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## Diseases of Quail

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There are over 100 species and subspecies of small partridge-like birds called quail found on all continents except Antarctica. Only the gallinaceous quail, especially the two semidomesticated species that are most commonly kept (the bobwhite quail [*Colinus virginianus*] and the Japanese quail [*Coturnix coturnix japonica*]), will be considered in this article. The other species of quails are either not found in captivity or are uncommon in zoologic or private aviaries or are maintained as companion birds. The major groups of quails are presented in Table 1.

Both the bobwhite and Japanese quails are widely grown for a number of purposes, including meat and egg production, release on hunting preserves, and research on physiology, nutrition, genetics, and health. The Japanese quail has been grown commercially since the 16th century.<sup>27</sup> It matures in 6 weeks at a growth rate that exceeds that of small chicken breeds by three and a half times. The Japanese quail is marketed as pharaoh quail in the United States. A number of different strains of both quails, including color varieties, have been bred. Production of the birds occurs in laboratory environments, small-scale "backyard" units, and on large commercial farms where over 100,000 birds may be grown annually. The anatomy,<sup>19</sup> physiology,<sup>86</sup> and use of Japanese quail as a laboratory animal<sup>40,63</sup> have been reviewed. Biologic data for both types of quail are presented in Table 2.

Specially formulated diets for quail (game-bird feeds) are available; however, feeds available for commercial turkey and chicken production are suitable for both species.<sup>52</sup> With the possible exception of a need for thiamine supplementation,<sup>73</sup> turkey diets are adequate for Japanese quail and chicken diets are adequate for bobwhite quail. Quail have higher requirements for choline than chickens and turkeys.<sup>52</sup> Nutrient deficiencies can occur in quail and are similar to those of chickens and turkeys. Japanese quail have been used extensively to examine the effects of deficient, or otherwise abnormal, diets on growth and reproduction.<sup>73</sup>

In our laboratory, we have found better livability in young bobwhite if a water-miscible form of vitamin A is provided at the recommended level for the first 2 weeks. Similar supplementation of Japanese quail is unne-

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Table 1. *Classification of Quails*<sup>27,82</sup>

Order:	Galliformes (gallinaceous birds, chicken-like)
Family:	Phasianidae (chickens, grouse, pheasants, partridges, peacocks, francolins, snowcocks, tragopans, monals, ptarmigans, prairie chickens, and quails)
Tribe:	Perdicini Bush quail ( <i>Perdica</i> , <i>Cryptoplectron</i> ) found in India Mountain quail ( <i>Ophrysia</i> ) found in Southeast Asia
Tribe:	Coturnicini (Old World Quails) Rain quail, New Zealand quail, Harlequin quail, Common quail, European quail, Ussuri quail, Japanese quail, African quail, <i>Coturnix</i> species and subspecies found in Europe, Asia, Africa, and Australia (only migratory galliforme) Chinese Painted quail ( <i>Excalfactoria</i> ) found in Africa, southeast Asia, and Australia (smallest gallinaceous bird, adult weight 45 gm) Snow Mountain quail ( <i>Anurophasis</i> ) found in New Guinea
Tribe:	Odontophorini (New World quails) Mountain quail ( <i>Oreortyx</i> ) found on Pacific coast of United States Scaled quail ( <i>Callipepla</i> ) found in southwestern United States and Mexico California quail, Gambel's quail, Douglass' quail ( <i>Lophortyx</i> ) found in Western United States Barred (banded) quail ( <i>Philortyx</i> ) found in southwestern Mexico Bobwhite quail, Black-throated quail, Crested quail ( <i>Colinus</i> ) found in North and South America Montezuma quail ( <i>Cyrtonyx</i> ) found in southwestern United States and Mexico Wood quails ( <i>Odontophorus</i> ) found in Mexico and Central America Singing quail ( <i>Dactylortyx</i> ) found in Mexico and Central America Banded quail ( <i>Rynchortyx</i> ) found in Central and South America
Order:	Gruiformes (cranes, rails, and similar birds)
Family:	Turnicidae (Buttonquails, Hemipodes) Buttonquails ( <i>Turnix</i> ) found in Asia, Australia, and Africa Lark quail ( <i>Ortyxelos</i> ) found in Africa

essary. Also, losses from "starve-out" will be markedly reduced if commercially available starter rations for turkeys and chickens are ground into a mash for very young quail. This can be easily accomplished in a heavy-duty blender or manually with a mortar and pestle.

Being galliformes, quail are susceptible to most of the diseases that affect chickens and turkeys. General reference texts on poultry diseases are useful for diagnosing and understanding quail diseases.<sup>21,35,37</sup> Comprehensive studies on the causes of mortality in flocks of Japanese quail,<sup>4,74</sup> a checklist of parasites and diseases of bobwhite quail,<sup>44</sup> and a recent review of the common diseases of quail have been published.<sup>71</sup> Procedures used for diagnosis of diseases in poultry are equally appropriate for quail.<sup>35,89</sup> The jugular vein has been found to be the best site for collecting blood samples.<sup>1</sup> A significant increase (20 per cent to 40 per cent) in both wet and dry liver weights of Japanese quail within 30 minutes after death compared with livers removed immediately after death has been reported.<sup>80</sup> Because Japanese

Table 2. *Biological Data for Coturnix and Bobwhite Quails*<sup>17</sup>

	COTURNIX	BOBWHITE
Adult weight		
Male	100-140 gm	180-250 gm
Female	120-160 gm	190-230 gm
Age of		
Sex differentiation	3 wk	8 wk
Sexual maturity	6-7 wk	16-24 wk
Life span	2.5-3.5 yr	7-10 yr
Egg production	1-2 times daily, continuous	90 in 6 mo, clutches
Light requirement	14-18 hr	14-17 hr
Space requirements		
Floor	16-25 sq in	0.5-1 sq ft
Feeder	0.5-1.0 in/bird	Same
Waterer	0.25 in/bird	Same
Egg weight	10 gm	8 gm
Incubation		
Duration	14-17 d	23-24 d
Incubator	99.5°F, 60% RH	99.8°F, 84-86% RH
Hatching	99.0°F, 70% RH	99.8°F, 87-98% RH

quail are commonly used for toxicology studies and residues are often reported on the basis of organ weights, lack of attention to the time of tissue collection relative to death could result in large errors.

As a general rule, Japanese quail appear to be somewhat more resistant to infectious diseases than the chicken, whereas bobwhite quail are generally more susceptible. For example, adenoviruses that cause little or no disease in chickens can produce a highly fatal disease known as quail bronchitis in bobwhites. It is an extremely poor management procedure to raise quail together with chickens or turkeys. Practicing good biosecurity as one moves between flocks of different avian species or different flocks of the same species and rearing only one group of birds on a premise at a time ("all in-all out" production) will contribute more to the health of the flock than any drug or vaccine. Biosecurity and adequate management are the bases for flock health and can never be overemphasized.<sup>89</sup>

## NONINFECTIOUS DISEASES

Mortality from noninfectious causes is highest in young birds and most often results from errors in management. Even those diseases caused by infectious or parasitic agents often occur because of mismanagement. Use of a medicated starter feed containing 200 gm per ton oxytetracycline for 1 to 6 weeks after hatching resulted in a marked reduction in mortality of masked bobwhite quail (*C.v. ridgwayi*), a rare subspecies.<sup>70</sup> Table 3 lists common noninfectious causes of mortality that can be reduced or prevented by management practices.

Reproductive disorders may have an infectious cause or may result from mismanagement. Poor fertility and hatchability can result from inadequate

Table 3. *Common Causes of Mortality that Can Be Reduced or Prevented by Management Practices*

PROBLEM	CAUSES	PREVENTION
Excess chick mortality (Fig. 1)	Chilling	Provide 95°F brooder and 80°F room temperatures; decrease temperature 5°F per wk; insulate buildings
	Dehydration	Provide waterer with adequate space and light (24 hr) for first 2 weeks
	Starve-out	Provide feeder of adequate height and space, light, feed size
	Drowning	Provide small, shallow waterers
Injuries/abscesses (Fig. 2)	Environment	Provide adequate housing with "hiding" area; use dim lighting after 2 wk; do not "spook" birds during chores, handling; cranial and leg injuries are most commonly seen
	Entrapment	Cover large wire floors with fine plastic mesh
Picking/abscesses/mortality	Overcrowding	See above
	Feed deprivation	See above; leads to toe and nose picking
	Water deprivation	See above
	Excess light	Provide dim light; after 2 weeks, use red light
	High temperatures	Maintain cool to neutral temperature (66–75°F for adults)
	Diet	Provide balanced ration
Piling/hysteria/smothering	Environment/activities	Do not mix different ages, groups of birds, especially do not place one bird into established group; separate males during breeding, beak trimming
		Maintain proper temperature; avoid complete darkness and sudden, unusual noise
Eye inflammation	High ammonia	Maintain proper litter moisture (25–35%); provide adequate ventilation
Crop impaction <sup>39</sup>	Fibrous diet	Provide adequate balanced ration

Figure 1. Vent pasting in this quail chick has led to obstruction and distension of the lower gut. Poor management usually causes this condition; however, *Salmonella* infection must also be considered.



nutrition, improper holding of hatching eggs, or improper incubation and hatching conditions (see Table 2). Ideally, fertile quail eggs should not be stored longer than 7 days to obtain optimum hatching. A storage temperature of 55°F and relative humidity of 70 per cent are considered optimum for Japanese quail, whereas a higher humidity (80 to 85 per cent) is preferred for storing fertile bobwhite eggs.<sup>17,85</sup>

Oviduct impaction (Fig. 3), egg-binding, and uterine prolapse occur in hens in heavy production. Obesity predisposes the birds to these problems. Prolapses often lead to episodes of cannibalism. Prevention is based on providing space for adequate exercise and appropriate diets for laying hens. Affected birds should be culled as soon as they are observed.

