AVIAN DISEASE MANUAL
SIXTH EDITION

American Association of Avian Pathologists

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FUNGAL DISEASES

PARASITIC DISEASES

NUTRITIONAL DISEASES

MISCELLANOUS DISEASES

APPENDIX

AVIAN ADENOVIRAL INFECTIONS

DEFINITION

Adenoviral infections are very common in poultry and other avian populations. Although some infections such as those described in this section can be defined in terms of clinical and pathologic characteristics, most are either subclinical or associated with nondescript clinical syndromes.

OCCURRENCE

Serologic surveys indicate that nearly all poultry flocks experience infections with one or more adenoviral serotypes. Because most adenoviruses are egg transmitted, they can be present in developing embryos. The frequent presence of these viruses even in healthy birds means that their role in disease must be very critically examined. Adenoviruses must also be carefully excluded from chicken, duck, or turkey embryo-propagated vaccines as well as cellular and blood products used in diagnostic or research reagents.

HISTORICAL INFORMATION

1. Adenoviruses have long been recognized in humans and animals but they serve as primary pathogens in only a few instances. More often they accompany other more pathogenic agents or initiate pathologic effects in immunologically deficient animals.

2. The first recognized adenoviral infections of birds were quail bronchitis, a severe respiratory disease of quail; and an infection causing spontaneous mortality in chick embryos caused by an adenovirus known as chick embryo lethal orphan (CELO) virus. These two infections, described in 1949 and 1952, respectively, were later discovered to be caused by the same adenovirus serotype.

3. The exact role that adenoviruses play in avian diseases is unclear. Adenoviruses are suspected of playing a primary or secondary role in a variety of syndromes including inclusion body hepatitis in chickens; marble spleen disease of pheasants; hemorrhagic enteritis of turkeys; splenomegaly in chickens; egg production declines in laying chickens (egg drop syndrome—1976); and miscellaneous respiratory, arthritic, encephalitic, and enteric syndromes.

ETIOLOGY

1. Adenoviruses are DNA viruses that replicate and frequently produce inclusion bodies in the nuclei of infected cells. The viruses are unenveloped and range in size from 70 to 80 nm. They tend to be host specific.

2. The avian adenoviruses have been divided into three major groups or types. Although members of each type may share group antigens, there is no common antigen shared between types and there is no antigenic relationship between avian and mammalian adenoviruses. Type I avian adenoviruses include the CELO virus and numerous other serotypes among which are viruses associated with inclusion body hepatitis of chickens; type II avian adenoviruses encompass the viruses causing marble spleen disease in pheasants and hemorrhagic enteritis in turkeys; type III adenovirus is the hemagglutinating agent responsible for egg drop syndrome─1976.

EPIZOOTIOLOGY

1. Egg-transmitted adenoviruses may remain inactive in infected chickens or poults until maternal antibody wanes. Virus replication and shedding, especially in feces, occurs from 2 to 3 weeks of age onward.

2. In exposed birds the virus enters via the alimentary tract (and, in some cases, by the conjunctiva and nasal passages) and primary replication occurs in the nasopharynx and intestine. Frequently there is a viremic stage in the infection with widespread dissemination of virus to secondary sites of replication. As active antibody is produced viral activity wanes but the virus may persist in a latent state in some organs. There may be periods of virus reactivation throughout life especially during episodes of immunosuppression or stress.

3. Exposure to one serotype of type I avian adenovirus confers no immunity to other serotypes within this group. Similarly infections with type I will not protect against infection with types II or III. Thus, birds can and do suffer repeated infections with antigenically unrelated adenoviruses.

4. Adenoviruses tend to persist in a contaminated environment because they are relatively resistant to physical and chemical environmental factors. This group of viruses is susceptible to formaldehyde and iodine disinfectants.

CLINICAL AND PATHOLOGIC FEATURES

Diseases with reasonably well-established adenoviral etiologies, namely inclusion body hepatitis in chickens, quail bronchitis, hemorrhagic enteritis, and egg drop syndrome—1976 are presented in detail in this section. Reports of other diseases attributed to adenoviral causation should be scrutinized closely for solid evidence of a definitive etiologic role.

I. QUAIL BRONCHITIS
(QB)

DEFINITION

Quail bronchitis (QB) is an acute, contagious and sometimes highly lethal respiratory disease of bobwhite quail (*Colinus virginianus*) caused by an adenovirus (type I, serotype 1) and characterized by catarrhal tracheitis and airsacculitis.

OCCURRENCE

QB, originally recognized in 1950, has been documented sporadically in captive quail throughout the United States. There is evidence suggesting occurrence in wild quail as well.

ETIOLOGY

The adenovirus causing QB is closely related to the prototype CELO (chick embryo lethal orphan) virus, which is widespread in chickens. This suggests a potential hazard to quail in situations where there is direct or indirect contact with chickens and perhaps other avian carriers.

EPIZOOTIOLOGY

1. As with other birds, the probable sources of the causative adenovirus for susceptible bobwhite quail are infected breeders (via transovarial passage), carrier birds, or contaminated feces, or mucus mechanically carried from infected premises.

2. Once established in a flock the QB virus spreads rapidly primarily by the fecal-oral route. Morbidity usually reaches 100% in susceptible birds.

3. The disease frequently occurs in succeeding broods of quail reared on contaminated premises owing in great part to the resistance and persistence of the causative adenovirus.

CLINICAL SIGNS

1. QB occurs with sudden onset of severe respiratory signs including tracheal rales, coughing, and sneezing. Lacrimation, conjunctivitis, and neurologic disorders may also be seen but are less consistent signs.

2. The disease is most severe in young quail (under 4 weeks of age). Infections are milder or subclinical in birds over 8 weeks of age.

3. The incubation period of QB is 2-7 days, which explains the explosive spread of the disease in susceptible flocks. Morbidity and mortality can be substantial, ranging from 10 to 100% in young birds and the course of the disease in affected flocks varies from 1 to 3 weeks.

LESIONS

1. Excess mucus with thickening and roughening of the mucosa are the major lesions in the trachea [Fig. 1; Quail bronchitis; Cornell U] and bronchi. Air sacs may be mildly thickened and cloudy.

2. Clouding of corneas, conjunctivitis, and mucosal congestion in the nasal passages and infraorbital sinuses are occasionally noted.

3. Microscopically, the major lesions include mild to moderate epithelial deciliation and hyperplasia of respiratory epithelium and variable lymphocytic/plasmacytic infiltration in the tracheal or bronchial propria. Intranuclear inclusion bodies may be present in respiratory epithelium in early stages of infection.

DIAGNOSIS

1. Acute respiratory disease with high mortality in young quail chicks is highly suggestive of QB.

2. Confirmation of severe catarrhal tracheitis and bronchitis on histopathologic examination and demonstration of intranuclear inclusion bodies in respiratory epithelium establish a strong tentative diagnosis.

3. Isolation and identification of the causative adenovirus confirms the diagnosis of QB. Isolation is accomplished by inoculation of 9-11-day-old specific-pathogen-free embryonating eggs via the allantoic sac with triturates of trachea, air sacs, lungs, etc. Virus isolation may also be accomplished using cell cultures (chick embryo kidney or chicken kidney).

4. Serologic tests are of limited value unless flock sampling is done on both an acute and convalescent basis to demonstrate definitive seroconversion. Agar-gel precipitin and virus neutralization for type I serotype 1 avian adenovirus are applicable tests.

CONTROL

Little can be recommended except frequent monitoring to assure QB virus-free breeding stock and strict isolation of young growing birds to avoid introduction of the virus. No federally licensed vaccines are available.

TREATMENT

There is no effective treatment but increasing brooding house temperature, elimination of drafts, and expanding floor space may be helpful as supportive measures in the face of an outbreak.

II. INCLUSION BODY HEPATITIS

(IBH; Adenoviral Infection)

DEFINITION

Inclusion body hepatitis (IBH) is an adenoviral infection of young chickens characterized by sudden onset and sharply increased mortality, short course, anemia, and hepatitis, often accompanied by intranuclear inclusion bodies.

OCCURRENCE

IBH occurs in 3-15-week-old chickens but more frequently in 4-8-week-old chickens. IBH appears to be a secondary invader when there is immunosuppression caused by other diseases (infectious bursal disease or chicken infectious anemia). Many flocks of older chickens are solidly immune and serologic testing indicates that they carry antibody to the disease. IBH has been described in Canada, the United States, the United Kingdom, Italy, Iraq, and Australia. With the advent of breeder vaccination for infectious bursal disease (IBD), the incidence of IBH has been reduced substantially.

HISTORICAL INFORMATION

1. In 1963 hepatitis with inclusion bodies was described in chickens but the causative agent was not identified. That outbreak probably was the disease we now call inclusion body hepatitis (IBH). In the early 1970s a similar disease occurred in many flocks in Canada and the United States. Adenovirus was isolated from an Indiana outbreak and, eventually, from flocks in many other locations.

2. Hemorrhagic syndrome (aplastic anemia), a syndrome diagnosed frequently in the 1950s, is now suspected of being caused by adenoviral infection. However, there may be more than one cause of hemorrhagic syndrome. A virus designated “chicken infectious anemia (CIA) virus” is strongly suspect as an important causative factor.

ETIOLOGY

1. At least three serotypes of adenovirus have been isolated from chickens with IBH. In several IBH adenovirus isolates there is an accompanying adenovirus-associated parvovirus.

2. Although many features of IBH can be reproduced using adenovirus isolates in specific- pathogen-free chicks, many investigators conclude that the natural disease does not occur without the immunosuppressive effects of early infectious bursal disease.

EPIZOOTIOLOGY

1. Infected chickens shed adenovirus in their feces for at least a few weeks and infection is suspected of spreading slowly through a flock. The virus is resistant to many environmental influences and could be spread readily on fomites or mechanically. Farm-to-farm spread has been observed.

2. Circumstantial evidence suggests that the IBH adenovirus can be transmitted through the egg. The virus spreads laterally from infected to susceptible chickens, presumably through contaminated feed, water, and the environment.

CLINICAL SIGNS

1. A sudden marked increase in mortality often is the first indication of the disease. Mortality increases for 3-5 days, levels off for 3-5 days, and then decreases to normal levels over another 3-5 days. Total mortality may approach 10% but is usually considerably lower.

2. The morbidity in an outbreak is less than would be expected considering the mortality. Affected birds often show clinical signs for only a few hours and then die.

3. There are few specific signs. There may be pallor of the comb, wattles, and facial skin. The affected birds are depressed and listless. In some outbreaks the clinical signs are masked by other diseases in the flock.

LESIONS

1. The skin is pale and may be icteric and contain hemorrhages, particularly over the legs and breast. Internally, hemorrhages often are present in skeletal muscles and under serous membranes.

2. The liver is swollen, yellow to tan, and there may be mottling with focal soft areas [Fig. 1; Inclusion body hepatitis; Cornell U]. There are petechial and ecchymotic hemorrhages under the capsule and in the parenchyma.

3. The kidneys frequently are swollen and pale and may contain cortical hemorrhages.

4. The bone marrow is often pale yellow and the blood is thin and watery. The bursa of Fabricius and spleen are usually small. Pericardial fluid is often increased and there may be gray to white patches on the heart.

5. Microscopically there is extensive degeneration and necrosis in the liver and there may be intranuclear inclusions in parenchymatous cells [Fig. 2; Inclusion body hepatitis; Cornell U] during the early stages of the disease. There is hypoplasia of the bone marrow.

DIAGNOSIS

1. In young, growing flocks a sudden increase in mortality accompanied by low morbidity is suggestive of IBH. Typical gross lesions and a history of prior outbreaks in the area or on the premises are helpful.

2. Demonstration of typical microscopic lesions in the liver, including intranuclear inclusions, is often used as a basis for a diagnosis. The adenovirus often can be isolated from the respiratory or digestive tract.

3. Isolation of an adenovirus or the demonstration of a titer to adenoviral group antigen do not prove infection in ailing flocks; adenoviruses and antibody to them are widely distributed in poultry. Nevertheless, an agar-gel precipitin test is widely used to demonstrate antibody. It may be useful if the reactor rate can be shown to increase between onset and convalescence.

4. IBH should be differentiated from aplastic anemias caused by toxic agents.

5. One should attempt to determine if the birds were previously infected with IBD or CIA virus. Inclusion

body hepatitis is now suspected of occurring in immunologically deficient flocks as a consequence of earlier infection with IBD or CIA virus.

CONTROL

1. Because IBH virus is suspected of being transmitted through the egg, eggs from primary breeding flocks whose progeny have consistently had IBH should not be used for hatching.

2. Application of quarantine and good sanitary practices appears to be the best defense against infection. Wild birds should be kept out of poultry houses because they possibly may serve as disseminators of virus.

3. No vaccine is available. It appears that only polyvalent vaccines would be of value because at least three serotypes of adenovirus have been isolated from birds with IBH.

4. Prevention must begin with the control of IBD and CIA. Vaccination of breeders for IBD virus and natural or controlled exposure of breeder pullets for CIA virus is the most practical method for preventing IBH.

TREATMENT

There is no effective treatment for chickens with IBH. Good husbandry and care usually suppress mortality.

III. HEMORRHAGIC ENTERITIS OF TURKEYS

(HE; Bloody Gut)

DEFINITION

Hemorrhagic enteritis (HE) is a viral disease of young turkeys characterized by sudden onset, depression, bloody droppings, and variable but often high mortality. A subclinical form characterized by an enlarged, mottled spleen occurs and is more common than the acute form.

OCCURRENCE

HE typically occurs in 6-12-week-old turkeys but has been seen in poults as young as 2 weeks and in older turkeys. It is rare in turkeys less than 4 weeks of age, presumably because of maternal antibody. The disease is more prevalent in the summer and in turkeys on range. HE has been reported from most turkey-raising areas of the United States and appears to be increasing in incidence. The disease has a worldwide distribution.

HISTORICAL INFORMATION

HE was first reported in 1937 but the cause was unknown. Only a few reports of the disease were published during the next 30 years. In 1972 the disease clearly was demonstrated to be caused by a viral infection. Since 1970 there have been numerous reports on research and field aspects of the disease. HE now is recognized as a common and important disease of turkeys.

ETIOLOGY

1. The etiologic agent is a type II adenovirus. Thus far it has not been possible to propagate the agent in embryos but recently it has been propagated in tissue culture.

2. Marble spleen disease (MSD) in pheasants and splenomegaly in chickens are caused by the same or similar viruses. The latter has been confused with Marek's disease and resulted in high condemnations of affected flocks.

EPIZOOTIOLOGY

The epizootiology of HE is not known. The virus possibly is transmitted by chickens or other birds, rodents, or on contaminated equipment, shoes, boots, etc. Once introduced, the virus spreads laterally, presumably through ingestion of feces from infected turkeys. Infection frequently reoccurs on the same farm in successive flocks. There is no evidence of egg transmission. Infection of turkeys with HE virus results in a transient immunosuppression, often involving secondary colibacillosis.

CLINICAL SIGNS

1. Sudden deaths are often the first sign of HE in a flock. A concurrent drop in feed and water consumption may be noted. Droppings containing fresh blood or melena can be seen, especially around waterers.

2. A few birds exhibit signs of depression and have bloody feces. Blood may be seen oozing from the vent of dead or moribund birds or may be adhered to feathers around the vent. Blood may be expelled from the vent if the abdomen is squeezed. Most birds with bloody feces die.

3. The disease usually runs its course in a flock in 10-14 days. Most mortality occurs over a 10-day period. Mortality may exceed 60% but averages 5-10%.

4. Outbreaks of colisepticemia often follow clinical and subclinical infections with hemorrhagic enteritis virus 12-14 days later. Colisepticemia may be the only indication of prior subclinical infection.

LESIONS

1. Skin of dead poults often exhibits pallor. The birds are well fleshed.

2. The intestinal tract, especially the small intestine, is distended, dark purple, and filled with bloody material [Fig. 1; Hemorrhagic enteritis; Cornell U]. The mucosa is congested, especially in the duodenum, and may be covered with a yellowish layer of fibrinous debris. An intact spleen and bursa of Fabricius have been shown to be necessary for development of intestinal hemorrhage.

3. Small hemorrhages have been reported at various sites including: subcutaneous tissues, breast and thigh muscles, on the epicardium of the heart or surface of the liver, the gizzard, the proventricular-ventricular junction, at the pyloric opening, and on the kidneys.

4. The spleen is strikingly mottled and enlarged [Fig. 2; Hemorrhagic enteritis; NCSU]. Experimentally infected birds have splenic enlargement only during the first 4 days of illness. Afterwards the spleen is shrunken and silver-gray. Similar splenic lesions are seen in affected chickens and pheasants.

5. Microscopically, white pulp of the spleen is hyperplastic and there may be small, necrotic foci. Large acidophilic to basophilic intranuclear inclusions bodies are present in reticuloendothelial cells. The condensed nuclear chromatin around inclusions often resembles a signet ring. Similar inclusions can occur in cells in the lamina propria of the intestine and in many other organs but are less numerous.

6. The only lesion of MSD in pheasants not seen in turkey poults is marked congestion and edema of lungs. In affected pheasants this lesion often appears to be the probable cause of death. Pheasant lungs may also have focal areas of necrosis and reticular cell hyperplasia and the intranuclear inclusions may be found in reticular cells.

DIAGNOSIS

1. Typical history and gross lesions strongly suggest the diagnosis. Demonstration of intranuclear inclusions in reticuloendothelial cells in the spleen or intestine confirms the diagnosis.

2. The disease can be reproduced in 6-week-old or older, susceptible poults by giving minced splenic tissue or its supernate intravenously, orally, or intracloacally. Typical intestinal content also will reproduce the disease when given orally or cloacally.

3. If known-positive antiserum and known-infectious splenic tissue are available, it is possible to use the agar-gel diffusion test to demonstrate antigen in the spleen of an infected turkey or to demonstrate antibody in the convalescent sera of recovered birds.

4. HE must be differentiated from other causes of severe enteritis, from acute coliform septicemia, and, rarely, from reticuloendotheliosis viral infection in turkeys. Occasionally septicemic diseases including streptococcosis, colisepticemia, and fowl cholera produce an enlarged, mottled spleen. Adenoviral splenomegaly in broilers must be differentiated from Marek's disease.

CONTROL

1. Use a live tissue culture vaccine by water administration to prevent HE in turkeys. This vaccine can also be used to prevent MSD of pheasants.

2. Good biosecurity is helpful in preventing spread of infection from flock to flock.

3. Good care and management will reduce mortality and economic loss. Radical changes in feed or management should be avoided.

4. Antibiotic therapy should be started within 1 week after onset of the disease to prevent secondary colisepticemia.

TREATMENT

1. There is no practical satisfactory treatment of HE in turkeys.

2. The subcutaneous inoculation of 0.5-1.0 ml of immune antiserum from recovered turkeys may decrease the severity of a flock outbreak. Inoculation is done as soon as possible after onset of the disease.

3. Antibiotic therapy should be started within 1 week after onset of the disease to prevent secondary colisepticemia.

IV. EGG DROP SYNDROME—1976

(EDS76)

DEFINITION

Egg drop syndrome (EDS76) is an infectious disease of laying hens caused by a hemagglutinating adenovirus and characterized by loss of color in pigmented eggs and failure to achieve production targets or by production of thin-shelled or shell-less eggs in otherwise healthy birds.

OCCURRENCE

EDS76 is known to affect only laying hens although the causative virus has been recovered from ducks, geese, and a variety of other waterfowl. It appears that the EDS76 virus is a well-adapted duck or goose adenovirus and the chicken is not a natural host. Reproductive disorders are not associated with infection in waterfowl. EDS76 has been documented in numerous countries throughout the world, but not in the United States.

HISTORICAL INFORMATION

This syndrome was first described as a unique problem in laying hens in Holland in 1976. Isolation of a hemagglutinating adenovirus and much of the research defining the disease and the circumstances accounting for establishment of the virus in chickens were reported from Northern Ireland in the 1970s. Reports documenting the infection in other locations around the world are numerous.

ETIOLOGY

EDS76 virus is classified as an adenovirus on the basis of its morphology, replication, chemical composition, and resistance to chemical and physical agents. However, the virus appears not to be related to 11 fowl and 2 turkey prototype adenoviruses based on serum neutralization or hemagglutination inhibition tests. Only one serotype of EDS76 virus is known. The virus is readily propagated in cell cultures derived from ducks and duck embryos but grows poorly in turkey cells and not at all in a wide range of mammalian cells. The virus replicates to very high titers in embryonated duck and goose eggs but no growth has been detected in embryonated chicken eggs.

EPIZOOTIOLOGY

It appears that the EDS76 virus was first introduced to chickens through a contaminated vaccine. Initially, major transmission was vertical from breeders to progeny chicks and the virus often remained latent until birds approached peak production. In many cases infected chicks do not excrete the virus or develop detectable antibodies until the flock is between 50% and peak production. At this stage the virus is excreted and spread of the virus occurs. Lateral spread to susceptible contacts is slow in cage houses but faster in birds on litter. Infected chickens develop a viremia and the virus is distributed in various internal organs including the alimentary canal, respiratory tract, spleen, liver, and oviduct. Virus is shed primarily from the pharynx and in feces but spread seems to depend on contact with feces. Virus can also be spread by contaminated needles when infected birds are in the viremic phase. Although ducks and other waterfowl are the natural hosts of the EDS76 virus, natural transmission from waterfowl to chickens either does not happen or is extremely rare. There appears to be no age or breed predilection in chickens and effects of infection are not apparent until pullets achieve peak production.

CLINICAL SIGNS

There are no reliable clinical signs other than effects on the ovary and oviduct. Alterations in egg production vary depending on the serologic status of the infected flock. In flocks with a low rate of serologic reactors the first sign is loss of color in pigmented eggs followed rapidly by production of thin-shelled or shell-less eggs. Other than shell quality there are few other problems with eggs; internal quality, fertility, or hatchability are not frequently affected. In flocks with a substantial level of antibody before the virus is unmasked the major reproductive effects are failure to achieve projected egg production or delay in the onset of lay. The drop in production associated with EDS76 may be rapid or extended. Depressed production lasts 4-10 weeks and production is reduced up to 40%.

LESIONS

Gross lesions other than inactive ovaries and atrophied oviducts are not seen in natural infections. Edema and swelling of the mucosal folds of the uterus have been described in experimentally infected hens. Histologically, oviductal changes include proprial edema, infiltration of mononuclear leukocytes (lymphofollicular aggregates in some cases), atrophy of tubular glands, and degeneration/desquamation of uterine epithelium. Intranuclear inclusion bodies may be seen in epithelial cells of the uterus, isthmus, and vagina.

DIAGNOSIS

Reduction in production with the occurrence of depigmented, soft-shelled eggs in the absence of other clinical signs should trigger consideration of EDS76. Isolation and identification of the virus is best achieved using EDS76-free embryonated duck or goose eggs or cell culture of duck or goose origin. Harvested allantoic fluid or cell culture supernatant can be checked for hemagglutinating activity, which is inhibited by specific EDS76 antiserum. Use of immunofluorescence with labeled EDS antiserum is perhaps the most direct means of identifying the virus in cell cultures. Application of serology in suspect flocks is most helpful immediately after egg changes are observed because many infected flocks do not have demonstrable antibody during the growing period. The hemagglutination inhibition test or the serum neutralization tests are the most helpful in serodiagnosis.

CONTROL

An oil-adjuvant inactivated vaccine is widely used at 14-16 weeks of age in replacement pullets. Although the vaccine confers good protection against clinical disease, vaccine-induced antibody titers may not be high or uniform. As with other diseases, avoidance of infection is the best prevention. Knowledge of the infection status of breeder flock sources of replacement chicks is essential. If chicks are reliably determined to be free of EDS76 virus, traffic control to avoid introduction is critical as is avoidance of contact with waterfowl.

AVIAN ENCEPHALOMYELITIS

(AE; Epidemic Tremor)

DEFINITION

Avian encephalomyelitis (AE) is a viral infection of chickens, turkeys, pheasants, and coturnix quail characterized in young birds by ataxia progressing to paralysis and, usually, by tremors of the head and neck. Infected adults usually show no signs.

OCCURRENCE

Clinical outbreaks are usually observed in chickens and most outbreaks are in 1-3-week-old chicks. Turkey poults, pheasants, and coturnix quail are also infected naturally. Experimental infection has been induced in ducklings, guinea fowl, and pigeon hatchlings. Infection can occur in older birds but usually is clinically inapparent. AE is worldwide in distribution.

HISTORICAL INFORMATION

1. In 1930 AE was first seen in 2-week-old Rhode Island Red commercial chicks. Within a few years the disease was present in most of the other New England states and was referred to as "New England disease". Between 1955 and 1970 the disease was described successively in coturnix quail, pheasants, and turkeys.

2. A nationwide testing program for AE antibody revealed that many chicken flocks in the United States have antibody to AE virus.

3. Hatcheries once replaced baby chicks that had AE or developed AE shortly after delivery. This practice caused considerable loss to the hatcheries. Vaccination of the breeders was first successfully implemented in the 1950s and AE largely became controlled in commercial flocks by the 1960s.

ETIOLOGY

1. AE is caused by a hepatovirus belonging to the Picornaviridae family. There appear to be no serologic differences among isolates although they vary in their tissue tropisms. All field strains are enterotropic but some strains are more neurotropic than others and pathogenicity varies.

2. The virus can be grown in the yolk sac of chick embryos free of maternal antibodies and in a variety of tissue culture systems. Embryo-adapted strains are not infectious by the oral route, are highly neurotropic, and cause muscular dystrophy in inoculated embryos.

3. Virus is present in the feces of infected birds and will survive there for at least 4 weeks.

4. The virus is fairly resistant to various environmental conditions.

EPIDEMIOLOGY

1. During the acute phase of infection in laying chickens, a period up to 1 month, some layers shed virus in some of the eggs they lay. Although vertically transmitted AE may affect hatchability, many of the chicks will hatch and can show clinical signs of the disease as early as the 1st day of age. The infected chicks will eliminate virus in their feces and virus may spread horizontally to other chicks of the hatch. Younger chicks shed for longer a longer period of time than older chicks.

2. The method of transmission of AE to susceptible adult flocks is unknown but is probably via fomites. Multiage farms are more likely to be infected than those with single age groups.

CLINICAL SIGNS

1. In chicks, signs may be present at the time of hatch but usually occur between the 1st and 2nd week. Age resistance is marked if exposure is after 2-3 weeks of age.

2. In chicks, signs include dull expression, ataxia progressing to paralysis and prostration [Fig. 1; Avian encephalomyelitis; Cornell U], and tremors of the head and neck. Tremor may be inapparent but often can be accentuated if the bird is frightened or held inverted in the hand. Prostrate birds are soon trampled and killed by the other birds.

3. The morbidity in chicks is quite variable but may go as high as 60%. If most chicks in the flock come from immune dams, morbidity is usually low. Mortality averages 25%. Few birds with signs recover completely. Those that survive often fail to grow or produce eggs normally. Many survivors later develop a bluish opacity to the lens and have impaired vision [Fig. 2; Avian encephalomyelitis; Cornell U].

4. Layers seldom show signs when infection is going through the flock. However, good production records often reveal a significant decline in egg production generally lasting no more than 2 weeks.

LESIONS

1. Generally, there are no gross lesions. In chicks, whitish areas in musculature of the ventriculus can sometimes be observed. No gross lesions are seen in adult birds.

2. Microscopic lesions, if typical, have special diagnostic value. There is a disseminated, nonpurulent encephalomyelitis with widespread and marked perivascular cuffing. Two microscopic changes are especially helpful: swelling and chromatolysis of neurons [Fig. 3; Avian encephalomyelitis; NCSU] in nuclei (nucleus rotundus and nucleus ovoidalis) in the midbrain and cerebellum, and dense lymphoid aggregates in the muscle of the proventriculus and/or gizzard.

DIAGNOSIS

1. In chicks the history, age of the birds, and typical signs of central nervous system (CNS) lesions permit a strong presumptive diagnosis. The diagnosis can often be strengthened by histopathologic examination. Alternatively, the direct fluorescent antibody technique can be used to demonstrate AE viral antigen in infected chicks.

2. Isolation and identification of the virus from the brains of infected chicks is possible but is time consuming and expensive. Also, there must be a source of susceptible chick embryos and this usually necessitates a layer flock that has never been exposed to AE.

3. Antibodies to AE can be detected as early as 4 days postinfection and persist for at least 28 months. Serologic assays include the ELISA, immunodiffusion test, virus neutralization test, passive hemagglutinin test and the indirect FA test. Rising titers in sequential samples are highly suggestive of active infection.

4. AE must be differentiated from other diseases that cause signs of CNS disease in young birds. These include:

Newcastle disease	Mycotic encephalitis
Arboviral infection	Brain abscesses
Vitamin deficiencies	Marek's disease
(E, A and Riboflavin)	Toxicities
Equine Encephalomyelitis Virus	(salt, some pesticides, etc.)

CONTROL

1. Chicks from immune hens are usually protected by parental immunity during the critical first few weeks after hatching. Breeding flocks can be vaccinated to provide maximum protection to their chicks. Although vaccination is usually conducted prior to the onset of lay, some killed vaccines can be used during production.

2. Both killed and live vaccines are used for vaccination and are effective. Live virus vaccines must not be embryo adapted as they lose their ability to infect orally and can cause clinical disease when administered parenterally. Live vaccine is given by the wing web stick method in combination with pox, via the drinking water, or by spray. Birds that will serve as breeders should not be vaccinated until they are at least 8 weeks old. One vaccination is usually adequate for the life of the bird. Live vaccines should be applied at least 4 weeks prior to production; vaccines used in chickens can be protective for turkeys.

3. Chicks from flocks that have been naturally infected will probably receive enough parental immunity so that they will not develop the disease.

TREATMENT

Treatment is of no value.

AVIAN INFLUENZA

(AI; Influenza)

DEFINITION

Avian influenza (AI) is a viral disease characterized by respiratory signs, depression and reduced feed and water intake. In egg laying birds there is a decline in egg production and quality. There are two pathotypes of AI virus: the most common is low pathogenic AI (LPAI) and the other is highly pathogenic AI (HPAI).

The most virulent form (HPAI) was once called fowl plague. At the 1981 International Symposium on Avian Influenza, the term fowl plague was replaced with the term "highly virulent" influenza virus infection. The AI epidemic of 1983-1984 required yet new terms to describe relative pathogenicity of different isolates of the same serotype (nonpathogenic, low-pathogenic, and highly pathogenic).

OCCURRENCE

AI outbreaks have occurred throughout the world. LPAI is common in large turkey-producing areas, particularly where semi-confinement or range-rearing is still widely practiced. Outbreaks are more sporadic in other areas of the United States. AI can occur in most, if not all, species of birds. In the United States, most outbreaks have been in turkeys. A few outbreaks have occurred in chickens. Humans, horses, pigs, and some wildlife species may be infected with influenza viruses.

A Pennsylvania chicken outbreak of LPAI in 1983 mutated into HPAI in 1983-1984 resulting in a federal-state eradication program that required the depopulation of 17 million birds. Similar outbreaks of LPAI in Mexico in 1992 and Italy in 1999 also mutated into HPAI causing severe losses.

HISTORICAL INFORMATION

Highly pathogenic AI (fowl plague) was first documented in Italy more than 100 years ago. Highly pathogenic AI first occurred in the United States in 1924-1925 and again in 1929 but was eradicated both times. The epidemic of HPAI in the northeastern United States in 1983-1984 cost over 70 million dollars eradicate. The United States has been HPAI free since 1986, although LPAI viruses are present and some have caused significant losses in poultry. In 2004 a virus was detected in Texas that did not meet the pathological criteria for HPAI, but on the basis of molecular characteristics of the cleavage site of the hemagglutinin the virus was designated high path.

It was in 1964 that influenza viruses of low to moderate pathogenicity were first detected in poultry and they have been detected somewhere in United States poultry every year since 1964.

An International Commission for Control of Avian Influenza monitors outbreaks of HPAI and has designated certain laboratories as reference labs for diagnosis. In the United States, the National Veterinary Service Laboratory in Ames, Iowa, can identify influenza virus serotypes and pathotypes.

ETIOLOGY

Avian influenza is caused by a type A influenza virus belonging to the Orthomyxoviridae family. The agar gel immuno-diffusion (AGID) test identifies antibody to type A antigen.

Influenza viruses have two important surface antigens, hemagglutinin (H) and neuraminidase (N) that give rise to subtype names for specific viruses (eg. H4N6). There are 16 hemagglutinins and 9 neuraminidases making 144 possible virus subtypes. Influenza viruses are subtyped by hemagglutination inhibition and neuraminidase inhibition tests. Cross-protection does not occur between subtypes.

Influenza viruses vary widely in pathogenicity and ability to spread among birds. Two pathotypes are recognized: LPAI and HPAI. These pathotype designations are derived from laboratory inoculation of 8 susceptible chickens; HPAI isolates cause death in 6 or more chickens while LPAI viruses usually do not result in death of any chickens following experimental inoculation. Although most H5 and H7 isolates are low path viruses, so far all HPAI outbreaks have been due to H5 or H7 viruses.

All influenza viruses hemagglutinate chicken red blood cells. Most grow readily in embryonating chicken eggs and tissue culture. They are susceptible to detergents, disinfectants and heat.

EPIDEMIOLOGY

Wild waterfowl and shorebirds are the major natural reservoir of influenza viruses. Wild waterfowl are asymptomatic, may excrete virus in the feces for long periods, may be infected with more than one subtype, and often do not develop a detectable antibody response. Influenza virus has been recovered directly from lake and pond water utilized by infected wild ducks. Contact of these birds with range-reared commercial flocks is an important factor in some outbreaks. This source of infection often results in a seasonal incidence in some states.

Two man-made reservoirs worth mention are live bird markets and commercial swine facilities. Live bird markets have existed in large cities forever, but they are an emerging phenomenon in some areas. They serve as a focal point for gathering and housing many species of birds that are then sold in or around large cities. These facilities are usually neither cleaned nor depopulated. The continuous supply of susceptible poultry in such markets enhances opportunity for viral replication and mutation, and this in turn enhances the opportunity for viruses to be carried back to susceptible poultry flocks. Swine have been known to be infected with swine flu (H1N1) since the 1930s, but recently another subtype (H3N2) has been spreading in swine populations. Transmission of influenza from swine to turkeys has been documented.

AI viruses have been isolated from imported exotic birds. These infected birds are a potential threat to cage birds, wild birds, and poultry.

Although waterfowl shed virus in their droppings for long periods, most viral shedding from infected gallinaceous poultry stops after seroconversion. Influenza virus is released in respiratory secretions and excretions and droppings of infected birds where it is protected by organic material. The virus is labile in warm conditions, but can survive for months in a cold environment. Influenza virus has been isolated from turkey eggs and semen, but there is no evidence of egg transmission. Improper disposal of infected eggs could potentially expose other susceptible birds, but such transmission has not been observed.

Once AI is introduced into the poultry industry it is transmitted from farm to farm by direct and indirect contact. AI viruses can be transmitted on contaminated shoes, clothing, crates, and other equipment and by movement of birds and manure.

CLINICAL SIGNS

Most outbreaks are caused by LPAI viruses. The LPAI signs vary greatly and depend on many factors, including the age and species infected the virulence of the virus, concurrent infections, and husbandry. In most outbreaks, signs are predominantly those of a respiratory disease with coughing, sneezing, rales, lacrimation, sinusitis, and depression. In egg layers decreased egg production and quality are seen.

In young growing turkeys the disease may be subclinical or severe, particularly where secondary infection with live pasteurella vaccine, *E. coli*, or bordetella occurs. Outbreaks in egg laying turkeys often reduce production markedly and frequently are associated with abnormal eggshell pigmentation and quality.

Morbidity and mortality are highly variable, depending upon the same factors that determine clinical signs noted above.

HPAI is a severe form of influenza usually seen in chickens. Viruses of high pathogenicity may cause fatal infections preceded by few signs. Onset is sudden, the course is short, affected birds are quite ill, and mortality may approach 100%. Signs may relate to the respiratory, enteric, or nervous systems. There may be diarrhea, edema of the head and face, or nervous disorders.

LESIONS

With LPAI outbreaks in poultry there is mild to moderate inflammation of the trachea, sinuses, air sacs and conjunctiva. In laying birds there often is ovarian atresia and involution of the oviduct. Various degrees of congestive, hemorrhagic, transudative, and necrotic lesions have been described.

In HPAI infection, gross lesions in chickens are the most extensive and severe. Fibrinous exudates may be found on the air sacs, oviduct, pericardial sac, or on the peritoneum. Small foci of necrosis may be apparent in the skin, comb, and wattles or in the liver, kidney, spleen, or lungs. Indications of vascular damage often include congestion, edema, and hemorrhages at many sites.

Classical lesions of HPAI in chickens include cyanosis and edema of the head [Fig. 1; Avian influenza; NCFAD, Canada], [Fig. 2; Avian influenza; NCFAD, Canada], vesicles and ulceration on the combs, edema of the feet, blotchy red discoloration of the shanks [Fig. 3; Avian influenza; NCFAD, Canada], petechiae in the abdominal fat and various mucosal and serosal surfaces, and necrosis or hemorrhage in the mucosa of the ventriculus and proventriculus [Fig. 4; Avian influenza; UC Davis].

Lesions of HPAI in turkeys are not well described, but encephalitis and pancreatitis have been reported.

DIAGNOSIS

History, signs, and lesions may be suggestive of LPAI, but are similar to other diseases. With HPAI, lesions are more useful in a presumptive diagnosis. Confirmation of AI requires laboratory tests including serology and virus detection. Confirmation of HPAI requires inoculating susceptible chickens with the virus and molecular characterization of the cleavage site.

Influenza virus usually can be isolated in chick embryos from tissue or swab samples of trachea, lung, air sac, sinus exudate, or cloaca. The virus hemagglutinates chicken red blood cells. The AGID test can be used to identify type A internal antigen of the virus or to demonstrate an increase in antibody titer between acute and convalescent sera.

