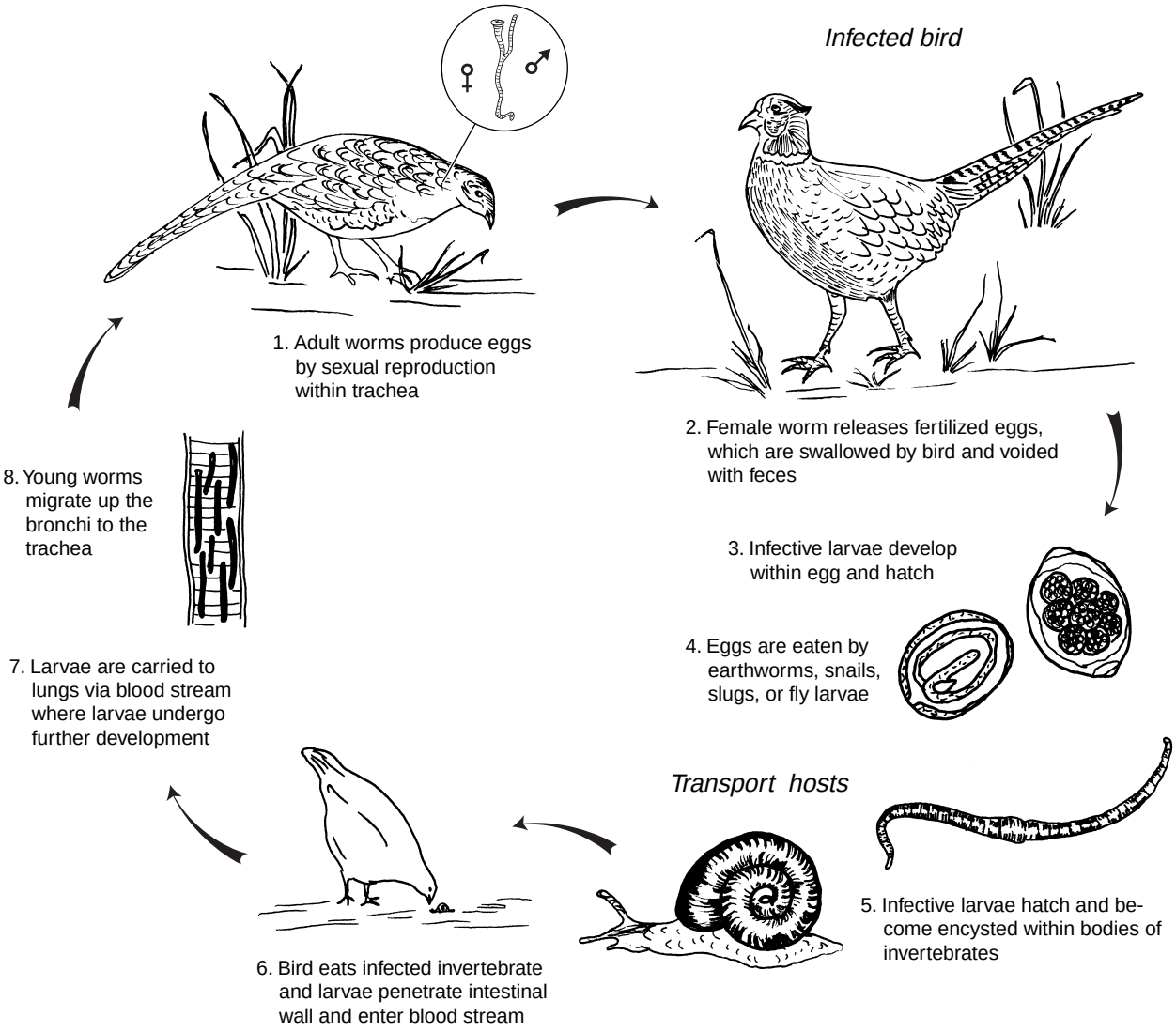

Figure 30.1 *Tracheal worm life cycle.*

the trachea cause irritation and excess mucus production. This often results in agitated bouts of coughing, head shaking, and sneezing as the birds attempt to dislodge the parasites. Severely infected birds may have most or all of the tracheal opening obstructed by worms, may stop feeding, and may rapidly lose body condition.

Gross Lesions

Severely affected birds experience severe weight loss and have poorer development of body mass than uninfected birds, and they often die from starvation (Fig. 30.2). Anemia may also be present due to the blood-feeding habits of the parasites.

Diagnosis

Identification of the worms (Fig. 30.3) and evaluation of any associated disease signs are required for a diagnosis. Clinical signs are not diagnostic because similar signs can be seen with some mite infections, aspergillosis, and wet pox.

Control

There is no feasible method for controlling tracheal worms in free-ranging birds. Disease prevention should be practiced by minimizing the potential for captive-propagation and release programs to infect invertebrates that are then fed upon by free-ranging birds. Land-use practices that provide direct contact between poultry rearing and wild birds and the disposal of bird feces and litter should also be considered because environmental contamination with infective larvae is a critical aspect of the disease cycle.

Human Health Considerations

There are no reports of these nematodes infecting humans.

Rebecca A. Cole

Photo by Milton Friend

Figure 30.2 *Comparative breast muscle mass of noninfected pheasants (left) and those with* ***Syngamus trachea*** *infections (right).*

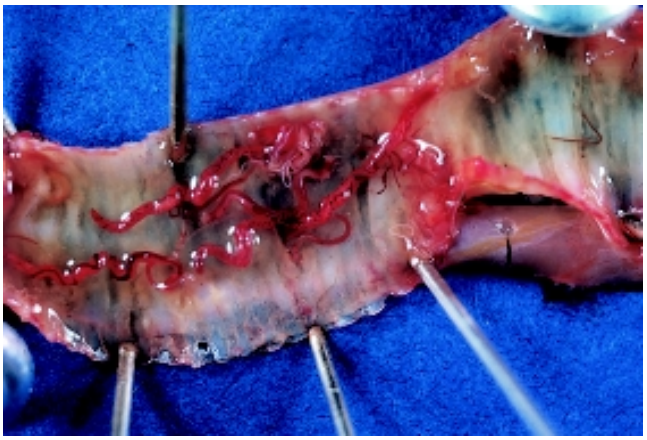

Photo by Milton Friend

Figure 30.3 ***Syngamus trachea*** *in the trachea of a ring-necked pheasant.*

Supplementary Reading

Anderson, R.C., 1992, Nematode parasites of vertebrates: Their development and transmission: Wallingford, England, CAB International, 578 p.

Cram, E.B., 1927, Bird parasites of the nematode suborders Strongylata, Ascaridata, and Spirurata: U.S. National Museum Bulletin No. 140, 465 p.

Fernando, M.A., Hoover, I.J., and Ogungbade, S.G., 1973, The migration and development of *Cyathostoma bronchialis* in geese: Journal of Parasitology, v. 59, p. 759–764.

Threlfall, W., 1965, Life-cycle of *Cyathostoma lari*: Nature, v. 206, p. 1,167–1,168.