Japanese quail are widely accepted as a nonmammalian species for research on toxic substances, both as a model for intoxications in other avian species, such as aflatoxicosis, or as an example of how a toxic substance (for example, insecticide toxicities) may affect nontarget avian species.<sup>26</sup> Strains of resistant and susceptible quail have been developed and used to understand better the mode of action of toxic substances such as aflatoxin.<sup>60</sup> Japanese quail embryos have been found useful in determining teratogenicity of toxic substances.<sup>81</sup>

Reports of naturally occurring toxicities are not common, especially in commercially reared quails. California quail (*Callipepla californica*) died following ingestion of anticoagulant pellets containing chlorophacinone intended for control of voles. The quails did not die because of the anticoagulant, however, but because the paraffin pellets used as the carrier impacted the gizzard, which led to starvation.<sup>5</sup>

Nonviral neoplasia appears to be uncommon even in aged quail. A





Figure 2. Injuries of the feet and legs are frequent causes of economic loss in quail and also provide a portal of entry for bacterial diseases. (Provided courtesy of Dr. Laddie Munger.)

mixed-cell sarcoma involving the lungs, kidneys, and thigh was found in a 25-month-old moribund Japanese quail.<sup>30</sup> Sertoli cell tumors occurred in the testicles of 3 of 33 Japanese quail older than 3 years of age. Neoplastic testicles were enlarged, firm, round, and contained variable cystic spaces. Either both testicles were involved or atrophy of the contralateral testicle had occurred. Histology was typical of that seen in mammalian Sertoli cell tumors.<sup>24</sup>

Other noninfectious disorders are more important for their value in comparative medicine and understanding degenerative disease processes than as a cause of economic loss in commercially reared flocks. Cataracts that develop without concurrent disease and increase in prevalence and severity with age have been reported in bobwhite<sup>47</sup> and mutant albino Japanese quails.<sup>75,76</sup> Retinal degeneration also occurs in the latter that closely resembles changes seen in glaucoma. Another important model of degenerative disease that has been developed in susceptible lines of Japanese quail is atherosclerosis.<sup>72</sup> Other noninfectious disorders have been identified in various strains of quail, such as chondrodystrophy<sup>31</sup> and a generalized type II glycogen storage disease.<sup>49</sup>

## INFECTIOUS DISEASES

### Viral Infections

Natural and experimental viral infections in Japanese quail have been reviewed.<sup>63</sup> From a clinical viewpoint, viral infections in quail result in (1)

respiratory diseases (quail bronchitis, influenza, Newcastle disease, coronavirus infection); (2) nervous diseases (avian encephalomyelitis, Newcastle disease, equine encephalitis); (3) neoplasia (Marek's disease, leukosis, reticuloendotheliosis, transmissible sarcomas); and (4) pox. With the exception of Marek's disease, quail appear to be relatively resistant to herpesvirus infections, although a herpesvirus was recovered from livers of quails with concurrent quail enteritis.<sup>42</sup> In contrast to commercial poultry, viral infections have not been found to be important causes of enteritis in quail. Quail reovirus was isolated from quail with severe enteritis,<sup>66</sup> but cryptosporidia were found to be the cause of the disease, with the virus acting only as an intensifier.<sup>28</sup> Also, quail have been found to be refractory to infectious bursal disease,<sup>83</sup> although quail × chicken hybrids are moderately susceptible.<sup>25</sup>

Quail bronchitis is a highly contagious acute upper respiratory disease caused by type I avian adenoviruses.<sup>87</sup> Bronchitis in quail is unrelated to infectious bronchitis in chickens, which is caused by coronaviruses. Quail bronchitis occurs primarily in captive bobwhite quail and less commonly in Japanese quail.<sup>7</sup> Onset is sudden, spread is rapid, morbidity approaches 100 per cent, and mortality can range from 10 per cent to 100 per cent. The disease is most severe in quail under 4 weeks of age. Clinical signs consist of rales, coughing, sneezing, conjunctivitis, huddling, and inactivity. Nervous signs are occasionally seen. Nasal discharges and sinusitis are not characteristic of quail bronchitis. Catarrhal nasal exudate has been reported in quail coronavirus infection.<sup>58</sup> The clinical disease becomes milder in older birds and may even be inapparent in adults. Depressed egg production with abnormal white eggs is seen in laying birds. At necropsy, inflammation of the nasal passages, trachea, and bronchi with accumulation of excess mucus can be seen.

A presumptive diagnosis of quail bronchitis is possible based on history and signs. A definitive diagnosis and differentiation from the other viral respiratory diseases of quail that are similar clinically require demonstration of seroconversion between acute and convalescent sera or isolation and identification of the virus. Virus isolation is best done early in the course of the disease. Trachea and lung are good tissues to examine for virus, although it has been isolated from many sites in infected birds, including the intestinal tract.

Similar type I adenoviruses commonly infect chickens and turkeys (often referred to as chick embryo lethal orphan virus, or CELO), causing inapparent or mild respiratory disease. Research strongly suggests that these adenoviruses are similar, if not identical, to quail bronchitis virus. Antibodies to these viruses have been found in a number of free-living avian species. Quail bronchitis has not been identified in free-living bobwhite quail. However, a closely related virus was identified as a cause of inclusion body hepatitis (a known manifestation of type I adenovirus infection in chickens) and mortality in wild quail in Florida.<sup>45</sup> The virus has been isolated from chicken and quail embryos. These findings indicate that other avian species, including poultry, can be a source of the virus for quail and that egg transmission from infected hens is likely.

There is no specific treatment for quail bronchitis or the other viral respiratory diseases. Supportive therapy such as providing the best possible



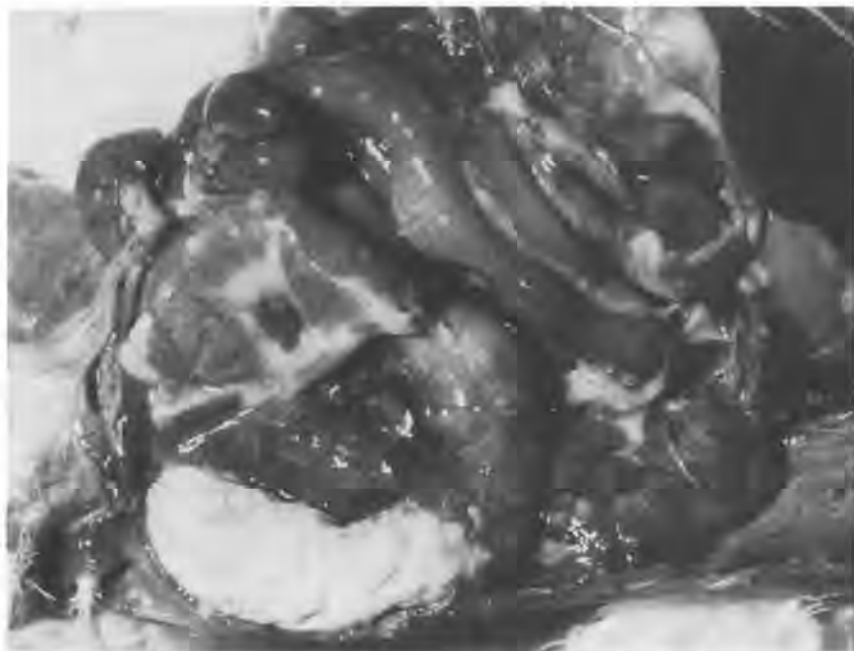


Figure 3. Impaction of the oviduct (lower left corner) occurs in heavily producing hens such as this albino Japanese quail. Secondary infection with *Escherichia coli* is common.

environment, using vitamins in the water, and broad-spectrum antibiotics to prevent secondary bacterial infections can help reduce mortality. Strict isolation of affected flocks and refraining from obtaining additional birds under 4 weeks of age through hatching or purchase will help limit spread.

