Chapter 26

Intestinal Coccidiosis

Synonyms

Coccidiosis, coccidiasis

Cause

Coccidia are a complex and diverse group of protozoan (single-celled organisms) parasites; the coccidia group contains many species, most of which do not cause clinical disease. In birds, most disease-causing or pathogenic forms of coccidia parasites belong to the genus *Eimeria*. Coccidia usually invade the intestinal tract, but some invade other organs, such as the liver and kidney (see Chapter 27).

Clinical illness caused by infection with these parasites is referred to as coccidiosis, but their presence without disease is called coccidiasis. In most cases, a bird that is infected by coccidia will develop immunity from disease and it will recover unless it is reinfected. The occurrence of disease depends, in part, upon the number of host cells that are destroyed by the juvenile form of the parasite, and this is moderated by many factors. Severely infected birds may die very quickly. Often, tissue damage to the bird's intestine results in interrupted feeding; disruption of digestive processes or nutrient absorption; dehydration; anemia; and increased susceptibility to other disease agents. In cranes, coccidia that normally inhabit the intestine sometimes become widely distributed throughout the body. The resulting disease, disseminated visceral coccidiosis (DVC) of cranes, is characterized by nodules, or granulomas, on the surface of organs and tissues that contain developmental stages of the parasite.

Collectively, coccidia are important parasites of domestic animals, but, because each coccidia species has a preference for parasitizing a particular bird species and because of the self-limiting nature of most infections, coccidiosis in free-ranging birds has not been of great concern. However, habitat losses that concentrate bird populations and the increasing numbers of captive-reared birds that are released into the wild enhance the potential for problems with coccidiosis.

Characteristics of Intestinal Coccidiosis

All domestic birds carry more than one species of coccidia, and pure infections with a single species are rare.

Different coccidia species are usually found in a specific location within the intestinal tract of the host bird.

After initial exposure to the parasite, the host bird may quickly develop immunity to it but immunity is not absolute. A bird can be reinfected by the same or a different species of the parasite.

Infections do not generally cause a problem of free-ranging birds; instead, coccidiosis is considered a disease of monoculture and of the raising of birds in confinement.

Life Cycle

Most intestinal coccidia have a complex but direct life cycle in which the infective forms of the parasite invade a single host animal for development to sexual maturity; the life cycle is completed in 1–2 weeks (Fig. 26.1). A mature female parasite in the intestine of an infected host bird produces noninfective, embryonated eggs or oocysts, which are passed into the environment in the feces of the host bird. The oocysts quickly develop into an infective form while they are in the environment. An uninfected bird ingests the infective oocysts while it is eating or drinking, and the infective oocysts invade the bird's intestine. Within the intestine, the oocysts may or may not undergo several stages of development, depending on the parasite species, before they become sexually mature male and female parasites. The complex life cycle for *Eimeria* (Fig. 26.2) illustrates the exponential rate of infection and destruction of the intestinal epithelial cells, which are the cells that provide the covering of the intestinal lining. The mature female parasites release noninfective oocysts to the environment, and, thus, the cycle begins anew.

Species Affected

Many animal species, including a wide variety of birds (Table 26.1) may harbor coccidia. Although disease is not common in free-ranging wild birds, several epizootics due to *E. aythyae* have been reported among lesser scaup in the United States. During those events, predominantly females have died, which suggests that female lesser scaup may be more susceptible to the disease than male lesser scaup. Lesions of DVC were first seen in captive sandhill cranes in the late 1970s. Since then, mortality of captive sandhill and whooping cranes has been attributed to DVC, and the disease has been found in wild sandhill cranes, including the endangered Mississippi sandhill crane.

Distribution

Coccidia are found worldwide. The few reported outbreaks of coccidiosis in free-ranging waterfowl have all occurred in the Midwestern United States (Fig. 26.3). Recurrent epizootics have broken out at a single reservoir in eastern Nebraska, and coccidiosis is also believed to be the cause of waterfowl die-offs in Wisconsin, North Dakota, Illinois, and Iowa. DVC has been found in migratory sandhill cranes at several locations, and it is a recurring problem in the only free-ranging population of the nonmigratory Mississippi sandhill crane. These birds reside at the Mississippi Sandhill Crane National Wildlife Refuge in Mississippi.