Influenza must be differentiated from other poultry diseases including Newcastle disease, other paramyxovirus infections, mycoplasmosis, chlamydial infections, and fowl cholera. Highly pathogenic AI should be differentiated from velogenic viscerotropic Newcastle disease. Because AI viruses causing highly pathogenic AI are considered exotic to the United States, they are reportable to the USDA, and confirmation by virus isolation is essential.

PREVENTION

Prevention of LPAI is largely through prevention of exposure to influenza viruses by direct or indirect contact with waterfowl and shorebirds, live bird markets and swine farms.

CONTROL

Once non H5 or H7 LPAI is introduced into the poultry industry, control is largely dependent on voluntary, industry efforts since there are no official state eradication programs.

1. Routine serologic monitoring of blood or egg yolk antibody is used in areas where AI has been a problem. This effort provides early detection of an outbreak and permits other measures such as isolation and sanitation to be used early.

2. Reporting outbreaks to industry personnel who are in direct or indirect contact with poultry is necessary so that people can take appropriate measures.

3. Voluntary isolation of infected flocks is the responsibility of the owner and is necessary to prevent transmission to other flocks. (Often doing nothing is the single most important thing to reduce the spread of disease.) Rigorous measures to prevent the contamination of and control the movement of people and equipment are required in order to stop this disease.

4. Different states and industries take different approaches to the next step. Controlled marketing of flocks after they have recovered from infection is common in the turkey industry. In some broiler producing states, voluntary destruction of infected flocks is encouraged.

5. Rescheduling flocks is necessary to make sure there is no active AI virus on the farm before another flock is placed.

6. Vaccines - Immunity is hemagglutinin subtype specific. Because birds are susceptible to all 15 hemagglutinins preventive vaccination is not practical. Once an outbreak occurs and the subtype is identified, however, vaccination is a tool that may be used to help bring the infection under control. Because influenza viruses are unstable little research has been done on live influenza virus vaccines for poultry. Only killed, injectable vaccines are available and currently USDA controls use of H5 or H7 vaccines.

   H5 or H7 LPAI results in a response that varies according to the response plan developed by each state. Generally all outbreaks of H5 or H7 LPAI will result in a rapid aggressive response, although the means that are used to bring it under control will vary according to species involved, density and the state plan.

   H5 or H7 HPAI results in a uniform response plan under the direction of the USDA APHIS VS although there will be input from public health, occupational health and pollution control agencies.

   History has proven that prevention of HPAI is based on successful control of H5 or H7 LPAI.

   Current USDA quarantine measures reduce the possibility of introducing highly pathogenic influenza through importation of poultry or exotic birds.

   All outbreaks of influenza should be reported immediately to the state veterinarian or other appropriate health authorities.

TREATMENT

There is no effective treatment. However, good husbandry, proper nutrition, and broad-spectrum antibiotics may reduce losses from secondary infections.

ZOONOTIC POTENTIAL

Although infection of humans with AI virus is rare, in 1997 18 people in Hong Kong fell ill due to a HPAI H5N1 virus. Six people died. Eradication of the HPAI outbreak was successful and no further human infection occurred.

More recently, in 2002 a pathogenic H5N1 virus believed to be related to the 1997 virus caused the death of one of two infected people in Hong Kong. This virus was the precursor of the viruses that subsequently spread in poultry and wild birds over most of Asia and parts of Europe and caused over 70 human deaths by January 2006.

AVIAN PNEUMOVIRUS INFECTION

DEFINITION

Avian pneumovirus (APV) infection is an infectious respiratory disease of turkeys, characterized by coughing, swollen sinuses and nasal discharge and lowered feed and water consumption. It has not been observed in broiler or layer type chickens in the U.S. In other parts of the world APV infection of turkeys has been called turkey rhinotracheitis (TRT), and APV infection of chickens has been called swollen head syndrome (SHS).

OCCURRENCE

1. APV infections have been described through out the world. Only Canada and Australia report no avian pneumovirus.

2. The host range for APV has not been established. The virus has been isolated from turkeys in many parts of the world. Experimental studies have shown chickens, ducks, guinea fowl and pheasants to be susceptible. Serological studies have detected antibody in ostriches and herring gulls. Using RT-PCR, APV RNA has been detected in geese, coots, sparrows, swallows, starlings and an owl. A seasonal incidence with peaks in the spring and fall has been observed.

HISTORICAL INFORMATION

1. Turkey rhinotracheitis, caused by an avian pneumovirus, was first identified in South Africa in the late 1970s and is now known to be present in many European, Asian and Central and South American countries.

2. In the U.S.A. APV was first identified in Colorado turkeys in 1997 by workers at the National Veterinary Services Laboratories (NVSL) in Ames, Iowa. Subsequently the disease was detected serologically, using an ELISA developed at NVSL, in Minnesota turkeys in the spring of 1997. It is likely that the infection was present in Colorado and Minnesota prior to 1997. It has remained in Minnesota turkeys infecting 40 to 50% of the flocks each year since 1997, and there has only been limited spread to neighboring states.

ETIOGENESIS

1. Avian pneumovirus is a member of the Paramyxoviridae family, subfamily pneumovirinae, genus metapneumovirus. Avian pneumoviruses are difficult to isolate, but once recovered from affected birds APVs grow in embryos and tissue culture systems. Unlike other members of the paramyxovirus family, pneumoviruses do not hemagglutinate.

2. Two pneumoviruses from Europe have been characterized as Type A and Type B avian pneumoviruses. The recent U.S.A. isolates, antigenically and genetically distinct from the two European types, have been tentatively called Type C APV. Pneumoviruses have a fusion (F) protein and a glycoprotein (G) as surface antigens. Limited studies indicate that there might be partial cross protection between APV types.

3. Pneumovirus is present in respiratory secretions and excretions of infected birds where it is protected by organic material. The virus is susceptible to detergents and disinfectants, survives drying for at least a week, survives many freeze-thaw cycles, survives in a pH range of 5 to 9 and survives for long periods of time under cool and moist environmental conditions. In poultry litter it has been shown to survive for three days at 20 to 25 C and for 14 to 30 days at 8 C.

EPIDEMIOLOGY

1. The natural reservoir of APV is unknown. Limited studies have implicated wild and domestic birds. Whether the virus persists in recovered turkey flocks is also unknown. TRT virus has been detected in oviduct tissue, and neonatal infection has been observed, but no proof of egg borne transmission has been shown.

2. The virus is transmitted by direct contact between infected and susceptible birds and is believed to be transmitted by indirect contact including exposure to aerosol droplets or to virus-contaminated boots, clothing or equipment. Airborne transmission has been demonstrated in the laboratory, but such transmission from farm to farm is unproven.

CLINICAL SIGNS

1. The clinical signs are extremely variable and seem to depend on the age, gender, concurrent infections, and environmental factors. Silent infections are possible. Listlessness, huddling, coughing, sneezing, rales, swollen sinuses, nasal discharge and stained shoulder feathers may be observed. As clinical signs subside birds may begin to die. Mortality rates in market turkeys range from nil to 80%, but death is usually due to secondary infections. FSIS condemnation rates are usually elevated if turkeys are infected within two weeks of processing.

2. Turkey breeder hens may have a decline in egg production of 10 to 30% and lay increased numbers of cull eggs. Mortality in breeders is usually 0 to 2% but may be higher if live pasteurella vaccine has been used in the flock.

LESIONS

There are few striking lesions because the disease is mild. There may be mild tracheitis, airsacculitis, lung congestion and inflammation of the turbinates. With early APV strains detected in U.S.A. microscopic lesions have been limited to infiltration of turbinates with lymphocytes, macrophages and plasma cells and excess mucus secretion, but more recently deciliation has also been observed. Secondary infections with *E. coli* or other bacteria may cause more severe lesions.

DIAGNOSIS

The history and signs of coughing, nasal discharge and swollen sinuses may be suggestive of APV infection but are similar to other respiratory infections. Confirmation of the diagnosis requires laboratory tests. The virus is difficult to isolate from swabs or tissues of affected birds so other laboratory tests used today are: immunohistochemistry on formalin fixed turbinate tissues, RT-PCR to detect viral RNA in tracheal swabs, choanal swabs or turbinates, and ELISA to detect pneumovirus-specific antibodies.

CONTROL

1. The reservoir of pneumovirus in nature is not known but wild birds are suspected. Whether or not infected flocks remain a source of virus for their whole life is also not known, but they should be considered a potential source for life. Prevention of pneumovirus infection requires preventing the introduction of the virus by direct or indirect contact from these possible reservoirs (wild birds and infected flocks).

2. Since the disease is spread by direct and indirect contact, strict biosecurity and a good sanitation program are imperative. A minimum biosecurity program for controlling APV would include:

    A. Crews that handle birds (vaccination, moving, live haul, insemination) must be controlled. Crew members should wear disposable or freshly laundered clothing including footwear.
    B. Equipment that moves from farm to farm and comes in contact with poultry or birds (rendering, moving, live haul trucks and dumpsters, loaders, vaccination equipment) should be washed with detergent and disinfectant.
    C. Poultry facilities should be wild bird proofed.

3. A live attenuated vaccine is available in the Midwestern USA. Killed autogenous oil emulsion vaccine has been used after live vaccines in turkey breeders.
4. A routine monitoring program is suggested for areas where APV infection has been a problem. Serological screening of blood samples and PCR testing of choanal swabs can provide early detection so that other control measures can be instituted.

TREATMENT

There is no treatment for pneumovirus infections other than good care. Reduced density, increased supplemental heat and good management conditions are associated with reduced financial loss due to the disease. Antibiotic treatment has been used to reduce the effects of concurrent bacterial infections.

AVIAN VIRAL TUMORS

DEFINITION

The several viral neoplastic diseases of chickens and turkeys, although previously considered a "complex", are actually distinct disease entities. In some cases a single tumor virus strain can induce multiple disease syndromes, thus causing uncertainty whether these neoplasms should be classified by etiology or by lesion type. Furthermore, some of the lesion types are so rare as to be of little concern.

In an attempt to simplify this situation, we will consider here only the four neoplastic disease syndromes that have economic importance: Marek's disease, a common lymphoproliferative disease of chickens caused by an alpha herpesvirus; avian leukosis/sarcoma, common retroviral diseases characterized by lymphoid or other neoplasias and lowered egg production in adult chickens; reticuloendotheliosis, which includes a runting disease and a chronic lymphoma in turkeys, chickens, and a variety of other avian species caused by a nondefective retrovirus; and lymphoproliferative disease, a retrovirus-induced disease of turkeys characterized by chronic lymphomas that although not yet reported in the United States, is found elsewhere and must be considered in a differential diagnosis.

I. MAREK'S DISEASE

DEFINITION

Marek's disease (MD) is a herpesvirus-induced neoplastic disease of chickens characterized by infiltration of various nerve trunks and/or organs with pleomorphic lymphoid cells.

OCCURRENCE

Marek's disease is important primarily in chickens, to a much lesser degree in quail, and has been rarely observed in turkeys, pheasants and jungle fowl. Turkeys and other species have limited susceptibility. The disease most commonly occurs in young, sexually immature chickens 2-7 months old, but can occur at virtually any age beyond 3 weeks. The disease occurs throughout the world and virtually all flocks are exposed to the causative virus.

HISTORICAL INFORMATION

A report in 1907 by a Hungarian veterinarian, Jozsef Marek, of paresis in roosters is the first description of the disease now called MD. The disease was first reported in the United States in 1914. Although forms of MD were an important cause of mortality in chickens prior to 1950, a sudden increase in mortality in the late 1950s and 1960s accelerated research. Reliable experimental transmission was achieved in 1962 and the causative herpesvirus was isolated and identified in 1967. Vaccines became available for use in the United States by 1970 and have been very effective in preventing the disease. However, sporadic losses and the fear of increased virulence of the virus have kept MD among the most important poultry diseases.

ETIOLOGY

1. Marek’s disease virus is a cell-associated alpha herpesvirus of subgroup a3. The herpesviruses associated with MD are classified into three serotypes. Serotype 1 isolates are ubiquitous in chickens and pathotypes vary from very virulent plus (vv+) (oncogenic) to nearly avirulent (mild). Serotype 2 isolates are common in chickens and are nononcogenic. Serotype 3 isolates, also known as turkey herpesvirus, are ubiquitous in turkeys and are nononcogenic. The three serotypes have considerable antigenic cross-reactivity.

2. The serotype 1 virus can be grown in cultured chick kidney cells prepared from 1-3 week old chicks and in duck embryo fibroblasts. It produces a distinct cytopathic effect with intranuclear inclusions in those cells.

Embryonal chick kidney cells and chick embryo fibroblasts are less effective for low-passage virus. Serotype 2 and 3 viruses can be isolated and propagated in chick embryo fibroblasts. The virus is usually tightly bound to living cells and in this form is very labile, but cell-free virus is released from the feather follicle epithelium and is relatively resistant to environmental factors. Both cell-associated and cell-free viruses are susceptible to a number of common disinfectants.

EPIDEMIOLOGY

Infected chickens shed virus-containing feather follicle dander, which is a source of infection for other chickens by the respiratory route. Infected carriers may or may not be clinically ill, and carrier birds can sporadically shed virus throughout their lifetimes. The disease is very contagious and infectious dander can be disseminated over long distances. Although excretions and secretions of infected chickens may contain virus, dander containing infectious enveloped virus particles is the most important means of transmission. Transmission of the virus through the egg does not occur. Hatchery transmission through shell contamination is also unlikely due to adverse environmental conditions for the virus.

CLINICAL SIGNS

Clinical signs occur in chickens affected with MD but are of little help in establishing a diagnosis. Birds with visceral tumors are depressed and often cachectic prior to death. Birds with lymphoid infiltration of peripheral nerves may demonstrate asymmetric partial paralysis [Fig. 1; Marek's Disease; NCSU] and / or dilation of the crop due to vagus nerve paralysis. Blindness is associated with lymphoid infiltration of the iris [Fig. 2; Marek's Disease; UC Davis]. Clinical signs usually do not appear prior to 3 weeks of age and peak between 2 and 7 months.

LESIONS

1. At least four different lesion patterns are recognized: gross enlargement [Fig. 3; Marek's Disease; UC Davis] and/or yellowing and loss of cross-striations of peripheral nerves [Fig. 4; Marek's Disease; NCSU]; discoloration of the iris [Fig. 5; Marek's Disease; NCSU]; enlargement of feather follicles [Fig. 6; Marek's Disease; Cornell U] with reddening (skin leukosis); and visceral tumors [Fig. 7; Marek's Disease; UC Davis] involving the liver [Fig. 8; Marek's Disease; UC Davis], heart [Fig. 9; Marek's Disease; UC Davis], spleen, gonad, kidney [Fig. 10; Marek's Disease; UC Davis], proventriculus [Fig. 11; Marek's Disease; NCSU], and other organs and tissues. Visceral tumors are the most frequent lesions, but combinations of lesion patterns are common.

2. Microscopically, the lymphomas are characterized by a mixture of pleomorphic lymphocytes [Fig. 12; Marek's Disease; UC Davis]. Some of these probably are true tumor cells that carry T-cell surface antigens and a tumor-associated antigen (MATSA). Others are probably host cells reacting against viral or tumor antigens and represent both T- and B-cells.

DIAGNOSIS

1. A diagnosis can usually be made after careful consideration of the history, the ages of the birds affected, and the location of the neoplastic lesions in a generous sample of typically affected chickens. Few epornitic diseases resemble MD with the exception of lymphoid leukosis and reticuloendotheliosis.

2. Marek's disease often occurs in 2-5-month-old (sexually immature) chickens but can also occur after the onset of egg production. Outbreaks after the onset of egg production in vaccinated stock have been called "late Marek's" and are often associated with newer, more highly virulent vv+ pathotypes.

3. Characteristics of MD lesions of importance in differential diagnosis include nerve involvement (when present), the absence of bursal lesions or, rarely, diffusely thickened bursas, and pleomorphic lymphocytes comprising lesions, some of which exhibit MATSA and only few of which are positive for immunoglobulin M (IgM). The ubiquitous nature of MD virus renders virology and serology of little value in diagnosis.

CONTROL

1. Commercial flocks are usually immunized via injection at 18 days of embryonation or at hatching. Care must be taken to insure that an effective dose is administered to every embryo or chicken. Because immunity from vaccination is not fully developed for 7-10 days, it is crucial to minimize early exposure. This requires careful sanitation and disinfection, particularly because MD virus survives well for months in poultry houses. Revaccination is not necessary and immunity is usually life-long. Appearance of the disease at older ages has been attributed to immunodepression due to environmental stress or infection with vv+ pathotype.

2. The most common vaccines consist of turkey herpesvirus (HVT), a serotype 3 virus, as a cell-associated preparation or bivalent vaccine consisting of turkey herpesvirus and a serotype 2 virus (SB-1 or 301 B). Attenuated serotype 1 vaccines are also used. Care must be taken in handling cell-associated vaccines as they are highly susceptible to adverse environmental conditions.

3. Genetic differences associated with the major histocompatibility (B) complex can aid both in resistance to MD as well as the response to vaccination.

TREATMENT

There is no effective treatment for MD. Birds with tumors or multiple skin lesions are condemned at slaughter.

II. AVIAN LEUKOSIS/SARCOMA VIRUSES

DEFINITION

The avian leukosis (ALV)/sarcoma group are retrovirus-caused, neoplastic diseases of semimature or mature chickens. The most common, lymphoid leukosis (LL) is characterized by a gradual onset in a flock, persistent low mortality, and neoplasia of the bursa of Fabricius with metastasis to many other internal organs, especially the liver, spleen, and kidney. A relatively new strain of ALV, "J", probably resulting from the recombination of endogenous and exogenous viruses, primarily causes myeloid leukosis (myelocytomatosis).

OCCURRENCE

Lymphoid leukosis associated mortality is most common in chickens 16 weeks of age or older. The disease is worldwide in distribution and widespread in the United States. Virtually all flocks are considered to be exposed to the virus but infection rates within some flocks have decreased due to efforts at eradication by primary breeder companies. Overall, the incidence of LL is low (1 or 2%), although occasional heavy losses can occur. A higher incidence of bursal disease virus may be associated with a reduced incidence of LL. With ALV-J, meat-type chickens appear to be more susceptible than layers.

HISTORICAL INFORMATION

The first report of LL is attributed to Roloff in 1868. However, the disease was not well characterized until a basis for its separation from MD was established in 1962.

ETIOLOGY

Avian leukosis is caused by a family of retroviruses known as avian leukosis viruses (alpha retroviruses), which have been classified into 10 subgroups—A, B, C, D, E, F, G, H, I and J. In the United States, subgroup A viruses are most common and are most frequently associated with LL with ALV-J myelocytomatosis next in frequency. Subgroup B viruses are occasionally isolated, whereas subgroups C and D are rare. Subgroup E viruses are common and are considered "endogenous" because they are derived from proviral genes permanently integrated into the host cell DNA; they rarely are associated with neoplasms. Subgroup F, G, H and I viruses primarily cause leukosis in species other than chickens. The viruses produce a group-specific antigen that can be detected in albumen of eggs and body tissues or fluids. ALV-J viruses have extensive antigenic variation within the strain. The avian leukosis viruses can be cultured in chicken embryo fibroblasts but most produce no cytopathology and are detected by antigen tests. Simple tests for antigen detection are available and are used in eradication programs in breeders. Antibody tests are also available and are used to monitor the status of flocks from which the virus has been eradicated.

EPIDEMIOLOGY

Egg transmission is an important mechanism of spread of avian leukosis viruses. The frequency of infected eggs is usually low but chicks hatched from infected eggs are permanently viremic (immune tolerant), do not develop antibody, have an increased risk of death from LL, may lay fewer eggs, and will probably shed virus into their own eggs thus perpetuating the infection. Chickens also can become infected by contact exposure, particularly with ALV-J, which is efficient at horizontal transmission. In meat type chickens, ALV-J viremia negative/antibody positive birds can shed virus and post hatch infected birds become tolerant shedders. Some chickens, particularly those of greater susceptibility due to endogenous virus infection or absence of maternal antibody, may transmit virus to progeny as a result of contact infection soon after hatch.

CLINICAL SIGNS

Chickens with LL may present with nonspecific or no clinical signs of disease. Many birds with tumors are unthrifty or emaciated and have pale combs and wattles. Enlargement of the abdomen may result from massive enlargement of the liver. Some birds with tumors can be detected prior to death by palpation of an enlarged and lumpy bursa of Fabricius by insertion of a finger into the cloaca. Birds with skeletal myelocytomatosis may have observable masses on the shanks, head and thorax. Osteopetrosis of the long bones [Fig. 1; Leukosis; Cornell U] or “boot” shanks may occur. Flocks with high infection rates experience depressed egg production.

LESIONS

1. There are no unique external lesions. Lymphomas [Fig. 2; Leukosis; Cornell U] are seen in many organs in chickens 16 weeks of age or older, but are especially common in the liver, kidney, ovary, and bursa of Fabricius [Fig. 3; Leukosis; Cornell U]. The white-to-gray neoplastic lesions can be diffuse or are sometimes focal. If the bursa of Fabricius is incised, small nodular lesions can often be detected that would not otherwise be obvious. Myelocytomatosis [Fig. 4; Leukosis; UC Davis] is most common with ALV-J; however, other tumor types such as hemangiomas can also be seen.

2. Microscopically, the neoplastic cells in lymphoid tumors are uniformly lymphoblastic and the cells are pyroninophilic. Also, they are nearly all positive for surface immunoglobulin M (IgM). The tumors originate from bursal lymphocytes (B-cells) in which the proviral DNA of the virus integrates during the process of replication at a site in the host cell genome close to the *c-myc* gene, a normal host cell gene with homology to an oncogene present in avian retroviral strain MC29. Activation of this oncogene is believed to be the primary event in starting the neoplastic process.

DIAGNOSIS

1. Lymphoid Leukosis can usually be diagnosed after careful consideration of the age of the affected chickens, the course of the disease and the pattern of mortality in the flock, and the location of gross lesions

in a moderate number of typically affected chickens. Involvement of the bursa of Fabricius is nearly always present, although the lesions may not be detected without incision of the organ and examination of the epithelial surface. In contrast to MD, bursal tumors are intrafollicular, generally causing a more nodular enlargement. The characteristic tumor cell has B-cell and IgM surface markers. Molecular methods are available in research laboratories to detect in the DNA of tumor cells the proviral DNA of ALV located in close proximity to the *c-myc* gene.

2. Diagnosis is made more difficult because the lesions of LL often appear similar to those of MD, and can appear identical to those induced experimentally by reticuloendotheliosis virus. Because ALV is very widespread in chickens, virological and serological methods offer little help in confirming a diagnosis.

3. Diagnosis of ALV-J is achieved by gross and histopathologic examination of tumors and by virus isolation from cloacal or vaginal swabs or tumors. Although PCR tests have been developed, the virus mutates frequently requiring the production of new primers.

CONTROL

1. With LL, because egg transmission is so important and the disease is not very contagious, eradication is the preferred method of control. Most of the eradication efforts have been conducted by the primary breeding companies. Many breeders of egg-type chickens have reduced markedly the rate of congenital transmission in their primary breeders and grandparent stocks through a program of testing dams prior to egg production and removal of those considered likely to transmit virus to progeny. Some breeders have flocks from which the virus apparently has been eradicated. Commercial progeny from such breeders should have lower infection rates and thus should experience less tumor mortality and greater egg production.

2. Although LL is not a disease of commercial broilers, ALV-J is a problem and breeders have made significant progress in their eradication programs. However, due to the efficient horizontal transmission of ALV-J, control by eradication is more difficult.

3. Genetic resistance to infection with subgroup A viruses is common in meat-type stocks, but quite rare in egg-type stocks. When present, this resistance offers an alternative approach to control.

4. There is no vaccine that can protect against tumor mortality. Congenitally infected chicks are immunologically tolerant and cannot be immunized. Vaccines to immunize parent stock where ALV has been eradicated is being considered as a means to provide maternal immunity to progeny chicks.

TREATMENT

There is no effective treatment for LL.

III. RETICULOENDOTHELIOSIS
(RE)

DEFINITION

Reticuloendotheliosis (RE) is a term used for a variety of syndromes caused by retroviruses that may be either defective or nondefective for replication in cell culture. Only a runting syndrome and a chronic lymphoma, both caused by nondefective RE virus, are of economic importance.

OCCURRENCE

Nondefective RE virus is not ubiquitous, but infection is fairly widespread in chickens and turkeys, particularly in the southern states. The disease is uncommon. Runting disease has been associated with the use

of RE virus-contaminated vaccines in chickens. Chronic lymphomas occur naturally in turkeys, including wild turkeys, ducks, quail, pheasants, geese, peafowl, prairie chickens and chickens but are rare. Exportation of seropositive birds to some countries is not permitted.

HISTORICAL INFORMATION

A virus was isolated from a field case of turkey lymphomas in 1958 that, after rapid serial passage in chickens and turkeys caused high neoplastic mortality within 3 weeks. Although this isolate, strain T, has been considered a prototype, it is not typical of field strains. Other isolates from ducks and chickens were recognized in 1974 to comprise a family of RE viruses.

ETIOLOGY

RE virus is a retrovirus with an unusually wide host range. It can be grown in cells from chickens, ducks, turkeys, quail, and other species, even some mammalian cells. It infects a variety of avian species. Non avian species are resistant to infection. All isolates are of a single serotype, but minor subtype differences have been noted.

EPIDEMIOLOGY

The virus is transmitted horizontally. Mosquitoes have been incriminated as passive carriers. Fowl pox viruses have also been found to harbor infectious REV. Transmission through the egg has also been identified, but usually occurs at a very low rate.

CLINICAL SIGNS

The runting syndrome, usually induced by inoculation of chicks at 1 week of age or less with RE virus-contaminated biologics, produces severe stunting and a feather abnormality characterized by compression of barbules to the shaft in its proximal portion. Signs associated with chronic lymphomas are few but birds may become depressed prior to death.

LESIONS

1. The runting syndrome is characterized by severe atrophy of the thymus and bursa of Fabricius. The birds are immunodepressed and may show lesions of concurrent infections. Generally no tumors are noted but some birds may have enlarged nerves, proventriculitis, enteritis, and anemia.

2. The chronic lymphoma syndrome as produced experimentally in chickens with nondefective RE virus is identical in all respects with lymphoid leukosis. A different lymphoma has been induced experimentally in certain chicken strains that closely resembles MD because of nerve lesions, tumors in the liver [Fig. 1; Reticuloendotheliosis] thymus, and heart, and its occurrence as early as 6 weeks of age. Chronic lymphomas in species other than chickens are characterized by tumors of the liver and spleen, but bursal tumors are not particularly common.

DIAGNOSIS

1. A diagnosis of RE is probably best made on the basis of typical lesions and demonstration of infection with the causative agent by virus or antibody tests. Currently, the PCR test, an immunoperoxidase plaque assay, and an enzyme immunoassay are available.

2. In chickens, the disease must be distinguished from both LL and MD. Thus far, however, naturally occurring chronic lymphomas in chickens have not been documented. The runting syndrome must be distinguished from other immunodepressive conditions, especially infectious bursal disease and chicken infectious anemia.

3. In turkeys, the disease must be distinguished from lymphoproliferative disease in countries of occurrence. This can usually be accomplished by noting the age of occurrence, the absence of greatly enlarged spleens, and the uniform lymphoblastic morphology of the tumor cells on histology and using PCR assays.

CONTROL

No methods for control or treatment have been reported. Strict sanitation and insect control may help prevent infection from environmental sources. Eradication programs patterned after those developed for LL may be useful in breaking the egg transmission cycle.

DIFFERENTIAL DIAGNOSIS OF AVIAN TUMORS

The differential diagnosis of tumors in chickens and turkeys is difficult and requires an adequate history and a careful postmortem examination of a representative sample of birds with typical lesions. In some cases, additional tests such as histology, immunofluorescent tests for surface antigens, and molecular hybridization will be helpful. The characteristics in the following table may be helpful in arriving at the correct diagnosis.

	Chickens			Turkeys	
Characteristic	MD[A]	LL	RE[B]	RE	LPD
Gross lesions					
Liver	+++	+++	+++	+++	+++
Spleen	+++	+++	+++	+++	+++
Nerves	+++	-	++	+++	+
Skin	+++	+	+	+	+
Gonads	+++	+++	+++	+++	+
Heart	+++	+	+++	++	+
Bursa	+	+++	+++/--	+++	+
Intestine	+	++	+++	+++	+++
Lungs	+++	++	++	++	+++
Kidneys	+++	+++	+++	+++	+++
Histology					
Pleomorphic cells	+	-	-/+	-	+
Uniform blast cells	-	+	+/-	+	-
Antigens					
MATSA	+	-	-	?	?
IgM	+	+++	+/-	?	?
B-cell	+	+++	+/-	?	?
T-cell	+++	+	-/+	?	?
Age of occurrence (months)					
Peak time	2-7	4-10	2-6	4-6	2-4
Limits	> 1	> 3	> 1	> 4	> 2

[A]Abbreviations: MD = Marek's disease, LL = lymphoid leukosis, RE = reticuloendotheliosis, LPD = lymphoproliferative disease.

[B]Two experimental syndromes are recognized: a bursal lymphoma with characteristics similar to LL, and a nonbursal lymphoma with characteristics similar to MD.

CHICKEN INFECTIOUS ANEMIA

(CIA; Chicken Anemia Virus; Blue Wing Disease)

DEFINITION

Chicken infectious anemia (CIA) is a disease of chickens caused by a circovirus and characterized by aplastic anemia, generalized lymphoid atrophy, subcutaneous and intramuscular hemorrhage, and immunodepression.

OCCURRENCE

CIA is probably ubiquitous in all major chicken-producing countries in the world.

HISTORICAL INFORMATION

CIA virus was first isolated by Yuasa in Japan in 1979. It has also been called chicken anemia agent, chicken anemia virus, and parvovirus-like virus. The clinical signs and lesions previously described as blue wing disease, anemia-dermatitis syndrome, and hemorrhagic anemia may have been caused by CIA virus.

ETIOLOGY

1. CIA virus is the only member of the genus Gyrovirus of the *Circoviridae.*

2. CIA virus is difficult to isolate due to the restricted cell lines suitable for propagation. Most chicken embryo cell lines and chick embryos are resistant to infection or produce low virus yields.

3. Bioassay in susceptible 1-day-old chicks is the most specific method for primary isolation.

4. CIA virus is extremely resistant.

5. No antigenic differences have been noted amongst strains.

EPIDEMIOLOGY

1. CIA virus is thought to be ubiquitous in poultry-producing areas of the world.

2. Chickens are the only known hosts.

3. All ages are susceptible to infection but clinical disease is seen only during the first 2 to 4 weeks. However, age resistance is delayed by simultaneous infection with infectious bursal disease virus.

4. The most important method of transmission is vertical from infected hens. Antibody-negative chicks are most susceptible to clinical disease. CIA virus also easily spreads via feces among birds in a population.

CLINICAL SIGNS

1. The only specific sign of CIA is anemia characterized by hematocrit values ranging from 6 to 27% (normal hematocrit value is 35%)

2. Nonspecific clinical signs include depression, pale tissues, depressed weight gain, and secondary bacterial, mycotic, and viral infections.

3. Morbidity and mortality rates vary depending upon altered immune status due to other infections such as infectious bursal disease, Marek's disease, or reticuloendotheliosis. Mortality is usually 5-10% but can be 60%.

4. Early infections with CIA virus can interfere with vaccination against Marek's disease or infectious bursal disease.

LESIONS

1. Thymic atrophy is the most consistent lesion.

2. Fatty yellowish bone marrow, particularly in the femur, is characteristic [Fig. 1; Chicken infectious anemia; Cornell U].

3. Bursal atrophy can also be seen.

4. Hemorrhages in the mucosa of the proventriculus, subcutis, and muscles and a swollen mottled liver can also be observed.

5. Lesions of secondary bacterial infections, such as gangrenous dermatitis and blue wing disease can be seen in commercial flocks.

DIAGNOSIS

1. A presumptive diagnosis is based upon clinical signs and gross lesions.

2. Isolation and identification of the virus from most tissues, buffy coat cells, and cloacal contents.

3. Serologic assays to detect antibodies such as the ELISA, virus neutralization test, and indirect immunofluorescence.
4. PCR is the test of choice for identification of CIA virus in cell cultures and chicken tissues.

CONTROL

1. Best prevention is by immunization of breeder flocks prior to the onset of egg production (between 13-15 weeks of age but no closer to egg production than 4 weeks).

2. Where vaccines are not available, nonimmune breeder flocks are exposed to the virus during the growing period by rearing them on litter transferred from previously infected breeder houses. This is an effective but obviously risky practice.

TREATMENT

1. There is no satisfactory treatment for the viral infection.

2. Use supplemental vitamins via drinking water, including B vitamins, and A, E, and K.

3. Treat secondary gangrenous dermatitis with penicillin, bacitracin, lincomycin, or other antibiotics effective against *Clostridium* spp. or *Staphylococcus* spp.

CORONAVIRAL ENTERITIS OF TURKEYS

(Transmissible Enteritis of Turkeys; Blue Comb)

DEFINITION

Coronaviral enteritis is an acute, highly infectious disease of turkeys, especially poults, characterized by anorexia, diarrhea, dehydration, and variable mortality.

OCCURRENCE

Coronaviral enteritis occurs in turkeys of all ages but is seen more frequently in young turkeys. Chickens are not infected. The disease occurs throughout the year. It is recognized in the United States, Canada, and Australia; however, the incidence has substantially decreased since the 1970s.

HISTORICAL INFORMATION

1. Coronaviral enteritis was first reported in 1951 in Washington and shortly thereafter in Minnesota. The disease has not been reported frequently from other states.

2. For many years this disease was reported as the most costly disease of the turkey industry in Minnesota. There it accounted for 23% of total turkey mortality in 1966. The last reported outbreak in Minnesota occurred in 1977.

3. Transmissible enteritis is distinct from hemorrhagic enteritis, another disease of turkeys.

ETIOLOGY

1. A coronavirus is now accepted as the cause of transmissible enteritis. There is no relationship between this virus and other avian or mammalian coronaviruses. In the past the disease has been attributed to many different agents, including vibrio and a variety of viruses.

2. Under experimental conditions the virus is readily destroyed in batteries and cages. Destruction of the virus probably is more difficult under natural conditions because it survives well in frozen feces. Under field conditions, cleaning and disinfection of buildings and equipment, combined with at least 3-4 weeks of depopulation, is advisable. Virus survival in ranges can be extensive despite depopulation.

3. A number of other enteric viruses including rotavirus, reovirus, astrovirus, enterovirus, and calicivirus have been identified from turkey feces. Their role in enteritis is uncertain.

EPIZOOTIOLOGY

The virus is shed in the feces of recovered carrier birds for several months. Further, the virus persists in frozen feces for several months. The virus can be spread by contact of susceptible birds with infected birds or their feces. Once introduced into a flock, the disease spreads rapidly among susceptible birds. There is no evidence that the virus is transmitted through the egg.

CLINICAL SIGNS

1. In young poults the signs appear suddenly after an incubation period of 1-5 days. Signs include anorexia, depression, frothy diarrhea, subnormal temperatures, darkening of the head and skin, and loss of weight. Birds tend to huddle around heat sources. Spread is rapid and morbidity is close to 100%.
2. The signs seen in young poults may also be observed in laying turkeys but usually are less marked. Moreover, there is a sudden decrease in egg production and some eggshells are chalky.

3. Good husbandry and supplemental heat tend to suppress mortality. Mortality varies with the age of the birds affected and can range from 5 to 50% in natural infections.

4. The course of the disease in a flock is around 2 weeks. Recovery may be prolonged, particularly in males, and the flock may become uneven in size.

LESIONS

1. There is marked dehydration and emaciation, especially in older birds.

2. Enteritis [Fig.1; Coronaviral enteritis; Cornell U] is a consistent lesion and there may be petechial hemorrhages in the mucosa. Watery, gaseous ingesta with excess mucus and occasional casts are often observed. Ceca are distended with odiferous, watery contents.

3. The pancreas has a whitish, chalky appearance. This change is not entirely uniform but is usually present to some degree.

4. Excessive urates are frequently found in the kidneys and ureters. The spleen often is small.

5. Microscopically, the villus-to-crypt ratio is decreased, particularly in the jejunum. There are large numbers of round cells in the adrenals. In the pancreas there is focal hydropic degeneration and some cells contain large eosinophilic intracytoplasmic inclusions, which are aggregates of intracytoplasmic granules.

DIAGNOSIS

1. The history, signs, and lesions are suggestive of the diagnosis but are duplicated by certain other diseases. Filtered intestinal material can be given to a few susceptible poults and will reproduce the disease in a few days.

2. A direct fluorescent antibody (FA) technique can be used to identify the virus in the cytoplasm of intestinal mucosal cells and in the bursa. An indirect FA technique can be used to detect antibodies in the serum 14 days or more after exposure to the virus.

3. The virus can be isolated in embryonating turkey eggs and can be identified by electron microscopy (EM). EM can also be used to detect virus particles in intestinal contents.

4. In differential diagnosis one should consider the following diseases:

Young Poults (Less than 7 Weeks)	Growing and Mature Turkeys
Salmonellosis	Fowl cholera
Hexamitiasis	Erysipelas
Starve outs (very young birds only)	Blackhead
Coccidiosis	Trichomoniasis
Water deprivation	Hemorrhagic enteritis

CONTROL

1. Turkeys should be reared under the all-in, all-out system. Use quarantine and a high standard of sanitation to prevent introduction of the virus.

2. If the disease has been present in prior broods, thoroughly clean and disinfect the premises after complete depopulation. Leave the premises empty for at least a month, perhaps longer during the winter season.

3. No vaccine is available for immunization.

TREATMENT

1. Treatment is largely empirical. Broad-spectrum antibiotics often appear to suppress mortality by preventing secondary infections. Therapeutic agents that have been reported to be of value include penicillin, streptomycin, tetracyclines, bacitracin, and neomycin.