Quail bronchitis can be prevented by depopulating affected farms and waiting at least 4 weeks before repopulating, quarantining any new additions to a flock for at least 4 weeks, obtaining breeding stock free of antibodies (an agar gel precipitin test is most commonly used), isolating quail from contact with free-living birds and poultry, and practicing strict biosecurity, especially when moving from older quail, which may be inapparent carriers, to younger flocks. No vaccine is generally available, although products are sometimes locally available in intense quail-rearing areas. Should Newcastle disease or influenza virus be the cause of viral respiratory disease, vaccination using products prepared for use in poultry would be theoretically possible. The safety and efficacy of these vaccines for quail have not yet been determined.

Viral diseases characterized by nervous signs in quail are not common. High mortality and a marked drop in egg production have also been seen in flocks with Newcastle disease.<sup>29,32</sup> Signs of avian encephalomyelitis were typical of the disease in other avian species and occurred only in young chicks under 4 weeks of age.<sup>33</sup> Eastern equine encephalomyelitis virus caused tremors, partial paralysis, and depression that rapidly progressed to complete paralysis, torticollis, and death within a few hours. The disease spread rap-



Figure 4. Nodular tumors in the intestines are typical of lymphoproliferative disease caused by reticuloendotheliosis virus in quail. (Provided courtesy of Dr. Laddie Munger.)

idly, causing 40 per cent to 90 per cent mortality in all groups of birds over 2 weeks of age. Total losses exceeded 90,000 quail on a farm in South Carolina. Cannibalism was identified as the principle mode of spread in this outbreak after the disease was introduced, presumably from blackbirds roosting nearby and mosquito vectors.<sup>16</sup>

Diagnosis of viral diseases of quail affecting the central nervous system depends on isolation and identification of the causative agent. There is no useful treatment. One should also remember that equine encephalomyelitis viruses can infect human beings and care should be taken if this disease is suspected. Vaccines for each of the viral diseases affecting the central nervous system of quail are available for other species, but their use in quail has not been explored.

Quail are susceptible to many of the tumor viruses that cause diseases in poultry and have been used, along with quail  $\times$  chicken hybrids, as experimental hosts for Marek's disease, reticuloendotheliosis, leukoses, and transmissible sarcomas. Lymphoproliferative diseases have also been found to occur naturally<sup>4,6,53,62,84</sup> in quail but are not common except, perhaps, in areas where quail are intensively raised.<sup>46</sup>

Quail affected with tumor viruses are generally in poor health, and mortality is elevated over a period of a few weeks to 5 or 6 months. Mortality can reach 100 per cent. Necropsy of affected birds reveals tumors in many body tissues. Nodular tumors in birds with reticuloendotheliosis are common in the digestive tract<sup>6</sup> (Fig. 4), whereas those of Marek's disease occur most frequently in the spleen, liver, proventriculus, and duodenum.<sup>46,62</sup> Considerable variation exists in the gross appearance and distribution of tumors caused by these viruses in quail, and one cannot distinguish among them on the basis of gross pathology. Tissues should be collected from affected birds for histology and virus isolation. Reticuloendotheliosis tumors are composed of sheets of highly invasive, extremely anaplastic mononuclear cells, whereas the tumors of Marek's disease contain polymorphic, more differentiated lymphocytes. Nerves are more often affected in quail with Marek's disease, but the frequency of lesions is less than that seen in affected chick-



Figure 5. Proliferative wart-like lesions around the beak and on the eyelids, face (A), feet, and legs (B) are characteristic of quailpox. Biting insects transmit the virus and feed on these unfeathered areas, which accounts for the characteristic pattern of lesions.

ens. Most of the reports of naturally occurring lymphoproliferative diseases in quail do not completely document which virus is responsible. Additional studies on this group of virus diseases are definitely needed in order to understand them better.

Both Marek's disease and reticuloendotheliosis viruses can be spread to quail from infected hosts. Marek's disease virus is airborne, whereas reticuloendotheliosis virus may also be carried by arthropod vectors. Infected chickens present the highest risk of Marek's disease for quail.<sup>15,62</sup> Strict isolation of quail from other avian hosts should prevent viral tumors. Vaccination with turkey herpesvirus at 7 and 19 days has been found to be useful in reducing Marek's disease in quail.<sup>46</sup> Depopulation of affected flocks and thorough disinfection before restocking should also be considered.

Pox in quail is a serious disease that may result in very high mortality.<sup>71</sup> Losses as high as 90 per cent have occurred in bobwhite flocks in the southeast United States. The disease is seasonal, being most prevalent during the warmer periods of the year when ornithophilic biting arthropods are most numerous. It also appears to be cyclic because the degree of occurrence from year to year can vary greatly. In spite of its importance and common occurrence, quailpox has received little attention, and not much is known about the epidemiology of the disease or the nature of the viruses.

Affected birds are presented from flocks with a history of increasing mortality, unthriftiness, poor growth, and "infected eyes." Proliferative lesions on the eyelids, around the beak, in the oral and nasal cavities, and on the legs and feet are seen early in the disease (Fig. 5A and B). Later, the ocular and oral lesions ulcerate and become covered with yellow to brown, sticky, diptheretic membranes. Death occurs from starvation, suffocation when respiratory passages are occluded, and effects of secondary infections with bacteria and/or fungi.

Diagnosis can be made on the basis of gross lesions, cytology (Fig. 6A and B), demonstration of the typical intracytoplasmic inclusion bodies in proliferating, hypertrophic epithelial cells by histopathology (Fig. 7), or virus isolation. Treatment of severely affected birds is probably not worthwhile. General supportive care may help reduce losses when birds are mildly af-



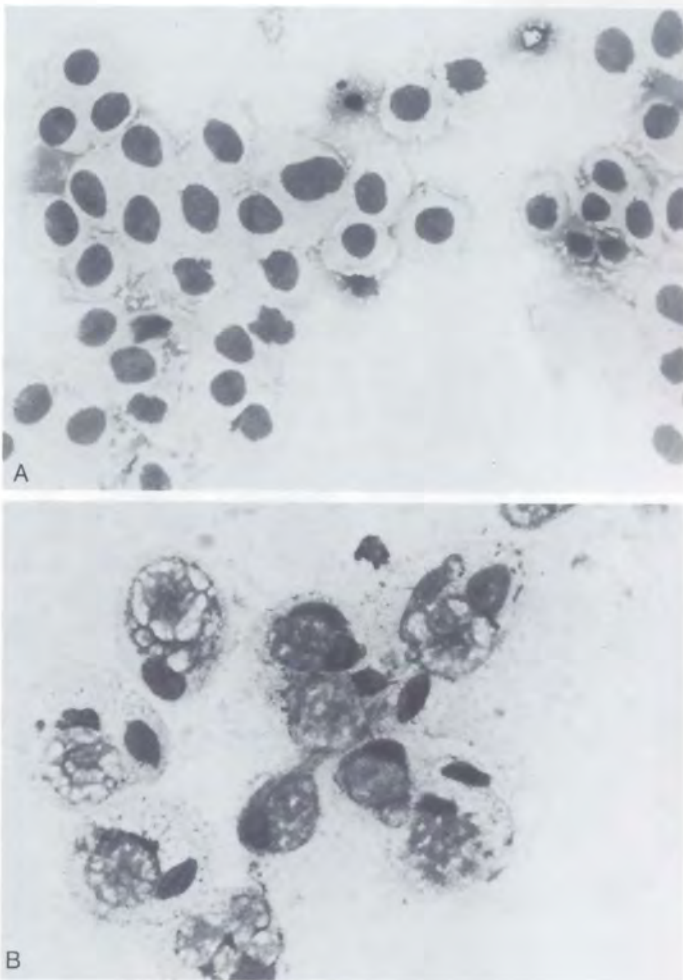


Figure 6. Avian pox infections can be diagnosed by cytologic examination of lesions. In these Giemsa-stained smears from a normal conjunctiva (A) and one with pox lesions (B), the grossly hypertrophied infected cells with vacuolated cytoplasmic inclusions can be seen.