Seasonality

Birds may be infected with coccidia at any time. Although little is known about the conditions that may lead to the development of clinical disease in wild birds, birds may become diseased more frequently during periods of stress. Most epizootics of intestinal coccidiosis in waterfowl in the Upper Midwest have broken out in early spring, during a stressful staging period of spring migration. Mississippi sandhill cranes also die from DVC most frequently during the spring.

Field Signs

Field signs for free-ranging wild birds have not been reported. Nonspecific clinical signs reported for captive birds include inactivity, anaemia, weight loss, general unthrifty appearance, and a watery diarrhea that may be greenish or bloody. Tremors, convulsions, and lameness are also occasionally seen. Rapid weight loss may lead to emaciation and dehydration followed by death. Young birds that survive severe infections may suffer retardation of growth.

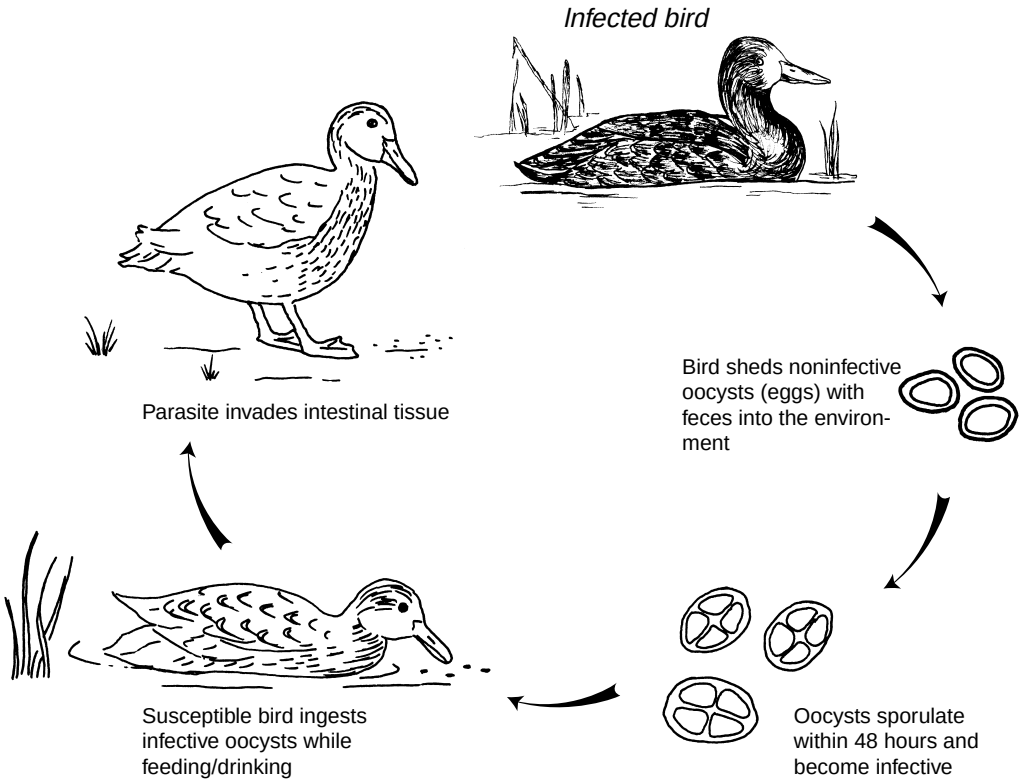
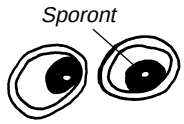
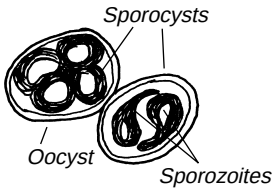


Figure 26.1 *Direct life cycle of* ***Eimeria*** *infection in birds.*

A. Noninfective parasite oocysts (eggs) containing a single cell referred to as the sporont are passed via feces into the environment.