2. Calf milk replacer (25 lb/100 gal) or potassium chloride (450 g/100 gal) may be added to the drinking water for a few days. In mature birds molasses is sometimes added to the drinking water (1 pint/5 gal) for a 1-day flush before administration of a course of antibiotics.

DUCK VIRUS ENTERITIS

(DVE; Duck Plague)

DEFINITION

Duck virus enteritis (DVE) is an acute viral disease of ducks, geese, and swans characterized by weakness, thirst, diarrhea, short course, high mortality, and by lesions of the vascular, digestive, and lymphoid systems.

OCCURRENCE

1. Wild and domestic ducks, geese, and swans (order *Anseriformes*) are affected. All age groups and many varieties are susceptible; however, mostly adults are affected. The blue-winged teal is the most susceptible and the pintail duck is the least susceptible.

2. In the United States the disease has occurred in New York, Pennsylvania, Maryland, California, and South Dakota. The disease has been reported in the Netherlands, France, China, Belgium, and India. Because wild waterfowl are migratory, it seems likely that the disease may occur in other countries that have migratory waterfowl.

HISTORICAL INFORMATION

1. The disease was first observed in the Netherlands in 1923. Initially it was mistaken for avian influenza but in 1942 it was clearly differentiated from avian influenza and was termed duck plague. Subsequently the disease was identified in many other countries.

2. In 1967 DVE appeared in white Pekin ducks raised commercially on Long Island, New York. It also was identified in wild waterfowl. An effort to eradicate the disease in the domesticated white Pekin duck appeared to be successful.

3. Multiple outbreaks of DVE have been recognized in California and it is classified as a reportable disease in this state. In the spring of 1973 the disease appeared in congregated wild waterfowl in South Dakota and resulted in the death of approximately 48,000 waterfowl, mostly ducks.

4. DVE is now considered to be enzootic in North America. Prior to the 1973 outbreak, DVE was considered an exotic disease by the USDA. It is being watched with interest because its ability to kill congregated, susceptible waterfowl is recognized.

ETIOLOGY

1. The etiologic agent is a herpesvirus. Although strains vary in pathogenicity, all appear to be identical immunologically.

2. The virus is nonhemagglutinating. This differs from the viruses of Newcastle and avian influenza, which do hemagglutinate and which must be differentiated in diagnostic work.

3. The virus grows best on the chorioallantoic membrane of 9-14-day-old embryonating duck eggs or on duck embryo fibroblasts. Initially it does not grow in chicken eggs although it can be adapted to them. The virus also can be isolated in ducklings, with Muscovy ducklings being the most sensitive.

4. The virus produces intranuclear inclusion bodies in a variety of cells of infected waterfowl.

DUCK VIRUS ENTERITIS

EPIZOOTIOLOGY

1. The virus can be transmitted when susceptible birds contact infected birds or an environment (particularly water) contaminated by them. Natural infection is limited to ducks, geese, and swans.

2. A carrier state for as long as 1 year has been demonstrated in wild ducks. Perhaps carrier birds under stress shed virus intermittently, thus exposing susceptible birds.

3. Because viremia occurs in affected birds, arthropods feeding on those birds may transmit the disease. However, this method of transmission is unproven.

4. Vertical transmission has been reported experimentally.

CLINICAL SIGNS

1. In young commercial ducklings, signs appear 3-7 days after exposure. Ducklings have diarrhea, a blood-stained vent, dehydration, and a cyanotic bill. Death usually occurs in 1-5 days.

2. In domestic breeder ducks there is a sudden, high, persistent mortality and a marked drop in egg production (25-40%). Sick birds show inappetence, weakness, ataxia, photophobia, adhered eyelids, nasal discharge, extreme thirst, prolapsed penis, and watery diarrhea. They soon become exhausted and unable to stand. They then maintain a position with drooping outstretched wings and with the head down. Tremors may be apparent. Morbidity and mortality are usually high but vary from 5 to 100%. Most birds that develop clinical signs die. Wild waterfowl are said to have similar signs. They often conceal themselves and die in vegetation near the water.

LESIONS

1. Hemorrhages are present at many sites and there may be free blood in body cavities, gizzard, or intestine. Hemorrhages often occur on the liver, in the mucosa of the gastrointestinal tract (including the esophageal-proventricular junction), throughout the heart, and in the pericardium and ovary. There may be edema in the cervical region.

2. There is severe enteritis. There may be elevated, crusty plaques in the esophagus, ceca, rectum, cloaca, or bursa of Fabricius. In young ducklings the esophageal mucosa may slough.

3. There is hemorrhage and/or necrosis in the annular bands or discs of lymphoid tissue along the intestine. The spleen is usually of normal or reduced size.

4. Initially the liver may be discolored and contain petechial hemorrhages. Later it may be bile-stained and contain scattered small, white foci as well as many hemorrhages.

5. Microscopically there may be intranuclear inclusion bodies in degenerating hepatocytes, epithelial cells of the digestive tract, and in reticuloendothelial cells.

DIAGNOSIS

1. Typical signs and lesions, along with epizootic losses, are highly suggestive of duck plague. The diagnosis can be strengthened if intranuclear inclusion bodies can be demonstrated or if the virus can be demonstrated in the tissues through fluorescent antibody tests.

2. The virus should be isolated and identified for confirmation. The virus will grow initially in embryonating duck eggs but not in chick embryos. Using known antisera to DVE, the virus can be identified by a neutralization test.

3. Retrospectively, it is possible to identify an outbreak of DVE if acute and convalescent sera are used to demonstrate an increasing antibody titer to duck plague virus.
4. DVE must be differentiated from duck viral hepatitis, pasteurellosis, Newcastle disease, avian influenza, coccidiosis, and other causes of enteritis.

CONTROL

1. Owners should prevent cohabitation or contact of their waterfowl with wild waterfowl. All appropriate quarantine and sanitary practices should be followed to prevent disease.

2. All suspected outbreaks should be reported immediately to state authorities. They, with federal authorities, will decide how an outbreak is to be handled. In commercially raised waterfowl, outbreaks were once controlled by combining slaughter with indemnification and by the application of quarantine measures. Presently, slaughter with indemnification has been discontinued and vaccination has been authorized on certain premises.

3. A vaccine is available for prevention but approval by animal health authorities is required before it can be used. It has not been authorized for general use.

4. A monitoring system for detection of DVE has been established in the United States. Suspected outbreaks should be processed through official state or federal diagnostic laboratories.

TREATMENT

There is no effective treatment.

DUCK VIRUS HEPATITIS

(DVH)

DEFINITION

Duck virus hepatitis (DVH) is a peracute, rapidly spreading viral infection of young ducklings characterized by a short course, high mortality, and by punctate or ecchymotic hemorrhages in the liver. Three different viruses are known to cause DVH.

OCCURRENCE

DVH type 1 occurs primarily in commercially raised Pekin ducklings and is seen almost exclusively in ducklings less than 5 weeks of age. Natural outbreaks have not been reported in other species. The disease is probably present in all major duck-raising areas of the world. DVH type 2 is seen exclusively in the United Kingdom and affects ducklings up to 6 weeks of age. The United States is the only country in which DVH type 3 has been observed. Ducklings up to 5 weeks of age are susceptible to DVH type 3.

HISTORICAL INFORMATION

A disease that probably was DVH type 1 first appeared in New York in 1945. A similar disease, called duck viral hepatitis, appeared on Long Island in 1949 and killed an estimated 750,000 ducklings. Subsequently, the disease was reported in many other states and from many countries throughout the world. In the United States the disease remains one of the major diseases of the duck-raising industry. DVH type 2 was first reported in DVH type 1-vaccinated ducklings in Great Britain in 1965. In 1969, DVH type 3 was reported to occur in DVH type 1-immune ducklings on Long Island.

ETIOLOGY

1. The etiologic agent of DVH type 1 is an enterovirus in the family Picornaviridae. It is chloroform resistant and does not hemagglutinate; features that help separate it from most other viral diseases of ducks. The virus is rather stable and difficult to eliminate from contaminated premises. Serologic variants of DVH type 1 have been reported.

2. DVH type 2 has been identified as an astrovirus. As with DVH type 1, the virus is fairly resistant. DVH type 3 is caused by a picornavirus unrelated to DVH type 1.

3. DVH type 1 can be isolated from typically affected livers in embryonating chicken or duck eggs, 1-day-old ducklings, or duck embryo kidney or liver cell cultures. DVH type 2 is difficult to isolate, whereas DVH type 3 can be isolated on chorioallantoic membranes of 9-10-day-old duck embryos.

4. DVH viruses stimulate a high degree of immunity in ducklings that survive infection and in inoculated adult ducks. A potent antiserum can be made from the blood of such ducks. The blood can be collected at slaughter and the sera harvested. Antibodies for prophylactic use may also be obtained from the yolk of eggs produced by immune breeders, or from the eggs of chickens hyperimmunized with the virus.

EPIZOOTIOLOGY

1. DVH type 1 is a highly contagious disease. The virus is excreted by recovered ducklings for up to 8 weeks after onset of infection. Susceptible ducklings can be infected by contact with infected ducklings or their contaminated pens. The virus can survive for 10 weeks in contaminated brooders and for 37 days in feces. DVH type 2 is transmitted via both the oral and cloacal routes. Survivors excrete virus for up to 1 week postinfection. DVH type 3 is similar to but less severe than DVH type 2.

2. Wild birds have been suspected of acting as mechanical carriers of virus over short distances. The viruses do not appear to be transmitted through the egg and there are no known vectors of the disease.

CLINICAL SIGNS

DVH type 1

1. The incubation period is very short, often around 24 hours in experimental birds, and morbidity is close to 100%. Onset and spread within a flock are very rapid and most mortality occurs within 1 week of onset.

2. Affected ducklings at first lag behind the flock. Within a short time they squat with their eyes partially closed, fall on their side, kick spasmodically, and soon die. They often die in the opisthotonus position [Fig. 1; Duck viral hepatitis; Cornell U]. Death often occurs within 1 hour of the appearance of signs.

3. Mortality is age related and occurs as follows: ducklings less than 1 week old—up to 95% mortality; ducklings 1-3 weeks old—up to 50% mortality; ducklings over 4 weeks and older ducks—negligible mortality.

4. In older or partially immune ducklings, signs and losses may be so limited that the disease may go unrecognized.

DVH type 2

Affected ducklings die within 1-2 hours of being sick. Clinical signs usually appear within 1-4 days postinfection. Signs include convulsions and opisthotonus. Mortality ranges from 10 to 50% and nearly all birds with clinical signs die. DVH type 3 is similar to DVH type 1 but mortality is rarely over 30% and morbidity is higher.

LESIONS

1. The lesions observed with all three viruses are similar.

2. The cadaver may be in opisthotonus, the position in which many of the ducklings die.

3. The liver is swollen and contains punctate or diffuse hemorrhages [Fig. 2; Duck viral hepatitis; Cornell U]. The kidneys may be swollen and the spleen enlarged. Microscopically, there may be areas of hepatic necrosis, bile duct proliferation, and some degree of inflammatory response.

DIAGNOSIS

1. The sudden onset, rapid spread, short course, and focal, hemorrhagic hepatitis in young ducklings suggest a diagnosis of DVH.

2. DVH type 1 can usually be isolated in embryonating chick or duck embryos or 1-day-old susceptible ducklings. Once the virus is isolated, it can be identified by serum neutralization using known hepatitis antiserum. Identification is also possible by inoculation of the virus into both susceptible and immune ducklings. DVH type 2 can be identified through electron microscopy on liver or blood. DVH type 3 cannot be isolated in chicken embryos and is difficult to reproduce in ducklings. The chorioallantoic membranes of duck embryos are the preferred route. A direct fluorescent test on duckling liver has been reported.

3. The disease must be differentiated from duck viral enteritis, Newcastle disease, and avian influenza. In contrast to the viruses of DVH, the viruses causing those diseases are susceptible to chloroform; also the viruses of influenza and Newcastle disease hemagglutinate erythrocytes.

CONTROL

DVH type 1

1. In the initial stages of outbreak, all susceptible ducklings should be inoculated intramuscularly with duck hepatitis viral antiserum. One inoculation should be adequate if the antiserum is potent.

2. Unexposed ducklings can be actively immunized using a chicken embryo-adapted apathogenic vaccine. However, young ducklings with parental immunity may not respond to vaccination.

3. Many duck breeders prefer to vaccinate their breeding stock at 3-4-month intervals to maintain a high antibody titer. Those birds then will transmit antibody through their eggs to the progeny. The progeny will usually be protected through the critical early weeks. Breeder birds should be vaccinated at least 2 weeks before their eggs are to be saved for hatching. Both live and inactivated vaccines are available.

DVH type 2 and 3

Vaccines for breeders are in the experimental stage only. Strict biosecurity procedures must be employed. Information concerning vaccines or antiserum can be obtained by contacting the Duck Research Laboratory, Eastport, Long Island, NY 11941.

TREATMENT

Treatment is of no value.

EQUINE ENCEPHALITIS VIRAL INFECTION

(Alphaviral Infection; Arboviral Infection)

DEFINITION

Equine encephalitis viral infection is an acute disease of pheasants, chukar partridges, turkeys, ducks, pigeons, or wild birds caused by any one of a number of alphaviruses; it often is characterized by signs related to lesions in the central nervous system. In ratites the disease is characterized by bloody diarrhea.

OCCURRENCE

In the United States infection with eastern equine encephalitis (EEE) occurs naturally in pheasants, chukar partridges, turkeys, ducks, pigeons, and ratites. Western equine encephalitis (WEE) occasionally occurs in many of those same species. Chickens can be infected naturally but seldom show clinical signs. Many wild and migratory birds are susceptible but may or may not show signs when infected.

Most outbreaks in captive game birds and poultry occur in birds less than 6 months old. Outbreaks usually occur during the mosquito season. Most outbreaks reported have been in the United States, usually in the Atlantic coastal states or in the upper Midwest.

HISTORICAL INFORMATION

Just a few years after EEE and WEE were recognized in horses, those infections were recognized in birds. Most outbreaks were in captive game birds, especially pheasants. Wild birds and captive game birds were soon recognized as important links in the epizootiology of the disease in horses. Outbreaks still occur in wild and captive game birds but are rare in domestic poultry. The disease has recently become important to the ratite industry in some regions of the United States.

ETIOLOGY

EEE and WEE are now classified as alphaviruses in the family Togaviridae. They, along with many other viruses, were once classified as arboviruses. As originally defined, arboviruses are viruses that naturally infect arthropods that consume infected vertebrate blood. In many instances those viruses multiply in the arthropod and are transmitted by bite to susceptible vertebrates. The term arbovirus is still useful in that it implies this type of epizootiology.

The viruses of EEE and WEE readily infect humans, and many laboratory workers have had severe or fatal infection. This should be kept in mind in dealing with outbreaks in birds and poultry and in virus isolation and identification efforts.

EPIZOOTIOLOGY

Birds acutely ill with alphaviral infection are viremic, at least initially. They may or may not show clinical signs. Certain mosquitoes, primarily *Culiseta melanura*, feed on the viremic birds and become infected with virus, oftentimes for life. The virus may increase in titer within the mosquitoes, although this is not necessary for transmission. Infected mosquitoes then transmit the infection to other susceptible birds, horses, or people while feeding on them. Birds are the major source of virus for mosquitoes because they carry a higher titer of virus than most mammals.

Cannibalism of viremic, sick, or dead birds by other susceptible birds is an important method of transmission of virus within infected flocks. Also, certain biting insects (gnats, deerflies, horseflies, etc.) may transmit the virus mechanically.

EQUINE ENCEPHALITIS VIRAL INFECTION

CLINICAL SIGNS

1. Many infected wild birds and poultry flocks have transient alphaviral infections and show no clinical signs. Antibodies can be demonstrated in their sera.

2. Infected poultry flocks (especially captive game birds) show marked signs of disease of the central nervous system. Signs often include ataxia, paresis, paralysis, inability to stand or hold up the neck, circling, and tremors. Morbidity and mortality often are very high. Few birds that show signs recover.

LESIONS

There are no significant gross lesions. Microscopic lesions occur in the brain of most clinically ill birds but are not pathognomonic.

DIAGNOSIS

In flocks with clinically ill birds, the signs are suggestive of a disease of the central nervous system. Definitive diagnosis is made by isolating and identifying the virus. Isolation is usually via chicken embryos, laboratory mice, or tissue culture. Specific identification is usually made via virus neutralization or complement fixation tests. This is a reportable disease in many states, so even suspected cases should be reported to the state veterinarian and public health authorities.

CONTROL

1. Protect birds against mosquitoes by raising them where mosquitoes do not thrive, or by the use of screens, sprays, or other mosquito control methods.

2. Trim beaks of captive birds and practice all other known methods of preventing cannibalism, including:

    A. Avoid overcrowding and keep the houses or pens at a comfortable temperature.

    B. Keep the houses rather dark and use only red light bulbs.

    C. Use "spectacles" or other mechanical devices that prevent the birds from seeing ahead but that permit feeding.

    D. Prevent or treat louse infestation, which often leads to feather picking and cannibalism.

3. Pheasants in enzootic areas may be immunized before the mosquito season begins. Use the appropriate equine encephalitis vaccine in the dilution recommended by the manufacturer. Although vaccination has been used in pheasants, its efficacy is in question.

TREATMENT

Treatment of sick birds is of no value.

FOWL POX

(Pox; Avian Pox)

DEFINITION

Fowl pox is a slow-spreading viral disease of chickens, turkeys, and many other birds characterized by cutaneous lesions on unfeathered skin of the head, neck, legs, and feet and/or by diphtheritic lesions in the upper digestive and respiratory tracts.

OCCURRENCE

Among poultry, pox occurs frequently in chickens and turkeys. Among other birds, pigeons, canaries, and psittacines are frequently infected and the disease is seen occasionally in many wild birds. Perhaps all birds are susceptible. The disease occurs in all age groups except the recently hatched and is worldwide in distribution.

HISTORICAL INFORMATION

Fowl pox is an ancient disease and in the distant past was mistakenly thought to be related to small pox and chicken pox of man. The characteristic pox inclusion bodies (Bollinger bodies) and the smaller elementary bodies within them (Borrel bodies) were studied by Drs. Bollinger and Borrel, respectively, in 1873 and 1904. In the United States, pox has been a common and frequently reported disease of poultry. In recent years there has been increased interest in pox in wildlife and caged birds, which are being submitted to diagnostic laboratories in increasing frequencies.

ETIOLOGY

1. Fowl pox is caused by a large DNA Avipoxvirus of the family Poxviridae. Many strains of virus are recognized and naturally infect the species given in their name. Some common examples are:

fowl poxvirus (type species)	quail poxvirus
turkey poxvirus	mynah poxvirus
pigeon poxvirus	psittacine poxvirus
canary poxvirus	

2. Poxviruses appear to be closely related, however, a strong host specificity is found with most poxvirus strains. In some instances, exposure to one of the viruses in the group engenders immunity to that virus and one or more of the other viruses in the group. Poxvirus isolates from Hawaiian forest birds (alala and apapane poxvirus strains) are more related to each other than to fowl poxvirus indicating genetically distinct poxviruses in this region. Perhaps all strains are host-modified variants of what was once a single virus.

3. The various strains of avian poxvirus are morphologically identical. Strain classification has traditionally depended upon the cross-protection test in birds but these are not practical for routine diagnosis. Restriction endonuclease analysis of DNA has been successful in differentiating strains.

4. Recovery from poxvirus infection usually results in a strong, enduring immunity to later exposure to the same virus. Also, in turkeys and chickens vaccination is usually quite effective in preventing pox. Recently, however, several outbreaks of fowl pox have occurred in vaccinated chickens.

5. Virus is present in lesions and in desquamated scabs. Poxvirus is quite resistant to environmental factors and persists in the environment for many months.

6. Most poxviruses stimulate the formation of inclusion bodies in infected epithelium. Intracytoplasmic inclusions (Bollinger bodies) contain elementary bodies (Borrel bodies). Bollinger bodies are quite large and readily identified microscopically.

EPIDEMIOLOGY

1. The virus-containing crusts (scabs) formed on the skin are desquamated into the litter. Virus persists in the environment and may later infect susceptible birds by entering the skin through minor abrasions. Mechanical transmission via cannibalism is thought to play a significant role in some outbreaks. Respiratory tract infection can result from inhalation of aerosolized virus-containing feathers and scabs.

2. Certain mosquitoes, and possibly other blood-sucking arthropods, can transmit virus from infected to susceptible birds. Mosquitoes remain infective for several weeks. Mosquito-transmitted outbreaks may result in rapid spread.

3. Poxvirus infection can result from mechanical transmission from toms to turkey hens via artificial insemination.

CLINICAL SIGNS

1. In poultry, onset often is gradual and the disease may go undetected until cutaneous lesions are numerous and obvious in the flock. The disease spreads slowly and severe outbreaks may last many weeks. Turkey pox infection is generally more chronic than fowl pox infection. Canaries can have systemic infection with high mortality. Signs vary somewhat with the two overlapping forms of pox:

   A. Cutaneous form

      This form predominates in most outbreaks. Birds often show few signs other than a mild to moderate reduction in rate of gain, a temporary loss in egg production, or a lack of flock vigor. Mortality is low if the disease is uncomplicated.

   B. Diphtheritic form

      Lesions in the upper respiratory or digestive tract may result in dyspnea or inappetence, respectively. Lesions in the nasal cavity or conjunctiva lead to nasal or ocular discharge. Mortality is low to moderate and is often due to suffocation or starvation and dehydration.

LESIONS

1. Cutaneous lesions vary in appearance according to whether the papule, vesicle, pustule, or crust (scab) stage is observed. In most outbreaks the terminal reddish brown to black scab stage [Fig. 1; Fowl Pox; UC Davis] is present on at least some of the birds presented for diagnosis. Papules, the initial lesions, are light-colored nodules in the skin. Vesicles and pustules are raised, usually yellow. Occasionally, small papilloma-like lesions occur. Lesions usually occur on the unfeathered skin of the head [Fig. 2; Fowl Pox; UC Davis] and neck but may occur around the vent or on the feet or legs. Cage birds and wild birds often have lesions on the feet or legs and these may appear as horny growths.

2. Diphtheritic lesions are raised, buff to yellow plaques on mucous membranes. They usually predominate in the mouth [Fig. 3; Fowl Pox; NCSU] but may be present in the sinuses, nasal cavity, conjunctiva, pharynx, larynx, trachea, or esophagus. Diphtheritic lesions often accompany cutaneous lesions but may occur alone in some birds.

3. Turkey pox [Fig. 4; Fowl Pox; UC Davis] has been observed in turkeys previously vaccinated with fowl pox vaccine. Occasional birds develop lesions on the conjunctiva, mouth, and upper digestive tract. Economic loss is often due to poor feed conversion.

4. Microscopically, epithelial hyperplasia with eosinophilic cytoplasmic inclusion bodies [Fig. 5; Fowl Pox; UC Davis] and surrounding inflammation are observed.

DIAGNOSIS

1. Typical skin lesions are very suggestive of the disease. The diagnosis can be confirmed by demonstrating intracytoplasmic inclusion bodies in stained sections or in scrapings of the lesions.

2. Typical skin lesions can be reproduced in a susceptible bird of the same species. Ground lesion material should be inoculated into scarified skin or empty feather follicles and should produce a typical pox "take" at the application site in about 5-7 days.

3. Virus-containing lesion material will produce pocks on the dropped chorioallantoic membrane of embryonated chicken eggs. The lesions contain typical intracytoplasmic inclusion bodies.

4. Some poxvirus strains, particularly turkey pox, may not have demonstrable inclusion bodies in tissue sections. Electron microscopy may be helpful in these cases.

CONTROL

1. Pox can be prevented in chickens, turkeys, pigeons, canaries, and quail by vaccination. Vaccination is usually done when the birds are 4 weeks of age but can be done at any age if necessary. Pullets should be vaccinated 1-2 months before production begins.

2. Chickens and pigeons usually are vaccinated by the wing web-stick method. An applicator with two slotted needles is dipped in vaccine and thrust through the wing web. Turkeys may be vaccinated by the wing web route but lesions may be transferred to the head from the vaccination site. Vaccination by a drumstick-stab method when 2-3 months old is the recommended route. Turkeys retained as breeders should be revaccinated.

3. Pigeon pox vaccine is now widely used in chickens either alone or in combination with fowl pox vaccine. Chickens purchased as replacements for layers should be revaccinated if the initial vaccination occurs prior to 10 weeks of age. Pigeon pox vaccine can cause severe reactions in pigeons if not applied properly.

4. Turkeys are usually vaccinated with fowl pox vaccine. Turkey pox, quail pox, and canary pox vaccines are commercially available when circumstances indicate that these strains are the causative agents. Fowl and pigeon pox vaccines are not cross-protective with these strains. Fowl pox vaccine should not be used to vaccinate pigeons.

5. Vaccination produces a small lesion ("take") at the site of vaccination. A generous sample of the birds should be examined for vaccination lesions about 5-7 days after vaccination. Takes caused by turkey pox vaccine generally appear later (8-10 days after vaccination) than those caused by fowl pox. A large percent of those birds should have takes or revaccination is necessary.

6. Broilers are not vaccinated unless there is pox in the area. Broilers may be vaccinated with a mild tissue culture fowl pox vaccine administered subcutaneously at 1 day of age. This vaccine does not produce a visible take, but may result in a small number of birds that exhibit central nervous system (CNS) signs at 4-12 days postvaccination. *In-ovo* injection of this vaccine may magnify the number of chicks exhibiting CNS reactions.

7. Control cannibalism with proper beak trimming and reduced environmental light intensity.

8. Fowl pox is currently being employed as a vector for recombinant vaccines.

TREATMENT

There is no satisfactory treatment for pox.

INFECTIOUS BRONCHITIS

(IB)

DEFINITION

Infectious bronchitis (IB) is an acute, highly contagious, virus-caused disease of chickens characterized by respiratory signs (gasping, sneezing, coughing, and nasal discharge), severe renal disease associated with nephrotropic strains, and a marked decrease in egg production.

OCCURRENCE

1. IB occurs naturally only in chickens. All ages are susceptible, assuming they have not had prior exposure to the virus or are not passively immune.

2. The disease is present in all countries where chickens are raised in large numbers. In the United States the disease occurs frequently and throughout the year, even in vaccinated flocks.

HISTORICAL INFORMATION

1. In 1930, IB was first observed in young chicks. By the 1940's, IB was a serious disease of laying flocks causing marked loss in egg production. Nephropathogenic IB was first observed in the 1960s.

2. The virus was first isolated by Beach and Schalm in 1936 and multiple serotypes were first reported in 1956.

3. Vaccination became commercially available in the 1950's and is currently practiced worldwide.

ETIOLOGY

1. IB is caused by a coronavirus. The virus is fairly labile and can be destroyed by many common disinfectants.

2. The virus is resistant enough to survive in 50% glycerin in most mailed tissue specimens. The fresh trachea and lung of an infected chicken can be submitted this way for virus isolation.

3. IB virus without enzyme treatment does not hemagglutinate erythrocytes as do Newcastle and influenza viruses.

4. There is great antigenic variation among IB viral strains and many serotypes of the virus have been identified. Four common serotypes (Connecticut, Massachusetts, Arkansas 99 and O72) are used in U.S. vaccine preparation. Cross-protection among serotypes is highly variable.

5. Some IB viral strains have a distinct predilection for renal tissue and these nephrotropic strains can induce significant mortality.

6. IB virus retains a great capacity to mutate, thus making classification of strains difficult.

EPIZOOTIOLOGY

1. Transmission of IB is by inhalation of virus-containing droplets expelled by infected, coughing chickens. Aerosol transmission is suspected of occurring over considerable distance. Spread of infection throughout a flock is explosively rapid.

2. The virus of IB may persist on contaminated premises for approximately 4 weeks or longer under favorable conditions. Susceptible birds brought on the premises during that interval may contract the disease.

3. A few birds may remain carriers and shedders of the virus for months after infection. They intermittently eliminate virus in secretions and discharges, thus exposing susceptible chickens or contaminating premises.

4. Vertical transmission has not been documented.

CLINICAL SIGNS

Baby chicks

1. Signs include coughing, sneezing, rales, and nasal and ocular discharge [Fig. 1; Infectious bronchitis; Univ Montreal]. Morbidity is virtually 100%, although severity of signs varies. Signs can develop within 48 hours postinfection.

2. There is weakness, depression, and huddling near heat sources.

3. Mortality in young chicks is usually negligible unless the disease is complicated by another infectious agent. Nephrogenic strains may cause high mortality.

Laying chickens and broilers

1. There is coughing, sneezing, and rales. Seldom is there nasal or ocular discharge.

2. Egg production drops markedly (up to 50%). Effects on production can last 6-8 weeks or longer. Eggs are often soft-shelled or misshapened [Fig. 2; Infectious bronchitis; Univ Montreal]. Egg albumin may be watery. Low egg quality and shell irregularities may persist long after an outbreak of IB. Chickens that had IB or a severe reaction to IB vaccine when less than 2 weeks old may suffer permanent damage to the oviduct resulting in poor-to-no egg-laying capacity.

3. Chickens that have IB or a severe reaction to IB vaccination may develop airsacculitis, due to an increased susceptibility to secondary infectious agents (especially *E. coli* or *Mycoplasma gallisepticum*). This complication can be very severe and may accentuate respiratory signs, especially in young chickens.

4. Mortality associated with swollen pale kidneys and urolithiasis is induced by nephrotropic IB strains in pullets and even in mature birds.

LESIONS

1. There is mild to moderate inflammation of the upper respiratory tract. There may or may not be airsacculitis. Severe airsacculitis is manifested as a marked thickening and opacity of the air sac membranes and often is accompanied by much exudate in the air sacs. Airsacculitis can result in high mortality in young, growing birds, especially if husbandry is poor. Older birds are usually more resistant.

2. The kidneys sometimes are swollen and the ureters and tubules contain uric acid crystals, especially in young birds, including broilers.

3. Yolk material frequently is present throughout the peritoneal cavity and the ovarian follicles appear flaccid. These lesions are not specific for IB but accompany many acute diseases of layers.

4. In layers that had IB or a severe vaccination reaction while less than 2 week old, there may be abnormalities of the oviducts (particularly the middle third) in occasional birds. Oviducts may be hypoplastic or cystic and such birds may deposit yolks or fully formed eggs in the abdominal cavity and are referred to as internal layers.

DIAGNOSIS

1. Tests of paired acute and convalescent serum can be very useful in demonstrating a specific immune response. Several procedures including serum-virus neutralization, enzyme-linked immunosorbent assay (ELISA) and modified hemagglutination inhibitions are available.

2. For diagnosis it is necessary to isolate and identify the IB virus. This usually is done in 9-12-day-old chick embryos. Trachea, lungs, air sacs, and kidneys are good sources of virus. In infections beyond 1 week duration, cloacal swabs are preferred. Confirmation of IB virus and its serotype can be done by various antibody methods using monoclonal antibodies. PCR, RTPCR and nested PCR have been used to identify IB viral serotypes.

3. Nine to 12-day-old chick embryos inoculated with supernatant containing IB virus develop lesions that are useful in diagnosis. The mesonephros of living embryos surviving 5-7 days postinoculation contains excessive urates. IB virus causes dwarfing and stunting of some inoculated chick embryos. Also, the amnion and allantois are thickened and closely invest the embryo. After initial isolation it may be necessary to passage the virus three to five times to obtain embryo lesions. The alterations are duplicated by some lentogenic strains of Newcastle virus.

4. Egg fluids from inoculated embryos do not hemagglutinate erythrocytes if IB virus is present but will hemagglutinate if Newcastle virus is present.

5. The fluorescent antibody technique or electron microscopy can be used on tracheal samples for rapid diagnosis of IB but do not differentiate the serotype.

CONTROL

1. Modified live virus vaccines are used in young chickens for prevention. Vaccines are effective only if they contain the right serotypes of virus for a given area. If given to chicks carrying parental immunity, vaccination should be repeated at least once. Polyvalent bronchitis vaccines are sometimes used but can cause more severe vaccine reactions in naive chicks. Infectious bronchitis vaccine is often combined with Newcastle vaccine in the same vial but can cause interference with the Newcastle vaccine if not commercially prepared as a combination vaccine. Vaccines are generally applied via the drinking water or by spray. Utmost care needs to be taken to preserve the vaccine integrity as the vaccine virus can be prone to inactivation under adverse conditions.

2. Vaccinated birds should be watched carefully for possible onset of airsacculitis following vaccination. If signs or lesions of airsacculitis are detected, broad-spectrum antibiotics added to the feed or water will usually minimize the airsacculitis and reaction.

3. Killed virus vaccines (oil emulsion base) are now widely used. They are administered by injection (subcutaneous or intramuscular) to breeders or layer replacement pullets from 14 to 18 weeks of age. They induce high and sustained antibody levels.

TREATMENT

1. No effective treatment of IB is known although broad-spectrum antibiotics may control the complications. If there are no complications of IB infection or vaccination, medication following vaccination or infection is not indicated.

2. For baby chicks with IB, it may be helpful to increase the room temperature, encourage the birds to eat by using a warm moist mash, and correct any apparent management deficiencies.

INFECTIOUS BURSAL DISEASE

(IBD; Gumboro Disease)

DEFINITION

Infectious bursal disease (IBD) is an acute, contagious, viral disease of young chickens characterized by diarrhea, vent picking, trembling, incoordination, inflammation followed by atrophy of the bursa of Fabricius, and by variable degrees of immunosuppression.

OCCURRENCE

IBD occurs primarily in chickens. Clinical signs and mortality are generally more severe in birds 3-6 weeks old. However, IBD may occur in chickens as long as they have a functional bursa of Fabricius (1-16-weeks of age). Birds infected at less than 3 weeks of age do not have clinical signs. However, destruction of the bursa results in immunosuppression. The younger the bird at the time of infection, the more severe the immunosuppression, resulting in a high degree of susceptibility to subsequent pathogens. Once a premise has been contaminated with IBD virus, the disease tends to recur, usually as a subclinical infection.

In turkeys, subclinical infection with IBD virus occurs without immunosuppression. However, there is no known disease associated with IBD viral infection. Most of the IBD viruses from turkeys are serologically distinct from those isolated from chickens. Ducks can also be subclinically infected with no resultant immunosuppression.

IBD now occurs in all of the major poultry-producing countries of the world.

HISTORICAL INFORMATION

In 1962, “avian nephrosis”, a condition now believed to be IBD was reported to be occurring on farms near Gumboro, Delaware. Initially the disease was confused with a variant form of infectious bronchitis accompanied by nephrosis, but the disease is now well delineated and well characterized. The immunosuppressive effects of IBD were first reported by Allan in 1972. Variant strains of serotype 1 IBD were found in the Delmarva region in the 1980’s. Very virulent strains of IBD have been reported in The Netherlands, Africa, Asia, and South America. In the United States the disease is a persistent problem in the broiler industry despite vaccination.

ETIOLOGY

1. IBD is caused by a virus belonging to the family Birnaviridae. The viral genome has two double-stranded RNA segments. Other similar viruses occur in fish and mollusks. The virus may be propagated in chicken embryos or chicken embryo cell cultures. Two serotypes exist, with only serotype 1 being pathogenic. Within serotype 1, there are six unrelated or partially related strains.

2. The virus is very resistant to most disinfectants and environmental factors. It persists for months in contaminated houses and for weeks in water, feed, and droppings. It can be transmitted by fomites. It has some susceptibility to formalin and iodide disinfectants. Invert soaps with 0.05% sodium hydroxide may kill IBD virus.

3. The virus is lymphocidal (immunoglobulin-bearing lymphocytes) and severely damages the bursa of Fabricius. The thymus, spleen, and cecal tonsils are also damaged but less severely.

4. It has been demonstrated that IBD can severely damage the humoral responsiveness of susceptible chicks when they are infected at less than 3 weeks of age. Those chicks then do not respond properly when vaccinated against other diseases. There is evidence that inclusion body hepatitis and gangrenous dermatitis occur frequently in such flocks. Some live vaccines may have a similar potential for damage as field infections.

5. The passive transfer of maternal antibodies to baby chicks is very important for the prevention of early infections with the virus. Breeder flocks must receive vaccines or field exposure to the virus followed by booster vaccinations to stimulate high levels of maternal antibody. Progeny from well-immunized breeder flocks will resist infection for 2-4 weeks. Passive immunity will interfere with vaccinations and it is necessary to vaccinate chickens after maternal immunity has fallen to a point that the vaccine will overcome the lower levels of maternal antibody. If chicks are from nonimmune flocks, vaccinations should be administered at hatch.

EPIZOOTIOLOGY

1. The virus spreads rapidly from infected chicks and from contaminated premises or fomites to susceptible chicks. The disease is highly contagious.

2. Transmission of virus through the egg does not appear to occur and there is no evidence for a carrier state.

3. The lesser meal worm (*Alphitobius diaperinus*) harbors the virus for weeks after an outbreak and may transmit it to susceptible birds. The worm lives in poultry litter.

4. The incubation period is very short with clinical signs evident 2-3 days post exposure.

5. Subclinical infection (before 3 weeks of age) is economically important due to suppression of humoral immunity and subsequent secondary infections.

CLINICAL SIGNS

1. Clinical disease is observed only after 3 weeks of age. There is a sudden onset, particularly with the first outbreak. There may be tremor or unsteadiness. There is depression, anorexia, ruffled feathers, and a droopy appearance that resembles coccidiosis.

2. Diarrhea and dehydration are usually present. Occasionally there is voiding of blood and straining during defecation. Vent picking is common and may be self-inflicted.

3. Morbidity is very high. Mortality is usually low although it can be substantial (approaching 30%) if husbandry is poor or if strains are particularly virulent. Mortality in a flock has usually peaked and receded within a week of onset. IBD tends to be more severe in leghorn strains than in broiler stock.