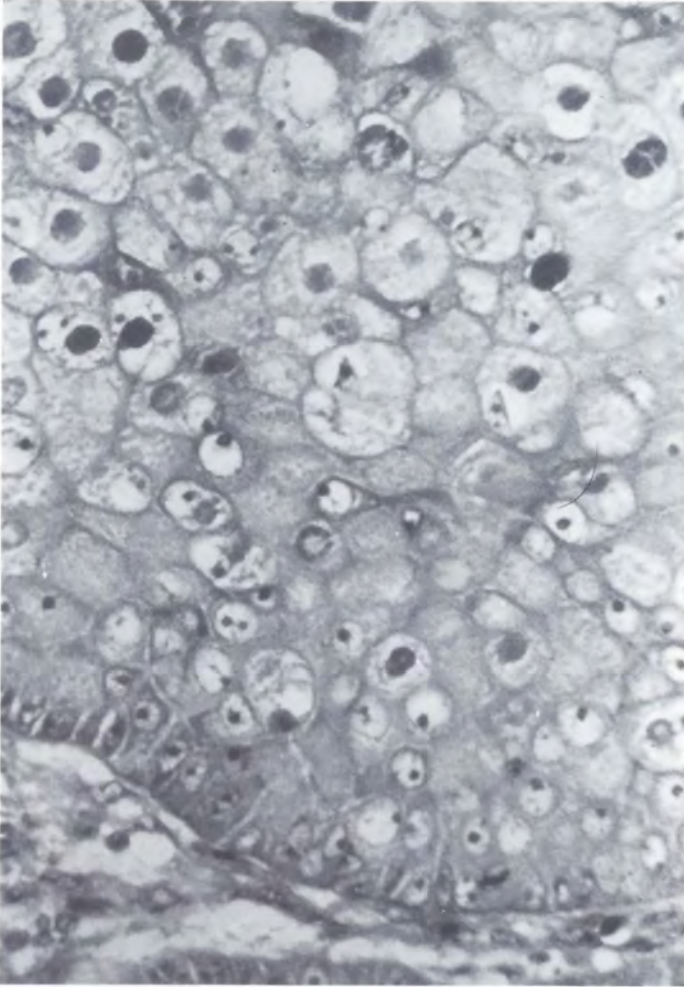


Figure 7. The typical cell changes described in Figure 6 are also apparent on histopathologic examination and can be used to confirm a suspected outbreak of avian pox. Note the intracytoplasmic inclusion bodies.



fect. Vaccination of affected flocks in the face of an outbreak is useful if done early. A specific quailpox vaccine should be used because recent studies indicate that at least some poxviruses infecting quail are distinct from either pigeonpox or fowlpox viruses, and use of these vaccines provides little if any protection.<sup>88</sup> Although not available nationally, locally produced quailpox vaccines can often be obtained in intense quail-rearing areas. Combined quailpox, fowlpox, and pigeonpox vaccines would also be useful and confer protection against a broader spectrum of avian poxviruses.

### Bacterial Diseases

For purposes of treatment, control, and prevention, it is convenient to think of bacterial diseases in quail as being either primary or secondary. The latter are generally caused by opportunists and typically result from mismanagement or complicate other diseases, especially those caused by viruses. Nonspecific omphalitis (not caused by *Salmonella*), nonspecific enteritis (no known cause identified), coliform septicemia, and cellulitis or abscesses are examples of commonly encountered secondary bacterial diseases in quail. Treatment of secondary bacterial diseases is usually not highly effective. Progress in controlling and preventing these diseases can only be made by ferreting out the management problems and correcting them.

Nonspecific omphalitis is a major cause of early quail mortality.<sup>4,70,71</sup> Gram-negative bacteria such as *Proteus*, *Pseudomonas*, and *Escherichia coli* are typically recovered when affected chicks are cultured. Gram-positive bacteria, especially *Streptococcus* and *Staphylococcus*, can also be found in some cases but are less common. Reduced hatchability and weak chicks are often associated with omphalitis. Control and prevention of omphalitis require that one provide clean nesting material; set clean eggs; fumigate eggs, incubator, and hatcher with formaldehyde; allow chicks to remain in hatcher to "dry"; and place newly hatched chicks in a clean, disinfected environment.

Nonspecific enteritis tends to be a chronic problem on certain farms. Repeated attempts to identify a known cause of enteritis have been unsuccessful. Management factors contributing to nonspecific enteritis are unsanitary brooding and rearing areas, overcrowding and underventilation of birds leading to high environmental contamination, and consumption of polluted water and/or contaminated feed. Frequent cleaning and disinfection of waterers and provision of chlorinated water containing 2.5 to 3 ppm chlorine at point of consumption will help reduce nonspecific enteritis. Test kits for determining chlorine levels in water are generally available from firms carrying swimming pool supplies.

Coliform septicemia can be the cause of significant losses.<sup>4,71</sup> Its control and prevention depend on providing a good environment aimed at reducing the level of contamination of air, food, and water and preventing primary respiratory diseases that increase the susceptibility of the birds to opportunist infections.

*Staphylococcus* and *E. coli* are most frequently isolated from quail with cellulitis and abscesses. These infections follow injuries. Taking the measures presented in Table 2 to reduce picking, accidents, and injuries and providing a clean, sanitary environment will aid in the reduction and prevention of these lesions.

Primary bacterial diseases of quail occur as (1) septicemia (fowl cholera,<sup>34,56</sup> salmonellosis,<sup>4</sup> erysipelas,<sup>57</sup> staphylococcosis,<sup>71</sup> streptococcosis,<sup>71</sup> pseudotuberculosis, *Proteus* infection,<sup>69</sup> *Pasteurella anatipestifer* infection<sup>80</sup>); (2) enteritis (quail disease, for review see Ref. 59); (3) omphalitis (salmonellosis—pullorum, fowl typhoid, and paratyphoid);<sup>4</sup> (4) sinusitis (infectious coryza,<sup>9,64</sup> infectious sinusitis,<sup>48,51,78</sup> other mycoplasma infections);<sup>4</sup> (5) botulism;<sup>71</sup> (6) tularemia;<sup>44</sup> and (7) tuberculosis.<sup>38,43</sup> Environment and management are also important contributors to primary bacterial diseases. Introduction of the diseases into a flock often occurs following lapses in recognized good management procedures.<sup>89</sup>

Septicemic diseases are characterized by acute onset with rapidly rising mortality. Affected quail are inactive, anorexic, have closed eyes, and a huddled, withdrawn, rough-feathered appearance. In contrast to normal quail, septicemic birds are readily captured and handled. Feathers around the vent and on the caudal abdomen are typically wet with adhering urates. Droppings are fluid and yellow to green. The crop in peracutely affected quail may still contain feed but, more typically, it is empty or contains odorous, turbid fluid that may be regurgitated when the bird is handled.

Lesions of septicemia in quail are similar regardless of cause and include splenomegaly, hepatomegaly, catarrhal enteritis, and pale swollen kidneys, often with excess urates in the tubules and ureters. Because quail do not have a large spleen normally, splenomegaly—a key feature of septicemia—may go unrecognized by the inexperienced diagnostician. Edema and/or petechial and ecchymotic hemorrhages in serous membranes are often noted. Focal areas of necrosis in the liver and spleen occur occasionally. Such lesions are prominent in pseudotuberculosis (unpublished observations). Fibrinopurulent to purulent polyserositis manifested as pericarditis, pleuritis, air sacculitis, peritonitis, and perihepatitis is seen in quail that survive for a few days. Chronic arthritis is a common sequel to septicemia.

Differentiation of the septicemic diseases of quail depends on isolating and identifying the causative organism. Because of the high mortality and acute onset, septicemic diseases can initially be diagnosed as a poisoning. These features of septicemic disease in quail necessitate a rapid diagnosis and institution of appropriate antibacterial therapy. Impression smears of liver, spleen, and blood stained with a blood stain and Gram stain can provide a rapid, presumptive diagnosis and basis for beginning treatment. This should be accompanied by culture and antibiotic sensitivity testing. If it is impractical to treat the individual bird, which is usually the case, mass medication is best done through the water because affected birds will continue to drink after they stop eating.

Treatment with an appropriate antimicrobial is generally effective, although chronically affected birds may remain. Sulfaquinoxaline given for 2 days was used to control outbreaks of fowl cholera in quail.<sup>56</sup> Long-term use of the drug resulted in toxicity manifested as kidney damage and visceral gout. Penicillin and streptomycin controlled an outbreak of erysipelas.<sup>57</sup> Vaccination as a means of controlling septicemic bacterial diseases in quail apparently has not been examined. However, the CU strain of *P. multocida*, widely used as a live vaccine in poultry to provide protection against fowl



Figure 8. This "sick" quail has ulcerative enteritis but could be suffering from a number of diseases caused by other bacteria, viruses, fungi, and parasites.

cholera, was found to be either highly virulent or ineffective in both bobwhite and Japanese quail.<sup>10</sup>

Ulcerative enteritis, also known as quail disease although the disease occurs in other avian species, is a widespread, common, and significant disease of quail, especially bobwhite. It is caused by *Clostridium colinum* or, less commonly, by *C. perfringens*.<sup>41</sup> Clinical course of the disease in a flock and signs in acutely affected individual birds are similar to those described for the septicemic diseases (Fig. 8). Chronically affected birds show weight loss and have an unthrifty appearance. Mortality may approach 100 per cent in young quail.

Necropsy of affected birds will reveal the characteristic lesions of enteritis, an enlarged, hemorrhagic spleen and multifocal necrosis of the liver (Fig. 9). In the early stages of ulcerative enteritis, focal to diffuse hemorrhagic enteritis is found in the proximal small intestine. Later, multiple ulcers with depressed centers and raised borders are present in intestines and ceca, being most numerous in the ileum and rectum. Although they occur in the mucosa, ulcers are readily visible through the serosa. Ulcers become covered with a diphtheretic membrane and may coalesce in severely affected quail. Perforation can occur, leading to localized peritonitis.