B. Oocysts become infective after 2 days in the environment at ordinary temperatures through sporolation (sporogony), which is a developmental process that results in the sporont dividing and forming four sporocysts each containing two infective sporozoites.

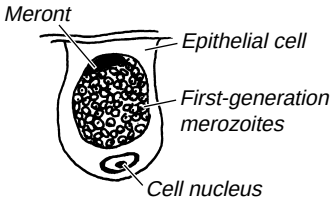


C. Infective oocysts are ingested by birds in contaminated feed, water, soil, or other ingesta.

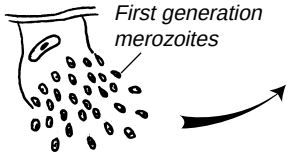
D. The oocyst wall breaks within the gizzard of the bird and releases the sporocysts.

E. The sporozoites escape from the sporocysts in the small intestine and enter the epithelial cells, which are cells that line the internal and external surfaces of the body of the intestine.

F. The sporozoites develop within the epithelial cells, and asexual multiple fission results in the formation of first-generation meronts, each of which produces about 900 first-generation merozoites.

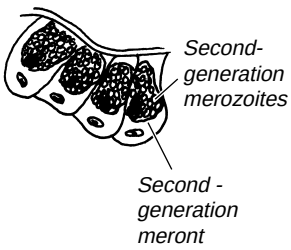


G. Merozoites break out of the epithelial cells into the intestinal canal about 2.5–3 days after infection. The merozoites enter new host cells and undergo developmental processes resulting in the formation of second-generation meronts. By dividing many times, each of these meronts produce about



200–350 second-generation merozoites that are 4–8 times larger in size than the first-generation merozoites and that are produced about 5 days after initial oocyst ingestion.

H. The cycle may continue with a third generation of a small number (4–30) of merozoites of intermediate size (between those of the first and second generation). However, many of the second-generation merozoites enter new host cells and begin the sexual phase of the life cycle referred to as gamogony.



I. Most of the second-generation merozoites develop into female gametes or macrogamonts and some become males or microgamonts. The females grow until they reach full size while a large number of tiny microgametes are formed within each of the microgamonts. The macrogamonts are fertilized by the microgametes and new oocysts result.

J. Seven days after ingestion of infected coccidia, the oocysts break out of their host cells and enter the intestinal canal to be passed from the body via feces to continue the cycle.

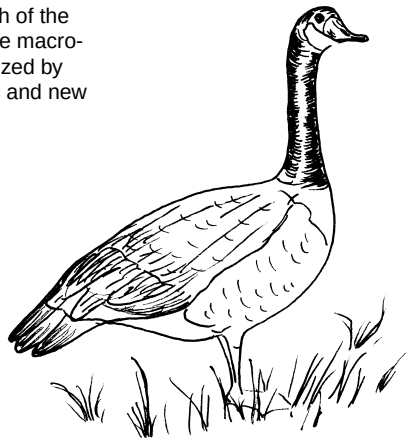


Figure 26.2 *A typical life cycle of* ***Eimeria*** *sp. in birds. (Adapted from* ***Eimeria tenella*** *in chickens.)*