LESIONS

1. Initially the bursa is enlarged to about twice normal size, severely edematous [Fig. 1; Infectious bursal disease; AAAP], and reddened. It may contain hemorrhages [Fig. 2; Infectious bursal disease; Cornell U]. The swelling recedes about the 5th day and the bursa atrophies rapidly until 8-10 post infection. There is increased mucus in the intestine. Bursal lesions are evident in both clinically and subclinically infected birds.

2. In field outbreaks, hemorrhages are common in thigh and pectoral muscles and, perhaps, at the junction of the proventriculus and gizzard.

3. The parenchymatous organs, especially the kidneys, may be swollen. The ureters may contain urates.

4. Necrotic lesions/atrophy may also be found in other lymphoid organs such as the thymus and the spleen, particularly with highly virulent IBD strains.

5. Microscopically, in the bursa there is lymphoid follicle depletion and destruction followed by atrophy. Similar changes occur in the spleen, thymus, and cecal tonsils but they recover more rapidly and completely than does the bursa.

6. Some variant strains of the virus cause few clinical signs and minimal gross acute changes in the bursa. However, these variant strains may induce rapid bursal atrophy and severe immunosuppression.

DIAGNOSIS

1. In an acute outbreak in susceptible chicks, the short course and bursal lesions are very suggestive of IBD. Signs and lesions can be are less apparent in subsequent outbreaks and in chicks with parental antibody.

2. Paired serologic testing with rising titers using the ELISA, agar-gel precipitin, or virus neutralization will usually confirm the diagnosis. However, the virus neutralization test is the only serological assay that will identify the infecting serotype or strain (subtype).

3. If susceptible chicken embryos and known-positive antiserum are available in a laboratory, the virus can be isolated from the bursa or spleen and then identified by neutralization. The virus neutralization assay, PCR and antigen-capture enzyme immunoassay with monoclonal antibodies can be used to differentiate serotype 1 subtypes.

4. Microscopic bursal lesions are said to be specific for IBD in the early stages of the disease. The direct fluorescent antibody technique and electron microscopy also can be applied to virus-containing tissues.

5. Coccidiosis, hemorrhagic syndrome, and adenoviral infection must be differentiated from IBD infection.

CONTROL

1. Vaccination of breeders to confer immunity to progeny is an effective method of reducing the disease in young chicks. Killed oil-emulsion vaccines are effective in producing high levels of antibody in breeders, after priming with a live vaccine applied prior to the onset of lay.

2. Chicks can be vaccinated against the disease but timing the vaccination in maternally immune chicks can be difficult. When maternal antibodies wane use of “hot” vaccines in nonimmune chicks may result in bursal atrophy. Vaccination with milder vaccines will not be effective in birds with high levels of maternal antibody. Therefore, knowledge of passive antibody levels and correct timing are necessary for successful vaccination.

3. An in ovo immune complex vaccine is available that results in decreased vaccine pathogenicity without loss of immunogenicity.

4. Sanitation programs are rarely successful due to the highly resistant nature of the virus.

TREATMENT

Treatment is of no value. However, good husbandry and adequate temperature may reduce the severity of the disease.

INFECTIOUS LARYNGOTRACHEITIS

(ILT; LT; Laryngotracheitis)

DEFINITION

Infectious laryngotracheitis (ILT) is an acute viral disease of chickens, and, rarely, pheasants, and peafowl characterized by marked dyspnea, coughing, gasping, and expectoration of bloody exudate.

OCCURRENCE

ILT is worldwide in distribution. Most outbreaks in chickens occur in broilers more than 4 weeks of age or in mature or nearly mature chickens, although all age groups are susceptible.

ETIOLOGY

1. ILT is caused by a herpesvirus. The virus is readily destroyed by many disinfectants and is not highly resistant outside of the host. There appears to be only one immunologic strain, although strains vary considerably in pathogenicity. Most strains are rather pathogenic, although there are some strains of low pathogenicity.

2. Herpesvirus infection leads to the formation of type A intranuclear inclusions during the first few days of infection. The inclusions may be observed in scattered groups of tracheal epithelial cells and, occasionally, in conjunctival epithelium. Similar inclusions occur in the infected chorioallantoic membrane of embryonating chicken eggs and in chick embryo cell cultures.

EPIZOOTIOLOGY

Some recovered chickens and vaccinated chickens become carriers and shed virus for long periods of time or much later can shed virus following stress-induced reactivation of latent infections, thus exposing other susceptible birds. Mechanical transmission of virus via fomites also is possible. The disease spreads horizontally via the respiratory tract after it has been introduced. However, spread is often less rapid than with other viral respiratory diseases of chickens. There is no evidence of vertical transmission.

CLINICAL SIGNS

Signs of markedly pathogenic ILT

1. There is marked dyspnea, often with loud gasping sounds and coughing. Severely affected chickens often raise and extend their head and neck during inspiration [Fig. 1; Infectious Laryngotracheitis; AAAP] and make loud wheezing sounds.

2. Expectoration of bloody mucus [Fig. 2; Infectious Laryngotracheitis; Cornell U] may occur as a consequence of coughing and head shaking. Beaks, faces, or feathers of occasional birds may be bloody.

3. High morbidity and considerable mortality are common. Morbidity as high as 50-70% has been reported but mortality usually is in the 10-20% range. There is also lowered egg production. The disease often persists for as long as 2-4 weeks in the flock, a course longer than that of most viral respiratory diseases of chickens.

Signs of ILT of low pathogenicity

Signs often include hemorrhagic conjunctivitis with watery eyes, lacrimation, persistent nasal discharge [Fig. 3; Infectious Laryngotracheitis; AAAP], swollen infraorbital sinuses, generalized unthriftiness and lowered egg production.

LESIONS

1. Lesions are most common in the conjunctiva, larynx and trachea. Severe ILT often presents with bloody exudate or diphtheritic changes in the trachea [Fig. 4; Infectious Laryngotracheitis; AAAP]. Lesions may vary from mucoid inflammation to severe degeneration of the mucosa with hemorrhage. Inflammation may extend into bronchi and air sacs. Dead birds may have an occluding pseudomembrane or caseous plugs in the trachea, with death having occurred from suffocation.

2. Infected birds often have a bloody beak or blood on the face, head, or feathers.

3. In less pathogenic outbreaks, mild conjunctivitis and sinusitis may be the only lesions.

4. Microscopic examination of the trachea of birds killed during the first few days (1-5) of the disease may reveal intranuclear inclusion bodies in epithelial cells [Fig. 5; Infectious Laryngotracheitis; AAAP]. Similar inclusions can be demonstrated in the chorioallantoic membrane of infected chicken embryos and in infected tissue culture cells. Inclusions disappear as the disease progresses due to necrosis and desquamation of the epithelial cells.

DIAGNOSIS

The signs and lesions of the pathogenic type of ILT are distinctive enough to incite suspicion of ILT. However, there may be few signs and lesions with ILT of low pathogenicity. ILT can usually be confirmed by one or more of the following steps:

1. Demonstration of intranuclear inclusions in the trachea (by histopathologic means) during early stages of the disease.

2. Demonstration of viral antigen in clinical samples, usually tracheal epithelium, by the use of the fluorescent antibody, immunoperoxidase, electron microscopy, DNA hybridization techniques, antigen capture ELISA and PCR.

3. Growth of the virus on the chorioallantoic membrane of embryonating chicken eggs. Typical plaques are produced and inclusion bodies can be demonstrated in them by histologic means and by the fluorescent antibody technique.

4. Exposure of known-immune and known-susceptible chickens to virus. The virus can be inoculated into either the infraorbital sinus, trachea, or the bursa of Fabricius and the reaction or lack of reaction can be observed.

CONTROL

1. Avoid adding vaccinated, recovered, or exposed birds to a susceptible flock because these birds may include recovered carrier birds with latent infections. Better yet, raise susceptible flocks in strict quarantine and never add birds of any kind.

2. Premises contaminated with laryngotracheitis virus should be depopulated, cleaned, disinfected, and left vacant for 4-6 weeks before being used again. Due to the heat-labile nature of the virus, virus destruction is enhanced by heating the contaminated poultry house (95-100 F for 72 hours).

3. In areas where ILT is endemic, vaccination of layers is frequently practiced and is quite effective. Attenuated vaccines are available and can be administered by eye drop, in the drinking water, or by aerosol spray. Drinking water vaccination may not be reliable because it depends upon the vaccine contacting nasal epithelium with high virus titers. Birds vaccinated prior to 10 weeks of age should be revaccinated at

10 weeks of age or older to confer lifelong immunity. Vaccination of broilers, when indicated, should be done before 4 weeks of age to minimize losses from severe vaccine reaction. Do not mix ILT vaccines with other vaccines.

4. Rarely, clinical ILT, indistinguishable from the natural disease, may occur 1-4 weeks after vaccination. These vaccine-related episodes are usually characterized by low morbidity and mortality.

5. ILT is a reportable disease in some states.

TREATMENT

Treatment is of little or no value and not practical. However, vaccination of unaffected birds and those in other houses on an infected farm may provide protection and stop the outbreak.

NEWCASTLE DISEASE

(Newcastle; Avian Pneumoencephalitis)

DEFINITION

Newcastle disease (ND) is a viral disease of many kinds of poultry, and wild and cage birds characterized by marked variation in morbidity, mortality, signs and lesions. Because of the variable nature and high incidence of ND in chickens and in wild and cage birds, the following salient points about ND clarify the definition:

1. In chickens, ND caused by slightly pathogenic (lentogenic) strains of virus may produce few or no signs and little or no mortality.

2. In young chickens, Newcastle disease caused by moderately pathogenic (mesogenic) strains of virus is characterized by respiratory signs, with concurrent or closely following central nervous system (CNS) involvement, and high mortality. In layers, ND causes a marked, sudden drop in egg production, accompanied by few or no signs and little or no mortality.

3. In chickens the most pathogenic (velogenic) form of ND is usually characterized by a short course, marked respiratory signs, diarrhea, and paralysis followed by death of most affected birds.

4. In wild and cage birds, ND often is inapparent. Signs, when apparent, are variable but often include gasping respiration, diarrhea, and later signs of CNS involvement. Sudden deaths are often the first indication of ND.

OCCURRENCE

The disease usually occurs in chickens or (less often) in turkeys, although most poultry and many wild and cage birds are susceptible. All age groups are susceptible. The disease occurs in all poultry-raising countries. ND is relatively common.

Humans who come in close contact with Newcastle virus for the first time may develop a temporary, localized eye infection (conjunctivitis).

HISTORICAL INFORMATION

1. ND first appeared in Java, Indonesia and Newcastle-upon-Tyne, England in 1926. Within 10 years it had spread to many countries throughout the world. The disease persists in many countries and in its velogenic form is one of the most devastating diseases of poultry, with mortality up to 100% in chickens.

2. ND of low to moderate virulence has been present in the United States since about 1940. In chickens these forms of ND are well controlled by often-repeated vaccinations. These forms have seldom been a major problem except in chickens.

3. Velogenic ND, the most pathogenic type, occurred in the United States in 1941, 1946, and 1951 but was quickly eradicated. Extensive outbreaks began in California (and other locations) in 1971 and 2002 and were eradicated at great expense ($52,000,000.00 and 170,000,000, respectively).

4. It has become apparent that velogenic ND is usually introduced by imported cage birds or fighting cocks, in many instances by illegally introduced birds. These sources of infection must be controlled if the poultry industry is to remain free of the disease.

ETIOLOGY

1. Newcastle disease is caused by paramyxovirus type 1. The many known strains vary greatly in pathogenicity. They often are classified or referred to as:

    A. Lentogenic—these are mildly pathogenic (examples: B-1, F, LaSota).

    B. Mesogenic—these are moderately pathogenic.

    C. Velogenic—these are markedly pathogenic (examples: Milano, Herts, Texas GB).

    Most enzootic strains are lentogenic or mesogenic. Vaccines prepared from lentogenic strains tend to produce immunity that is weak and of short duration so that frequent revaccination is necessary to maintain immunity. Conversely, more pathogenic (mesogenic) strains used for vaccine tend to produce longer, stronger immunity but may produce mortality in unthrifty chickens and are not used in the United States. All velogenic strains are classified as select agents in the US.

2. Newcastle virus hemagglutinates the erythrocytes of many species, including those of many birds. This unique characteristic is useful in that hemagglutination and hemagglutination inhibition (HI) tests are helpful in virus identification.

EPIZOOTIOLOGY

1. Virus-containing excretions from infected birds, including aerosols and feces, can contaminate feed, water, footwear, clothing, tools, equipment, and the environment. Exposure of susceptible birds to any of these sources of virus can result in transmission via inhalation or ingestion. Also, infected poultry may spread the virus if their tissues are used without proper processing in rendered products.

2. Eggs laid by infected hens may contain virus. Such eggs seldom hatch and few are laid due to cessation of production caused by ND. If they are accidentally broken in the hatcher, the entire hatch of chicks may be exposed. The exposed, apparently normal chicks may then be divided into small lots of birds and widely disseminated before the disease becomes apparent.

3. Live-virus vaccines may constitute a reservoir of Newcastle virus. Chickens often shed the vaccine virus. There is no evidence that attenuated viruses regain their virulence through passage.

4. At various times Newcastle virus has been isolated from sparrows, pigeons, doves, crows, owls, and waterfowl. Recent experience suggests these birds do not play a significant role in the spread of ND.

CLINICAL SIGNS

Newcastle strains contrasted

1. Adult chickens

    Lentogenic ND

    May not produce any clinical signs at all, or may produce mild respiratory signs and decreased egg production in laying flocks. A few eggs may be soft-shelled, roughened, or deformed.

    Mesogenic ND

    A. Sudden onset with mild depression and anorexia. Respiratory signs usually occur but may be in a mild or inapparent form. Mortality is low or absent.

B. Signs suggestive of CNS disease may occur in a few birds but often do not appear.

C. In layers, production almost completely ceases within a few days. Eggs laid are of low quality and may be soft-shelled, roughened, or deformed [Fig. 1; Newcastle Disease; Univ Montreal]. Production is resumed slowly, or not at all, depending on the stage of lay at the time of infection.

Velogenic ND

Signs vary according to tropism of the virus. Dyspnea often is marked. There is violent diarrhea, conjunctivitis, paralysis, and death in 2-3 days in many chickens. There may be swelling and darkening of tissues about the eyes with sticky ocular and nasal discharge. Some birds that survive a few days may exhibit signs of CNS involvement (e.g., tremors, twisting of the head and neck, circling, paresis, paralysis, terminal clonic spasms). Morbidity and mortality are high—up to 100%.

2. Young chicks

Lentogenic ND

Broilers may show sudden onset of respiratory signs including gasping, sneezing, coughing, rales, and nasal and lacrimal discharge. Some birds may have swollen heads. Even mild B1 strain vaccines may cause these signs in broilers with low immunity.

Mesogenic ND

A. Sudden onset with marked depression and prostration. Marked respiratory signs that include gasping, coughing, hoarse chirping, and nasal discharge.

B. Signs of CNS disease may accompany or closely follow the onset of respiratory signs. Abnormal positions of the head and neck ("star gazers") are common. Usually only a modest number (0-25%) show CNS signs.

C. Eventually there is paralysis, prostration, trampling by pen-mates, and death. Mortality can be very high (up to 50%) regardless of whether CNS signs occur.

Velogenic ND

Signs are similar to those induced with mesogenic strains in young birds but mortality is very high (50-100%) and the course is more acute.

LESIONS

Lentogenic and mesogenic ND

Usually gross lesions are minimal in young or old birds although there may be mild airsacculitis, conjunctivitis, and tracheitis. The absence of lesions is of some diagnostic value.

Velogenic ND

1. Hemorrhage and necrosis of the trachea [Fig. 2; Newcastle Disease; UC Davis]
and inflammation of the air sacs is usually severe, especially if respiratory signs have predominated.

2. Hemorrhagic or necrotic focal lesions are present in the mucosa of the entire digestive tract. The oral-pharyngeal cavity and esophagus lesions can be prominent [Fig. 3; Newcastle Disease; UC Davis]. Intestinal lesions often involve lymphoid tissue in the mucosa [Fig. 4; Newcastle Disease; UC Davis]. The

larger foci may be visible through the unopened intestine but smaller ones are not. Cecal tonsils (lymphoid tissue) often are necrotic and hemorrhagic [Fig. 5; Newcastle Disease; UC Davis] (cecal tonsils are at the opening of the ceca into the intestine).

3. Hemorrhages occasionally occur on the mucosal surface of the proventriculus [Fig. 6; Newcastle Disease; UC Davis] or in the gizzard. They may be present at many sites under serous membranes and in the mucosa of the esophagus.

4. Cage birds or wild birds often reveal no gross lesions or have mild, nonspecific lesions. Signs and lesions in turkeys are similar to those in chickens but milder.

5. Microscopic lesions have been described in detail (*Diseases of Poultry*, 11th ed.). In the CNS they include neuronal degeneration, perivascular cuffing with lymphocytic cells and endothelial hypertrophy. These must be differentiated from those of avian encephalomyelitis and other encephalitides. Microscopic lesions are similar in all forms of ND but are not always present.

DIAGNOSIS

1. Clinical diagnosis based on history, signs, and lesions may establish a strong index of suspicion once ND has been positively identified in an area, but laboratory confirmation should always be pursued in order to identify the strain.

2. It is essential to isolate (usually in chicken embryos) and identify the virus. Tests of value in identification include the following:

    A. Hemagglutination and hemagglutination-inhibition tests with the virus.

    B. Virus neutralization test (using known Newcastle antiserum).

    C. Plaque neutralization test (in a tissue culture system).

    D. Inoculation of the virus into known-immune and known-susceptible chickens.

    E. Fluorescent antibody technique using a conjugated Newcastle antiserum.

    F. Demonstration of increasing titer of Newcastle antibody in the flock from onset to convalescence.

    G. PCR and RTPCR

CONTROL

1. Chickens and turkeys can be immunized against ND by proper vaccination. The method of vaccine administration has considerable influence on the immune response. Low-virulence live-virus vaccines are administered by a variety of routes, including in ovo, and schedules from hatching through grow-out. Killed-virus oil-emulsion vaccines are administered parentally as a final vaccine prior to the onset of egg production. Although proper vaccination protects the birds from serious clinical disease it does not prevent virus replication and shed, which could be a source of infection to other flocks.

2. Stringent laws are in effect pertaining to the importation of poultry, poultry products, and cage birds. However, enforcement is often a difficult and nebulous problem.

3. A standby group of people trained in eradication of velogenic ND has been established. This force is deployed immediately when a new outbreak occurs and may be able to quickly contain new outbreaks.

4. ND is a reportable disease. All suspected outbreaks of ND must be reported to animal health authorities immediately. Do not attempt to differentiate between enzootic and velogenic forms of the disease. Report any highly pathogenic outbreaks of respiratory disease in poultry.

TREATMENT

Treatment of ND is of no value.

TURKEY VIRAL HEPATITIS

DEFINITION

Turkey viral hepatitis is a contagious, often subclinical disease of turkey poults characterized by lesions in the liver and pancreas.

OCCURRENCE

The clinical disease is seen only in turkey poults less than 5 weeks old. Other birds and mammals appear not to be affected. The disease has been observed in most turkey-producing areas of the world.

HISTORICAL INFORMATION

Turkey viral hepatitis was first reported in 1959. Subsequently, the disease has been reported in a number of states and countries but has seldom been associated with severe mortality. Incidence and distribution is difficult to evaluate due to the subclinical nature of the disease.

ETIOLOGY

The etiologic agent is a virus that has been suggested to be a picornavirus based on its morphologic features. It can be grown in the yolk sac of 5-7-day-old chicken or turkey embryos. Embryo mortality occurs in 4-10 days. The virus rarely achieves high titers despite repeated embryo passages. Young turkey poults can be infected following parenteral inoculation of infective tissue suspensions.

EPIZOOTIOLOGY

Up to 28 days after infection, the virus can be isolated consistently from feces and liver of infected poults. Transmission readily occurs by direct or indirect contact of susceptible poults. There is clinical evidence suggestive of egg transmission of virus.

CLINICAL SIGNS

The disease is usually subclinical. It may be that signs are apparent only if there are other concurrent diseases or stresses on the poults. Infected flocks in the early stages of the disease show variable depression with sporadic deaths of well-fleshed birds. Morbidity varies greatly. Mortality is usually very low (>5%) but has been as high as 25% during a 7-10 day period in occasional flocks.

LESIONS

1. In the liver, there are focal gray areas 1 mm or more in diameter that may coalesce [Fig. 1; Turkey viral hepatitis; NCSU]. Lesions are often slightly depressed and can be concealed by congestion and focal hemorrhages. Bile staining may be apparent.

2. In the pancreas, lesions are less consistent and appear as focal gray to pink areas [Fig. 2; Turkey viral hepatitis; NCSU]. They are usually more evident later in the course of the disease and on the dorsal side of the pancreas.

3. Microscopically, inclusion bodies have not been identified. The lesions observed grossly are found to be focal areas of necrosis, which subsequently become infiltrated with mixed inflammatory cells in which lymphocytes and reticular cells predominate; heterophils are present but not in high numbers. Syncytial cells arising from hepatocytes can often be found along lesion margins.

DIAGNOSIS

1. Typical lesions in both liver and pancreas are diagnostic. If turkey viral hepatitis is suspected, both liver and pancreas need to be submitted for histopathologic examination even if there are no visible lesions in the latter; often microscopic lesions can be found in the pancreas in the absence of gross lesions. If lesions are present only in the liver, they will have to be differentiated from those of blackhead and systemic bacterial infections.

2. Isolation and identification of the virus can be used for confirmation. An agar-gel precipitin test using rabbit antisera to the virus has been developed but is not in general use.

CONTROL

1. A high standard of sanitation along with proper nutrition and good husbandry should minimize the effects of the disease. There is no vaccine for prevention. Treatment of concurrent diseases is important.

2. Eggs from infected flocks should not be used for hatching because there may be transovarian transmission of virus.

TREATMENT

There is no proven effective treatment. Fortunately, most well cared for flocks recover spontaneously in a few weeks.

VIRAL ARTHRITIS

(Tenosynovitis; Ruptured Gastrocnemius Tendon; Reovirus Infection)

DEFINITION

Viral arthritis is a reovirus infection primarily of meat-type chickens characterized by arthritis and tenosynovitis (primarily of the tarsus and metatarsus) and, occasionally, by rupture of the gastrocnemius tendon(s).

OCCURRENCE

Viral arthritis occurs primarily in meat-type chickens with rare reports of the disease in egg-type chickens and turkeys.

HISTORICAL INFORMATION

Viral arthritis was first reported in 1957. Since then there have been numerous reports on the disease and much has been learned about it and the virus that causes it. Viral arthritis has assumed greater importance as other diseases of chickens have been brought under better control. Viral arthritis is of special importance to the broiler industry because broilers frequently are infected.

ETIOLOGY

1. The etiologic agent is a reovirus. Several serotypes have been identified. It grows well in susceptible chicken embryos, in chicken kidney cell culture, and young chickens.

2. All serotypes of the reovirus share some precipitin antigens. An agar-gel precipitin test can be used for identifying most birds that have been infected for a few weeks.

3. The reovirus is quite resistant to many environmental factors.

EPIZOOTIOLOGY

Reovirus is discharged in the feces of infected chickens and may contaminate eggshells. It is transmitted laterally to susceptible chickens. Egg transmission has been demonstrated. Reovirus is known to persist in infected birds for at least 289 days.

CLINICAL SIGNS

1. Lameness and swelling of the tendon sheaths of the shanks and of the gastrocnemius tendon above the hock are early signs. The shanks of affected chickens are enlarged. Many infected birds are in good condition but some are unthrifty and stunted. Mortality usually is quite low.

2. If the gastrocnemius tendon has been ruptured, the affected foot cannot be extended and the bird cannot bear weight on the affected leg. If both tendons are ruptured, the bird is immobilized [Fig. 1; Viral arthritis; AAAP]. There usually is swelling and discoloration of the skin over the site of tendon rupture.

LESIONS

1. In the acute phase of the disease there is swelling and inflammation of the tendons and tendon sheaths just above the hock and along the posterior aspect of the shanks [Fig. 2; Viral arthritis; NC Dept of Ag]. The hock joints and tendon sheaths contain inflammatory exudate that may be

bloody [Fig. 3; Viral arthritis; Cornell U]. The articular cartilages of the hock may be eroded [Fig. 4; Viral arthritis; AAAP] and the synovial membranes of the hock and tendons may contain hemorrhages.

2. Chickens that cannot extend the hock often have rupture of the gastrocnemius tendon, usually just above or over the hock joint. This is especially true of older, heavier birds.

3. In chronic cases there is usually less inflammatory exudate and more fibrosis of affected tendons and tendon sheaths. Affected tendons and tendon sheaths may be fused by adhesions.

DIAGNOSIS

1. A tentative diagnosis often can be made on the basis of history and bilateral enlargement of the tendon sheaths of the shanks. It may be necessary to confirm inflammation of the tendon sheaths and tendons by microscopy. Also, infiltration of heterophils, lymphocytes, and plasma cells among myocardial fibers is a constant microscopic finding.

2. If available, the direct fluorescent antibody test can be used to demonstrate reovirus antigen in the cytoplasm of infected cells of synovial membranes.

3. If convalescent sera can be obtained from a few birds of the infected flock, it may be possible to demonstrate antibody to the reovirus using the agar-gel precipitin test or enzyme-linked immunosorbent assay (ELISA). Antibody may disappear as early as 4 weeks postinfection in some birds but it persists in birds with joint involvement.

4. Isolation and identification of the virus in chick kidney cell culture or chicken embryos may be possible. Intracytoplasmic inclusions have been reported in kidney cells and in cells of the chorioallantoic membrane but are not relied upon for diagnosis.

5. It is essential to exclude other causes of lameness. These include mycoplasmosis (especially infectious synovitis), staphylococcal and other bacterial arthritis, salmonellosis, Marek's disease, pasteurellosis, erysipelas, deformities, and certain nutritional diseases. It should be remembered that dual infections can exist.

CONTROL

1. Due to the age-associated resistance to disease after 2 weeks, the ubiquitous distribution of the virus in poultry-raising areas, and the resistance of the virus to inactivation, prevention of this disease should be directed at preventing early infection.

2. Vaccination of breeder flocks with live and inactivated vaccines results in protection of 1- day-old chicks.

3. Mild vaccines are available for spray or subcutaneous injection of 1-day-old chicks and drinking water administration to older chicks. Older chickens can also be vaccinated by subcutaneous injection.

4. Chicks derived from reovirus-free flocks should be raised under the all-in, all-out system. They should be started as a unit and raised in quarantine. No birds should be added to the started flock

TREATMENT

There is no satisfactory treatment.

AVIAN CHLAMYDIOSIS

(Psittacosis; Ornithosis)

DEFINITION

Avian chlamydiosis is a reportable, acute or chronic infectious disease of poultry, many caged birds and wild and migratory birds. In clinically ill birds, the disease is characterized by systemic, pulmonary, or enteric signs and lesions. The latent, inapparent chlamydial infection has long been recognized as the predominant and most important state in the zoonotic relationship between chlamydial agent, birds and humans.

In the Psittacidae (parrots, parakeets, cockatoos, macaws, etc.) and humans, avian chlamydiosis is called psittacosis. Historically, avian chlamydiosis has been called ornithosis in other avian species.

OCCURRENCE

Chlamydiosis occurs in many kinds and ages of birds. Most acute outbreaks are in young birds. Parrots, parakeets, and pigeons frequently are infected. In poultry, occasional outbreaks occur in turkeys. Chickens seldom are affected. Severe outbreaks occasionally have been reported in shorebirds and migratory birds. Important outbreaks of psittacosis occasionally occur in humans, usually following exposure in poultry processing plants. The presence of wild pigeons in some cities in North America, Europe, and Asia poses a major problem in efforts to control human chlamydiosis.

HISTORICAL INFORMATION

1. Psittacosis is a significant public health concern. From 1999 to 2003, 78 human cases of psittacosis were reported to the United States Center for Disease Control. In the United States most human cases were related to exposure of people to infected cage birds, especially parrots, or to sick turkeys in processing plants.

2. Some of the first recognized outbreaks of avian chlamydiosis were in pigeons. Important outbreaks soon were recognized in turkeys and ducks.

3. Interest in chlamydiosis waned somewhat with the introduction of antibiotics that control mortality in people with the infection.

4. During the last decade *Chlamydophila psittaci* outbreaks have occurred relatively infrequently among birds and humans. However, in 1974-1975 there were at least 11 outbreaks in turkey flocks, mostly in Texas. People were infected in at least seven of the outbreaks. These outbreaks again have focused attention on the public health aspects of avian chlamydiosis.

5. During the past few years chlamydiosis has been recognized as a common and major problem in imported and domestic exotic birds.

ETIOLOGY

1. In birds the etiologic agent is *Chlamydophila psittaci* (*Chlamydia psittaci*). Chlamydia is closely related to rickettsia.

2. *C. psittaci* can be grown in chicken embryos, cell culture, mice, and guinea pigs. Chlamydia form intracytoplasmic inclusion bodies in many kinds of cells, including macrophages, and inclusions can be demonstrated in stained smears [Fig. 1; Avian chlamydiosis; Cornell U] and histologic sections. All chlamydia are highly susceptible to tetracyclines. They are obligate intracellular organisms and cannot be grown in artificial media.

3. Isolates of *C. psittaci* vary greatly in pathogenicity. Concurrent infection, especially with *Salmonella*, sometimes enhances the pathogenicity of *C. psittaci*. Younger birds are more susceptible. Crowding or otherwise unfavorable environmental conditions and stress from shipping, racing, and handling contribute to the severity of the disease.

4. A common group-specific antigen is present in all chlamydia. Antibody to that antigen can be demonstrated in the sera of exposed or sick birds after an appropriate interval of time has elapsed.

EPIDEMIOLOGY

1. It is believed that wild carrier birds (and cage birds) transmit chlamydia to their nestlings and some surviving nestlings in turn become carriers. A delicate host-parasite relationship is established so that stressed carriers intermittently shed chlamydia in their secretions and excretions thus exposing other susceptible birds.

2. Chlamydiosis may become epizootic when large numbers of birds, including disseminating carriers, are in close contact. Transmission is primarily by inhalation of chlamydia in fecal dust but also can result from ingestion of *C. psittaci*.

3. It is suspected that wild birds may transmit chlamydia to poultry. Pigeons are strongly suspected of being important disseminators. Wild migratory birds such as gulls, egrets, and ducks are known to excrete chlamydia under certain conditions. Little is known of possible transmission of chlamydia between infected mammals and poultry but the chlamydia found in mammals are believed to be distinct from those in birds.

CLINICAL SIGNS

1. Mild outbreaks of avian chlamydiosis result in few signs and may go unrecognized. Alternatively, mild respiratory signs or diarrhea may be noted.

2. In more pathogenic outbreaks in turkeys there is depression, weakness, inappetence, and loss of weight and there may be nasal discharge and respiratory distress. Frequently there is marked, yellowish-green diarrhea. Similar signs occur in many other kinds of birds such as ducks, geese, and pigeons and may reflect systemic, pulmonary, or enteric involvement. A watery diarrhea is noted in ducks, geese, and pigeons. An unbalanced gait or transient paralysis has been reported in ducks, geese, and pigeons.

3. In pigeons, unilateral or bilateral conjunctivitis often occurs and should lead the diagnostician to suspect chlamydiosis. Other signs include depression, anorexia, diarrhea, or rales. The latter signs resemble those seen in many cage birds with psittacosis.

LESIONS

1. The basic lesions in chlamydiosis are characterized by proliferation of acute and chronic inflammatory cells and necrosis. These basic tissue responses may lead to pneumonia, airsacculitis, hepatitis, myo- and epicarditis, nephritis, peritonitis, and splenitis.

2. In turkeys the severity of lesions is in proportion to the pathogenicity of the strain of *C. psittaci*. In turkeys that succumb, there is wasting, vascular congestion, fibrinous pericarditis [Fig. 2; Avian chlamydiosis; Cornell U], fibrinous airsacculitis, and perhaps fibrinous perihepatitis. The lungs are congested and often there is a fibrinous pneumonia. The spleen is enlarged and congested, and may be the only lesion.

3. In pigeons there is conjunctivitis with encrusted, swollen eyelids. There may be hepatomegaly, airsacculitis, and enteritis.

4. In cage birds that succumb, the spleen frequently is enlarged and contains white foci. Often there is hepatomegaly with focal necrosis and yellowish discoloration, airsacculitis, pericarditis, and congestion of the intestinal tract.

DIAGNOSIS

1. A tentative diagnosis can be made on history, signs, and lesions and the demonstration of intracytoplasmic inclusions [Fig. 3: Avian chlamydiosis; Cornell U] on impression smears made from fresh exudates (monocytic cells) from the surface of the air sac (epithelial cells), spleen, liver, lung, serous surface, and pericardium. Where available, the fluorescent antibody technique may be useful in demonstrating chlamydia. A definitive diagnosis is usually obtained by isolation and identification of chlamydia organisms or by demonstration of a fourfold rise in antibody titer to chlamydial group antigen.

2. Every precaution should be taken to avoid self-infection. Psittacosis is highly contagious and many laboratory workers have been infected while handling infected birds or their tissues. Dead birds should be completely immersed in effective disinfectant solutions because the highly infectious nasal and fecal secretions dried on feathers can give rise to infectious dust in the air.

3. Chlamydiosis in turkeys must be differentiated carefully from *Mycoplasma gallisepticum* infection, influenza, aspergillosis, and cholera. Lesions in turkeys closely resemble those of *M. gallisepticum* infection. In general, diagnostic specimens from birds for chlamydia diagnosis should also be cultured for species of *Salmonella, Pasteurella, Mycoplasma*, and other bacteria as well as viruses.

4. Spleen, liver, lung, fibrinous exudate, air sacs, nasal washings and mucosa, fecal samples, or intestinal loops should be used for microbiologic or pathologic evaluation.

CONTROL

1. Because there is no effective vaccine against chlamydiosis, prevention of the disease in poultry depends upon avoidance of exposure. Facilities should be cleaned and disinfected prior to use. Flocks should be started and raised as units and no birds should be added to a started flock.

2. Poultry should not be exposed to other birds, animals, or their excreta. A preventive level of tetracycline can be added to poultry rations if exposure of the flock is suspected or anticipated. Poultry farm workers should not own any pet birds or poultry.

3. Federal law specifies how commercial birds imported for resale, research, breeding, or public display must be handled. During the quarantine period, all exotic birds of the psittacine family are treated with chlortetracycline as a precautionary measure against psittacosis. The quarantine period, designed to prevent the introduction of velogenic Newcastle disease, is 30 days and an effective treatment for chlamydiosis requires 45 days.

TREATMENT

Avian chlamydiosis is a reportable disease in most states and must be reported to the state veterinarian or other designated officials. Flocks should be treated only under supervision. Infected turkey flocks often have been treated with tetracyclines and slaughtered under supervision without human infection.

AVIAN TUBERCULOSIS

(Avian TB; TB)

DEFINITION

Avian tuberculosis is a slow-spreading, usually chronic, granulomatous infection of semimature or mature birds, characterized by progressive weight loss and, ultimately, by emaciation and death.

OCCURRENCE

Avian tuberculosis occurs in many kinds of birds, including poultry, game birds, cage birds, wild birds, and zoo birds. Most outbreaks are encountered in old backyard chickens. Avian tuberculosis also occurs in mammals, including swine, sheep, mink, cattle, and, rarely, humans. Among mammals, swine are more frequently infected. Avian tuberculosis is worldwide in distribution.

HISTORICAL INFORMATION

1. Tuberculosis in chickens was first recognized as a separate disease about 1884. Once identified, it soon was recognized in many countries. Avian tuberculosis eventually was found to be transmissible to certain other birds and mammals, especially swine, and was shown to sensitize cattle to tuberculin and johnin.

2. In the United States there once were many farm flocks of chickens and a farm flock often was kept for years. In old flocks avian tuberculosis was a very common disease. Later, farm flocks largely were replaced by large commercial flocks, which are sold after one laying cycle, a practice that greatly restricts the spread of avian tuberculosis.

3. Avian tuberculosis is seldom seen today in poultry species. However, there is a tendency toward the reestablishment of small farm flocks, which probably will be kept long enough for tuberculosis to develop.

4. Avian tuberculosis commonly infects swine, interferes with the eradication of bovine tuberculosis, and sometimes infects humans. However, there presently is no formal eradication program for this disease.

ETIOLOGY

1. The etiologic agent is *Mycobacterium avium*, a highly resistant, acid-fast bacillus. It resists heat, cold, water, dryness, pH changes, and many disinfectants and survives in soil for years.

2. Destruction by disinfection is impractical on most poultry farms. *M. avium* is distinct from the bacilli that cause human and bovine types of tuberculosis, although all three types share many similar characteristics

3. *M. avium* is present in large numbers in avian tubercles. Stained impression smears or histologic sections made from the centers of tubercles readily reveal the acid-fast rods. Their demonstration permits a strong presumptive diagnosis of tuberculosis.

EPIDEMIOLOGY

1. In chickens (and many other birds) small round to oval nodules (tubercles) develop as diverticuli along the intestine. These tubercles discharge viable tubercle bacilli into the intestine. Infectious feces and other excretions contaminate feed, water, litter, and soil and survive in the environment for months to years. Transmission of the organism is predominantly through ingestion of contaminated feed, water, litter, and soil.

2. During intermittent periods of bacteremia, tubercle bacilli spread from the intestine to most other organs and tissues. If bacteremic or dead infected poultry are cannibalized or consumed by other susceptible poultry or mammals (e.g., swine) the bacillus can be transmitted.

3. Other sources of dissemination of the bacilli include offal from infected chickens, excretions from wild birds (pigeons, sparrows, starlings, etc.), contaminated shoes or equipment, and the feces of infected mammals, especially swine.