Diagnosis is based on history, signs, and lesions. The causative organism is difficult to isolate and identify because it requires strict anaerobic conditions for survival and growth. It can be seen in gram-stained smears of blood and lesions in the liver and gut as a large gram-positive bacillus with subterminal spores. Treatment with bacitracin, streptomycin, tetracycline, or furazolidone in the water or feed have all been used effectively. Bacitracin is considered the drug of choice. Prevention hinges on avoiding ingestion of large numbers of the organism that contaminate the environment, water, or feed and controlling coccidiosis, which may predispose birds to ulcerative





Figure 9. Necropsy of the quail in Figure 8 reveals ulcers in the lower small intestine that are visible even in the unopened gut and pale yellow foci along the margins of the liver. These lesions are typical for ulcerative enteritis.

enteritis. Housing birds on raised wire floor will help. Depopulation followed by thorough cleanup and disinfection before repopulating is necessary to break the disease cycle. Because of the marked resistance of the spores, even this procedure may not be completely effective, and it is not unusual for the disease to recur in previously treated flocks and successive broods once it becomes established on a farm.

Quail are susceptible to the species of *Salmonella* that affect poultry. Early chick mortality because of omphalitis is typical of salmonellosis. The organism may come from many different sources, including transovarially from infected hens, by shell contamination in the hen's cloaca or contaminated nest material, airborne in the hatcher, through contact with infected hatchmates, or from contamination of environment, feed, and water. Rodents are frequent carriers of the organism. Recovery of *Salmonella* from experimentally infected quail was substantially improved by use of a double-enrichment procedure.<sup>61</sup>

Quail infected with *S. gallinarum*, the causative agent of fowl typhoid, could not be detected by the whole blood plate agglutination test that has been used successfully in chickens and turkeys to eliminate this disease in commercial poultry in the United States. Routine testing of quail breeders by this procedure should not be relied on to assure they are free of fowl typhoid and, presumably, pullorum disease, which is also detected by the same procedure.<sup>2</sup>

Losses from salmonellosis were sharply reduced in quail chicks by incorporating furazolidone in the starter ration.<sup>4</sup> Prevention and control are accomplished by using breeders free of *Salmonella* infection, eliminating



Figure 10. Neither *Mycoplasma* nor *Haemophilus* organisms were recovered from the infected sinuses of this Japanese quail, although the lesion is typical of infectious sinusitis or infectious coryza. Cultures taken from the lesion at an earlier date may have been more informative.

environmental sources of the organisms, and preventing direct or indirect exposure to other animals, especially wild birds and rodents.

Respiratory diseases in quail caused by bacteria and mycoplasmas include infectious coryza caused by *Haemophilus paragallinarum* (formerly *H. gallinarum*) and infectious sinusitis caused by *Mycoplasma gallisepticum* and perhaps other mycoplasmas. Both diseases are characterized by swollen sinuses and nasal and ocular discharges (Fig. 10). Quail with infectious coryza also experience conjunctivitis and keratitis.<sup>64</sup> Air sacculitis occurs in quail infected with *M. gallisepticum*.<sup>48,51</sup> The organisms require special culture conditions for isolation and identification. Recovery of the causative agent is best accomplished early in the disease outbreak. Secondary bacteria may prevent finding them in the later stages of the disease. Serology is widely available for *M. gallisepticum* because this organism is an important pathogen in poultry. Seroconversion of paired sera can also be used to establish a diagnosis. Isolation of quail from poultry is important in preventing both of these diseases in quail.

Other bacterial infections in quail include botulism, tularemia, and tuberculosis. Botulism may occur as an autointoxication or from ingestion of preformed toxins. Affected birds exhibit flaccid paralysis while being otherwise alert. No lesions are found at necropsy. Plasma or serum from the birds will reproduce botulism in mice when they are inoculated. Protection of mice by pretreatment with botulism antitoxin is confirmatory. Control is based on "flushing" the affected flock with blackstrap molasses or epsom salts and timely disposal of dead birds, grossly contaminated water, any source of fly maggots, and spoiled feed.<sup>21</sup> Tularemia has been reported in wild quail and was the source of human infection.<sup>44</sup> Tuberculosis is uncom-





Figure 11. This bird has crop mycosis. The crop is empty and the mucosa is thickened because of a diffuse, flocculent, yellow-white pseudomembrane on the mucosal surface.

mon but could occur in older quail on farms or in zoo collections. The typical lesions of the disease in avian species are found in the intestine, spleen, and liver. Both granulomatous and nongranulomatous lesions have been described in experimentally infected quail.<sup>38,43</sup> A rapid diagnosis of tuberculosis can be achieved by examination of acid-fast stained smears prepared from the lesions.

Current recommendations for use of antibacterials in quail have been summarized.<sup>71</sup>

### Mycotic Diseases

Quail are susceptible to the common mycotic diseases that affect poultry, including crop mycosis, aspergillosis, and dactylariasis.<sup>55,71</sup> Young birds under 4 weeks of age are most commonly affected with diseases in this group. Mortality is more common in outbreaks of aspergillosis and dactylariasis; unthriftiness and poor growth with occasional mortality occur in flocks with crop mycosis.

The salient lesion of crop mycosis is a thickened, usually empty crop that has a focal to diffuse flocculent white, gray, or pale yellow pseudomembrane on the mucosa (Fig. 11). With experience, the thickened crop can be palpated in the live bird while it is examined for presence of feed. A similar lesion occurs in capillariasis, but this disease is more common in older quail. The two diseases can be rapidly differentiated by examining wet or periodic acid-Schiff (PAS) stained smears of the crop lesions. In crop mycosis, the typical pseudohyphae and yeasts that are seen are PAS-positive. Biopericulate eggs will be seen if the crop lesions are caused by *Capillaria*. Histopathologic examination may also be used (Fig. 12 A and B).

Crop mycosis occurs following ingestion of *Candida albicans*, the caus-

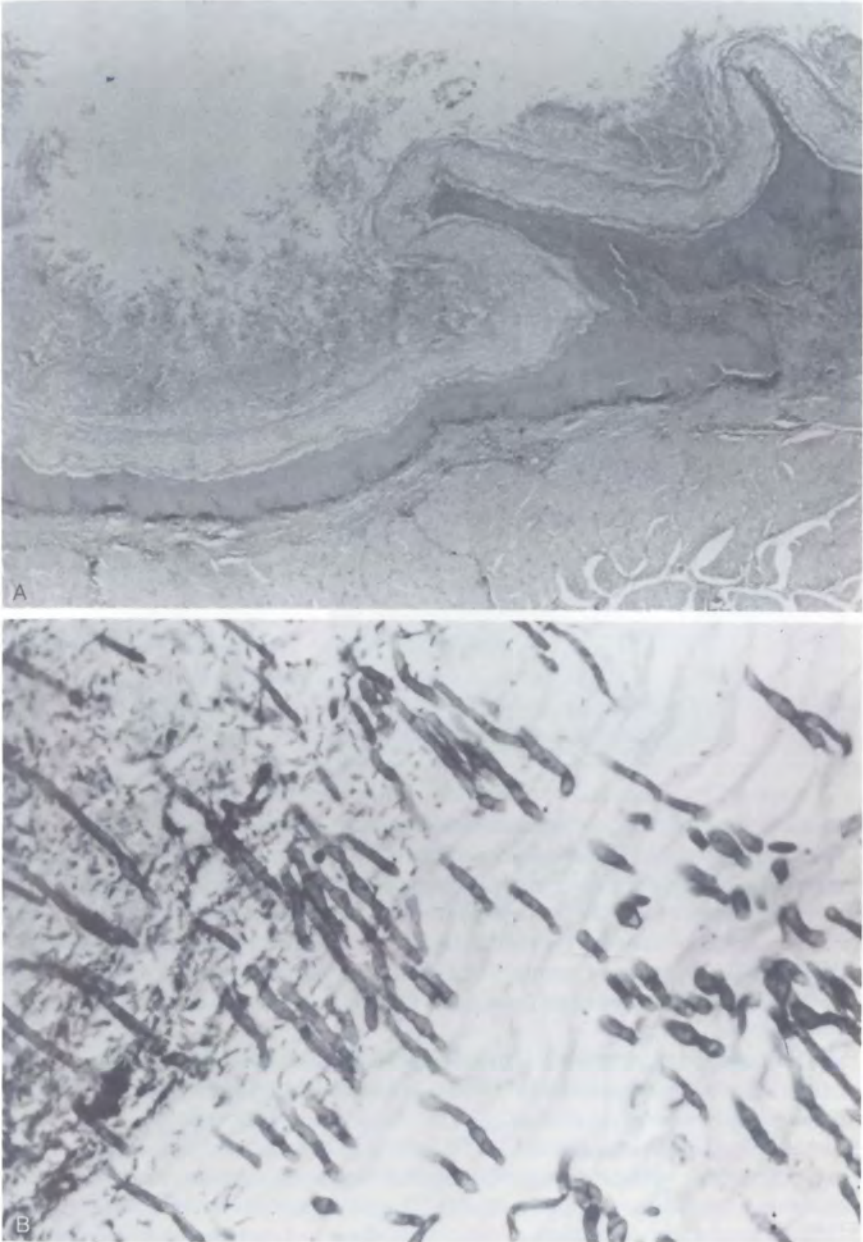


Figure 12. On histopathologic examination, the thickened crop mucosa is a diagnostic feature of crop mycosis (A). By using a PAS stain, one can easily see the large numbers of *Candida albicans* pseudohyphae in the mucosa perpendicular to the mucosal surface (B).