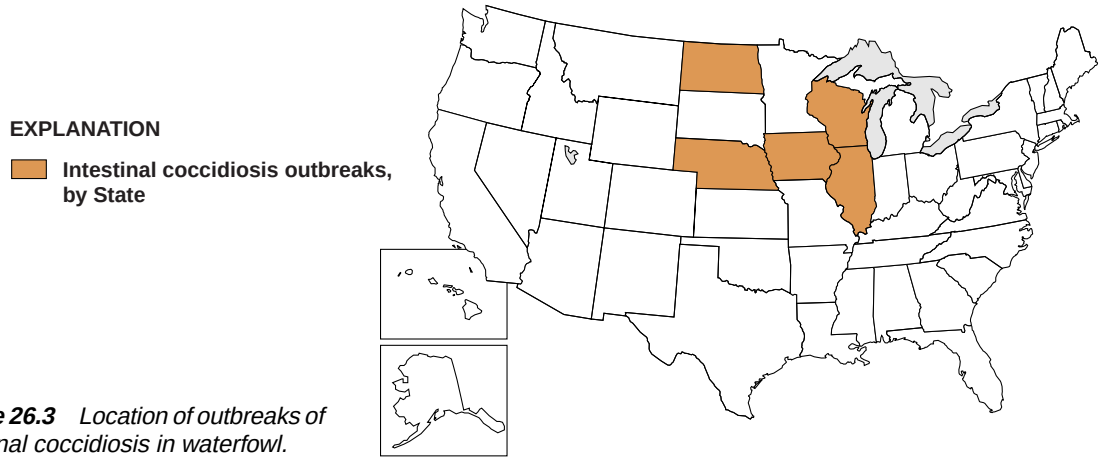


Figure 26.3 *Location of outbreaks of intestinal coccidiosis in waterfowl.*

Table 26.1 *Relative occurrence of coccidia in different groups of birds. [Frequency of occurrence: ● occasional, ● common, — not reported]*

Bird types (and examples)	Coccidia species				
	Eimeria sp.	***Isospora*** sp.	***Tyzzeria*** sp.	***Cryptosporidium*** sp.	***Wenyonella*** sp.
Poultry (Chicken, turkey)	●	—	—	●	—
Anseriformes (Ducks, geese)	●	●	●	●	●
Charadriiformes (Gulls, shorebirds)	●	—	—	—	—
Columbiformes (Pigeons, doves)	●	—	—	—	●
Coraciiformes (Kingfishers)	—	●	—	—	—
Falconiformes (Hawks, falcons)	—	●	—	—	—
Galliformes (Pheasant, quail)	●	●	—	●	—
Gruiformes (Cranes, rails)	●	—	—	—	—
Passeriformes (Songbirds)	—	●	—	—	—
Pelicaniiformes (Pelicans)	●	—	—	—	—
Piciformes (Woodpeckers)	—	●	—	—	—
Psittaciformes (Parrots)	●	●	—	●	—
Strigiformes (Owls)	—	●	—	—	—
Struthioniformes (Ostriches)	—	●	—	—	—

Gross Lesions

The location of lesions varies with the species of coccidia and the severity and intensity of infection. In acutely-affected lesser scaup, bloody inflammation or enteritis is commonly seen in the upper small intestine (Fig. 26.4A). In scaup that survive for longer periods, dry crusts form on the mucosal (internal) surface of the intestinal tract. The severity of this lesion decreases from the small intestine to the large intestine (Fig. 26.4B). Chronic lesions of intestinal coccidiosis take other forms in different species, sometimes appearing as rather distinct light-colored areas within the intestinal wall (Fig. 26.5).

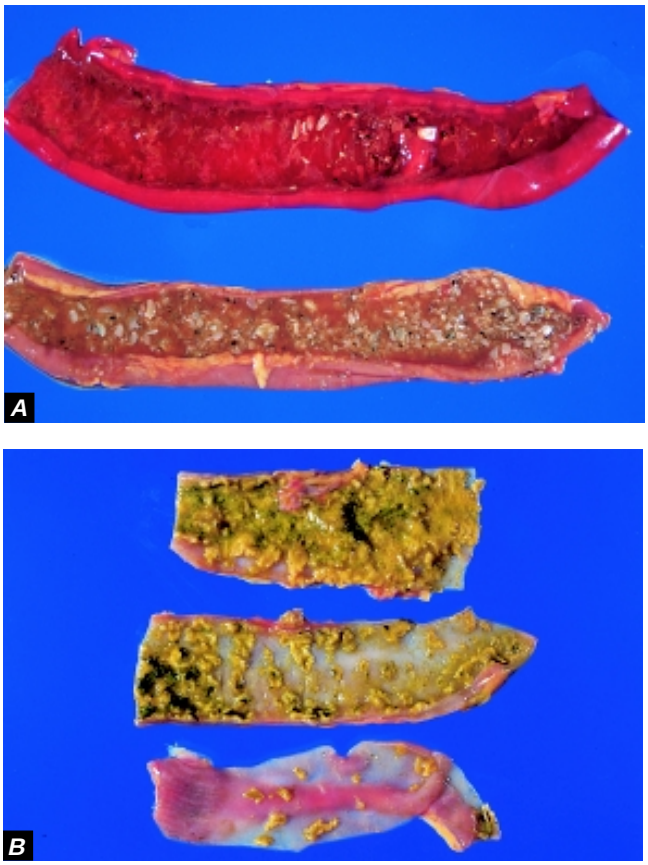
Lesions of DVC in cranes typically consist of small (usually less than 5 millimeters in diameter), raised, light-colored granulomas. These nodules may be found on any surface within the body cavity, but they are commonly seen on the lining of the esophagus near the thoracic inlet area and on the inner surface of the sternum (Fig. 26.6A–C). Light-colored patches may also appear on and within organs such as the heart and liver (Fig. 26.7A, B).