4. With the increased popularity of exotic birds as pets, *M. avium* has become increasingly important as a potential zoonotic agent. Of particular concern is the disseminated disease that *M. avium* can cause in humans with immunosuppressive disease conditions (e.g., AIDS).

CLINICAL SIGNS

1. In chickens there is progressive wasting leading to emaciation, although the appetite is usually maintained. Diarrhea is common and there may be lameness in occasional birds. The skin of the face, wattles, and comb often appears pale.

2. The course in the individual bird and in the flock is prolonged. Total morbidity and total mortality are high, although both are spread over a period of many months and hence are misleading unless records are kept.

LESIONS

1. A bird with advanced tuberculosis is very light in weight and there is marked emaciation of the cadaver. Few other diseases result in such extreme emaciation. These features are unique enough that they should alert the prosector to the possibility of avian tuberculosis.

2. In chickens, gray to yellow nodules (tubercles) often are attached to and scattered along the periphery of the intestine [Fig. 1; Avian tuberculosis; Cornell U]. Smaller, discrete granulomas usually are present in parenchymatous organs, especially the liver and spleen. In advanced cases few organs are spared and tubercles often can be demonstrated in the bone marrow of the femur. Surprisingly, the lung often has few or no gross lesions.

DIAGNOSIS

1. A history of a chronic disease and persistent mortality in an old flock is suggestive of tuberculosis. Diagnosis often can be confirmed by the postmortem demonstration of typical gross lesions and the demonstration of acid-fast bacilli in impression smears or sections of tubercles. The tubercle bacillus should be cultured and identified.

2. Tuberculin testing was once utilized as a flock test and is still available but has fallen into disuse. Chickens are tested by inoculating avian tuberculin into one wattle. The other wattle is used as a control. Turkeys are tested by wing web inoculation. Skin tests are read in 48 hours. The tuberculin test has been used in other avian species with some success.

3. An enzyme-linked immunosorbent assay (ELISA) has been developed for the detection of mycobacterial antibodies in serum and this test has greater promise in the detection of avian tuberculosis in individual exotic birds and aviaries.

CONTROL

1. All chickens should be maintained in single-age groups. This will help control the disease by eliminating infected birds that might be disseminators. Thoroughly clean and disinfect buildings between broods. Maintain a high standard of sanitation at all times.

2. Young birds should be raised away from old birds on clean premises. Insofar as is possible, raise them in quarantine, thus avoiding exposure to all possible carriers including wild birds.

3. The use of tuberculin testing or ELISA serology monitoring may be of value in aviaries to identify and remove infected birds before widespread dissemination of the disease occurs.

TREATMENT

Treatment of avian tuberculosis is not recommended because the disease is infectious for humans. Furthermore, *M. avium* is resistant to many of the drugs used in treating other types of tuberculosis.

BORDETELLOSIS

(Turkey Coryza; Bordetella avium)

DEFINITION

Bordetellosis is an acute, persistent, contagious upper respiratory disease of turkeys characterized by ocular exudation and rhinitis in young turkeys and tracheitis in older turkeys caused by *Bordetella avium*.

OCCURRENCE

1. Bordetellosis occurs most commonly in turkeys 1-6 weeks of age. All ages of turkeys are susceptible, including breeders.

2. Outbreaks of the disease occur in most turkey-producing areas of the United States. Similar diseases have been reported from Canada, Germany, France, England, Italy, Israel, and South Africa.

3. Farms with continuous confinement production and multiage flocks have the greatest problems with bordetellosis. Bordetellosis occurs most commonly in the summer and fall.

4. *B. avium* has been recovered from chickens and occasionally other avian species. Presence of *B. avium* has been associated with increased severity of respiratory disease in broilers, especially when flocks are concurrently infected with infectious bronchitis virus, but its role as a primary pathogen in chickens is less obvious than in turkeys.

HISTORICAL INFORMATION

1. The term turkey coryza (TC) was first used in Canada in 1967 to describe a clinically distinct, acute respiratory disease of turkeys. TC was recognized in Iowa in 1971. Following greater awareness of TC, others recalled similar disease outbreaks that occurred in turkey- producing areas for at least the last three decades. As the number of turkeys being reared in confinement has increased, TC has been identified as an increasingly important respiratory disease and cause of economic loss.

2. The terms alcaligenes rhinotracheitis and turkey bordetellosis were introduced following preliminary identification of the causative agent as *Alcaligenes faecalis* or *Bordetella bronchiseptica*-like, respectively.

3. In Europe, a virus has been shown to cause a clinically similar disease, which has been named turkey rhinotracheitis. This disease is caused by a pneumovirus.

ETIOLOGY

1. *B. avium* has been identified as the cause of bordetellosis. *B. avium* can be distinguished from other species of *Bordetella* and nonfermenting, Gram-negative bacteria. Hemagglutination of guinea pig erythrocytes is associated with pathogenicity and is useful in distinguishing B. avium from *B. hinzii* (formally *B. avium*-like).

2. Strains vary greatly in virulence but virulence does not appear to be related to the presence or absence of plasmids.

3. *B. avium* produces hemagglutinin, and heat-stable and heat-labile toxins that can be neutralized by antiserum.

4. Presence of other infectious agents, notably Newcastle virus, other paramyxoviruses, *Mycoplasma gallisepticum, Pasteurella* and *Escherichia coli* increase the severity of bordetellosis.

EPIDEMIOLOGY

1. *B. avium* is susceptible to most disinfectants and environmental conditions, especially drying.

2. Older flocks serve as recovered carriers and are thought to be the most important source of infection for younger susceptible flocks on multiage farms. Transmission between flocks occurs as a result of human activity. There is no evidence of egg transmission.

3. Litter and contaminated water have been shown to be sources of infection. The organism has been found to persist for at least 6 months in moist litter but not dry litter. Contaminated water can remain in water lines and be a source of infection for new flocks.

4. Infection of flocks less than 10 days of age strongly suggests the environment as the source of the organism. Infection between 2 and 4 weeks may result either from the environment if poults had substantial maternal immunity or introduction from an outside source. Outbreaks in flocks over 4 weeks of age result from introduction of *B. avium*.

CLINICAL SIGNS

1. Onset is abrupt 4-7 days after exposure, with high morbidity and low mortality. Growth rate is decreased.

2. In young turkeys, initial clinical signs are clear, mucoid, nasal discharge and frothy ocular exudate accompanied by sneezing, "snicking", and flicking of the head. Activity is reduced and heat sources are sought out.

3. Exudates become progressively thicker with pasting of nostrils and matting of eyelids. The palpebral opening often assumes an almond shape. There are voice changes or loss in more severely affected birds, accompanied by tracheal rales. Birds show mouth breathing. The intermandibular tissue tends to balloon giving the profile a baggy appearance [Fig. 1; Bordetellosis; NCSU]. Poults may scratch at matted eyes causing trauma to eyelids. Dried exudate is commonly found on the upper wings and lower neck where the bird wipes off nasal-ocular exudates. Swollen infraorbital nasal sinuses are not typical of bordetellosis but are occasionally seen in a few birds.

4. Tracheal rales persist for several weeks after apparent recovery. Turkeys have been found to be culturally positive for at least 4 months after infection.

5. In uncomplicated outbreaks, mortality remains low. In bordetellosis outbreaks complicated by other respiratory disease agents mortality usually begins 10-14 days after onset of clinical signs and may be high (10-60%). *E. coli* is the most common cause of mortality. Flocks in poor environments, especially if ammonia levels are high, have higher mortality and greater production losses.

6. In older turkeys, nasal and ocular exudation does not occur. Typically the only sign observed in these birds is tracheal rales.

LESIONS

1. Catarrhal rhinitis, sinusitis, and tracheitis with hyperemia of the trachea are the only consistent lesions. In severely affected birds, there is distortion of tracheal rings in proximal segments of the trachea, which leads to narrowing of the tracheal lumen and retraction of the larynx. Cross sections through an affected segment will reveal the characteristic flattening or dorsal infolding of the trachea [Fig. 2; Bordetellosis; UC Davis]. Death occurs by suffocation from an obstructed trachea.

2. A variety of other lesions can be found in complicated outbreaks, depending upon the etiologic agents present.

3. *B. avium* attaches readily to ciliated epithelial cells of the upper respiratory tract [Fig. 3; Bordetellosis; UC Davis]. This leads to deciliation, altered mucus production, impairment of mucociliary clearance, and mucus accumulation. Inflammatory changes are not pronounced but are chronic, which leads to distortion of tracheal rings and hyperplastic bronchial-associated lymphoid tissue.

4. Infection with *B. avium* has been shown to interfere with vaccination for fowl cholera but the mechanism is unknown.

DIAGNOSIS

1. The bacterium is readily isolated from the trachea. Typical nonfermenter colonies occur on MacConkey agar in 48-72 hours.

2. If high populations of fermenting organisms are present on the plate, *B. avium* may be inhibited. This situation often occurs when the disease has been going on for several weeks. Early in the outbreak, almost pure, dense growths of *B. avium* are readily obtained.

3. *B. avium* should be looked for in any respiratory disease of turkeys even if another cause is identified because it is a significant predisposing factor to severe respiratory disease outbreaks.

4. A variety of serological tests including rapid plate agglutination, microagglutination, and enzyme-linked immunosorbent assay (ELISA) tests have been developed to detect antibodies to *B. avium*. The microagglutination and ELISA tests are commonly used for diagnostic purposes.

CONTROL

1. Clean out and disinfect the brooder house and all equipment between flocks. Make sure house and equipment are thoroughly dry. Depopulate problem farms.

2. Flush water lines with disinfectant between flocks.

3. Control traffic patterns. Traffic should always move from younger to older flocks without backtracking. Ideally only one person who has no other contact with poultry should care for a single brooder house (isolation brooding).

4. Prevent contact between wild birds and young turkeys.

5. An oil-emulsion bacterin is available for use in breeder hens. This will provide poults with maternal immunity for up to 4 weeks, the interval when infection generally results in a more severe disease.

6. A live vaccine prepared from a temperature-sensitive mutant of *B. avium* is available for use in poults. Two doses are recommended, the first given via spray cabinet in the hatchery with a booster administered through the drinking water at 2-3 weeks of age.

TREATMENT

Although *B. avium* is susceptible to most antibiotics on sensitivity tests, treatment with antibiotics is generally ineffective. This is thought to be due to failure of the antibiotic to reach effective levels in the respiratory tract where the organism is located. Aerosol administration of oxytetracycline is effective in reducing clinical signs during the treatment period but has little long-term benefit. The best management for an infected flock is to move the birds to range if possible. If not, increase ventilation, increase house temperature, and frequently stimulate the flock to move around encouraging them to eat and drink. Higher density "stress" rations and use of vitamins and electrolytes in water are useful adjuncts to general support of sick birds.

BOTULISM

(Limberneck; Western Duck Sickness)

DEFINITION

Botulism is an intoxication caused by ingestion of the toxins of *Clostridium botulinum*.

OCCURRENCE

1. In birds, botulism occurs frequently in captive pheasants and wild ducks and occasionally in chickens. Except for vultures, most birds are susceptible. Most outbreaks in birds occur in semimature or mature chicken flocks. Many mammals, including humans, are susceptible.

2. In waterfowl (especially wild ducks) occurrence is related to shallow water conditions in lakes with alkaline water and much decaying vegetation.

3. In some intense broiler rearing areas there is a recurring form of botulism on certain premises. Outbreaks occur in almost every new flock with a seasonal high incidence in the warmer months.

HISTORICAL INFORMATION

1. The first report of botulism in chickens was made in the United States in 1917. Within 25 years the disease had been reported frequently in chickens and in turkeys and waterfowl.

2. During the first half of this century, humans and chickens sometimes died from eating improperly canned foods containing the toxins of *C. botulinum*. Small farm flocks and home canning are now out of vogue and botulism is seldom seen in farm flocks or humans. However, botulism is still an important disease of wild waterfowl, especially ducks. Botulism seldom occurs in well-managed commercially raised poultry.

ETIOLOGY

1. Botulism is caused by ingestion of the preformed toxins of *C. botulinum* in feeds, foods, dead poultry, or toxin-containing maggots. Although *C. botulinum* itself is not pathogenic and is commonly found in the environment and in the intestinal tract, under ill-defined circumstances it colonizes the intestines, produces toxin and causes botulism.

2. The toxin of *C. botulinum* is extremely potent. The minimum lethal dose (MLD) for guinea pigs is 0.00012 mg/kg subcutaneously. (The MLD for cobra venom is 0.002 mg/kg). The toxin is relatively heat stable.

3. Based on the specific toxins produced and certain other criteria, there are eight types of *C. botulinum*. Type C is most common in poultry outbreaks although other types have occurred

4. Botulism should not be confused with pseudobotulism of chickens. Pseudobotulism closely resembles botulism except that affected birds almost invariably recover within 24 hours. Pseudobotulism is now considered to be a transient manifestation of Marek's disease.

EPIDEMIOLOGY

1. *C. botulinum* is ubiquitous in nature and commonly present in feeds. When ideal conditions for growth occur, large amounts of exotoxin may be formed. If adequate toxin is consumed, botulism will develop. Improperly sterilized canned fruits and vegetables, spoiled animal feeds, and decaying poultry carcasses can contain enough exotoxin to be highly lethal, even when taken in small amounts.

2. It is speculated that wild waterfowl contract botulism in the following ways:
    A. The toxin may be consumed in decaying vegetation in shallow, alkaline lakes as they dry up or are created by irrigation during the summer. Alternatively, it may be that the toxin is in larvae or crustaceans in the vegetation. Invertebrates killed by anaerobic conditions contain toxin from growth of *C. botulinum* within them and may be consumed by some waterfowl.

    B. Ducks that die from various causes may be invaded after death by *C. botulinum* normally present in their intestine. Toxins are formed in the cadavers. Ducks that feed on bits of the cadavers or on maggots from the cadavers may be poisoned.

3. A growing body of evidence suggests that *C. botulinum* type C can produce toxin within the intestinal tract of the live broiler chicken. This type of botulism has been termed toxico-infectious botulism.

CLINICAL SIGNS

Signs appear within a few hours to a few days. In chickens signs include drowsiness, weakness, and progressive loss of control of the legs, wings, and neck [Fig. 1; Botulism; Cornell U]. Paresis soon progresses to paralysis and the recumbent bird closes its eyes and appears to be in a deep coma. Fine tremors of muscles and feathers occur in some birds. Death may occur shortly or may be delayed for a few hours. Most visibly affected birds die.

LESIONS

Most birds with botulism are free of gross lesions. Rarely, in birds that have lived for some time, there may be mild enteritis. The upper digestive tract (especially the crop) may contain putrid ingesta or maggots but is usually empty.

DIAGNOSIS

1. In chickens and turkeys diagnosis is based largely on history, signs, the presence of putrid feed or maggots in the digestive tract, looseness of feathers (chickens only), and the absence of lesions. Finding a decaying cadaver upon which birds have been feeding may assist in diagnosis.

2. Saline gizzard or intestinal washings or blood serum from an affected bird can be tested for toxicity. Either can be injected into mice that have been inoculated with protective antiserum and into mice without antiserum. Results should clarify diagnosis.

3. A group of affected birds can be treated with polyvalent antitoxin. Recovery in a high percent of the birds tends to confirm a diagnosis of botulism. Unfortunately, commercial availability of antitoxins may be a problem.

CONTROL

1. The disease can be avoided by preventing access of poultry to any source of toxin. Sick and dead birds should be picked up regularly and frequently because they are a common source of toxin.

2. Type C toxoid can be used to immunize birds, although this is seldom done.

3. Wild ducks can be baited or frightened away from shallow lakes. Water sometimes can be pumped into shallow lakes to raise the water level and botulism may not occur.

4. On broiler farms where botulism is enzootic, the prophylactic use of selenium and antibiotics has been effective. They also aid in treatment of affected flocks.

TREATMENT

1. Antitoxin can be given to valuable affected birds. Results often are good although this will depend on the specificity of the antisera. Type C antitoxin is usually given. Polyvalent antisera (especially types A and C) are preferred but often difficult to obtain. It is important that the treated birds have access to fresh, clean, nonalkaline water.

2. Because toxico-infectious botulism has not been experimentally induced, treatment of this condition is based solely on the apparent response to therapy during field outbreaks of this condition. Treatment of flocks with sodium selenite and vitamins A, D, and E has been reported to reduce mortality. Treatments with bacitracin, streptomycin, chlortetracycline, and penicillin have also been reported to be efficacious.

COLIBACILLOSIS

(*Escherichia coli* Infections)

DEFINITION

Avian colibacillosis is an infectious disease of birds in which *Escherichia coli* is the primary or secondary pathogen. Infections include airsacculitis, cellulitis, omphalitis, peritonitis, salpingitis, synovitis, and coligranuloma.

OCCURRENCE

Colibacillosis occurs in all types and age groups of poultry as well as in other birds and many kinds of mammals. Most reported outbreaks in poultry have been in chickens, turkeys, and ducks. Many outbreaks occur in poultry raised under a low standard of sanitation, poor environmental conditions, or after a respiratory or immunosuppressive disease. Infection is more frequent in young than mature birds. Colibacillosis is common throughout the world.

HISTORICAL INFORMATION

Colibacillosis was first described in chickens in 1894. Since then, there have been numerous reports on colibacillosis in poultry and considerable research on the disease has been completed. Many investigators doubt that *E. coli* is a primary pathogen. Others are convinced that certain serotypes are primary pathogens and their opinion seems to prevail. Most investigators agree that *E. coli* frequently can be isolated from a variety of well-defined syndromes in poultry.

ETIOLOGY

The etiologic agent is *E. coli*. The O (somatic) antigen serotypes most commonly associated with disease outbreaks are O1, O2, O35, and O78. The K (capsular) antigens most commonly associated with virulence are K1 and K80. In the intestinal tract of normal poultry, nonpathogenic serotypes far outnumber pathogenic serotypes, with 10% to 15% of intestinal coliforms being potential pathogens.

EPIDEMIOLOGY

1. *E. coli* is present in the intestine of birds and mammals and is disseminated widely in feces. Birds are continuously exposed through contaminated feces, water, dust, and environment. Any time a bird's resistance to disease is impaired, pathogenic or facultative pathogenic strains may infect the bird. Sequestered *E. coli* in such sites as the intestine, nasal passages, air sacs, or reproductive tract may be a latent source of infection. Certain pathogenic serotypes may have the ability to infect a normal bird.

2. *E. coli* has been isolated from the eggs of normal hens. Its presence has been attributed to ovarian infection, oviduct infection, and to eggshell contamination followed by penetration. Chicks may hatch with a latent infection; however, active infection will typically only occur if some environmental stress or lesions initiates the disease process.

CLINICAL SIGNS AND LESIONS

A variety of syndromes from which *E. coli* has been isolated include:

1. Airsacculitis

   Respiratory signs occur and vary in severity. This syndrome may be associated with dusty litter, poor ventilation, stress, or adverse environmental conditions. It may accompany or follow vaccination or infection with mycoplasmas, infectious bronchitis virus, Newcastle disease virus, or laryngotracheitis virus.

Thickened air sacs and in severe cases, caseous exudate in the air sac is present [Fig. 1; Colibacillosis; UC Davis]. There often is an accompanying adhesive pericarditis and fibrinous perihepatitis. Airsacculitis occurs chiefly in 3-7-week-old broilers, probably peaking at 5-6 weeks.

2. Pericarditis

Most serotypes of *E. coli*, after a septicemia, cause a pericarditis [Fig. 2; Colibacillosis; UC Davis]. A myocarditis and an alteration of the pericardial sac (opaqueness) are usually associated with this and the epicardium becomes edematous. Pericarditis can also be caused by chlamydiosis.

3. Omphalitis and yolk sac infection

E. coli is often isolated in pure culture from organs or the yolk sac of recently hatched birds having depression, septicemia, and variable mortality. With omphalitis the navel is swollen and inflamed [Fig. 3; Colibacillosis; NCSU] and the bird feels wet. Abnormal yolk material and peritonitis is typically seen on necropsy of birds with an *E. coli* infection of the yolk sac.

A great variety of other organisms such as species of *Aerobacter, Proteus, Klebsiella, Pseudomonas, Salmonella, Bacillus, Staphylococcus,* enteric *Streptococcus*, and *Clostridia* are frequently isolated from yolk sacs of embryos and navels of chicks, most likely as mixed infections.

4. Coliform septicemia of ducks (new duck syndrome; duck septicemia)

E. coli, Salmonella, and *Pasteurella anatipestifer* produce respiratory signs, airsacculitis, pericarditis, perihepatitis, and peritonitis. In outbreaks of *P. anatipestifer*, involvement of the respiratory tract and a dry, thin transparent covering over visceral organs are present. In coliform septicemia (*E. coli*) usually a moist, granular to coagulative exudate of varying thickness is present on abdominal and thoracic viscera and surfaces of air sacs. The spleen and liver are swollen and dark with bile staining of the liver.

5. Acute septicemia

An acute septicemic disease caused by *E. coli* resembles fowl typhoid and fowl cholera. Birds are in good flesh and have full crops suggesting acuteness of the disease. This can occur in young or mature birds. There are sudden deaths, and variable morbidity and mortality. Parenchymatous organs are swollen with congested pectoral muscles. Livers are green in color and may have small necrotic foci. There may be petechial hemorrhages, pericarditis, or peritonitis. Acute systemic disease may also be caused by various *Pasteurella, Salmonella, Streptococci*, and other organisms.

6. Enteritis

Enteritis caused by E. coli is considered rare but pathogenic attaching effacing E. coli have been reported. Diarrhea and dehydration are noted on clinical examination. At necropsy there is enteritis, often with excessive fluid in the intestines. *E. coli* may be isolated from parenchymatous organs.

7. Salpingitis

This lesion may occur following entry of coliform bacteria from the vagina in laying hens. It is also likely to develop when the left greater abdominal air sac becomes infected by *E. coli*, causing a chronic salpingitis. Affected birds usually die during first 6 months postinfection and never lay. The oviduct is distended with exudate [Fig. 4; Colibacillosis; UC Davis] that may be cheesy and has a foul odor. No specific signs are noted but there may be an upright (penguin) posture.

8. Coligranuloma (Hjärre's disease)

Signs vary in this uncommon disease of chickens and turkeys. Nodules (granulomas) occur along the intestinal tract, and mesentery, and in the liver [Fig. 5; Colibacillosis; Cornell U]. The spleen is not

involved. The lesions resemble those of tuberculosis. The agent is a mucoid coliform, possibly not *E. coli*. Granulomas of the liver have many causes, which would include the anaerobic genera *Eubacterium* and *Bacteroides*.

9. Synovitis and osteoarthritis

   Affected birds are lame or recumbent. There is swelling of one or more tendon sheaths or joints. Synovitis and/or osteoarthritis are frequently a sequel to a systemic infection. With synovitis many birds will recover in about 1 week. Osteoarthritis is a more severe and chronic condition where the joint is inflamed and the associate bone has osteomyelitis. These severe chronic infections make birds unwilling or unable to walk and necropsy findings often include dehydration and emaciation. Synovitis-arthritis may also be caused by reovirus, or species of Mycoplasma*, Staphylococci*, and *Salmonella*.

10. Panophthalmitis and meningitis

    Occasional birds have a hypopyon and/or hyphema, usually in one eye, which is blind. Likewise, meningitis is a rare sequelae to *E. coli* septicemia.

11. Cellulitis (Infectious process)

    This is an *E. coli*-related condition occurring with apparently increasing prevalence in broiler chicken flocks in the United States, some European countries, and Canada. It is recognized primarily as an inspection finding at slaughter, with no abnormality having been noted in live birds. The USDA Food Safety and Inspection Service designates cellulitis as "infectious process" or "IP". Gross lesions include variable yellowing and dimpling of the skin ventral to the vent and over the ventrocaudal aspect of the breast extending in severe cases over the thighs [Fig. 6; Colibacillosis; UC Davis]. On incising the skin a leathery grayish-yellow membrane of inspissated exudate [Fig. 7; Colibacillosis; UC Davis] is noted in the subcutis. Frequently this sheet of exudate can be removed through the incision. Histologically there is extensive deep dermatitis in the affected areas involving both dermis and subcutis. The inflammatory reaction includes edema and heterophil infiltration in active areas, whereas there is accumulation of a walled-off causative sheet of exudate surrounded by a zone of giant cells in more chronic areas of involvement. Coccobacillary bacteria can be seen in microcolonies within the exudate and E. coli is recovered quite consistently on culture. One recent study also demonstrated *Streptococcus dysgalactiae* in the exudate. This condition may affect up to 5% of entire flocks at slaughter resulting in extensive trim-out, downgrading, or whole-carcass condemnation. Total population prevalence ranges from 0.12 to 0.16%. The pathogenesis of cellulitis has yet to be determined but there is a correlation with certain broiler breeds, poor feathering, sex (males more susceptible), skin scratches, increased stocking density, litter type and diet.

DIAGNOSIS

Diagnosis of primary colibacillosis is based on the isolation and typing of a coliform into one of the serotypes recognized as pathogens. Diagnosis based merely on the isolation of *E. coli* is of questionable validity. The possibility of other infections (viruses, bacteria, fungi, chlamydia, and mycoplasmas) should have been eliminated through culture or other means. When *E. coli* is isolated secondary to some other primary disease, it should be diagnosed as secondary colibacillosis.

CONTROL

1. Measures should be taken to minimize eggshell contamination of newly laid eggs. Eggs should be disinfected on the farm prior to storage and should be stored under ideal conditions. Scrupulous hatchery sanitation, disinfection, and/or fumigation procedures should be practiced.

2. A vigorous sanitation program should be followed in raising poultry.

3. Insofar as is possible, all disease, parasitisms, and other stresses on a flock should be minimized. Dust should be controlled.

4. Only feeds free of fecal contaminations should be fed to poultry. Pelleted feeds are more likely to be free of contamination.

TREATMENT

Many different antibiotics and drugs have been utilized for treatment. These have included tetracyclines, neomycin, sulfa drugs and others. Antibiotic sensitivity testing is advisable where applicable. Treatment is usually effective if given early.

ERYSIPELAS

DEFINITION

Erysipelas is an acute septicemic disease occurring most commonly in older male turkeys, characterized by serosal, cutaneous, and muscular hemorrhages and splenomegaly. Chronic erysipelas (polyarthritis, endocarditis) occurs occasionally, usually after acute outbreaks.

OCCURRENCE

Erysipelas is of primary importance in turkeys although outbreaks sometimes occur in geese, ducks, pheasants, other game birds, wild birds, and (rarely) in chickens. Sporadic cases have been reported in many wild birds. Erysipelas also occurs in swine, sheep, sea mammals, fish, and many wild animals. In humans, erysipelas is caused by streptococci, whereas *Erysipelothrix* causes a localized inflammation designated erysipeloid. In turkeys erysipelas usually occurs in toms approaching market weight; it seldom occurs in turkeys less than 10 weeks old, although no age or sex resistance has been found experimentally. The peak incidence in tom turkeys roughly coincides with puberty and the disease occurs occasionally in hen turkeys following artificial insemination. Because the bacterium is ubiquitous in nature, erysipelas probably affects poultry and birds throughout the world.

HISTORICAL INFORMATION

The economic significance of erysipelas in turkeys was pointed out in 1939 and the disease was soon recognized as a major disease of turkeys. In the United States, recognition of erysipelas in turkeys as an important disease roughly paralleled the recognition of erysipelas as an important disease of swine. The disease is not common today because most turkeys are raised in confinement, reducing exposure to the organism.

ETIOLOGY

The etiologic agent is *Erysipelothrix rhusiopathiae*. It is a Gram-positive, slender, slightly bent, pleomorphoric rod. Filamentous, beaded forms that tend to decolonize easily are often seen in cultures. It grows well on enriched media especially when incubated in a 5-10% carbon dioxide atmosphere (candle jar). Colonies tend to be quite small and grow slowly, making them easily overgrown by faster growing bacteria. Selective and enrichment media assist in recovering the organism. Production of hydrogen sulfide in iron-containing media is a useful characteristic for presumptive identification of isolates. The organism is quite resistant to many environmental factors and disinfectants and remains viable in favorable (alkaline) soils for months to years.

EPIDEMIOLOGY

1. The organism is shed in the feces of some recovered turkeys for up to 41 days. It also is shed in the feces of infected swine and lambs. Because turkeys can be infected experimentally by the oral route, it is believed that oral exposure is a common route of natural infection. Infection may occur after ingestion of contaminated soil, water, fish meal, meat meal, or following cannibalism of infectious live or dead birds.

2. The organism can also infect turkeys through breaks in the skin or mucous membranes. Cutaneous wounds commonly occur in tom turkeys, which are inclined to fight as they reach puberty. A typical maneuver during fighting is to grab the snood of the opponent and shake him violently causing considerable trauma to this skin appendage. The snood is considered to be a prime site of infection with *Erysipelothrix* for this reason.

3. Stress often precedes outbreaks of erysipelas. A major outbreak of erysipelas occurred in a large goose flock after the birds were plucked. Other stressors include such things as poor sanitation, bad weather, vaccinations, changes in the ration, etc. The role of vectors, if any, is unknown.

4. Important outbreaks of erysipelas have been reported in hen turkeys following artificial insemination. Presumably, infectious semen comes from carriers that shed the organism in their semen.

5. Injecting numerous birds in a flock with the same needle may spread the organism if septicemic birds are present.

6. *Erysipelothrix* can persist for years in the soil. Outbreaks occur most commonly during the late fall and winter following periods of cold, wet weather. Repeated reoccurrence on a farm is common.

7. Feeding dead turkeys to swine has resulted in outbreaks of erysipelas in the pigs. There is clinical evidence indicating that people who move between swine herds and turkey flocks can spread the organism from the pigs to turkeys.

CLINICAL SIGNS

1. The onset of erysipelas in turkeys is usually sudden with a few birds being found dead. At that time careful examinations of the flock often reveals other turkeys that squat on the floor, and appear sleepy and depressed. They can be aroused but have an unsteady gait when forced to move. Occasional birds may exhibit respiratory signs or have yellow-green diarrhea.

2. Within a few days morbidity increases markedly. The course of the disease is short, often only a few hours or overnight and most visibly sick birds die.

3. Occasionally, infected turkeys have a swollen snood or irregular, dark red skin, and demarcated lesions on their dewlap, face, or head [Fig. 1; Erysipelas; NCSU]. In recently inseminated, infected hens there may be perineal congestion and hemorrhage.

4. Crippled birds with swollen joints are seen in chronic infections. These often occur after an acute outbreak.

LESIONS

1. The lesions are those of a septicemia. The carcass is congested and parenchymatous organs (liver, kidney, spleen) are swollen. Splenomegaly is often marked [Fig. 2; Erysipelas; Cornell U].

2. Petechial or suffusion hemorrhages often occur in heavy muscle masses, in pericardial fat, on the epicardium, under serous membranes, and in mucous membranes. Hemorrhages vary greatly.

3. There usually is a marked catarrhal enteritis, often more apparent in the duodenum, with excess mucus in the gut.

4. Skin lesions occur occasionally and are more apparent on the face, head, and neck. Inseminated hens may have peritonitis, perineal congestion, and hemorrhage.

5. Purulent arthritis, often in more than one joint, and vegetative valvular endocarditis are seen in chronic cases.

DIAGNOSIS

1. History, signs, and lesions may suggest erysipelas but the etiologic agent should be isolated and identified for confirmation. Erysipelas must be differentiated from fowl cholera. Helpful necropsy findings are the markedly enlarged spleen seen in erysipelas but not fowl cholera, and the pneumonia that often occurs in fowl cholera but not erysipelas. Erysipelas also should be differentiated from acute colisepticemia, salmonellosis, streptococcosis, chlamydiosis, and virulent Newcastle disease. Erysipelas can occur concurrently with other diseases including fowl cholera, chlamydiosis, and internal parasitism.

2. Gram-stained impression smears made from the cut surface of liver, spleen, and bone marrow will reveal Gram-positive, slightly bent, thin bacilli. If available, the fluorescent antibody technique can be used to identify the organism in smears or tissue sections.

CONTROL

1. Poultry should be raised separately from older turkeys, which may be carriers. The poults should be started and raised as a flock and no birds should be added. Contact should be prevented between the turkeys and animal carriers, especially sheep and swine. Raise turkeys in houses that were cleaned and disinfected and on ranges without a history of outbreaks of erysipelas.

2. If erysipelas is enzootic in the area, turkeys should be vaccinated with bacterin when 8-12 weeks old. Immunity is enhanced if bacterin inoculation is repeated at least once. Breeders should be revaccinated prior to onset of egg production. Use of a live oral erysipelas vaccine for swine given by injection or orally has been found to confer some protection against experimentally challenged turkeys.

3. Semen for artificial insemination should come from tom turkeys with no history of erysipelas infection.

4. Formerly, desnooding was a common hatchery practice, especially for males. This practice is now being discontinued because outbreaks of erysipelas are not as common and oral infection with acute systemic disease now appears to be more common than skin infection.

5. Selection for genetic resistance may be possible. Strains of turkeys selected for rapid growth have been found to be more susceptible to naturally occurring erysipelas than unselected lines or lines selected for high egg production.

TREATMENT

1. Penicillin and erysipelas bacterin often are inoculated simultaneously into all birds of an infected flock. Sick birds should be inoculated with a fast-acting form of penicillin. It may be necessary to repeat the inoculation. A longer acting form of penicillin can be used in birds not obviously sick.

2. Water-soluble penicillin used at a rate of 1.5 million units/gal is effective, but the disease often resumes after treatment is stopped. Depending on market conditions the cost of treatment may be greater than the value of the commercial birds.

FOWL CHOLERA

(Cholera; Pasteurellosis)

DEFINITION

Fowl cholera is an infectious disease of poultry, waterfowl, and many other birds, usually appearing in poultry as an acute septicemic disease with high morbidity and mortality. A chronic, localized form occurs in poultry and may follow the acute form, or may occur independently.

OCCURRENCE

Fowl cholera is a disease of many species of birds, including chickens, turkeys, geese, ducks, quail, canaries, and many wild and zoo birds. Perhaps all birds are susceptible under appropriate conditions. In poultry, most outbreaks occur in semimature or mature birds, although there are exceptions. The disease occurs more frequently in turkeys than in chickens. The disease occurs frequently in domesticated waterfowl and often causes extensive losses among wild waterfowl. Geese are highly susceptible. Fowl cholera is more likely to occur in birds that are stressed by such things as poor sanitation, parasitism, malnutrition, and other diseases. Fowl cholera occurs worldwide and is a relatively common disease. There is no relationship between cholera in humans and fowl cholera.

HISTORICAL INFORMATION

Fowl cholera has been recognized as a disease of poultry for more than 200 years. About 100 years ago, Pasteur isolated the organism and used it in one of the first vaccines. In the United States, Dr. Salmon studied the disease as early as 1880. Fowl cholera was one of four major livestock diseases that stimulated formation of the Veterinary Division of the United States Department of Agriculture. Although fowl cholera has been recognized and studied for almost 200 years, it still remains an important disease of poultry.

ETIOLOGY

1. The etiologic agent is *Pasteurella multocida*, a Gram-negative, bipolar-staining bacillus that grows readily on blood agar but not on MacConkey agar. Virulence among isolates is highly variable. Encapsulated strains are usually highly virulent; unencapsulated isolates are typically of low virulence.

2. The organism varies greatly in its antigenic makeup, a characteristic responsible for difficulties in producing effective bacterins and vaccines. The gel diffusion precipitin test has been used to describe 16 P. multocida serotypes, all of which have been isolated from avian hosts. Serotypes 1, 3, and 3X4 are most commonly isolated from poultry outbreaks.

3. *P. multocida* is easily destroyed by many disinfectants and by sunlight, heat, and drying. However, the organism persists for months in decaying carcasses and moist soil.

EPIDEMIOLOGY

1. Poultry flocks that have recovered from an outbreak of fowl cholera will remain carriers of *P. multocida* and spread the disease to susceptible flocks. These carriers harbor the organism in the choanal cleft and contaminate feed, water, and the environment with oral fluids. Likewise, wild birds may carry the organism and introduce it into the poultry flock if appropriate biosecurity practices are not followed.

2. Several mammalian species are carriers of *P. multocida* and may introduce the organism to poultry flocks. Swine and raccoons have been shown to be carriers of *P. multocida* and those isolated have been shown to be pathogenic in poultry.

3. Birds that die of septicemic cholera have the agent in most of their tissues. Cannibalism of sick or dead birds is an important method of dissemination of the disease.

4. Resistance to cholera is correlated to humoral immunity. Immunosuppression increases susceptibility.

5. *P. multocida* is resistant enough to be readily spread on contaminated crates, feed bags, shoes, equipment, etc.

CLINICAL SIGNS

1. With acute cholera, sudden unexpected deaths occur in the flock. Mortality often increases rapidly. Laying chickens may be found dead on the nest. Geese have been reported to just drop dead while walking across a barnyard. Poisoning is often initially suspected in outbreaks of acute cholera.

2. Sick birds show anorexia, depression, cyanosis, rales, nasal and oral discharge of mucus, and white watery or green mucoid diarrhea. The course of illness is short and often followed by death. Affected chickens often conceal themselves under equipment.

3. Chronic fowl cholera is most common in chickens. Often there is swelling of a joint, wattle [Fig. 1; Fowl Cholera; AAAP], foot pad, or tendon sheath. Exudate, often cheesy, may accumulate in a conjunctival sac or infraorbital sinus. There may be torticollis in a few birds [Fig. 2; Fowl Cholera; AAAP].

4. Abscesses of the infraorbital sinuses and middle ear infection resulting in torticollis, often occur in turkeys with chronic cholera.

5. In turkey breeders there is a drop in egg production and increased mortality following handling of hens during insemination. Affected toms produce thin, watery, poor quality semen.