Figure 13. This bird has both the respiratory and air sac forms of aspergillosis. One can see multiple small nodules in the lungs and plaques in the air sacs.

ative agent. Use of broad-spectrum antibiotics, vitamin preparations containing high amounts of sugar as a carrier, ingestion of spilled or damp feed, and unsanitary conditions contribute to the disease. Nystatin in the feed or water has been shown to be the most effective treatment and preventive procedure in experimentally infected quail.<sup>50</sup>

Aspergillosis can involve the brain, lungs, trachea, air sacs, or eyes. The disease follows exposure and inhalation of high numbers of infective spores. Often this occurs in the incubator or hatcher from fungal growth in contaminated eggs or on residual debris in the warm, moist environment, or during early brooding when chicks are placed on previously wet litter containing high numbers of spores. Incubating dirty or cracked eggs containing aspergillus growth is a prime source for hatchery contamination. Problems such as stress during brooding or concurrent infections increase the likelihood of aspergillosis.

Pulmonary aspergillosis ("brooder pneumonia") is the most severe form of the disease. Pale nodules that often are surrounded by a narrow red zone and randomly scattered throughout the lungs are seen in pulmonary aspergillosis (Fig. 13). When air sacs are involved, a flat, discolored plaque is formed, most commonly at the recurrent bronchi of the caudal air sacs, that frequently has a fuzzy gray, blue, or green center. The latter results from aerobic growth of the fungus and production of fruiting bodies and spores. Only hyphae are seen in tissue lesions. Caseous plugs are found occluding the distal trachea or primary bronchi in the tracheal form (Fig. 14). Abscesses in the brain and/or exudate in the ventricles are seen in the central nervous system form. Bilateral or unilateral keratoconjunctivitis and accumulation of caseous exudate are typical of ocular aspergillosis.



Figure 14. This bird has the tracheal form of aspergillosis. A caseous plug occluding the trachea is visible in the syrinx.



Diagnosis of aspergillosis is based on finding the typical lesions, demonstrating the fungus in smears (Fig. 15) or by histopathology (Fig. 16), or isolation and identification of the organism. There is no treatment for aspergillosis. The disease is controlled by preventing exposure to large numbers of spores and maintaining the general health of the flock. Good hatchery sanitation is paramount for aspergillosis prevention. Experimentally, *coturnix* were found to be more susceptible to aspergillosis than turkeys or chickens.<sup>23</sup>

Dactylariosis is much less common than the other fungal diseases. The fungus infects the brain and causes central nervous system signs in affected birds that resemble those seen in birds with nutritional encephalomalacia. A diffuse to focal red discoloration of the brain is found in affected quail at necropsy. Diagnosis depends on isolation and identification or demonstration of the fungus by histopathology (Fig. 17). Dactylariosis must be distinguished from the central nervous system form of aspergillosis. A very characteristic granulomatous encephalitis is seen microscopically in birds with dactylariosis (Fig. 18).

Little is known about the source of dactylariosis for birds. In other species, it has been associated with the use of hardwood shavings as litter, but a definite relationship has not been proved. Sanitation with clean-up

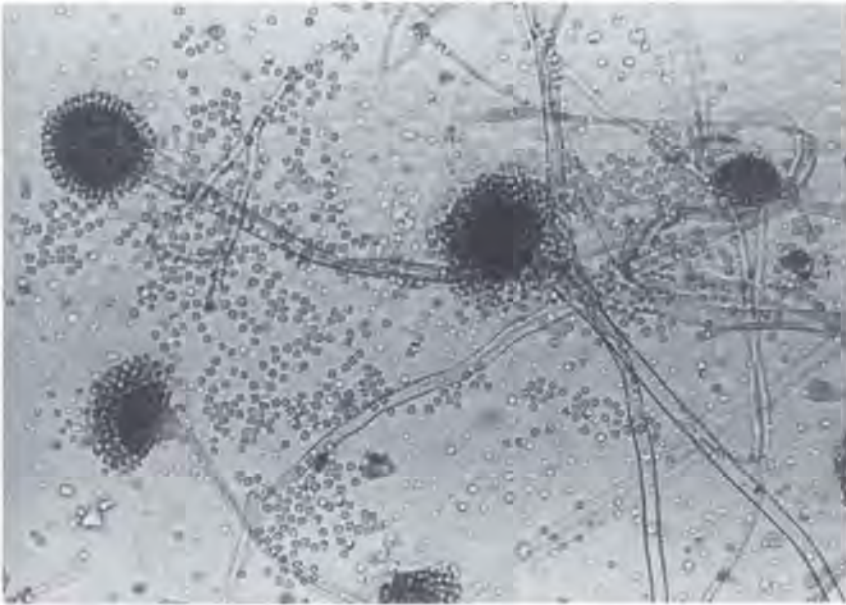


Figure 15. If the lesions are in air passages, the fungus can develop fruiting bodies and spores. These can be identified in wet smears to confirm a presumptive diagnosis. Lactophenol cotton blue, which was used in this instance, is an excellent stain to demonstrate the organisms.

and disinfection of premises having affected flocks are the only recommendations that can be made at this time.

### Parasitic Diseases

Free-living quail serve as hosts to a large number of protozoan, helminth, and arthropod parasites, none of which are considered to be significant causes of disease in wild quail. It is beyond the scope of this article to cover these organisms, and the interested reader is referred to reviews and checklists on these parasites.<sup>3,9,13,14,44</sup> Only the more common and important parasitic diseases of quail will be covered here.

When flocks of quail are reared in confinement with high stocking densities, some of the parasites in wild quail become significant. Those that do not require an intermediate host are most commonly found, whereas parasites with complicated, indirect life cycles are rare. In addition, parasites of other species may infect quail as either larvae or adults and cause disease. As with the viral, bacterial, mycotic, and noninfectious diseases discussed in the preceding sections, management, particularly raising birds on wire instead of on bare earth or litter, is the most important factor in prevention and control of parasitic diseases.

Because the effects of parasitism are often subtle but still of economic significance, quail submitted for diagnosis should always be examined for parasites. If possible, flocks should be placed on a routine monitoring program for evidence of parasitism. Ideally, this would include examination of



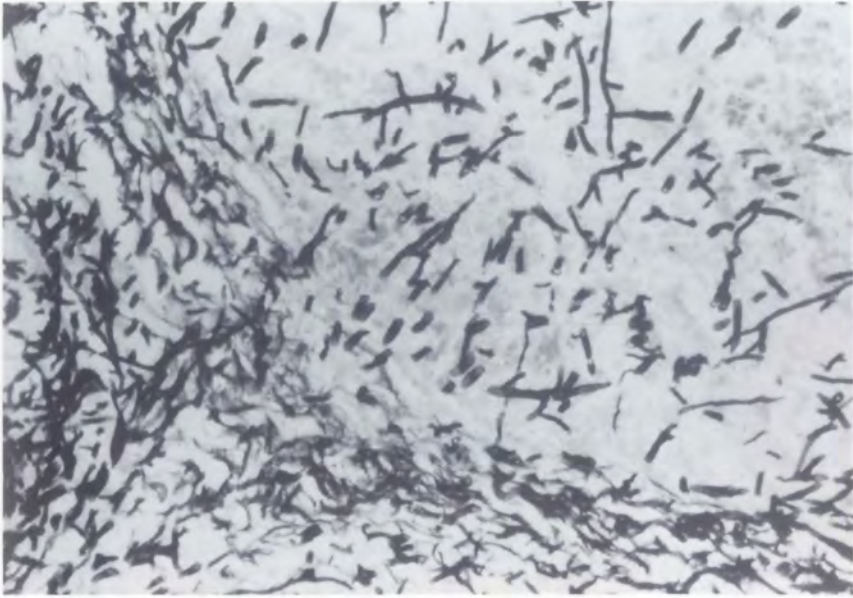


Figure 16. Fungal hyphae in tissues can be readily demonstrated by histopathology, especially if a special fungus stain such as this Grocott's stain is used (hyphae are black with light green background).