Diagnosis

When large numbers of oocysts are found in the feces of live birds concurrent with diarrhea, emaciation, and pallor or pale skin color, coccidiosis should be suspected as the cause of illness. However, a diagnosis of coccidiosis as cause of death requires a necropsy evaluation combined with identification of the causative coccidia. Fecal evaluations are not adequate for a diagnosis of coccidiosis because disease may develop before large numbers of oocysts are present in feces and because oocysts seen in the feces may not be those of pathogenic species. As with other diagnostic evaluations, submit chilled, whole carcasses for necropsy by qualified specialists. When carcasses cannot be provided, remove intestinal tracts and submit them chilled. If submissions will be delayed for several days or longer and carcasses cannot be preserved by freezing, remove the entire intestinal tract and preserve it in an adequate volume of neutral formalin (see Chapter 3).

Control

Oocysts can rapidly build up in the environment when birds are overcrowded and use an area for a prolonged period of time. The disease risk increases significantly when these conditions result in oocyst contamination of food and drinking water. In captive situations, good husbandry and sanitation, including continual removal of contaminated feed and litter, can minimize the potential for coccidiosis. Captive birds can be treated with therapeutic agents that control, but that do not eliminate, the level of infection. Therefore, oocyst shedding by those birds after they are removed from therapy should be considered if they are to be released or mixed with other birds. Light infections result in a substantial level of immunity to that species of coccidia and are use-



Photos by J. Christian Franson

Figure 26.4 ***(A)*** *Hemorrhage in the small intestine of a lesser scaup with acute intestinal coccidiosis (upper part of photo), compared with normal small intestine (lower part of photo).* ***(B)*** *Dry, crust-like lesions in the intestinal tract of a lesser scaup with chronic intestinal coccidiosis. The lesions are most severe in the upper small intestine (top section in photo). The severity decreases in lower parts of the intestine (middle and bottom sections in photo).*