LESIONS

1. Lesions may be absent if the disease is very acute. Usually there are petechial and ecchymotic hemorrhages at a few sites, for example, on the heart, under serous membranes, in mucous membranes, on the gizzard, or in abdominal fat. There is often a generalized hyperemia of the upper intestine. Acute lesions develop as a result of disseminated intravascular coagulation. In layers and breeder hens, free yolk in the peritoneal cavity, acute oophoritis with regressing follicles, and acute diffuse peritonitis are frequently seen. These lesions can accompany many other acute diseases.

2. In acute cases of cholera there often is enlargement of the liver. If the birds live a few days, there may be a few or many small necrotic foci in the liver [Fig. 3; Fowl Cholera; AAAP]. Consolidation of lungs is a common finding in affected turkeys [Fig. 4; Fowl Cholera; AAAP], [Fig. 5; Fowl Cholera; AAAP]. With time, these lesions become sequestered as necrotic areas in the lungs and these lung lesions often are extensive.

3. In chronic cases there may be localized inflammatory lesions. These often involve a joint, tendon sheath [Fig. 6; Fowl Cholera; AAAP], wattle, conjunctival sac, infraorbital sinus, the nasal turbinates, the middle ear, or cranial bones at the base of the skull. Caseous exudate in a localized lesion [Fig. 7; Fowl Cholera; AAAP] should arouse suspicion of cholera.

DIAGNOSIS

1. At necropsy, Gram-stained impression smears of liver or heart blood from septicemic cases often reveal bipolar-stained, Gram-negative rods suggestive of cholera. Use of blood stains or methylene blue readily demonstrates the bipolar morphology of the organism.

2. Although the history, signs, and lesions may strongly suggest fowl cholera, *P. multocida* should be isolated and identified for confirmation. Isolates should be tested for antibiotic susceptibility because of widespread

resistance and should be serotyped, especially if routine treatment and vaccination procedures appear ineffective.

3. Cholera must be differentiated carefully from erysipelas and acute colibacillosis in turkeys and other birds that are susceptible to both diseases. Erysipelas is caused by a Gram-positive rod. Cholera can be differentiated readily from most septicemic and viremic diseases of poultry by the isolation of *P. multocida*.

4. Cholera always should be suspected if there are epizootic losses in domesticated or wild waterfowl.

5. Related organisms can cause cholera like diseases or complicate other diseases. These include *Pasteurella gallinarum, P. haemolytica, P. anatipestifer, Moraxella osloensis*, and *Yersinia pseudotuberculosis.*

6. Several serological tests have been developed. Currently an enzyme-linked immunosorbent assay (ELISA) is commercially available and widely used. Serology is used primarily to evaluate efficacy of vaccination rather than for diagnosis of a disease outbreak.

CONTROL

1. *P. multocida* is not transmitted through the egg. Obtain clean birds and raise them in quarantine on disease-free premises and away from all birds and mammals that might be carriers. Never add birds to the flock as they may be carriers. Avoid stresses, insofar as is possible, and practice a high standard of sanitation.

2. Pick up and destroy all sick or dead birds before they can be cannibalized. Birds with cholera are teeming with *P. multocida* and are important in the transmission of the agent. Dispose of carcasses by burying or burning to prevent them from being fed on by scavengers (including dogs and cats).

3. Although bacterins are not always effective, in many instances they do a good job of immunizing birds, especially if they can be repeated at least once. They often are given when birds are about 8 and 12 weeks old. Bacterins do not provide good cross-protection between serotypes. Oil-emulsion bacterins are used to immunize breeders prior to production. They can cause serious drops in egg production if given to laying birds.

4. Live vaccines are given via wing web inoculation to chickens and via drinking water or wing web inoculation to turkeys. In the United States live vaccines are based on the Clemson University (CU) strain of *P. multocida*. This is a naturally occurring low-virulent organism. Since its introduction as a commercial product, two milder mutants of the original CU strain have been produced: PM-1 and M-9 strains. They frequently are given to turkeys at 2-6- week intervals beginning at 6-7 weeks of age in the drinking water. Some turkey breeders are vaccinated via the wing web. Layers and breeders are inoculated by wing web stick at 10-11 weeks of age, and revaccinated in 6-8 weeks. Fowl pox vaccine may be given concurrently in the opposite wing. The live vaccines have been shown to be safe but vaccine reaction problems can occur in the field, presumably because of immunosuppression, concurrent diseases, breed sensitivity, late vaccination, or management stress such as intentional feed restriction. Parenteral administration may result in a localized lesion, or, more seriously, arthritis. Live vaccines confer better resistance than killed bacterins and offer a broad spectrum of protection against most serotypes.

5. Following an outbreak, depopulation should be considered because many surviving birds become carriers and transmit *P. multocida*. Following depopulation, the premises and equipment should be thoroughly cleaned and disinfected and, if possible, kept free of poultry for a few weeks.

6. Continuous medication programs have been used but are generally more costly than a vaccination program.

7. Reduce rodents, scavengers, and predators in the farm environment and limit their contact with flocks.

8. Differing susceptibilities among genetic lines of turkeys have been shown, suggesting that selection for resistance to fowl cholera may be possible.

TREATMENT

1. Many sulfa drugs and antibiotics will lower the mortality from cholera but mortality may resume when treatment is discontinued. Most medications are given in the feed or water. Sulfaquinoxaline is one of the better treatments but will depress egg production in layers and may throw them completely out of production. Care should be taken to use only those products approved by the Food and Drug Administration for the class of poultry being treated. Drugs and antibiotics in common use include:

Sulfadimethoxine	Tetracyclines
Sulfaquinoxaline	Erythromycin
Sulfamethazine	Streptomycin
Penicillin	

2. Moving an infected flock to clean premises or markedly improving sanitation during an outbreak may slow the course of cholera. Use of live vaccine during the early course of an outbreak may be effective.

3. If cholera cannot be controlled, it may be necessary to market the flock early. Be sure to adhere to regulations relating to withdrawal of medication.

GANGRENOUS DERMATITIS

(Necrotic Dermatitis)

DEFINITION

Gangrenous dermatitis is a disease of young growing chickens characterized by necrotic areas of the skin and by a severe, underlying, infectious cellulitis.

OCCURRENCE

Most outbreaks have occurred in chickens 4-16 weeks old. Young birds of this age group may be poorly feathered. Outbreaks often occur in excessively warm, humid houses.

HISTORICAL INFORMATION

Gangrenous dermatitis was reported first in 1930 although most outbreaks have been reported since 1963. Some of the more recent reports have suggested that affected flocks may be immunologically deficient.

ETIOLOGY

It appears that primary skin lesions are secondarily invaded by various bacteria including *Clostridia sp*. (especially *C. septicum*), *Staphylococcus* sp. and *Escherichia coli*.

EPIZOOTIOLOGY

1. Cutaneous wounds probably occur initially as a result of cannibalism, mechanical trauma (from mechanical feeders, etc.), or other trauma. Bacteria invade the traumatized skin and underlying tissue and their toxins or metabolites cause cellulitis. Septicemia and toxemia follow, leading to death.

2. Increased susceptibility of affected flocks to infection is an important factor in the pathogenesis. This increased susceptibility is commonly related to immunosuppression secondary to infectious bursal disease or chicken infectious anemia virus.

3. Other factors that may enhance susceptibility include aflatoxicosis, nutritional insufficiency or imbalance, or poor sanitation.

CLINICAL SIGNS

A sudden, sharp increase in mortality is often the first indication of onset. When sick birds are observed, they are depressed, and sometimes prostrate or lame. Skin lesions, often crepitant, are apparent in live or dead birds. The course of the illness is often less than 24 hours. Mortality varies but can be quite high.

LESIONS

1. There are scattered patches of darkened, gangrenous skin, often with cutaneous sloughing or feather loss in affected areas. Marked emphysematous or serosanguineous cellulitis underlies some skin lesions, especially with clostridial infections.

2. Swelling and infarction may be apparent in parenchymatous organs. There may be foci of necrosis in the liver.

3. Severe atrophy of the bursa of Fabricius is usually present.

GANGRENOUS DERMATITIS

DIAGNOSIS

A tentative diagnosis often can be made on the basis of history and gross lesions. For confirmation, smears or histologic sections of affected tissues will reveal bacteria. Bacteria can be cultured from the area of cellulitis.

CONTROL

1. The cause of skin trauma should be found and eliminated. If cannibalism is a cause, it may be necessary to trim the beaks or improve the quality of previous beak trimming. Mechanical feeders should be examined carefully as a source of possible trauma.

2. Vaccinate the breeder flock for infectious bursal disease to prevent or reduce possible immunosuppression in the progeny.

3. Insofar as is possible, eliminate all stresses on the birds (e.g., parasitism, malnutrition, coccidiosis, etc.).

4. Improve sanitation in the house, particularly that of the feeders, waterers, and litter. A thorough cleaning and disinfection of the house may be helpful. If litter in the house stays wet, improve moisture control. Repeat problem houses may benefit from salting the floor at cleanout. Cheap grade feed salt is used on the soil at a rate of 60-63 lb/1,000 ft^2.

5. Broad-spectrum antibiotics (e.g., penicillin, erythromycin, and tetracyclines) can be added to the ration of the flock and will reduce mortality.

TREATMENT

In addition to adding broad-spectrum antibiotics to the ration, valuable birds can be treated individually with penicillin, tetracyclines, or other fast-acting antibiotics.

INFECTIOUS CORYZA

(Coryza)

DEFINITION

An acute or subacute disease of chickens, pheasants, and guinea fowl characterized by conjunctivitis, oculonasal discharge, swelling of infraorbital sinuses, edema of the face, sneezing, and sometimes by infection of the lower respiratory tract. Prolonged outbreaks are now believed to be outbreaks complicated by other diseases, especially *Mycoplasma gallisepticum* infection (chronic respiratory disease).

OCCURRENCE

Chickens are primarily affected, although the disease has been reported in pheasants and guinea fowl. All ages of chickens are susceptible although most natural outbreaks occur in chickens that are half grown or older. The disease is seen more frequently on chicken farms where facilities are used so intensively that they are never free of chickens. The disease has a worldwide distribution. Infectious coryza does not occur in turkeys and should not be confused with turkey coryza caused by *Bordetella avium*.

HISTORICAL INFORMATION

Infectious coryza was believed to be a separate disease of chickens as early as 1920 but this was not confirmed until 10-15 years later. The incidence of coryza has varied markedly. Presently coryza is a disease of considerable importance, especially on multiage egg production complexes.

ETIOLOGY

1. The etiologic agent, *Avibacterium paragallinarum* (formerly *Hemophilus paragallinarum* and *H. gallinarum*) is a Gram-negative, bipolar-staining, nonmotile rod with a tendency toward filament formation. *A. paragallinarum* requires V-factor (nicotinamide adenine dinucleotide), which is available in certain enriched medium (i.e. chocolate agar). It grows on blood agar (with a *Staphylococcus aureus* nurse colony) as dewdrop-like satellite colonies in a microaerophilic environment. V-factor independent isolates have been described from South Africa.

2. *A. paragallinarum* is not a very resistant organism and will persist outside of the host for only a few days. It is easily destroyed by many disinfectants and by environmental factors. The organism is susceptible *in vitro* to many chemicals and antibiotics, including spectinomycin, neomycin, novobiocin, and tetracycline.

3. *A. paragallinarum* is present in sinus exudate and is easily demonstrated in stained smears.

4. There are several strain classification schemes. The Page scheme recognizes three antigenic types (A, B, C) of *A. paragallinarum*, although all types share certain antigens. Hemagglutinins produced by the organism appear to be important antigens capable of inducing protection against infectious coryza. Bacterins are available that allow limited protection to laying chickens. Bacterins also have a positive influence of the success of chemotherapy.

EPIDEMIOLOGY

Chronically ill or apparently healthy carrier birds are the major reservoirs of infection and readily transmit the agent to susceptible chickens. Transmission probably occurs by inhalation of infectious aerosol coughed into the air or through ingestion of contaminated feed or water. The etiologic agent can be transmitted by fomites, although it soon perishes outside of the host. Recovered birds are frequently carriers.

CLINICAL SIGNS

1. Usually there is a rapid onset and morbidity is high in the flock. Feed consumption, egg production or growth are reduced noticeably.

2. There is oculonasal discharge, conjunctivitis with some adherence of eyelids, edema of the face [Fig. 1; Coryza; AAAP] (occasionally of the wattles [Fig. 2; Coryza; AAAP]), respiratory noises, and, perhaps, diarrhea. Later, some of the birds may have swollen infraorbital sinuses and/or exudate in the conjunctival sac. There is considerable variation in the severity and length of course in flock outbreaks.

3. Respiratory signs usually persist for only a few weeks. Persistence of signs occurs when complicated by fowl pox, *M. gallisepticum*, infectious bronchitis, *Pasteurella sp*., or infectious laryngotracheitis and unthrifty birds will become apparent. Persistence of signs was once attributed entirely to strains of *A. paragallinarum* of low virulence.

GROSS LESIONS

1. There is catarrhal inflammation of nasal passages and sinuses and nasal discharge often is apparent [Fig. 3; Coryza; AAAP]. One or both infraorbital sinuses may be distended with exudate (similar distension can occur with localized fowl cholera, pox, vitamin A deficiency, and staphylococcal infection).

2. There is conjunctivitis, frequently with adherence of the eyelids or with accumulation of cheesy exudate in the conjunctival sac.

3. There often is edema of the face and, occasionally, of the wattles. In complicated cases there may be tracheitis, pneumonia, or airsacculitis.

DIAGNOSIS

1. Typical history, signs, and lesions are suggestive of coryza, although other respiratory diseases of chickens must be differentiated.

2. A smear of sinus exudate should be made and Gram stained. It should reveal Gram-negative, bipolar-staining rods with a tendency toward filament formation and pleomorphism.

3. Aseptically collect sinus exudate and swab it on blood agar. On the same plate then make an S-shaped streak of *S. aureus* (use a strain that excretes V-factor), which will serve as a feeder colony. Incubate the culture in a candle jar. Tiny dewdrop satellite colonies [Fig.4; Coryza; AAAP] of *A. paragallinarum* will grow adjacent to the feeder colony. The organism can be further identified by biochemical means or by a PCR test specific for *A. paragallinarum*.

4. A nonpathogenic species, *Avibacterium avium*, (*Hemophilus avium*) may be cultured from the sinus, either alone or with *A. paragallinarum*. *A. paragallinarum* is catalase negative and the nonpathogenic species is catalase positive.

5. Put a small amount of sinus exudate in the infraorbital sinus of a few young susceptible chickens. Typical signs and lesions develop in 3-5 days (rarely less).

6. Hemagglutination inhibition and immunodiffusion tests can be used to detect *H. paragallinarum* antibodies in serum. Both tests are serotype specific.

CONTROL

1. Depopulate, if necessary, to eliminate all carrier birds. Leave the premises vacant for a few days after thorough cleaning and disinfection. Then restock with 1-day-old or other coryza-free chickens. Raise them, insofar as is possible, in quarantine.

2. Commercial bacterins can be used to immunize chickens and protect only for the serotype included in the vaccine. All pullets to be housed on multiage infected farms should receive two injections of the bacterin at 3-week intervals prior to being placed on the farm. The first vaccination should be given after the birds reach 14 weeks of age.

4. Controlled exposure of pullets prior to the onset of lay is sometimes used but has not been widely accepted by the poultry industry. Vaccination with bacterin 2 weeks prior to controlled exposure is sometimes used to improve immunity, reduce the severity of infection, and provide cross-protection against other serotypes.

TREATMENT

Various sulfonamides and antibiotics have been used, usually in feed or drinking water. Birds usually respond to treatment but relapses may occur when treatment is discontinued. Erythromycin and oxytetracycline are commonly used in layer operations.

MYCOPLASMOSIS

PREFACE

Mycoplasmas belong to the Order *Mycoplasmatales*, are the smallest prokaryotic organism (DNA content) cultivatable on artificial medium and are completely devoid of a cell wall. The absence of a cell wall accounts for their pleomorphic shape, fried-egg colony appearance and their resistance to penicillin-like antibiotics. Over twenty five species have been identified from avian hosts although several isolates are unidentifiable. Generally, mycoplasmas have a narrow host range. Four mycoplasma species are considered pathogenic to commercial poultry; *Mycoplasma gallisepticum, M. synoviae, M. meleagridis* and *M. iowae*. Pathogenic species generally infect the respiratory system but other systems maybe involved. Transmission is generally by direct contact although egg transmission, carrier birds and fomites are of importance. The cultivation of *Mycoplasma sp*. is somewhat demanding and requires specialized media containing 10 – 15% serum, yeast-derived components and unique factors for certain species. Colony morphology is variable but is characterized by a "fried-egg" appearance. In general, colony morphology, cultural characteristics or carbohydrate fermentation are not useful for speciation. Identification to the species level is usually based on immunologic tests utilizing species-specific antisera or amplified DNA type tests. Inoculation of 5-7 day-old embryos is an alternative isolation procedure utilized when artificial media is unrewarding. Alternatively, clinical material can be inoculated into young chickens or turkeys and a comparison of pre-inoculated sera with 3-5 week post-inoculated sera by the immunologic tests may provide clues as to which *Mycoplasma sp.* is involved.

I. MYCOPLASMA GALLISEPTICUM INFECTION

(MG; Chronic Respiratory Disease; CRD;
Infectious Sinusitis of Turkeys)
(See Table on Page 101)

DEFINITION

A mycoplasma infection characterized by respiratory signs and lesions, a prolonged course in the flock and primarily affecting chickens and turkeys. In turkeys the disease is frequently manifested by swelling of the infraorbital sinus (es) and called infectious sinusitis.

OCCURRENCE

Mycoplasma gallisepticum (MG) occurs primarily in chickens and turkeys but also has been reported in partridge, pheasants, peafowl, quail, guinea fowl, ducks, and pigeons. All ages of chickens and turkeys can have the disease although the very young are seldom submitted with MG. Since 1994, a serious *M. gallisepticum* infection of free-ranging house finches has been shown to cause periorbital swelling, conjunctivitis and mortality.

HISTORICAL INFORMATION

In the United States the disease was first described in turkeys in 1905, then in chickens in 1935. MG became of major importance as the poultry industry expanded over the last 25 years. The first of a series of national conferences on mycoplasmosis in poultry was held in 1962 and recognized the importance of MG. Considerable progress has been made in the control and eradication of MG, especially in turkeys, but the disease is still of major importance.

MG is one of the more costly poultry diseases, sharing that distinction in the United States with Marek's disease and Newcastle disease. A few years ago the annual loss from MG was estimated at 125 million dollars.

MYCOPLASMOSIS

ETIOLOGY

1. *M. gallisepticum* is the etiologic agent. However, this organism is often associated with one or more of the following agents and pathogenicity is enhanced by these associations: infectious bronchitis virus, Newcastle disease virus, *Escherichia coli*, *Pasteurella multocida*, and *Hemophilus paragallinarum*.

2. *M. gallisepticum* seldom survives for more than a few days outside of the host. Carrier birds are essential for its survival.

3. In chickens the organism may be present and cause no disease until triggered by stress, such as changes in housing, management, nutrition, or weather; vaccination against or infection with infectious bronchitis or Newcastle disease; or increased levels of dust or ammonia in the environment.

4. *M. gallisepticum* strain variability exists and accounts for the variability of host susceptibility, clinical presentation and immunologic response.

EPIDEMIOLOGY

M. gallisepticum is transmitted in some of the eggs (transovarian transmission) laid by inapparent carriers. Infected progeny then transmit the agent laterally, probably through infectious aerosols coughed into the air and through contamination of feed, water, and the environment. The agent probably can be transmitted by other species of birds, domestic or wild. In addition the agent can be transmitted mechanically on shoes, feed sacks, crates, etc.

CLINICAL SIGNS

Signs usually develop slowly in the flock. They vary in severity, depending on strain of organism, and may persist for weeks or months. Signs are the same as those observed with many other avian respiratory diseases. They include coughing, sneezing, snicks, rales, ocular and nasal discharge, and, in turkeys, swelling of the infraorbital sinus (es) in occasional birds. Additional signs are listed below.

1. Adult layers- Drop in feed consumption and egg production. Egg production continues at a lower level. Mortality is low but there may be many unthrifty birds.

2. Broilers (3-8 weeks old) - Signs are more pronounced than in adult birds and the disease is more severe. Feed intake and growth rate are reduced. Mortality is variable but may be high, particularly if poor husbandry, exposure, or other stress factors are present.

3. Turkeys- In addition to swelling of one or both infraorbital sinuses [Fig. 1; M. gallisepticum; AAAP], infected turkeys may have nasal exudate wiped on their wings. Alternatively, if the air sacs and lungs are primarily involved, mortality from pneumonia and airsacculitis may be very high although few birds have swollen sinuses.

LESIONS

1. Poor physical condition and loss of weight are usually apparent and suggest the presence of a chronic disease.

2. There is marked catarrhal inflammation of the nasal passages, sinuses, trachea, and bronchi. Air sacs often are thickened [Fig. 2; M. gallisepticum; AAAP], and opaque and may contain hyperplastic lymphoid follicles in their wall. Recent vaccination against Newcastle disease or infectious bronchitis may enhance opacity of air sacs. Air sacs often contain mucoid or caseous exudate.

3. The following classic triad of lesions is often the basis of extensive condemnation of infected birds at slaughter: airsacculitis, fibrinous perihepatitis, and adhesive pericarditis [Fig. 3; M. gallisepticum; AAAP]. These lesions are not pathognomonic and may occur with chlamydiosis or septicemia.

4. In infectious sinusitis of turkeys, lesions may be restricted to swelling of the infraorbital sinuses. Conversely, sinusitis may be absent although rhinitis, tracheitis, and airsacculitis occur and there may be a fibrinous pneumonia. Occasional turkeys and chickens may have the oviduct distended with exudate (salpingitis).

DIAGNOSIS

1. A history of chronic respiratory disease accompanied by lowered feed consumption, poor gains, or lowered egg production is suggestive of MG. Typical gross lesions are very suggestive.

2. Positive plate or tube agglutination tests for MG on sera from a few birds in the flock strengthen the diagnosis. Because sera from birds with *Mycoplasma synoviae* may cross-react, it is a good plan to confirm some of the agglutination tests with the hemagglutination inhibition (HI) test or MG ELISA test. Cross-reactions usually do not occur when the HI or ELISA test is used. Flocks recently vaccinated with oil-based vaccines may also produce false-positive agglutination tests.

3. A commercial PCR test specific for *M. gallisepticum* is available. Tracheal swabs from a number of birds can be tested.

4. Isolation and identification of *M. gallisepticum* can be cultured from exudate, trachea, sinuses, air sacs, or lungs on artificial media or in chick embryos. MG identified isolates can be compared by molecular techniques for epidemiologic purposes.

5. In many instances it will be necessary to differentiate MG from other respiratory diseases of poultry, usually by culture or serologic tests. Pulmonary and air sac lesions may be confused with colibacillosis and aspergillosis. In turkeys, fowl cholera is a frequent and important complication and may be accompanied by a fibrinous pneumonia. Sinusitis in turkeys can also be caused by avian influenza, *M. synoviae* infection, and cryptosporidiosis.

CONTROL

1. Depopulation of infected premises should precede establishment of a "clean" flock. Thoroughly clean and disinfect the houses and leave them vacant for a few weeks.

2. Prevention is based largely on obtaining chicks or poults hatched from eggs from MG-free breeder flocks. The MG-free progeny are then raised in quarantine. MG-free breeder flocks have been established for both chickens and turkeys. They are monitored by serologic testing to assure that they and their eggs are free of *M. gallisepticum*. Quarantine measures must be strictly enforced and good management and sanitation must be practiced to keep a flock free of infection.

3. The vaccination of replacement pullets scheduled to enter MG positive layer complexes is practiced in most states. Three live commercial vaccines (F-strain, TS 11 and 6/85) are available and are administered via fine spray or eye-drop to birds during the growing period to protect them from clinical disease during the laying period. The live F-strain MG vaccine is pathogenic in turkeys. An oil-emulsion- based bacterin is also available for use in replacement pullets destined for multiage egg production complexes where MG is established in older hens.

4. Many MG-free breeder flocks were established initially by identifying small flocks that were not infected and using that flock as a nucleus. In addition egg dipping (in antibiotic solutions), heat sterilization, and antibiotic treatment of hatching eggs have all been used in attempts to obtain disease-free progeny from infected breeder flocks. All of the latter three methods reduce the number of infected progeny that hatch from eggs from infected flocks. No antibiotic or drug given to infected breeders will prevent them from laying some infected eggs.

TREATMENT

1. Marketing an infected flock with a low incidence of the disease may be more economical than treatment because treatment is very expensive. Consider this possibility initially.

2. Improve the management, husbandry, or nutrition if possible. In particular, try to reduce the dust to which the birds are exposed if dust is excessive. Remove accumulated manure and improve ventilation if ammonia levels are excessive. Eliminate all possible sources of stress.

3. Many broad-spectrum antibiotics have been used for treatment and will suppress losses. However, relapses often occur when treatment is discontinued. Most antibiotics are given in feed or water, preferably in water. Tylosin and tetracyclines have been used extensively for treatment. Injectable antibiotics may be more effective if the disease is advanced and if the flock is small enough to be treated individually. Treated birds must be held off the market for a time to meet regulations designed to prevent residues in meat.

II. MYCOPLASMA MELEAGRIDIS INFECTION

(MM Infection)

(See Table on Page 105)

DEFINITION

An egg-transmitted mycoplasmosis of turkeys characterized by inapparent venereal infection in breeder turkeys, airsacculitis in recently hatched poults and late embryo mortality.

OCCURRENCE

Mycoplasma meleagridis (MM) infection is confined to turkeys and occurs in all age groups. The disease is usually inapparent except in nonhatching turkey embryos or recently hatched poults in which it causes airsacculitis. Most large turkey breeder flocks are now free of MM infection.

HISTORICAL INFORMATION

1. Airsacculitis in newly hatched poults was first noted in 1958 but was not considered a major cause of loss because the lesions usually regressed. Airsacculitis at time of slaughter was usually attributed to infection with *Mycoplasma gallisepticum* (MG) or *Mycoplasma synoviae* (MS).

2. After MG and MS infections were brought under control, airsacculitis still remained as a significant cause of condemnation at slaughter. MM infection was found to be the cause.

3. *M. meleagridis* has been eliminated from the major primary turkey breeders. Infection in commercial flocks is not uncommon.

4. Skeletal abnormalities have been referred to as TS-65 (Turkey Syndrome 65) and crooked necks abnormalities have been referred to as wryneck.

ETIOLOGY

1. The etiologic agent is *M. meleagridis*. The organism is fastidious in its growth requirements and, presumably, easily destroyed by environmental factors and most disinfectants.

2. Concurrent infection with other mycoplasmas can occur and increases the severity of lesions.

EPIDEMIOLOGY

1. Infected breeder hens lay some eggs containing *M. meleagridis*, transmitting mycoplasma to some of their progeny. Organisms are spread laterally to many hatchmates via aerosols from the respiratory tract or to the vent on contaminated hands during vent-sexing.

2. In some progeny, the organism later spreads to and localizes in the reproductive tract. In male turkeys localization is often in the cloaca and/or phallus and their semen may contain the agent.

3. Artificial insemination of turkey hens with pooled infected semen is an important method of MM spread.

4. Most tom and hen turkeys overcome MM infection after one breeding season. By then the infection has already been transmitted to many of their progeny.

5. MM has a distinct predilection for the bursa of Fabricius and can cause immunosuppression, which may explain why infected turkeys are more susceptible to other infections, especially colibacillosis.

CLINICAL SIGNS

Most of the following signs are mild or inapparent on casual examination and go unobserved.

1. Often there is impaired hatchability of eggs from infected flocks. Embryo mortality is highest after eggs are transferred to the hatcher and at pipping.

2. Poults hatched from infected lots of eggs may have a high incidence of starveouts and may make poor weight gains.

3. Young growing poults may show mild respiratory signs and occasional poults may have sinusitis.

4. Small numbers of poults may have skeletal abnormalities associated with a deforming osteomyelitis in cervical vertebrae (crooked neck) or leg deformities.

5. Adult breeders usually show no signs [Fig. 1; M. meleagridis; AAAP] of venereal or respiratory infection.

LESIONS

1. Infected, pipped, unhatched embryos and recently hatched poults have a variable degree of airsacculitis [Fig. 2; M. meleagridis; AAAP] manifested by thickening of air sac membranes and possibly by the presence of small amounts of yellow exudate in the air sacs [Fig. 3; M. meleagridis; NCSU]. In uncomplicated MM, lesions regress and disappear in most turkeys by marketing time.

2. Poults with wryneck may have a cervical airsacculitis and osteomyelitis of adjacent vertebrae that can be demonstrated microscopically.

3. Adult breeders are free of gross lesions of the genitalia. However, outbreaks of airsacculitis, synovitis, and sinusitis have been observed in mature and semimature turkeys from which only MM could be isolated.

4. MM can also cause a generalized skeletal disorder historically known as turkey syndrome 65 (TS 65) characterized by chondrodystrophy, or unilateral or bilateral varus deformities [Fig. 4; M. meleagridis; AAAP] and perosis.

DIAGNOSIS

1. Monitor pipped embryos and weak, cull poults for the presence of air sac lesions.

2. Diagnosis can be made by the isolation and identification of MM from infected tissues or exudates. The organism is fastidious and requires special media. Isolates must be differentiated from MG, MS, and other mycoplasmas by serologic methods or fluorescent antibody techniques.

3. Turkeys infected with MM develop antibodies in 3-5 weeks. These antibodies can be demonstrated by plate and tube agglutination tests. Positive reactors can be confirmed by hemagglutination inhibition tests. These tests alone are not adequate for eradication of infection but can indicate infection in a flock. Test antigens are commercially available.

CONTROL

1. Poults should be obtained from MM-free breeder flocks.

2. A breeder flock free of MM infection can be established by inoculating all of the turkey eggs for hatching with 0.6 mg of gentamicin sulfate and 2.4 mg of tylosin in a 0.2-ml volume. Injections are made into the albumen through a small hole made by a dental drill in the small end of the egg. The newly hatched flock is monitored by culturing pipped (unhatched) eggs and 1-day-old cull poults and by using the plate agglutination test on sera from the flock.

3. Dipping the eggs from infected breeder flocks into solutions of tylosin or gentamicin will substantially reduce the incidence of infection in the progeny. Dipping is often combined with temperature- or pressure-differential techniques.

4. Repeated serologic testing alone has not been successful in establishing clean flocks. However, both egg dipping and testing programs have value.

5. Poults are often injected with an antibiotic during servicing in the hatchery, which probably aids in reducing MM infections.

6. Treatment of semen with antibiotics results in an unacceptable decrease in sperm viability.

TREATMENT

Because MM is sensitive to tylosin and tetracyclines, they may be of value in controlling airsacculitis in infected turkeys. It is unlikely they would be effective in controlling venereal infection. Use of lincomycin/spectinomycin in the drinking water (2 g/gal) for the first 5-10 days of life reduced the incidence of airsacculitis and improved weight gains.

III. MYCOPLASMA SYNOVIAE INFECTION

(MS, Infectious Synovitis; Tenovaginitis)

(See Table on Page 105)

DEFINITION

Predominately a subclinical upper respiratory infection of chickens and turkeys. Systemic infection results in an acute or chronic condition of chickens and turkeys characterized by inflammation of synovial membranes and, usually, by exudate in the joints and tendon sheaths of many infected birds. Synovial involvement is referred to as infectious synovitis.

OCCURRENCE

Respiratory *M. synoviae* infections are common in multi-age commercial layer flocks and are predominately subclinical. The Infectious Synovitis form occurs in chickens, especially in broilers, but the disease also occurs in turkeys. The disease is usually seen in young (4-12-week-old) chickens or young (10-12-

week-old) turkeys. It has been seen in adult layers and in chicks as young as 6 days of age. Synovitis occurs throughout the year but is more severe during the cold, damp seasons or whenever the litter is wet. The disease is probably worldwide in distribution. The incidence of the disease has greatly decreased in recent years in the United States.

HISTORICAL INFORMATION

Infectious synovitis was first described in chickens in 1954 and in turkeys in 1955. Although the disease was uncommon initially, it is now well established. It is said to be less commonly encountered today than a decade ago.

ETIOLOGY

1. The etiologic agent is *M. synoviae* (MS), a fastidious organism that requires nicotinamide adenine dinucleotide for growth. There appears to be only one serotype of the organism, although isolates vary in pathogenicity.

2. *M. synoviae* is a fastidious organism. It can usually be grown in 5-7-day-old embryonating chicken eggs or in special mycoplasma media. A commercially available PCR test can rapidly identify a MS positive flock.

3. Convalescent sera from birds with *M. synoviae* will agglutinate commercially available *M. synoviae* plate antigen. During the early stages of synovitis the sera may also agglutinate *Mycoplasma gallisepticum* plate antigen. Cross-reactions usually do not occur when hemagglutination inhibition (HI) or ELISA tests are used to separate *M. synoviae* from *M. gallisepticum* infection.

EPIDEMIOLOGY

1. Transovarian transmission is an important means of spread of the infectious agent. Only a small number of eggs from reactor birds carry *M. synoviae* and most of them are laid during the earlier stages of infection.

2. Infection also spreads laterally via the respiratory tract. Such spread is slow and only part of the infected birds develop joint lesions.

3. The organism has a predilection to localize in synovial-lined structures such as joints, tendon sheaths and bursas (breast blisters). It also localizes in the ovary and, occasionally, in the air sacs or sinuses.

CLINICAL SIGNS

1. Lameness in many birds and a tendency of affected birds to rest on the floor are prominent early signs. Many affected birds have pale head parts and swollen hocks or foot pads. The feces of acutely affected birds often are green. Eventually, affected birds become dehydrated and thin because of failure to eat and drink regularly.

2. Morbidity is usually low to moderate but may be high if there is damp, cold weather or the litter is wet. Mortality is usually less than 10% unless there are other diseases present or the husbandry is poor.

3. A slight, transient egg production drop maybe observed in acutely infected layer flocks.

4. Respiratory tract infections are usually asymptomatic.

LESIONS

1. In the early phase of synovitis most synovial-lined structures (joints, tendon sheaths) contain a sticky, viscid, gray to yellow exudates [Fig. 1; M. Synoviae; AAAP]. This is usually more voluminous in swollen hock or wing joints or under swollen foot pads [Fig. 2; M. Synoviae; AAAP].

2. In later stages of the disease the birds may be emaciated or thin and there may be no lesions in internal organs. Exudate in joints and tendon sheaths may be inspissated or joint surfaces may be stained orange or yellow. Breast blisters often are present secondary to trauma from resting on the floor.
3. Respiratory lesions may be absent or consist of a mild mucoid tracheitis, airsacculitis, or sinusitis, lesions that are usually associated with *M. gallisepticum* infection (chronic respiratory disease). Such birds may not have the usual lesions of synovitis described above.

DIAGNOSIS

1. Typical signs and gross lesions, especially epizootic lameness and characteristic exudate in swollen joints or tendon sheaths, are suggestive of synovitis. The diagnosis can be strengthened by obtaining positive plate agglutination tests for synovitis on sera from a few birds in the flock. Three to 5 weeks are required for antibody formation to have occurred.

2. *M. synoviae* can be isolated on special media or in 5-7-day embryonating chicken eggs. Trachea, sinuses, air sacs or synovial exudate are preferable for culture. The isolated *Mycoplasma* can be identified by direct fluorescent antibody techniques applied to colony imprints. Alternatively, exudate may be inoculated into the foot pad of chickens and turkeys to reproduce typical lesions. Later, their preinoculation and convalescent sera may be tested against *M. synoviae* antigen and *M. gallisepticum* antigen. The HI test can be used to confirm agglutination test results.

3. A commercial PCR test specific for *M. synoviae* is available. Tracheal swabs from a number of birds can be tested.

4. Synovitis must be differentiated from arthritis caused by *Staphylococci*, fowl typhoid, pullorum disease, and viral arthritis. Agents of the first three are easily cultured. Viral arthritis should infect experimentally inoculated chickens but not turkeys.

CONTROL

1. In most areas it is now possible to get chicks or poults that were hatched from eggs from MS- free flocks. If possible, start with such chicks or poults.

2. Insofar as is possible, raise the birds in quarantine under the all-in, all-out system.

3. Synovitis can usually be prevented by continuously giving the birds a low-level antibiotic in the feed. This is an expensive procedure. Many antibiotics used for treatment can be used for prevention but are fed at a lower level.

4. Commercial *M. synoviae* vaccine is available and maybe beneficial in certain management situations.

TREATMENT

Treatment of lame birds with well-established synovitis is usually not very satisfactory. Relatively high levels of antibiotics are required and may be given in feed or water. Aureomycin and Terramycin have been widely used. Streptomycin has been used intramuscularly in small groups of birds where they could be handled individually.

THE MYCOPLASMOSES[A]

Name(s) of the Disease	Etiologic Agent	Nature of the Disease	Major Lesions
Chronic respiratory disease	*Mycoplasma gallisepticum*	A respiratory disease.	Airsacculitis, adhesive pericarditis, fibrinous perihepatitis. Occasionally causes synovitis or salpingitis.
Infectious sinusitis	*Mycoplasma gallisepticum*	Unilateral or bilateral sinusitis. May spread to or occur initially in the lower respiratory system.	Swollen infraorbital sinus (es) may or may not be followed by airsacculitis, pericarditis, and perihepatitis.
Infectious synovitis	*Mycoplasma synoviae*	Involves synovial lining of joints, tendon sheaths. Results in lameness, debility.	Swollen joints and tendon sheaths. Feet, shanks, hocks more obviously affected. Occasionally causes airsacculitis in broilers and turkeys.
Mycoplasma meleagridis infection; MM infection	*Mycoplasma meleagridis*	A venereal infection of turkeys, usually transmitted by infected pooled semen. Produces airsacculitis in many progeny.	Airsacculitis in nonhatching or newly hatched poults. May spread laterally to other young poults as an airsacculitis. May lead to airsacculitis in market birds.