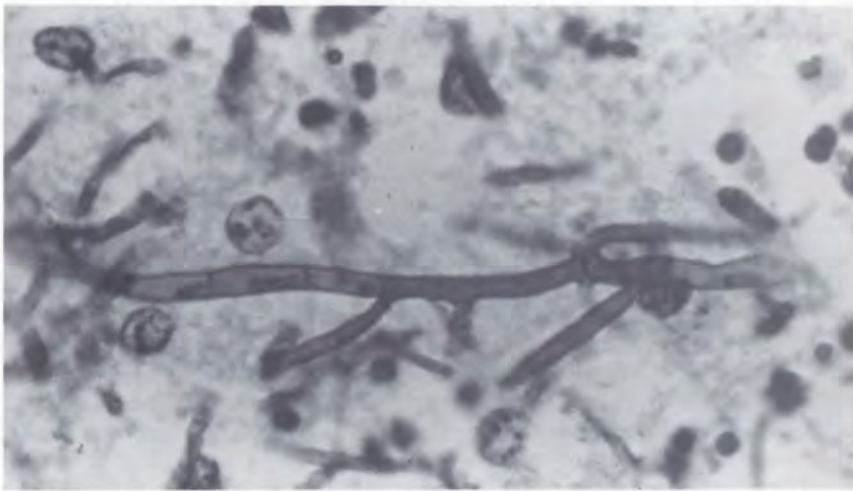


Figure 17. The fungus *Dactylaria* in a section of brain from an affected bird that has been stained with PAS.



Figure 18. The characteristic lesion of dactylariosis, which distinguishes it from the central nervous system form of aspergillosis, is a granulomatous encephalitis. In this section of brain from an affected bird, malacia is generalized and severe and there are numerous multinucleated giant cells that are often elongated. The fungus does not stain with routine hematoxylin and eosin procedures.

blood smears, fecal samples, and necropsy of a sample of birds on a regular basis. Procedures useful in the diagnosis of parasitic diseases in other species are equally useful for quail. A jeweler's loop or illuminated magnifier helps to recognize smaller organisms.

Important diseases caused by protozoa include coccidiosis, cryptosporidiosis, and histomoniasis. *Hexamita*, *Chilomastix*, and trichomonads have been associated with mortality and diarrhea in quail, but their significance and distribution have not been determined. A number of blood parasites naturally or experimentally infect quail but are not known to cause significant disease. A possible exception is an aberrant haemosporidian infection of unknown type that caused myositis and death in bobwhite quail. Linear hemorrhages in all muscles were apparent at necropsy, and the parasites were found by histopathologic examination.<sup>22</sup>

Although clinical coccidiosis is only diagnosed occasionally in quail, the disease is probably common but overlooked. Unlike coccidiosis in chickens, overt disease with prominent gross lesions is not typical in quail. An exception is coccidiosis in Japanese quail caused by *E. tsunodai*, which produces a hemorrhagic typhlitis similar to cecal coccidiosis caused by *E. tenella* in chickens.<sup>79</sup> Most cases of coccidiosis in growing quail are characterized by low to negligible mortality, slow growth, and poor feed utilization (higher feed conversions). Young quail are more severely affected than older ones. Egg production is decreased when adult quail contract coccidiosis, but weight is unaffected. Gross lesions are absent or consist of pallor and distension of the affected part of the gut.<sup>68</sup>

There are at least five species of *Eimeria* that infect coturnix quail (*E. coturnicis*, *E. bateri*, *E. taldykurganica*, *E. tsunodai*, and *E. uzura*) and three species that infect bobwhite (*E. colini*, *E. dispersa*, and *E. lettyae*).



Figure 19. Both quail are the same age. The one on the right is infected with cryptosporidia, and the one on the left is uninfected. Note the marked growth depression; dark, atrophied breast muscles; and the fluid- and gas-distended ceca and intestines in the infected bird. Cryptosporidiosis is a severe disease in young bobwhite quail. (Provided courtesy of Dr. David Ley.)

More species probably exist, but they have not been identified. Like other avian coccidia, the species in quail are highly host-specific and do not infect other unrelated avian hosts. An exception is *E. dispersa*, which can use other galliformes, including turkeys, as a host. Likewise, the eimerian species that infect chickens or turkeys (with the exception of *E. dispersa*) will not infect quail.<sup>54</sup>

Diagnosis of coccidiosis in quail depends on demonstrating high numbers of parasites in wet smears, fecal floats, or tissue sections from quail that are not performing as well as expected. Finding parasites in normal quail or in low numbers confirms infection and the potential of disease developing but is not sufficient for a diagnosis of coccidiosis. Anticoccidial drugs available for poultry have been used to treat affected quail with varied success. At the present time, the drug of choice for coccidiosis in Japanese quail would appear to be monensin. Both monensin and salinomycin have been found to be safe and effective for use in affected bobwhite.<sup>67</sup>

Prevention of coccidiosis in quail requires prohibiting the introduction of infective oocysts to new flocks that must come, either directly or indirectly, from other infected quail and minimizing exposure of flocks on contaminated premises. Routine use of medicated feeds to control coccidiosis is not common.

Cryptosporidiosis is an acute enteric disease of young bobwhite caused by *Cryptosporidium* spp. and is characterized by severe diarrhea, high mortality, and marked weight depression.<sup>18,28,36,66</sup> At necropsy, dark, atrophic





Figure 20. High numbers of cryptosporidia can be seen as small round parasites in the microvillus border of this section of intestine.

muscles, thin-walled, pale, fluid-filled intestines, and ceca distended with foamy, pale brown fluid and gas are seen (Fig. 19). Japanese quail infected with cryptosporidia showed respiratory signs and moderate mortality. These birds had excess mucus in the trachea, congested nasal passages, and bursal atrophy.<sup>77</sup> Diagnosis of cryptosporidiosis depends on demonstrating the tiny ( $5\ \mu\text{m}$ ) parasites in microscopic sections of gut (Fig. 20), Giemsa-stained impression smears, auramine-O-stained fecal smears, or finding the oocysts in fecal floats.

There is no treatment for cryptosporidiosis other than supportive treatment. Depopulation followed by cleaning and disinfection with 50 per cent bleach was effective in preventing recurrence of the disease in subsequent flocks.<sup>37</sup> Cryptosporidia are not host-specific. At the present time, there is an indication that the parasite in quail can also infect turkeys and perhaps chickens. Isolation of quail from other birds, especially turkeys, should be done to assist in preventing the disease. Concurrent infection with quail reovirus and cryptosporidia results in a more severe disease and enhances the effect of both organisms on the bird.<sup>28</sup>

Histomoniasis is caused by the ameboflagellate *Histomonas meleagridis*. Quail are moderately susceptible to the disease, typically experiencing a mortality of 10 per cent to 20 per cent,<sup>71</sup> although an outbreak with 100 per cent mortality has been reported.<sup>11</sup> Exposure of quail to the insecticide Sevin increased the susceptibility of quail to histomoniasis.<sup>90</sup> The protozoan exists within the eggs and larvae of *Heterakis gallinarum*, the common cecal worm of chickens and turkeys. Because of this, several benefits accrue to the protozoan, including protection from environment, survival for several years, and ability to use earthworms as transport hosts. Quail can become infected by ingesting fresh droppings from infected birds, eggs of the cecal worm, or earthworms containing *Heterakis* larvae. Control of histomoniasis





Figure 21. Lesions in the ceca and liver are indicative of histomoniasis. The changes in these tissues from an affected quail are typical. (Provided courtesy of Dr. Laddie Munger.)

depends on preventing ingestion of the preceding substances. Routine prophylactic medication is not practiced by quail growers. Quail should not be raised in premises or on ground previously occupied by chickens or turkeys for at least 5 years.<sup>21</sup>

Quail affected with histomoniasis are depressed, anorexic, and have the typical huddled, rough appearance of sick quail. Droppings are often bright sulfur yellow in color. The characteristic lesions seen at necropsy are cecal ulceration with caseous cores and multifocal necrosis in the liver (Fig. 21). Localized peritonitis may be found adjacent to severely affected ceca. Early lesions in the liver are circular, depressed, and have a targetlike appearance; later they become uniformly pale.

Dimetridazole is the preferred treatment for histomoniasis in quail. Other drugs used for the disease in turkeys may also be useful for quail.<sup>71</sup>

Infection with capillarids, ascarids, cecal worms, gapeworms, and visceral larval migrans caused by larvae of the raccoon roundworm (*Baylisascaris procyonis*) and *Physaloptera* spp. are either common or important diseases caused by helminths in quail. Tapeworms are occasionally seen and may be numerous, but because they cause little if any clinical disease, treatment is not warranted. Prevention of tapeworm infection requires isolating the birds from the cestode's intermediate hosts: beetles, ants, grasshoppers, other insects, slugs, and snails. Trematodes and acanthocephalids occur in wild quail but are rare in confined flocks.