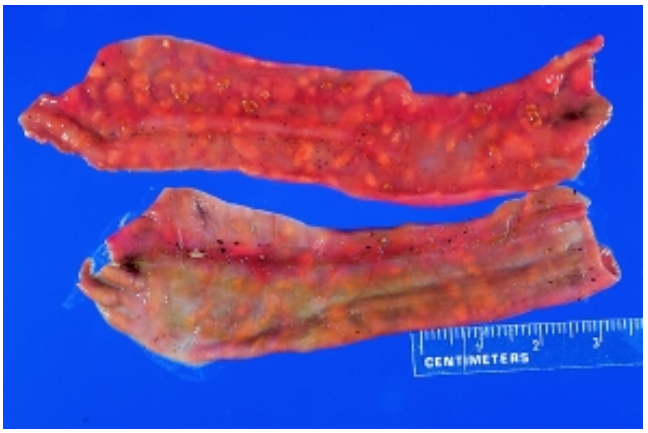
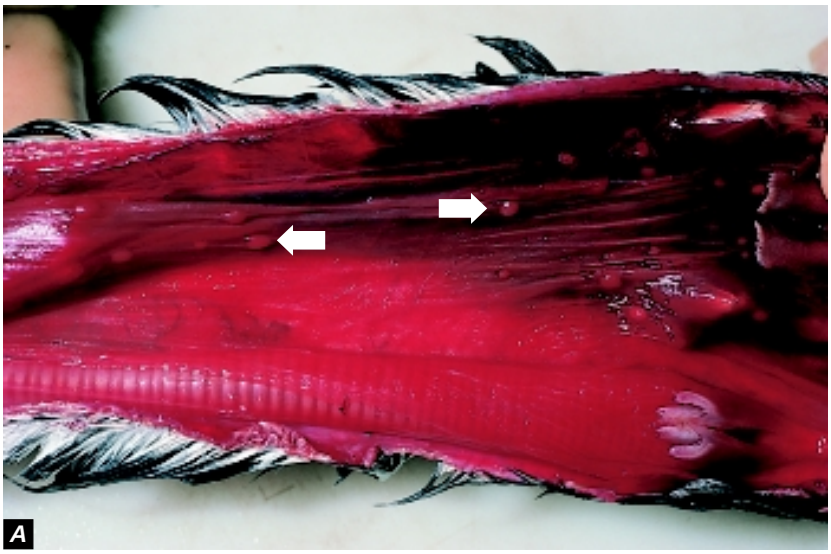
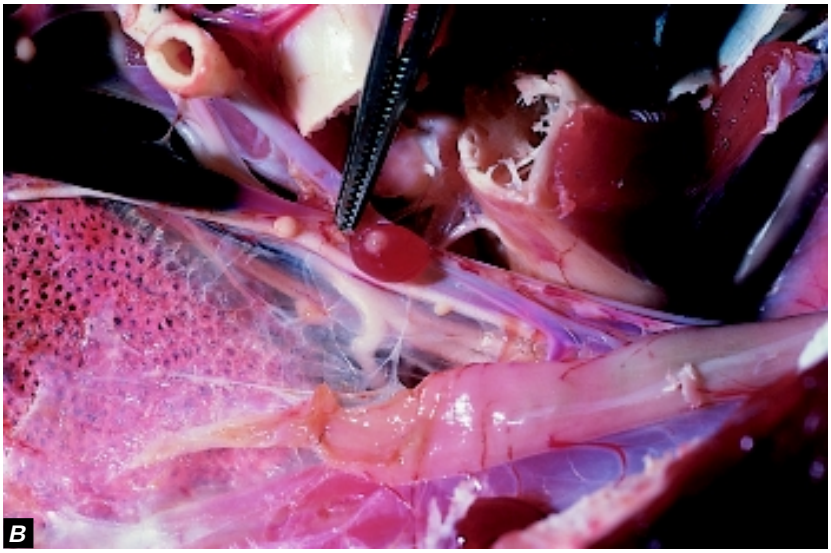
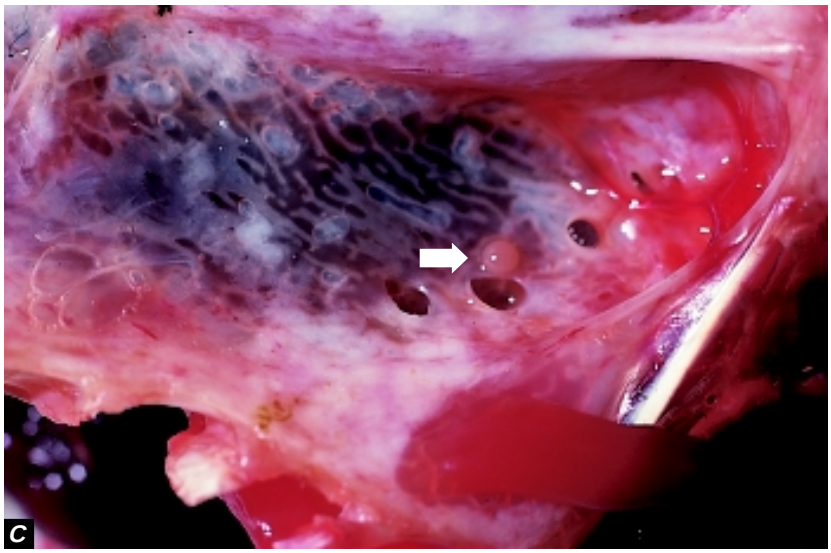