[A] More than 20 serotypes of *Mycoplasma* have been identified in chickens, turkeys, and ducks. These three are the most significant pathogens.

NECROTIC ENTERITIS

DEFINITION

Necrotic enteritis is an acute bacterial infection primarily of chickens and turkeys, although other avian species can be affected. The disease is characterized by sudden death, friable and distended intestines, and severe necrosis of the intestinal mucosal.

OCCURRENCE

Chickens 2-10 weeks of age raised on litter are most frequently involved. Turkeys that are 7-12 weeks of age are also affected. Both species usually have some predisposing enteric condition.

HISTORICAL INFORMATION

Necrotic enteritis was first reported in 1961 and has been reported to occur where ever poultry are produced.

ETIOLOGY

1. *Clostridium perfringens* (types A or C) and their toxins are the cause of necrotic enteritis. These bacteria are anaerobic Gram-positive rods and produce double-zoned hemolysis on blood-agar plates.

2. Alpha toxin is produced by *C. perfringens* type A and C and beta toxin is produced by C. *perfringens* type C and is responsible for the mucosal necrosis.

3. *C. perfringens* are ubiquitous and are normal inhabitants of the intestinal tract.

4. Intestinal mucosal damage is necessary for the *Clostridia* to proliferate and produce sufficient toxin. Coccidiosis, ascarid migration, hemorrhagic enteritis in turkeys, and severe salmonella infection are predisposing conditions for mucosal damage.

EPIDEMIOLOGY

Necrotic enteritis often develops as an acute terminal complication of other primary intestinal diseases or in situations where the intestinal microflora is disturbed or the host is severely immunosuppressed. A disturbed intestinal microflora can result from sudden changes in feed formulation such as addition of high levels of fish meal or wheat. Immunosuppression from infectious bursal disease or hemorrhagic enteritis frequently precedes necrotic enteritis.

CLINICAL SIGNS

The acute onset of depressed, ruffled birds occurs; these birds rapidly progress to death. There is a rapid increase in mortality.

GROSS LESIONS

1. Lesions are usually found in the mid-small intestines, which are distended and friable.

2. Intestinal contents consist of foul-smelling brown fluid and the mucosa is covered by a brownish diphtheritic membrane.

3. Severe dehydration with darkening of the breast muscle and swelling and congestion of the liver may also be present.

NECROTIC ENTERITIS

DIAGNOSIS

1. Intestinal mucosal appearance and typical history of acute and severe increase in mortality is strongly suggestive of necrotic enteritis.

2. Histologically, there is heavy clostridial colonization of the villous epithelium accompanied by coagulative necrosis of the mucosa.

3. Identification of a predisposing factor is necessary for successful treatment.

CONTROL

1. Good management practices of cleaning and disinfection of poultry houses prior to bird placement are essential. Repeat problem houses may benefit from salting the floor at cleanout. Cheap grade feed salt is used on the soil at a rate of 60-63 lb/1,000 ft^2.

2. All predisposing factors must be controlled.

3. Administration of appropriate feed medication may be warranted.

TREATMENT

Determination of predisposing condition will dictate specific medication. The clostridial component of this disease usually responds well to the same antibiotics specified for ulcerative enteritis (i.e., bacitracin, penicillin, and lincomycin).

ORNITHOBACTERIUM RHINOTRACHEALE INFECTION

(OR, ORT)

DEFINITION

Ornithobacterium rhinotracheale (OR) is a recently encountered bacterium which has been associated with respiratory disease in poultry. Certain bacteriologic and pathologic aspects of the organism and disease have only recently been investigated.

OCCURRENCE

O. rhinotracheale has recently been found with an increasing frequency in broiler and turkey operations experiencing respiratory problems. OR is frequently isolated with other respiratory agents (i.e. *E. coli, Bordetella avium, Mycoplasma sp.* and respiratory viruses). Broiler and meat turkey operations see primarily birds with respiratory signs, with no consistent mortality. Condemnation and decreased feed efficiency have been reported. Egg production can be decreased in layer or breeder flocks. Mortality appears more severe in turkey breeder operations.

HISTORICAL INFORMATION

O. rhinotracheale was first isolated in 1981 in Germany and in 1989 in the United States. In 1994, the bacterial organism was named by Vandamme.

ETIOLOGY

1. *O. rhinotracheale* is a pleomorphic Gram negative rod which grows well (but slowly) on blood agar plates. After 24 hours of incubation at 37 C in 7.5% CO_2, OR colonies are pinpoint in size and show no hemolysis. No growth is observed on MacConkey agar plates.

2. Key biochemical tests include the Gram's stain reaction and morphology (short plump rods, club shaped rods, or long filamentous rods), positive oxidase test, positive β-galactosidase (ONPG) test, and negative catalase test. No reaction is observed in most carbohydrates.

3. The api-ZYM system (Biomerieux, France) is most useful. This system gives fourteen positive reactions and five negative reactions (lipase, β-glucuronidase, β-glucosidase, α-mannosidase, and α-fucosidase).

EPIDEMIOLOGY

1. *O. rhinotracheale* has been isolated from broiler and layer chickens, turkey and chicken breeders, meat turkeys, game birds, pigeons, pheasants, partridges, and chukers. Isolates are most frequently obtained from respiratory sites such as the trachea, sinuses, and lungs. Occasionally, systemic involvement is indicated by isolations from the heart, spleen, liver, bone, and joint.

2. In most disease situations, other primary respiratory agents can be demonstrated (*Bordetella avium, Mycoplasma sp., Pasteurella sp., E. coli*, paramyxovirus, and infectious bronchitis virus).

CLINICAL SIGNS

Mild respiratory signs are most frequently observed with only a slight increase in mortality. Older birds may experience more severe respiratory signs with gasping, marked respiratory effort and an increase in mortality.

ORNITHOBACTERIUM RHINOTRACHEALE INFECTION

LESIONS

Mild sinusitis, tracheitis, or unilateral or bilateral lung consolidation may be observed. Turkeys frequently have blood-stained mucous in the mouth. Serofibrinous bronchopneumonia and fibrinous inflammation of the air sacs are noted histopathologically.

DIAGNOSIS

Bacterial culture is required to demonstrate *O. rhinotracheale'*s involvement in respiratory disease. Care must be taken to prevent its overgrowth by other bacteria. In turkeys, differentiation from fowl cholera requires bacterial culture.

CONTROL

Little is known on the prevention of *O. rhinotracheale*. It is frequently present in consecutive flocks on the same ranch. Currently, there is no commercial vaccine available but autogenous bacterins have been used with some apparent benefit.

TREATMENT

Treatments with tetracycline and amoxicillin have been reported in Europe. Limited success has been reported with enrofloxacin and trimethoprim/sulfa.

SALMONELLOSIS

PREFACE

Bacteria of the genus *Salmonella* have long presented serious challenges to the poultry industry and are responsible for significant health problems in nonpoultry avian species as well. In this section salmonella infections are presented in four parts covering pullorum disease, fowl typhoid, arizonosis, and paratyphoid. The essential background information on each disease is provided within these parts. It is noteworthy that although the host-specific salmonellae (*Salmonella pullorum* and *Salmonella gallinarum*) literally prevented intensive large-scale poultry production prior to the evolution of practical testing and eradication programs in breeders, it is now the paratyphoid infections that threaten public acceptance of poultry products by virtue of concern for food-borne infection. Paratyphoid salmonella infections are relatively common in poultry and all reasonable steps should be taken to minimize contamination of the finished product. The poultry industry is justifiably proud of its wide spectrum of economical and appealing products and every effort should be made to protect the industry from either implied unwholesomeness or true food safety problems.

I. PULLORUM DISEASE

DEFINITION

Pullorum disease is an infectious, egg-transmitted disease of poultry, especially chicks and turkey poults, often characterized by white diarrhea and high mortality in young birds and by asymptomatic adult carriers.

OCCURRENCE

Pullorum disease occurs primarily in young chicks and turkey poults. Many other species can be infected naturally but they usually play an insignificant role in the epidemiology of this disease. Pullorum disease occurs in all age groups of chickens and turkeys but causes greatest loss in those less than 4 weeks old and is worldwide in distribution.

HISTORICAL INFORMATION

1. The bacillus that causes pullorum disease was first described in 1900. Within a few years pullorum disease was recognized as a common, worldwide, egg-borne disease of chickens. A tube agglutination test that would detect carriers was developed in 1913 and a whole blood test was developed in 1931. These tests permitted development of eradication programs.

2. Losses from pullorum disease were once so severe that they impaired expansion of the poultry industry. Pullorum disease sometimes was spread through hatchery-infected chicks. Extensive losses from pullorum disease and fowl typhoid were partly responsible for stimulating the development of the National Poultry Improvement Plan; the plan contains measures for the control of hatchery-disseminated diseases.

3. Through the application of control measures now detailed in the voluntary National Poultry Improvement Plan, pullorum disease has been eliminated from commercial poultry in the United States. The disease still persists in small backyard flocks. It probably could be eradicated if proven control measures could be enforced for all poultry and exotic birds.

4. Pullorum disease still causes catastrophic losses when no effort is made to control it. This occurs repeatedly in developing countries trying to establish a poultry industry.

ETIOLOGY

1. The etiologic agent is *S. pullorum*, a nonmotile, Gram-negative bacillus adapted to poultry. This organism, like many other *Salmonella* spp., tends to infect young birds more frequently than older individuals and to establish a bacteremia. *S. pullorum* closely resembles *S. gallinarum*, the cause of fowl typhoid. They share certain antigens and usually cross-agglutinate on serologic tests.

2. The organism is rather resistant under moderate climatic conditions and can survive for months. However, it can be destroyed by thorough cleaning followed by disinfection. The organism can be killed by formaldehyde gas, which may be used in fumigation of fertile eggs and hatchers.

EPIDEMIOLOGY

1. *S. pullorum* is spread primarily through occasional infected eggs laid by infected carrier hens. Many of the infected chicks hatch and then transmit the organism laterally to other birds in the hatch through the digestive and respiratory systems. Sale of exposed but apparently healthy birds to many different purchasers can result in widespread dissemination of the etiologic agent.

2. Adult carriers also shed the organism in their feces. Slow lateral spread to other adults is possible through contamination of feed, water, and the environment. Also, contamination of nests and eggs therein can result in eggshell penetration and infection of chicks that hatch from those eggs.

3. Cannibalism of infected bacteremic birds can result in transmission.

CLINICAL SIGNS

Adults

Usually there are no signs. The infected adult may or may not appear unthrifty. An infected hen may or may not be a productive layer.

Young chicks and poults

1. In a setting of fertile eggs with a few infected embryos, there may be reduced hatchability. A few of the newly hatched birds appear weak or soon die. In others that develop bacteremia sudden death may occur. Mortality may be low during the first few days if only a few of the eggs contained the organism.

2. Morbidity and mortality begin to increase around the 4th or 5th day. Sick birds appear sleepy and weak. There is anorexia, white adherent diarrhea with pasting of the vent area, huddling near heat sources and shrill chirping. A few days later there may be respiratory signs in birds that inhaled the organism in the hatcher. Losses usually peak during the 2nd or 3rd week and then diminish. Survivors often are irregular in size and some are unthrifty, stunted, or poorly feathered. Many remain carriers and disseminators of the etiologic agent.

3. Mortality varies greatly but often is very high and can approach 100%. Mortality is increased by shipping, chilling, or poor husbandry. Conversely, mortality may be surprisingly low and the disease may go unrecognized.

LESIONS

Adults

Often there are no lesions. Occasionally there is a nodular myocarditis, pericarditis, or abnormal gonads. An abnormal ovary may have hemorrhagic, atrophic, or discolored follicles [Fig. 1; Pullorum; Cornell U]. Less frequently there is oviduct impaction, peritonitis, or ascites. Affected testes may have white foci or nudules.
Young chicks and poults

1. There may be few or no lesions in very young birds that die after a short septicemic course. Occasional dead birds feel wet. Many birds have pasted white feces in the vent area.

2. Classically there are gray nodules in one or more of the following sites: lungs, liver, gizzard wall, heart [Fig. 2; Pullorum; AAAP], intestinal or cecal wall, spleen, and peritoneum. Frequently there are petechial hemorrhages or foci of necrosis in the liver. Later there may be swollen joints in occasional birds.

3. When the intestine is opened, white plaques may be found in the intestinal mucosa and cheesy cores of debris may be found in the intestine or ceca. Plaques and cecal cores [Fig. 3; Pullorum; AAAP] occur more frequently in birds that die later in the course of the outbreak.

4. The spleen frequently is enlarged. (This lesion, along with mucosal plaques and cecal cores, also occurs frequently in *Salmonella* infections other than pullorum disease.) The ureters frequently are distended with urates.

DIAGNOSIS

1. In young chicks and poults, typical history, signs, and lesions may suggest pullorum disease. Positive agglutination tests using sera from surviving birds may strengthen the diagnosis. Chicks hatched by small, noncommercial operators are more likely to be positive for *S. pullorum*.

2. For a firm diagnosis, *S. pullorum* must be isolated and identified. Legal complications may occur so identification should be confirmed. The organism should be typed at a typing center.

3. The National Poultry Improvement Plan provides details for confirming infection in adult reactor birds. Specified organs are pooled and cultured for *S. pullorum.*

4. Diseases that must be differentiated from pullorum disease in young birds include:

    A. Chilling. Chilling is often associated with white diarrhea.

    B. Omphalitis (navel infection). Omphalitis occurs in this age group, often with diarrhea.

    C. Typhoid, paratyphoid, arizonosis, and colibacillosis. It will be necessary to isolate and identify the etiologic agent to separate these infections.

CONTROL

1. Prevention is based on establishment and maintenance of pullorum-free breeder and multiplier flocks by serologic testing and other measures. The following tests are used:

    A. The stained antigen, rapid whole blood test is typically performed in flocks in the field. This same antigen can be used for the rapid serum test in the laboratory.

    B. Tube agglutination test is performed on sera and is primarily used to confirm plate test reactions.

    C. An enzyme-linked immunosorbent assay (ELISA) has also been developed for the serologic diagnosis of pullorum disease.

2. Noninfected eggs from tested clean flocks should be hatched in a properly disinfected hatcher and raised on pullorum-free premises, preferably under quarantine.

3. Detailed regulations for control of pullorum disease are given in the National Poultry Improvement Plan. A copy of the plan can be obtained from The National Poultry Improvement Plan, USDA-APHIS-VS, Suite 101, 1498 Klondike Road, Conyers, GA 30094.

4. The poultry producer can avoid pullorum disease by purchasing chicks only from those hatcheries that participate in the National Poultry Improvement Plan or a similar eradication program. Exposure of the flock to carriers or a contaminated environment must be avoided.

TREATMENT

Insofar as chemotherapy perpetuates the carrier state, treatment of pullorum-infected birds is indefensible and should not be recommended under any circumstance.

II. FOWL TYPHOID

DEFINITION

Fowl typhoid is an infectious disease, primarily of chickens and turkeys, with many of the clinical and epidemiologic features and lesions that occur with pullorum disease.

In the following material only the differences between fowl typhoid and pullorum disease are emphasized. Most of the facts concerning pullorum disease (see Pullorum Disease) are applicable to fowl typhoid.

OCCURRENCE

Most outbreaks occur in chickens or turkeys but the disease occasionally occurs in other poultry, game birds, and wild birds. In chickens and turkeys most outbreaks occur in recently hatched, young birds, but unlike pullorum disease, the disease often continues for months. Many outbreaks occur in semimature flocks with no history of an earlier onset.

HISTORICAL INFORMATION

A disease that probably was fowl typhoid was recognized in 1888. By the early 1900s many outbreaks, both abroad and in the United States, had been reported. Between 1939 and 1946 there was a marked increase in outbreaks in the United States and fowl typhoid was a major disease of poultry. Application of testing and control measures (now detailed in the National Poultry Improvement Plan) greatly reduced the incidence of both fowl typhoid and pullorum disease. Fowl typhoid is seldom encountered today in the United States but persists as a challenging disease problem in several countries.

ETIOLOGY

The etiologic agent is *Salmonella gallinarum*. This organism shares many antigens with *Salmonella pullorum*, the agent that causes pullorum disease, and the two organisms usually cross-agglutinate. As a consequence, birds exposed to or infected with either disease can be identified by the same agglutination test.

EPIDEMIOLOGY

The epizootiology of fowl typhoid is similar to that of pullorum disease. Relatively speaking, transmission of infection through eggshell contamination may be of somewhat greater importance than with pullorum disease. Also, *S. gallinarum* is more frequently transmitted among growing or mature flocks and the incidence and mortality in older birds is usually higher.

CLINICAL SIGNS

Signs of fowl typhoid and pullorum disease are similar in birds less than approximately 1 month old. Semimature and mature birds with fowl typhoid often have pale head parts (comb, wattles, face), shrunken

combs and wattles, and diarrhea. Mortality can be substantial. In one extensive experiment, many broods of birds were hatched from eggs from a typhoid-infected flock of hens. Approximately one third of all hatched birds died with typhoid.

LESIONS

1. Lesions of fowl typhoid and pullorum disease are similar in chicks and young poults (see pullorum disease).

2. Lesions of acute fowl typhoid in older birds include:

   A. A bile-stained ("bronzed") enlarged liver with or without small necrotic foci [Fig. 1; Fowl Typhoid; AAAP].

   B. Enlargement of the spleen and kidneys.

   C. Pallor throughout the cadaver and thin watery blood.

   D. Enteritis in the anterior small intestine, often with ulceration.

3. In older birds, chronic fowl typhoid lesions resemble those seen in pullorum disease (see pullorum disease).

DIAGNOSIS

S. gallinarum should be isolated and identified for diagnosis. It should be carefully differentiated from other salmonella and paracolon organisms.

CONTROL

Control is as for pullorum disease. Fortunately, control of both pullorum disease and fowl typhoid is accomplished by the same program encompassed in the National Poultry Improvement Plan.

III. ARIZONOSIS

DEFINITION

Arizonosis is an egg-transmitted infection, seen primarily in young turkey poults, characterized by variable signs and lesions related to septicemia or to localization of infection in the intestine, peritoneal cavity, eye(s), brain, or other sites.

OCCURRENCE

Most outbreaks occur in turkeys. Although all ages are susceptible, the disease is most common in poults less than 3 weeks old. Chicks, ducklings, canaries, psittacines, and other birds occasionally have been found to be infected. Infection frequently occurs in reptiles, which can serve as reservoirs. Infection in humans has occurred but is not common. The disease probably is worldwide in distribution.

HISTORICAL INFORMATION

1. Arizonosis in chicks was reported in 1936, although it was not clearly differentiated from paratyphoid infection. In 1939 the etiologic agent of arizonosis was definitively characterized and found to cause a fatal septicemia in reptiles.

2. In the 1940s to 1960s arizonosis was recognized as an important and widely distributed disease of many turkey flocks.

3. Historically the etiologic agent of arizonosis was considered to be in the genus *Arizona*. Since 1984 the etiologic agent of arizonosis has been characterized as a subspecies of the genus *Salmonella* based on DNA relatedness to this genus.

ETIOLOGY

1. The etiologic agent is *Salmonella arizonae* (syn. *Arizona arizonae* and *Arizona hinshawii*). It is a non-spore forming, Gram-negative, motile bacterium in the family Enterobacteriaceae.

2. *S. arizonae* ferments lactose slowly, usually requiring a few days. Slow-fermenting *S. arizonae* may be mistaken for other *Salmonella* species unless the fermentation tubes are held for a sufficient period.

EPIZOOTIOLOGY

1. *S. arizonae* often localizes in the ovary of carrier birds. When this happens, the organism is included within eggs, infects the developing embryo, and results in infected progeny.

2. Infected adult birds are frequently intestinal carriers and intermittent shedders of *S. arizonae*. Contamination of eggshell surfaces with feces leads to eggshell penetration and infection of progeny.

3. Infected progeny that hatch from infected eggs transmit the organism laterally to uninfected birds in the hatch and later may become carriers and shedders of the organism.

4. Exposure to the agent can also occur via reptiles, rats, mice, and many other mammals, contaminated hatchers, or fomites. Transmission frequently is via fecal contamination of feed, water, or environment. The organism can persist in a contaminated environment for months.

5. As with many salmonellae, *S. arizonae* has few, if any, species barriers. Interspecies transmission occurs readily and there are many carriers.

CLINICAL SIGNS

In young poults there may be listlessness, diarrhea, pasting of feces in the vent area, huddling near heat sources, ataxia, trembling, torticollis [Fig. 1; Arizonosis; NCSU], excessive mortality (3-5% is most common, although losses up to 50% have been reported), and poor growth. Cloudiness (turbidity) and enlargement of the eye(s) causing blindness may occur in infected poults. Central nervous system signs occur in birds with brain lesions. Signs in young birds closely resemble those seen with paratyphoid. Moderate to marked uneven growth in the flock is seen even after the clinical disease has ended. Affected eyes undergo atrophy and are useful in identifying previously infected flocks. Adult carriers usually show no signs.

LESIONS

1. Typically, lesions of septicemia are observed, including an enlarged, mottled, yellow liver, a retained yolk sac and peritonitis. Occasionally there are cheesy plugs in the intestine or cecum and infected yolk sacs develop into abscesses in poults that survive the initial septicemia.

2. A small but significant number of poults have opacity or turbidity of the eye(s) (ophthalmitis) [Fig. 2; Arizonosis; NCSU]. This useful lesion is not pathognomonic because it also occurs with paratyphoid, aspergillosis, or colibacillosis. However, it occurs more frequently with arizonosis.

3. Purulent exudate in the meninges [Fig. 3; Arizonosis; NCSU], lateral ventricles of the brain, or in the middle and inner ear is seen in birds with central nervous system signs.

DIAGNOSIS

The etiologic agent must be isolated and identified. Signs and lesions are inadequate for separating arizonosis from salmonellosis. *S. arizonae* can usually be recovered from liver, spleen, heart blood, unabsorbed yolk, intestine, or other organs. It is readily recovered from infected eyes, ears, and brains. *S. arizonae* persists for several weeks in atrophied eyes, from which the organism can be easily recovered. Also, it may be cultured from nonhatching embryos, eggshells, or organs from infected breeder birds and environmental samples. Enrichment procedures used to isolate other salmonellae are equally effective for detecting *S. arizonae.*

CONTROL

1. If infected breeder flocks can be identified, they should not be used as a source of fertile eggs. Unfortunately, there is no readily available serologic test for identification of infected flocks or individual birds. Such flocks often are identified by culturing the agent from their eggs or progeny. Primary turkey breeder companies in the United States are now free of *S. arizonae* infection, but commercial breeder flocks are still occasionally affected.

2. One-day-old poults are usually inoculated at the hatchery with antibiotics to control mortality from arizonosis. Gentamicin is most commonly used. Strains resistant to gentamicin have been found and have caused high losses in poults. In these cases, the use of injectable tetracyclines or ceftiofur may be helpful.

3. Most of the measures used for prevention of paratyphoid are applicable for control of arizonosis (see under paratyphoid).

TREATMENT

Useful antibiotics and drugs include gentamicin, tetracyclines, and sulfonamides. Treatment does not prevent birds from becoming carriers and shedders of the organism. Experimentally, use of a bacterin in turkey breeder hens has been found to be helpful in reducing shedding and coupled with good management procedures, eventually eliminating the disease from breeder flocks.

IV. PARATYPHOID INFECTION

(Salmonellosis; Paratyphoid)

DEFINITION

Paratyphoid is an acute or chronic disease of poultry, many other birds, and mammals caused by any one of a large group of salmonellae that are not host specific.

OCCURRENCE

Paratyphoid infection occurs in many kinds of birds and mammals; it occurs frequently in poultry. It also occurs in rats, mice, and other rodents, in many reptiles, and in some insects. It is a frequent disease of humans. In most animals the young are more frequently and severely affected. Adults tend to be more resistant but can be infected, especially if stressed prior to exposure. Paratyphoid infection is worldwide in distribution.

These bacterial infections are of much more importance for public health impact than for economic losses in the affected animals. Poultry products have been repeatedly implicated in human outbreaks of salmonellosis and all health management personnel in the poultry industries need to be sensitive to this legitimate consumer concern. Although detailed epidemiology is outside the scope of this book, the technical details in this chapter should provide essential background on the control of avian Salmonella infections. The National Poultry Improvement Plan is involved in the regulatory aspects of *Salmonella enteritidis* infection and also has programming in place to allow poultry breeder farms to attain Salmonella monitored status.

SALMONELLOSIS

ETIOLOGY

1. The etiologic agents are over 200 species of *Salmonella*. They tend to be motile and are not host adapted. These characteristics contrast with *S. pullorum* and *S. gallinarum*, which are nonmotile and highly host adapted.

2. Approximately 10-20 species of *Salmonella* cause most outbreaks in poultry. Frequent isolates in the United States include:

S. enteritidis	*S. typhimurium*
S.hadar	*S.agona*
S. montevideo	*S. heidelberg*
S.muenstert	*S.kentucky*
S. senftenberg	*S. thompson*

3. Most paratyphoid organisms contain endotoxin, which is responsible for their pathogenic effects.

4. Paratyphoid organisms are moderately resistant in their natural environment but are susceptible to most disinfectants and to fumigation with formaldehyde gas.

EPIDEMIOLOGY

1. Paratyphoid organisms often localize in the intestine or gallbladder of carriers. They are intermittently shed in the feces and thus contaminate eggshells, feed and water. Poultry, other birds, reptiles, insects, and various mammals including humans can disseminate salmonella.

2. Infection of young chicks occurs primarily by fecal contamination of eggshells with paratyphoid organisms and subsequent penetration into the eggs. Some chicks are infected at the time of hatch and infection spreads laterally.

3. Localization of paratyphoid organisms in the ovary with subsequent transovarian transmission occurs in some instances. The frequency of this method of spread is unknown.

4. Contaminated animal proteins (tankage, meat scraps, etc.) can transmit the agents. These products often are contaminated after processing. Heated, pelleted products seldom contain living salmonella.

5. Interspecies transmission of paratyphoid organisms does occur, often through environmental contamination. Rodents are an important reservoir for paratyphoid organisms. Paratyphoid is an important public health problem and this aspect of the disease is by far the greatest challenge to the poultry industries.

CLINICAL SIGNS

1. Signs usually are seen only in young birds (less than 4 weeks of age). There is somnolence, profuse diarrhea followed by dehydration, pasting or wetting of the vent area, drooping wings, shivering, and huddling near heat sources.

2. There usually is high morbidity and mortality (especially during the first 2 weeks of brooding), although these are variable. The course often is short in individual birds.

LESIONS

1. There may be a few or no lesions in birds that die after a short septicemic course, perhaps only a few petechial hemorrhages.

2. There usually is dehydration and marked enteritis, often with focal necrotic lesions in the mucosa of the small intestine. Occasionally there are necrotic foci in the liver. In young birds there often is unabsorbed

yolk material in the yolk sac and overt omphalitis. Less frequent lesions include blindness, joint infections, or swollen eyelids, the latter two being common in pigeons.

3. Raised plaques in the intestinal mucosa and cheesy cecal cores are often seen in birds that survive for a few days or longer. These strongly suggest the presence of salmonellosis but are not pathognomonic for any one species of *Salmonella*.

DIAGNOSIS

The etiologic agent should be isolated from multiple organs and positively identified. Using polyvalent salmonella antiserum, most labs can identify any isolate as *Salmonella*. Typing centers can provide the service of complete species typing and should be utilized for this specialized work. Selective media often are utilized in isolating *Salmonella* from the gut.

CONTROL

1. Breeder flocks should be monitored bacteriologically for *Salmonella* infection in conjunction with efforts to minimize flock exposure.

2. If possible, all birds should be sold after one lay season thus eliminating carriers. While the premise is vacated, it should be thoroughly cleaned and disinfected. Eliminating rodents is an essential step in the eradication of *Salmonella* from a farm.

3. In flocks provided with nests the nests should be kept clean. Replace nesting material frequently as needed. Maintain a high standard of sanitation in all operations.

4. Gather eggs frequently and store them in a cool place. Separate dirty eggs from clean eggs at the time of gathering. Egg sanitation may be necessary at the poultry farm during the storage that precedes storage and incubation at the hatchery.

5. Fumigate hatching eggs as recommended during incubation (done routinely). Practice scrupulous hatchery sanitation.

6. Raise new broods of birds in the all-in, all-out system. Add no new birds to the started brood. Do not permit contact with wild birds, mammals, rodents, or reptiles. Control insect populations.

7. Provide uncontaminated animal proteins in the ration. Pelleted feeds are more likely to be free of salmonella.

8. If necessary, specific antigens may be prepared and used in an agglutination test for elimination of carriers. This has been done for *S. typhimurium* but not for most other paratyphoid organisms. Once developed, these test antigens can be used on a regular basis at the time of the annual pullorum-fowl typhoid test.

9. One-day-old chicks and poults are often inoculated at the hatchery with antibiotics to control mortality from paratyphoid and other bacterial infections. This practice may help control early mortality but the same positive result can be accomplished by the good breeder flock and hatchery management practices enumerated above.

10. A commercial bacterin is available for vaccination of commercial egg layers against *S. enteriitdis*.

TREATMENT

Treatment suppresses but does not eliminate infections. Appropriate treatment also minimizes mortality until the birds can develop immunity. Many drugs and antibiotics are reasonably effective. These include sulfaquinoxaline, sulfadimethoxine, ormetoprim and sulfadimethoxine, and tetracyclines. Treatment should be based on culture and antimicrobial susceptibility results.

SPIROCHETOSIS

DEFINITION

Spirochetosis or nonrelapsing borreliosis is a septicemic disease of most poultry and many other birds. Acute cases are characterized by depression, cyanosis, diarrhea, and leg weakness progressing to paralysis and death.

OCCURRENCE

Spirochetosis occurs naturally in chickens, turkeys, geese, ducks, pheasants, grouse, and canaries. Many other birds can be infected experimentally. In the United States the disease usually has occurred in turkeys, chickens, and pheasants. All age groups are susceptible if not previously exposed. The disease is widely distributed in tropical and temperate regions. In the United States it has been recognized in California, New Mexico, Texas, and Arizona.

HISTORICAL INFORMATION

1. Spirochetosis, one of the major scourges of poultry, was first reported in 1891. Spirochetosis has occurred only a few times in the Southwestern United States. It has been reported in California in 1946 and 1993 as well as in Arizona in 1961. The disease is of major importance in those countries where it is enzootic.

2. There is potential for spread of spirochetosis in the southwestern states because the presence of the tick vector, *Argas persicus*.

ETIOLOGY

1. The etiologic agent is *Borrelia anserina*, a spirochete with 5-8 spirals that is up to 30 microns long.

2. The organism is not very resistant outside the host and must be maintained in some vector between hosts.

EPIDEMIOLOGY

1. *B. anserina* can be transmitted through infectious droppings but usually is transmitted by blood sucking arthropods. *Argas persicus* is the usual vector and mosquitoes of the genus Culex may serve as vectors. Mites may serve as mechanical carriers.

2. *A. persicus* remains infective for up to 430 days after feeding on an infected host. Further, the tick passes the spirochete to its progeny.

3. Infectious vectors and mites transmit the spirochetes to susceptible birds when they feed upon them. Recovered birds clear the infection completely and do not become carriers.

4. Transmission can also occur through ingestion of infected ticks, cannibalism of moribund birds, or scavenging of infected carcasses.

CLINICAL SIGNS

Infected birds are depressed, cyanotic, thirsty, and often have a diarrhea that includes excessive white urates. The birds are weak, squat on the ground, and later may become paralyzed. Morbidity and mortality vary greatly depending on the virulence of the *B. anserine* strain. Morbidity and mortality may approach 100% in highly susceptible flocks.

LESIONS

1. There usually is marked enlargement of the spleen, which is mottled by ecchymotic hemorrhages.

2. The liver frequently is enlarged and may contain small hemorrhages, infarcts, or foci of necrosis. The kidneys and heart may be enlarged and pale. There usually is bile-stained mucoid enteritis. The histopathology has been well described but microscopic lesions are not diagnostic for the disease.

DIAGNOSIS

1. Spirochetosis should be suspected if the tick *A. persicus* is found on typical sick birds. However, nymphs and adult ticks live in the house and feed mostly at night.

2. The spirochetes can be identified in Giemsa-stained blood smears [Fig. 1; Spirochetosis; Cornell U] or by dark-field or phase- contrast microscopy of blood and other fluids. Spirochetes can be concentrated in the buffy coat of centrifuged blood. This may facilitate identifying birds with low spirochetemia. Spirochetes may not be observed during late stages of the disease.

3. In doubtful cases, the spirochete can be demonstrated by isolating it in six chick embryos inoculated with defibrinated blood from a typical early case. Alternatively, young chicks or poults can be inoculated with serum or tissue suspensions and their blood can be examined daily for spirochetes, which usually appear in 3-5 days.

4. The spirochete can be identified in specially stained tissue sections. Also, the fluorescent antibody test can be used to identify it in tissues or blood. Agar-gel precipitin tests have been used to detect spirochete antibodies and antigens.

CONTROL

1. Spirochetosis can be prevented by controlling or eradicating all the vectors and transmitters of *B. anserina*. It may be difficult to eradicate the fowl tick without destroying infested wooden buildings and all the birds in infected flocks. Isolating the roost by suspending it from wires or placing the supports of the roost in pans filled with oil is helpful in reducing tick feeding.

2. A wide variety of bacterins and vaccines has been prepared abroad but are not available in the United States. They appear to be reasonably effective although they produce a shorter, weaker immunity than is desirable unless revaccination is practiced.

TREATMENT

In countries where spirochetosis is enzootic, numerous drugs and antibiotics, including penicillin, streptomycin, tylosin and tetracyclines have been used successfully for treatment.

STAPHYLOCOCCOSIS

DEFINITION

Staphylococcosis is a systemic disease of birds characterized most frequently by purulent arthritis and tenosynovitis.

OCCURRENCE

Staphylococcal infections of poultry occur worldwide and affect all classes of birds. Outbreaks are most important in turkeys and broilers. The organisms are common in the environment and are especially associated with the skin. Most diseases produced by *Staphylococcus* sp. are associated with a break in the skin or beak (trauma, beak trimming, toe trimming, etc.) Avian infections tend to be caused by types occurring in birds rather than human strains. Isolates pathogenic for one class of poultry are usually pathogenic for other classes of birds. Toxigenic strains capable of causing food poisoning can contaminate the skin of processed poultry. The source of these strains is usually from the processing plant environment or workers.

HISTORICAL INFORMATION

Staphylococci were first discovered to be a cause of arthritis in geese in 1892. Since that time they have been identified as the cause of a variety of localized and systemic diseases in many different avian species and in most areas of the world. The disease was more common in turkeys when they were raised on range than it is now.

ETIOLOGY

1. Most staphylococci isolates have been identified as *Staphylococcus aureus*, a Gram-positive coccus occurring in clusters. Pathogenic isolates are usually coagulase positive.

2. Phage-typing is frequently used to distinguish strains. Isolates from different geographic areas tend to be different phage types. Particular phage types are often endemic on a particular farm and tend to reappear in successive flocks.

3. Organisms are moderately resistant to common disinfectants. Chlorine-containing disinfectants are efficacious in the absence of organic material.

4. Toxins produced by staphylococci can increase both the virulence and pathogenicity of a particular strain.

LESIONS

Diseases produced by staphylococcus infections include:

1. Omphalitis

   Although infections of the yolk sac occur, they are less common than omphalitis caused by other bacteria. Sources of the bacterium include the breeder flock, hatchery environment, and hatchery workers.

2. Gangrenous dermatitis

   Affected areas of the skin are dark red, moist, thickened, and clearly demarcated from adjacent normal skin. Usually traumatic lesions such as punctures or scratches are present. The serosanguinous fluid seen in clostridial infections is minimal or absent. Staphylococcal gangrenous dermatitis is typically secondary to immunosuppression caused by infectious bursal disease or chicken infectious anemia virus.

3. Cellulitis

   A purulent inflammation is present in subcutaneous tissues. Traumatic lesions may or may not be present. The overlying skin tends to be dry and discolored.

4. Abscesses

   There are localized purulent lesions in the skin. The plantar surface of the foot is a common site and results in bumblefoot. Abscesses result from puncture wounds.

5. Septicemia

   There is an acute increase in mortality with congestion of the internal organs. It is usually associated with a processing event of the bird such as beak trimming or some other trauma to the skin.

6. Arthritis/synovitis

   Any joint, tendon sheath, or synovial bursa can be affected. Arthritis/synovitis is seen clinically as swollen, hot joints, especially hock joints. It occurs as a sequel to septicemia and can be experimentally reproduced by intravenous injection of pathogenic strains. Initially, affected tissues are acutely inflamed and contain white to yellow soft fibrinopurulent exudate. Later, the exudate becomes caseous. Fibrosis of affected tissues occurs late. Affected birds often have bile stasis of the liver. High numbers of large mononuclear cells are seen in blood smears.

7. Discospondylitis (spondylitis)

   The joints of articulating thoracolumbar vertebrae are affected. The process spreads to affect adjacent vertebrae. Lesions may become so extensive that pressure on the spinal cord will develop causing paresis and paralysis.

8. Osteomyelitis

   This is a sequel to septicemia. Organisms localize in metaphyseal vessels invading the cartilage of the growth plate of actively growing bones. Initially, pale yellow, friable bone is seen in affected areas adjacent to the growth plate, especially in the proximal tibia and metatarsus. Necrotic areas, abscesses, and sequestra are seen later.

9. Endocarditis

   This is an uncommon sequel to septicemia. There are vegetations on the mitral and/or aortic valves. Emboli from valve lesions cause infarcts in the brain, liver, and spleen.

DIAGNOSIS

1. Gross lesions are suggestive. A rapid, presumptive diagnosis can be made by identifying the typical cocci in smears from lesions.