Two species of *Capillaria* infect quail: *C. contorta* and *C. obsignata*. Both have direct life cycles and cause poor growth, anemia, mortality, and



Figure 22. Mucosa of the crop from quail with capillariasis is thickened and covered with a pseudomembrane. An example of a moderately affected crop is seen here. (Provided courtesy of Dr. Laddie Munger.)

decreased production. *Capillaria contorta* burrows into the mucosa of the crop and esophagus, causing chronic inflammation that leads to thickening of the mucosa and formation of pseudomembranes on the luminal surface (Figs. 22 and 23A and B). *Capillaria obsignata* inhabits the mucosa of the small intestine but does not cause gross lesions other than hyperemia and thickening of the gut wall with a rough appearance to the mucosa. Capillarids are quite small. They are best found in washed mucosal scrapings examined with a dissecting scope or in scrapings squashed between two glass slides and examined under the microscope. The presence of characteristic eggs with bipolar opercula in the females will identify the parasites. Finding the distinctive eggs in fecal floats can also provide a diagnosis of infection. However, capillarids are not very fecund, and the diagnostician should not be misled into thinking a bird is only mildly parasitized if only low numbers of eggs are found. *Capillaria* infection is difficult to treat. Levamisole is the current drug of choice.<sup>71</sup>

The large roundworms (*Ascaridia* spp.) and cecal worms (*Heterakis* spp.) are seen in intensively raised flocks grown on litter or on the ground. These worms are readily recognized at necropsy or diagnosed by finding numerous typical eggs in fecal floats. Large numbers of roundworms will reduce productivity and may occasionally cause liver abscesses because of aberrant migration, or they may occlude the intestine. The importance of cecal worms stems more from their role in histomoniasis than from their effect on the host. Both parasites have direct life cycles. Infections can be treated with piperazine.<sup>71</sup> Isolating quail from fecal contamination will help control the parasites.

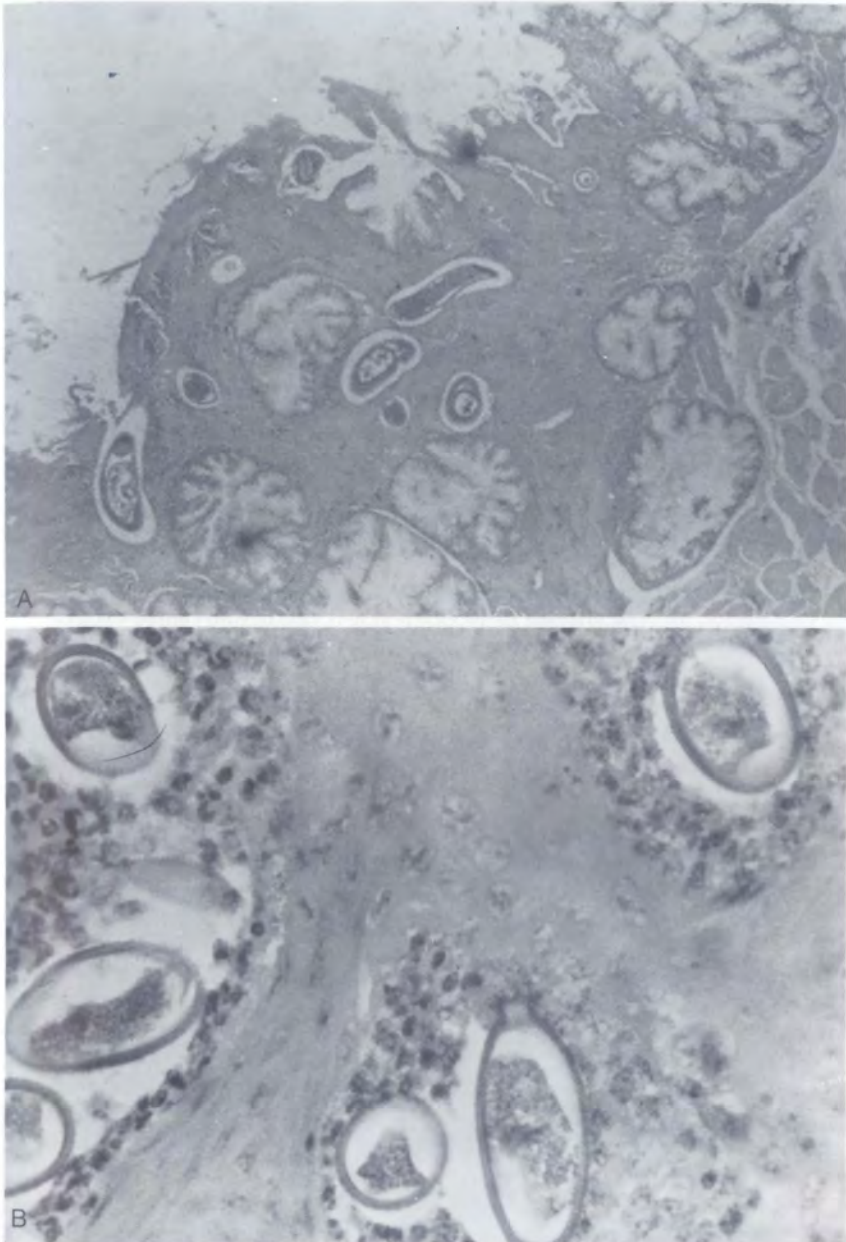


Figure 23. Microscopically, numerous parasites can be seen embedded in the crop mucosa (A) of quail with capillariasis. Presence of the bioperculated eggs confirms the identity of the parasites (B). Note that there is no inflammatory reaction against the parasite but that there is one against the eggs. This process probably helps the eggs move from the tissue into the crop lumen but leaves the parasites unaffected.



Figure 24. Severe dyspnea, commonly called the "gapes," is the most prominent clinical sign of gapeworm infection.



Figure 25. The large, bright-red worms in a "Y" configuration are easily found in the trachea at necropsy and establish the diagnosis. In this quail, the infection has been long-standing because the host has produced granulomatous nodules where the parasites have attached to the mucosa.





Figure 26. Initially diagnosed as *Sarcocystis* infection, the lesions in this quail from eastern Kansas were subsequently found to be the result of visceral larval migrans. The parasite could only be identified as a spirurid, indicating the quail was acting as a transport host.

Gapeworms (*Syngamus trachea*) occur in the trachea and cause marked respiratory signs ("gaping") (Fig. 24) and death from suffocation. They are relatively large bright-red helminths that are easily found at necropsy (Fig. 25). By extending the neck and transilluminating the trachea, they can usually be seen through the glottis of the live bird. Closer examination of the parasites will reveal the diagnostic "Y" configuration created by the smaller male being locked in permanent copulation with the larger female. Diagnosis can also be established by finding the parasite's eggs in fecal floats.

Gapeworms have a direct life cycle, but infection of quail is more likely from the ingestion of transport hosts, especially earthworms, in which the parasite larvae may remain viable for years. Epizootic outbreaks can occur if susceptible young birds are exposed to large numbers of infective transport hosts during a short period—for example, if they ingest large numbers of earthworms following a spring rain. Prevention of gapeworm infection is based on preventing quail from having access to transport hosts. Levamisole is an effective treatment.<sup>21</sup>

Not only do quail acquire parasite infections from transport hosts infected with nematode larvae, they can also serve as a transport host for spirurids that live in the stomach and intestines of carnivores. Larvae of *Physaloptera* have been found in muscle abscesses of bobwhites.<sup>12</sup> Although they have no apparent adverse effect on the quail, they make the carcass unfit for consumption. At necropsy, the linear tracts produced by the migrating larvae resemble sarcocystis (Figs. 26 and 27).

Visceral larval migrans in quail is caused by larvae of the raccoon roundworm *Baylisascaris procyonis*.<sup>65</sup> Affected quail may show respiratory signs early in the disease when the larvae migrate in the lungs. Later, they often migrate in the brain and spinal cord, causing pronounced nervous signs (Fig. 28). In contrast to the other diseases in quail affecting the central nervous



Figure 27. Histopathologic examination of the lesion from the quail in Figure 26 shows the inflammation along the migration path of the larval helminth in the muscle. The parasite can be seen within a cavity at the left end of the tract.



Figure 28. This quail had a history of pronounced central nervous system signs for the past 3 weeks. Others in the group of older breeders had been sporadically affected during the past several months. None of the young quail were affected. Visceral larval migrans was diagnosed following disclosure that the affected birds were being fed weeds pulled from around a pen containing three young raccoons.

Figure 29. Histopathologic examination of the brain from the bird in Figure 28 revealed larval ascarids. Granulomatous inflammation was intense in the migration tracts but not around the parasites themselves. (PAS stain.)



system, the signs in birds with visceral larval migrans are not progressive and tend to stabilize. Affected quail can survive for long periods of time. Also, the disease is typically sporadic and does spread among a flock like an infectious disease. Acute outbreaks can follow after quail have been placed in a building previously occupied by raccoons where they get exposed to large numbers of embryonated eggs during a short period. Clinical signs and a history of exposure to raccoons provide a presumptive diagnosis. In one outbreak seen by the author, exposure of quail resulted from feeding weeds that had been pulled around a cage containing three young raccoons. Confirmation of visceral larval migrans is achieved by demonstrating the presence of migrating larvae in tissues from affected quail by histopathology (Fig. 29). There is no treatment. Preventing exposure of quail to environments where raccoons have been will control the disease. Although there is no danger of human infection from diseased quail, there is a high risk to human beings if they are exposed to environments where raccoons had been or are being kept.

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