Photo by J. Christian Franson

Figure 26.5 *Intestinal coccidiosis in a common eider from Alaska, showing distinct light-colored areas within the wall of the intestine.*

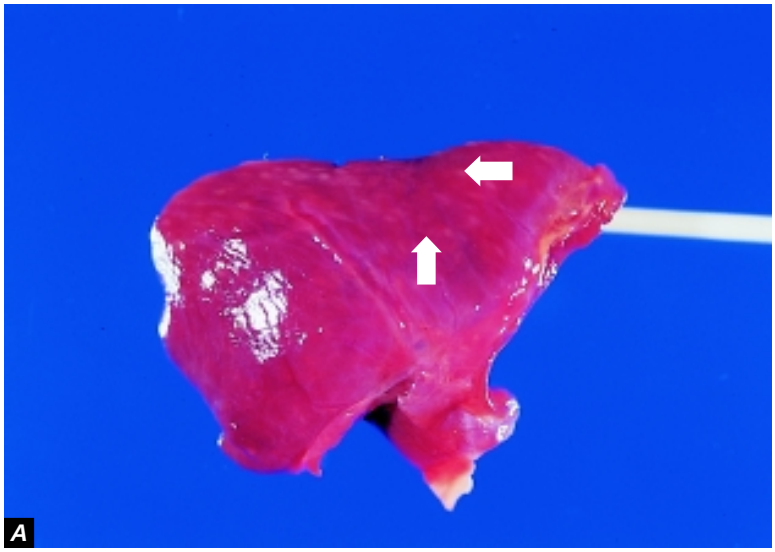






Photos by James Runningen

Figure 26.6 *Gross lesions of disseminated visceral coccidiosis of cranes.* ***(A)*** *Granulomas on the lining of the esophagus (arrows); and* ***(B)*** *in the area of the thoracic inlet [the tip of the forceps is between granulomas on the surface of a vessel and nerve (left) and on the thyroid gland (right)]; and* ***(C)*** *on the inside surface of the sternum (arrow).*



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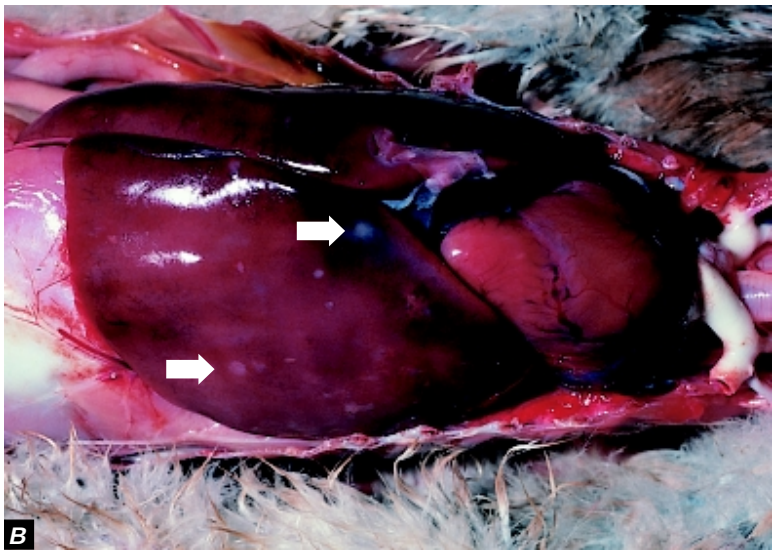

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Figure 26.7 *Lesions of disseminated visceral coccidiosis also may include light patches as seen here on the* ***(A)****, surfaces of the heart muscle and* ***(B)*** *on the liver (arrows).*

ful in preventing epizootics from this disease. Therefore, the objective is not to completely eliminate infection with coccidia; instead, the focus should be on preventing heavy infections and the establishment and persistence of high levels of environmental contamination with coccidia. For free-ranging birds, flock dispersal may be warranted when overcrowding continues for prolonged periods of time.

Human Health Considerations

None. Coccidia of birds are not infectious for humans.

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Supplementary Reading

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