2. Organisms can be readily cultured and identified from lesions and often from the livers of affected birds.

CONTROL

1. Because staphylococci are ubiquitous in the environment their presence cannot be prevented. When an outbreak is associated with a particular environment, the source should be sought and eliminated.

2. Protect broilers from infectious bursal disease with an appropriate vaccination program.

3. Take measures to reduce the occurrence of traumatic skin lesions, as well as any enteric disease which would damage the integrity of the intestinal mucosa.

4. The respiratory tract has also been identified as an important portal of entry for pathogenic staphylococci in turkeys. Exposing chickens or turkeys to strain 115 of *Staphylococcus epidermidis* by aerosol at 10 days and again at 4-5 weeks substantially reduced the incidence of staphylococcosis and improves overall flock livability.

5. Avoid overly severe feed restriction in breeder replacements which has been associated with an increased incidence of staphylococcosis.

TREATMENT

1. High levels of antibiotics effective against staphylococci may be helpful if given early in the course of the disease.

2. Resistance to antibiotics is common and isolates should be tested for sensitivity.

3. Usually treatment is not cost effective and preventive programs should be relied on.

ULCERATIVE ENTERITIS

(Quail Disease)

DEFINITION

Ulcerative enteritis is an acute bacterial infection of upland game birds, turkey poults, and young chickens characterized by ulcerations of the intestinal tract and by focal and/or diffuse hepatic necrosis.

OCCURRENCE

Ulcerative enteritis occurs frequently in young, captive, upland game birds and with increasing frequency in turkey poults and young chickens. Quail are the most susceptible host species. Young birds are affected more frequently than adults although the disease occurs frequently in adult quail. The disease is widespread in the United States and is known to occur in Europe and Asia. In chickens it frequently occurs in association with other diseases, including coccidiosis, chicken infectious anemia, and infectious bursal disease.

HISTORICAL INFORMATION

Ulcerative enteritis was reported in the United States in 1907 and had been observed prior to that time in Great Britain. It was first referred to as quail disease, a name retained for many years despite recognition of the disease in many other birds. The disease was recognized in chickens by 1934 and in domestic turkeys by 1944. The disease has increased in incidence and is now a well-recognized disease. At one time the agent was mistakenly believed to be *Clostridium perfringens*.

ETIOLOGY

The etiologic agent is *Clostridium colinum*, a Gram-positive, anaerobic, spore-forming bacillus. The organism is very resistant. It withstands boiling for 3 minutes or 70 C for 10 minutes. Boiling suspected material is useful in killing other contaminating bacteria during isolation attempts. *C. colinum* can best be isolated from the typically affected fresh liver. The preferred medium is tryptose-phosphate-glucose agar with 8% horse plasma. Cultures are incubated anaerobically.

EPIDEMIOLOGY

1. The etiologic agent is spread primarily through the droppings of acutely affected or recovered carrier birds and spores persist in the soil for years. Interspecies transmission can occur among susceptible birds.

2. Infection can be spread by flies that feed on infectious droppings. The disease is highly contagious, especially among quail.

CLINICAL SIGNS

1. In most species signs are similar to those seen with coccidiosis in chickens. These include listlessness, humped appearance, retracted neck, drooping wings, partially closed eyes, ruffled feathers, diarrhea, anemia, and perhaps bloody feces. In quail, white watery droppings are rather distinctive. In chicken flocks a course of 2 or 3 weeks is common and then the chickens recover slowly.

2. Sudden death may occur without signs being apparent, especially during onset. Birds that die suddenly may be well muscled and fat, especially in quail.

3. Mortality may be very high with quail, up to 100% within a few days. Mortality in chickens seldom exceeds 10%. Game birds other than quail usually have a higher mortality than chickens but less than occurs in quail.

ULCERATIVE ENTERITIS

LESIONS

1. Lesions are similar in most birds. Most cases presented for necropsy have deep ulcers scattered throughout the intestine, including the ceca, and the ulcers may be numerous enough to coalesce. Ulcers [Fig. 1; Ulcerative Enteritis; NCSU] may be round or lenticular, the latter shape being more common in the upper intestine. Deep ulcers often can be detected through the serosa of the unopened intestine [Fig. 2; Ulcerative Enteritis; UC Davis] and may penetrate it to induce peritonitis. The intestine may contain blood, thus mimicking coccidiosis. Acute cases have severe enteritis of the small intestine.

2. The affected liver usually contains large, yellow or tan areas or focal yellow lesions, or both. The lesions tend to be colorful and distinctive. The spleen is often enlarged and may be hemorrhagic.

DIAGNOSIS

1. Typical intestinal ulcerations and the distinctive colorful lesions in the liver strongly suggest ulcerative enteritis. Stained impression smears made from the cut surface of the liver may reveal the rod-shaped bacillus with its subterminal spore.

2. For confirmation the etiologic agent should be isolated and identified. The organism must be differentiated carefully from *Clostridium difficile* and *C. perfringens*.

3. Care should be taken to differentiate the disease in chickens from coccidiosis. Coccidiosis often is present in the same bird and assessing the relative importance of the two diseases may be difficult. Both diseases may be contributing to mortality.

CONTROL

1. Raise the flock in facilities and on ground where the disease has never occurred. Do not add birds. Prevent contact with all other species of birds. Raising birds on wire is of value if possibly feasible.

2. Keep old birds and young birds separated. If possible, do not have both age groups on the same premises. Practice careful sanitation including frequent cleaning and disinfection. Promptly remove and destroy all sick birds.

3. Streptomycin, bacitracin, penicillin, lincomycin, and tetracyclines have all been used intermittently in feed or drinking water for prevention of the disease. Rotating the use of these different antibiotics and chemicals will help prevent the emergence of *C. colinum* isolates which are resistant to antimicrobials.

TREATMENT

Most of the antibiotics and chemicals used in feed and water for prevention can be used at higher levels for treatment. These treatments should be administered in the drinking water.

VIBRIONIC HEPATITIS

(*Campylobacter* Hepatitis; Avian Infectious Hepatitis)

HISTORICAL NOTE

This syndrome occurred in the 1950s and 1960s, primarily in layers and was attributed to a vibrio-like organism and later to *Campylobacter jejuni*. The hepatic lesions were often distinct in appearance as stellate, asterisk-shaped, or cauliflower-like. Campylobacteriosis is an important zoonotic food-borne illness associated with a wide variety of sources including chicken. Currently, vibrionic hepatitis has completely disappeared from the United States and Western Europe. The reproduction of the hepatic lesions by *Campylobacter jejuni* has not been possible from strains isolated from either humans or chickens. The synergistic interaction of an unknown vibrionic appearing agent with co-pathogen may have been involved.

ASPERGILLOSIS

(Brooder Pneumonia; Mycotic Pneumonia; Pneumomycosis)

DEFINITION

Aspergillosis is an acute or chronic disease, primarily affecting the respiratory system. Peritoneal, visceral and systemic infections can also occur. The agent is *Aspergillus fumigatus*. Aspergillosis occurs frequently in turkeys, chickens, and game birds. This condition has also been reported in penguins, raptors, migratory waterfowl, psittacines and zoologic specimens, such as flamingos. All species of birds probably are susceptible. Aspergillosis was first described in a wild duck in 1833 and in turkeys as early as 1898. There are some other species and genera of fungi that may cause similar disease syndromes.

EPIDEMIOLOGY

1. Embryos. *Aspergillus fumigatus* can penetrate egg shells under ideal growth conditions and thus infect the embryos. Such eggs will often appear green when candled (the embryo will be dead). Infected embryos may hatch with well developed lesions.

2. Chicks and poults. If infected eggs break in the hatchery, large numbers of spores are released which contaminate the hatchery environment and air systems can lead to severe outbreaks in very young birds (less than 3 weeks of age). Eggs punctured for in-ovo injection are particularly susceptible to contamination. Even low-level contamination of hatchers or air systems can result in mortalities of 50% or greater when in-ovo injection is used.

3. Adults. Infection usually follows inhalation of large numbers of spores from heavily contaminated feed, litter or environment. Conjunctival infections may occur from heavy exposure to airborne spores following traumatic injuries. It is believed that healthy birds resist infection but that resistance can be overwhelmed by massive exposure combined with depressed host defenses. Debilitated and overcrowded birds are most susceptible. Market age tom turkeys and turkey breeders are commonly affected.

4. *Aspergillus fumigatus* is normally present in litter and feed. Enormous numbers of spores can be produced under ideal conditions. Sporulating colonies of *Aspergillus fumigatus* are blue-green and can often be observed grossly

5. Infections in the brain, posterior chamber of the eye or other visceral tissues result from systemic invasion from the respiratory tract.

CLINICAL SIGNS

1. Dyspnea, gasping, cyanosis and accelerated, labored breathing [Fig. 1; Aspergillosis; AAAP] frequently are observed. Rales do not usually accompany these respiratory diseases. Other signs include diarrhea, anorexia, somnolence, progressive emaciation, dehydration and increased thirst.

2. Morbidity is variable. Mortality is high in clinically affected birds. Increased mortality will be noted in affected flocks during loadout, hauling and following insemination. Affected birds often die during or just after handling especially if held by their legs.

3. Signs of central nervous system disturbance may occur in a small percentage are of the birds if there has been spread to the brain. Signs often include ataxia, falling, pushing over backwards, opisthotonos, paralysis, etc.

4. A gray-white opacity may develop in one or both eyes when there is eye infection. Ocular discharge occurs when the conjunctiva is infected and there can be corneal ulceration. A large mass of exudate typically accumulates in the medial canthus under the third eyelid.

LESIONS

1. Mycelial growth with sporulation may be apparent as fuzzy gray, blue, green or black material (sporulating fungus) on air sac lesions or in the main bronchi of the lungs.

2. Yellow or gray circumscribed nodules or plaques in the lungs [Fig. 2; Aspergillosis; AAAP], air sacs bronchi or trachea [Fig. 3; Aspergillosis; UC Davis] (usually the syrinx); less often in the peritoneal cavity, liver, brain or at other sites. In mature birds two patterns of air sac infection are found: disc-like plaques in the recurrent bronchi of the caudal thoracic and/or abdominal air sacs or markedly distended air sacs containing copious fluid and soft fibrinopurulent exudate.

DIAGNOSIS

1. The signs and gross lesions of aspergillosis are very suggestive of the diagnosis which can be confirmed by microscopic demonstration of fungus in fresh preparations made from the lesions or in histologic sections.

2. Microscopic examination reveals septate, branching hyphae within lesions. Hyphae can be seen in fresh preparations cleared with 10% KOH or stained with lactophenol cotton blue. If fungus is grossly visible in the lesions, the typical fruiting bodies [Fig. 4; Aspergillosis; AAAP] and spores can be easily found. In histologic sections, special stains (methenamine-silver, PAS, Gridley) are useful for demonstrating fungi in tissues. Nodules in the lungs usually appear as granulomas containing fungal hyphae.

3. Using sterile technique, the fungus can be cultured by tearing a nodule or plaque open and putting it on fungus media. Aspergillus will usually grow on blood agar in 24-48 hours. Sabouraud's dextrose agar [Fig. 5; Aspergillosis; AAAP] is a more selective medium. Since aspergillus spores are common laboratory contaminants, growth of only a few colonies may not be sufficient for a definitive diagnosis. For confirmation, tissue invasion should then be demonstrated.

4. Typical lesions of aspergillosis are unlike those of other avian respiratory diseases except pulmonary granulomas associated with complicated *Mycoplasma gallisepticum* infection. Histopathologic differentiation is usually easy.

5. Another fungus, *Dactylaria gallopava*, can cause lesions in the lungs or brain of young chickens and turkey poults. Signs and lesions resemble those caused by aspergillosis. The two fungi can be differentiated by culture. Numerous giant cells are characteristic of microscopic brain lesions caused by *D. gallopava*.

CONTROL

1. Collect clean eggs. Disinfect or fumigate eggs before setting. Do not set cracked eggs or eggs with poor shell quality.

2. Thoroughly clean, disinfect and fumigate incubators and hatchers. Inspect air systems and change air filters regularly in hatcheries. Monitor hatchery environment for mold contamination.

3. Use only dry, clean litter and freshly-ground, mold-free feeds. Store feeds and litters properly so as to inhibit growth of mold. Make sure feed bins and feed lines are kept clean, dry and free of mold growth. Do not permit feed to cake in feeders. Avoid wet litter under or around the waterers or feeders. Mold inhibitors may be added to feed to control fungus growth and prevent infection; however, this will add expense.

4. Optimize the ventilation and humidity in the poultry house to reduce air-borne spores. Humidity should be kept in the mid-range, neither too low nor too high. Alternating wet and dry conditions are an ideal situation for *Aspergillus*. The fungus multiplies during the wet period producing abundant spores which then become aerosolized when conditions become dry.

TREATMENT

1. If aspergillosis is diagnosed in a flock, cull clinically affected birds and remove any contaminated feed and litter. Clean and disinfect the house and then spray it with 1:2000 copper sulfate solutions or other fungicide and allow it to dry.

2. Valuable captive birds can be treated with Nystatin or Amphotericin-B or other anti-mycotic agents. Often antibiotics are given simultaneously to prevent secondary bacterial infection. Intravenous fluids may also be required. Ketaconizole, Miconozole and related drugs have been found effective for treating individual birds but are too expensive for commercial flocks.

CANDIDIASIS

(Thrush; moniliasis, crop mycosis, sour crop, muguet, soor, levurosis)

DEFINITION

Candidiasis is a disease of the digestive tract caused by the yeast-like fungus *Candida albicans*. The disease generally involves the upper digestive tract and usually occurs as a secondary infection.

EPIDEMIOLOGY

Candida albicans is a common yeast-like fungus that has been recognized as a commensal organism in poultry and mammals for many years. Candidiasis has been reported from a variety of avian species, such as, chickens, turkeys, pigeons, game birds, waterfowl, and geese. In poultry it seldom has been considered a disease of major importance. Young birds tend to be more susceptible than adult birds although all ages can be affected. When birds become debilitated or the normal digestive tract flora is altered, the ingestion of fungus in the feed and water can result in mucosal invasion. The production of a soluble endotoxin may also contribute to pathogenicity. Common predisposing causes include lack of good sanitation, prolonged treatment with antibiotics, heavy parasitism, vitamin deficiency, high carbohydrate diets, and immune suppressing or debilitating infectious diseases.

CLINICAL SIGNS

Signs are non-specific and include, listlessness, inappetence, retarded growth, and ruffled feathers. In advanced cases or diarrhea. The signs may be masked by the clinical signs of a primary disease. In advanced cases, the crop may not empty and may become fluid filled. The bird may regurgitate fluid with a sour, fermentative odor, i.e. the name “sour crop”.

LESIONS

1. Lesions vary greatly in severity. They are more common in the crop, mouth, pharynx and esophagus, but may involve the proventriculus and, less often, the intestine.

2. The affected mucosa is often diffusely or focally thickened [Fig. 1; Candidiasis; UC Davis], raised, corrugated and white, looking like terry cloth [Fig. 2; Candidiasis; UC Davis]. Lesions may also appear as proliferative white to gray pseudomembranous or diphtheritic patches and as shallow ulcers. Necrotic epithelium may slough into the lumen as masses of soft cheesy material.

3. Lesions of a primary predisposing disease may also be present and should be investigated. In particular one should search for evidence of coccidiosis, parasitism or malnutrition.

DIAGNOSIS

1. Characteristic gross lesions are generally adequate for diagnosis. Histopathologic examination of the affected mucosa usually will confirm invasion of the tissue by the septate fungal hyphae.

2. *Candida albicans* grows readily on Sabouraud's dextrose agar. However, since *Candida* is commonly present in normal birds, only the demonstration of massive numbers of colonies is of significance.

CONTROL

1. Practice a high standard of sanitation in the poultry operation. Phenolic disinfectants or iodine preparations should be used to sanitize equipment.

2. Prevent other diseases or management practices that might debilitate the birds.

3. Avoid over treatment of birds with antibiotics, drugs, coccidiostats, growth stimulants and other agents that might affect the bacterial flora of the digestive tract.

TREAMENT

1. Copper sulfate at a 1:2000 dilution in drinking water is commonly used both for prevention and treatment but its value is controversial. Nystatin in feed or water has shown efficacy against candidiasis in turkeys.

2. Routine addition of antifungal drugs to rations probably is a waste of money since elimination of contributing factors or other diseases usually will prevent candidiasis. However, if sanitation is at fault and cannot be improved, antifungal drugs may be advisable.

DACTYLARIOSIS

DEFINITION

A neurotropic, mycotic disease of turkey poults and young chickens with many of the clinical and pathologic features of aspergillosis. Signs of dactylariosis (incoordination, tremors, torticollis, circling, recumbency) are related to mycotic lesions in the brain [Fig. 1; Dactylariosis; NCSU]. Lesions also occur with less frequency in the air sacs, lungs, liver and eyes (globes). The etiologic agent, *Dactylaria gallopava*, grows naturally in old sawdust which often is used as poultry litter.

FAVUS

(Avian ringworm, Avian dermatophytosis)

DEFINITION

Favus is a mycotic infection found primary in gallinaceous birds. Favus is rare in commercial poultry today, but is occasionally reported in backyard flocks, especially exotic and game chickens. Characteristic lesions include white crusting on the comb and wattles [Fig. 1; Favus; Cornell U] that can extend to the feathered portion of the skin to form scutula around the bases of feather follicles. *Microsporum gallinae* is the agent most often isolated, although *M. gypseum* and *Trichophyton simii* have also been isolated. Topical treatment with nystatin has been efficacious on individual birds.

MYCOTOXICOSIS

DEFINITION

Mycotoxicosis is a disease caused by a toxic fungal metabolite. Mycotoxicoses may affect both humans and animals. Poultry mycotoxicoses are usually caused by fungi that colonize and invade grains and feeds, but other environmental aspects may be involved.

OCCURRENCE

1. Grains and forages used as foodstuffs support the growth of certain fungi when environmental conditions of temperature and humidity are suitable. Some of these fungi produce metabolites that are toxic to humans and animals and cause disease (mycotoxicosis) by either ingestion or cutaneous exposure.

2. Mycotoxicoses occur throughout animal-rearing regions of the world. Although specific mycotoxins form more frequently in certain geographic locations, interstate and international shipment of grains may result in widespread distribution of a mycotoxin problem.

DIAGNOSIS

1. A definitive diagnosis of mycotoxicosis should involve the isolation, identification, and quantitation of the specific toxin(s). This is usually difficult to accomplish in the modern poultry industry because of the rapid and voluminous use of feed and ingredients.

CONTROL

1. Prevention of mycotoxicoses requires the detection and control of mycotoxin contamination in feed ingredients and the application of feed manufacturing and management practices that prevent mold growth and mycotoxin formation.

2. Feeds and grains can now be screened for several mycotoxins (aflatoxin, T-2 toxin, ochratoxin, zearalenone) using monoclonal antibody detection kits. Many poultry companies already routinely test grain for aflatoxin contamination by a chromatographic procedure (minicolumn technique).

3. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by inspection of bins between flocks to certify absence of feed residue and by cleaning bins and feeders when necessary. Use of tandem feed bins on farms allows cleaning between successive feed deliveries.

4. Antifungal agents added to feeds to prevent fungal growth have no effect on toxin already formed, but may be cost-effective management in conjunction with other feed management practices. Several commercial products, most of which contain proprionic acid, should be applied according to manufacturers' instructions.

5. Zeolytes, a class of silica-containing compounds used as anticaking agents in feed formulation, and as aids in the improvement of eggshell quality, show promise as a practical and economical method of reducing the effects of certain mycotoxins. Hydrated sodium calcium aluminosilicate has been shown to bind aflatoxin B1, possibly by sequestration in the digestive tract, and reduce its toxicity to chickens.

TREATMENT

1. Remove the toxic feed and replace it with unadulterated feed.

2. Treat concurrent diseases (parasitic, bacterial) identified in the diagnostic evaluation.

3. Substandard management practices should be immediately corrected as they have increased detrimental effects in a flock stressed by mycotoxins.

4. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water-based treatment.

I. AFLATOXICOSIS

HISTORICAL INFORMATION

1. During the 1950s, a disease in dogs called hepatitis X occurred in the southeastern United States and was tied to the consumption of moldy dog food. It was later reasoned to have been caused by the same mycotoxin responsible for high mortality in turkeys due to hepatic toxicity (turkey X disease) in England in 1960. Peanut meal imported to England from Brazil was highly contaminated with fungi of the *Aspergillus flavus-Aspergillus parasiticus* group, which produced aflatoxins.

2. The aflatoxin story was historically important because unlike ergotism and alimentary toxic aleukia, which were sporadic and relatively localized phenomena, aflatoxicosis attracted global attention concerning the potential problems of mycotoxins in the food chain, and the ease by which these problems could be widely distributed.

ETIOLOGY

1. Mycotoxins of the aflatoxin group (B1, B2, G1, G2) are the cause of aflatoxicosis. Aflatoxin B1 is the most common in grains and is highly toxic. Aflatoxin forms in peanuts, corn, and cottonseed, and their products, in other grains, and in poultry litter. *A. flavus* is the primary producer of aflatoxin in grains, but not all strains of the fungus are toxigenic.

2. Like other mycotoxins, aflatoxin is produced only when substrate, temperature, and humidity are ideal. Favorable conditions for toxin formation may be localized within a volume of stored or transported grain creating toxic "hot spots". Once formed, the toxin is stable.

3. Grains damaged by insects and drought stress, and broken pieces of grain (screenings) are more likely to support fungal growth and toxin formation.

4. Aflatoxin B1 is a potent, naturally occurring carcinogen and thus has special public health considerations.

CLINICAL SIGNS

Aflatoxicosis in poultry is primarily a disease of the liver [Fig. 1; Aflatoxicosis; Univ Missouri] with important ramifications for other body systems, which may ultimately cause production problems and mortality. Affected birds have reductions in growth, carcass pigmentation, egg production, and immune function, and have increased nutrient requirements for protein, trace elements (selenium), and vitamins. The disease may be fatal.

LESIONS

At necropsy, lesions are minimal with either transient exposure or exposure to a low concentration of toxin. Jaundice, generalized edema and hemorrhages, tan [Fig. 2; Aflatoxicosis; Univ Missouri] or yellow discoloration of the liver, and swelling of the kidneys [Fig. 3; Aflatoxicosis; Univ Missouri] are seen with more severe intoxication. Microscopic changes in the liver occur as necrosis of hepatocytes, lipid accumulation in hepatocytes, bile duct proliferation, and fibrosis. These are common reactions of this organ to toxic insult and although they may be suggestive of aflatoxicosis, are not pathognomonic.

II. CITRININ MYCOTOXICOSIS

ETIOLOGY

Citrinin is a mycotoxin that was first isolated from *Penicillium citrinum* but is also produced by other species of *Penicillium* and by a few species of *Aspergillus*. Citrinin may be a factor in renal disease in food animals in Denmark, but no other documented case studies involving poultry are known.

CLINICAL SIGNS

Experimental citrinin mycotoxicosis in the chicken, turkey, and duckling has shown that chickens are relatively resistant, but all develop clinical illness of marked watery fecal droppings related to increases in water consumption and urine output. Metabolic alterations of electrolytes and acid-base balance occur. Young birds have reduced weight gain.

LESIONS

Citrinin produces marked functional changes in kidneys, however, gross lesions may be slight or overlooked. Swelling of kidneys and microscopic lesions of nephrosis may occur following severe exposure. In these circumstances, lymphoid tissues may be depleted and necrosis occurs in the liver.

III. ERGOTISM

HISTORICAL INFORMATION

1. Ergotism was recognized in central Europe in the Middle Ages and is the oldest known mycotoxicosis. Humans with ergotism (St. Anthony's fire) experienced an initial cold sensation in the hands and feet followed by an intense burning sensation. Gangrene of the extremities developed in both humans and afflicted animals. The disease occurred where bread was made from rye and other grains parasitized by toxigenic strains of the fungus *Claviceps purpurea*. The mold colonizes and replaces kernels of grain to form a hard, dark purple or black mass called an ergot or sclerotium.

2. Although the pharmaceutical properties of the ergot were recognized in China 5,000 years ago, it was not until 1875 that alkaloids present in the sclerotium were recognized as the cause of ergotism.

ETIOLOGY

1. The ergot alkaloids are a large family of compounds, and may cause constriction of blood vessels (vasoconstriction) those results in their pharmacologic and toxicologic effects.

2. *Claviceps* spp. that colonizes wheat, rye, and triticeal are the most common causes of ergotism of humans and animals.

CLINICAL SIGNS

In chickens, ergotism causes reductions in growth and egg production, and nervous incoordination.

LESIONS

Lesions include abnormal feather development, necrosis of the beak, comb, and toes, and enteritis.

IV. OCHRATOXICOSIS

HISTORICAL INFORMATION

1. Ochratoxin has been detected in kidneys of chickens with renal lesions in a processing plant in Denmark.

2. Three disease outbreaks in the United States involving 360,000 turkeys were associated with ochratoxin concentrations of up to 16 ppm.

ETIOLOGY

Ochratoxins A, B, and C are usually produced by toxigenic strains of *P. viridicatum* but may be produced by other species of *Penicillium* and by *Aspergillus ochraceus*. Ochratoxin A is the most toxic and is the greatest threat to poultry production.

CLINICAL SIGNS

1. Reductions in feed intake and increases in mortality.
2. Weight loss.
3. Drops in egg production have been reported from Ochratoxin A.

LESIONS

1. Gross and microscopic lesions in the kidneys and liver.

2. Experimental ochratoxicosis in chickens causes a dose-related reduction in weight gain, and gross and microscopic lesions occur in the target organs, liver and kidney. Visceral gout and reductions in plasma carotenoids, immune function, and certain blood coagulation factors also occur.

V. OOSPOREIN MYCOTOXICOSIS

ETIOLOGY

Oosporein is a toxic pigment produced by *Chaetomium* sp. and other fungi and is a contaminant of cereal grains and feedstuff.

CLINICAL SIGNS

Oosporein mycotoxicosis, studied in chickens and turkeys, causes a dose-related decrease in growth and an increase in water consumption. Chickens are more susceptible than turkeys. .

LESIONS

Visceral and articular gout as a result of nephrotoxicity.

VI. TRICHOTHECENE MYCOTOXICOSIS
(Fusariotoxicosis)

HISTORICAL INFORMATION

1. A disease called alimentary toxic aleukia occurred in the Russian people in the early 20th century, the 1930s, and especially during the Second World War. Labor shortages during the war necessitated the overwintering of grains (wheat, rye, and millet) in the fields and harvesting was delayed until spring. Bread made from the new grain caused acute gastroenteritis, followed by the formation of ulcers of the face and oral membranes, facial edema and lymph node enlargement, and in the later stages, bone marrow disorders, anemia, and uncontrolled hemorrhages. Morbidity and mortality greater than 50% occurred in some villages. Similar problems occurred in livestock and poultry in the region.

2. The disease is now recognized as a mycotoxicosis caused by colonization of grains by toxigenic species of *Fusarium*. These fungi produce mycotoxins of the trichothecene group, many of which cause caustic injury to mucous membranes and skin, the basis of the facial, oral, and gastrointestinal features of the disease. They also affect rapidly dividing cells (radiomimetic effect) manifested by disorders of the bone marrow (anemia, hemorrhagic disorders) and by abortions.

ETIOLOGY

More than 40 trichothecene mycotoxins are known to exist. T-2 toxin is one of the most toxic to poultry.

CLINICAL SIGNS

1. Chickens with fusariotoxicosis (trichothecene mycotoxicosis) have had reduced growth, abnormal feathering [Fig. 1; Trichothecene; Auburn Univ], severe depression, and bloody diarrhea.

2. In chickens, pigeons, ducks, and geese, the caustic properties of the trichothecenes have been manifested as feed refusal, extensive necrosis of the oral mucosa and areas of the skin in contact with the mold, and symptoms of acute gastrointestinal disease.

3. Experimental fusariotoxicosis, reproduced in chickens with pure T-2 toxin closely resembled the spontaneous disease but lacked the extensive hemorrhages.

LESIONS

1. Trichothecene mycotoxicosis may cause necrosis of the oral mucosa [Fig. 2; Trichothecene; Auburn Univ], reddening of the mucosa of the remainder of the gastrointestinal tract, mottling of the liver, distention of the gallbladder, atrophy of the spleen, and visceral hemorrhages.

VII. ZEARALENONE MYCOTOXICOSIS

HISTORICAL INFORMATION

In experimental studies, chickens have shown relative insensitivity to the effects of zearalenone. Zearalenone mycotoxicosis has been recognized since 1927 as the cause of a syndrome resembling estrogen stimulation in pigs and cattle in the United States and elsewhere.

ETIOLOGY

Zearalenone is a mycotoxin produced by *Fusarium roseum* (*Gibberella zeae*) and other *Fusarium* spp. A period of warm temperature and high humidity followed by low temperature is most conducive to toxin formation on grains

CLINICAL SIGNS

1. Zearalenone-contaminated feed has been associated with high mortality (40%) in a flock of 24,000 broiler breeder chickens. Affected birds had cyanotic combs and wattles and had difficulty walking.

2. Turkeys may develop swelling of the cloaca and reduced fertility.

3. Male geese may have reductions in sperm quantity and viability.

LESIONS

1. Affected chickens have had ascites and cysts both inside and outside of the oviduct. The oviducts were swollen and inflamed, and were obstructed with fibrinous fluid. Some oviducts had ruptured.

MYCOTOXICOSIS

	Aflatoxins	Fumonisins	Oosporein	Tricothecene	Ochratoxins	Citrinin	Ergotism	Zearalenone
Names	B1, B2, G1 and G2 are natural contaminants			T2, DAS, DON more than 100 fungal metabolites	Ochratoxins A, B & C		Ergot alkaloids (ergotamine, ergocristine)	
Major producers	*Aspergillus* mostly *Aspergillus flavus* and *A. parasiticus*	*Fusarium moniliforme*	*Chaetomium trilaterale*	Primarily isolated from *Fusarium* spp. Type A (more toxic to chickens) 14 PEB	Produced by several *Aspergillus penicillium viridicatum* and other species of *penicillium* Ochratoxin A –the most common toxic mycotoxin for poultry and the most toxic	*Penicillium citrinum*	*Claviceps purpurea* or other *claviceps* species	*Fusarium graminearum* *Fusarium roseum*
Toxic action	Target organ: Liver *potent hepatocarcinogen in humans	Disruption of sphingolipid synthesis Very low toxicity for poultry	Primary renal tubular damage	Primary inhibition of protein synthesis followed by secondary disruption of DNA & RNA synthesis Affect rapidly dividing cells such as those lining the GI tract, the skin, and lymphoid and erythroid cells Extensive necrosis of mucous membranes and skin in contact with the toxin Acute effects on digestive tract and bone narrow Immunosuppressive	Target organ: the kidney Interferes with DNA, RNA & protein synthesis Affects renal carbohydrate metabolism (gluconeogenesis) = damage to the epithelium of renal proximal convoluted tubules Decreased electrolyte absorption Increased water excretion	Reversible renal damage	Arterial and venous vasoconstriction Necrosis of peripheral tissues Decreased blood flow to extremities Possible endothelial damage	Potent estrogenic properties Low toxicity for poultry
Clinical signs	Decreased feed intake Decreased body weight poor skin Decreased egg production Decreased immunity	Decreased body weight	Dose-related decrease in growth Increased water consumption	Decreased feed intake Reduced growth Severe depression Abdominal feathering Bloody diarrhea	Decreased feed intake Weight loss Increased mortality Increased water consumption Humid litter Decreased egg production	Marked watery fecal droppings Increased water consumption Increased diuresis Reduced weight gain(young birds) Humid litter	Reduced growth Decreased egg production Nervous signs (incoordination)	Reduced fertility

MYCOTOXICOSIS

	Aflatoxins	Fumonisins	Oosporein	Tricothecene	Ochratoxins	Citrinin	Ergotism	Zearalenone
Lesions	Jaundice Generalized edema and hemorrhages tan or yellow discoloration of the liver Liver: Periportal necrosis with bile duct proliferation and fibrosis Depletion of lymphoid organs	Increased liver weight Increased kidney weight Liver hepatic necrosis with biliary hyperplasia	Dehydration Swollen and pale kidneys secondary visceral and articular gout	Circumscribed proliferative yellow caseous plaques in oral mucosa Reddening of GI mucosa Mottling of the liver Gallbladder distention Splenic atrophy Visceral hemorrhages	Pale and swollen kidneys Secondary visceral gout Pale and enlarged liver Regression and cellular depletion of lymphoid organs.	Swollen kidneys Degeneration and necrosis of tubular epithelial cells of both proximal and distal tubules.	Gangrenous-like lesions Necrosis of the beak, comb, toes Enteritis	Oviduct hypertrophy Cloacal swelling (turkeys) Reduction in sperm quantity and viability (geese)
Sources	Peanuts, corn, cottonseed, and their products, In other grains and in poultry litter	Corn and corn based feed	Cereal grains feedstuff	Fusarium spp. are important pathogens to plant producing cereal grains, (corn, wheat, barley, oats, rice, rye….)	Widespread natural contaminant of cereal grains (barley, oats, rye maize)	Often coexists in cereals with ochratoxin A (corn, wheat, barley, oats, rye and rice)	Open inflorescence of graminaceous plants (rye, wheat, triticale barley, oats, sorghum, corn, rice) and several grass species.	Corn, corn products, rice
Treatments				None	Vitamin C supplementation might reduce some adverse effects.			

EXTERNAL PARASITES

I. LICE

DEFINITION

Lice are insect ectoparasites. . Lice are generally species specific, meaning for each species of bird or mammal, there are particular species of lice.

ETIOLOGY

In domestic fowl, more than 40 species of lice have been reported. Some of the most important chicken lice include the Body Louse (*Menacanthus heterographa*), Head Louse (*Culclotogaster heterographa*), Shaft Louse (*Menopon gallinae*), Wing Louse (*Lipeurus caponis*), Fluff Louse (*Gonicocotes gallinae*) and the Brown Chicken Louse (*Goniodes dissimilis*). Also important are the Large Turkey Louse (*Chelopistes meleagridis*), and the Slender Pigeon Louse (*Columbicola columabae*). Birds may be parasitized simultaneously by more than one species.

EPIDEMIOLOGY

As is evident from the common names, certain lice prefer different regions of the bird. . They feed on scales of the skin and feathers. The life cycle is approximately 3 weeks. The entire life cycle is on the host. Lice will die in 5 to 6 days if separated from their host. Spread from bird to bird is dependent on close contact. Lice problems tend to be worse in the autumn and winter months.

CLINICAL SIGNS

Lice probably are not highly pathogenic for adult birds but the discomfort caused by the biting louse can have tremendous effects on flock performance. Heavy infestation of young birds is especially harmful due to the disruption of sleep

LESIONS

Careful examination of the vent area, the underside of the wings, the head (crest and beard) and legs will reveal these pests. Most bird lice are straw-colored and vary in size from 1-6 mm, but some may reach 10 mm. Louse eggs often can be found attached to feathers in clumps called “nits” [Fig. 1; Lice; Cornell U].

DIAGNOSIS

Diagnosis is based on gross observation of skin and feathers.

CONTROL

Pesticides treatments are highly regulated chemicals and all have hazardous potential for animal and human health tissue residues, and environmental contamination. A current listing of approved pesticides should be obtained from local agricultural authorities or university specialists. Examples of classes of products approved by the EPA for control of mites and lice include: organophosphates, carbamates, pyrethrins, and pyrethroids. Treatment is simplified by the fact that the louse is only found on the bird and not in the environment. In general, the efficacy of the treatment is dependent on the application of the chemical. The agent must penetrate to the skin in order to kill the lice. Also the entire bird must be treated as the lice are very mobile and will move away from the treated areas. Lice eggs are not affected by insecticide treatment, therefore a minimum of two treatments are required. Treatment should be performed on 7 to 10 day intervals. Egg-laden feathers should be removed from the premises. Routine examination for infestation should be performed for resident flocks on a bi-weekly or monthly basis.

II. MITES

DEFINITION

Mites are very small, barely discernible without magnification. They are not host specific and will infest any avian species. Mites feed on blood, feathers, skin or scales. Some mites do not spend their entire life on the bird; only visiting to feed, therefore, the bird and the environment must be treated to affect control. The most common mites of poultry species are listed below.

A. CHICKEN MITE –RED MITE (*Dermanyssus gallinae*)

Common red mites are up to 0.7 mm long x 0.4 mm and appear black or gray or red if engorged with blood. Red mites feed mostly at night and may not be found on the hosts during the day. Inspection at night is usually necessary to confirm infestation. Their life cycle can be completed in as few as 7 days. During the day, they may be found in colonies in the cracks or joints of roosts or nests. They can survive for over 30 weeks without food. This makes treatment of the facility imperative in control of this pest. Anemia and mortality can result from heavy infestations, especially in young birds. They have been reported to transmit the agents of fowl cholera and spirochetosis. They frequently parasitize caged layers. Treat both birds and facilities. Repeat treatment in one week.

B. NORTHERN FOWL MITE (*Ornithonyssus sylviarum*)

These mites are common bloodsuckers of a wide variety of poultry and other birds. They are known to or suspected of harboring agents that cause fowl pox, Newcastle disease, ornithosis and certain encephalitides. These mites stay on the birds continuously looking like moving red to black specks. Heavy infestations appear as blackened feathers, often near the vent with scabbed and cracked skin. Mite egg sacs are in white or off-white clusters located under the wings, above and below the vent, in the beard and crest and on the feathers of the proximal thigh. After handling or at necropsy they may transfer to the hands and arms of the handler or diagnostician. Northern fowl mites parasitize caged layers, especially during the wintertime, and often are seen crawling on eggs. All birds in the flock should be treated twice in a 5-7 day interval.

C. DEPLUMING MITES (*Knemidokoptes gallinae*)

A variety of feather mites live on the feathers or in the quills of domestic and wild birds. Feather mites tend to be somewhat host specific. They cause breakage or complete loss of feathers. The depluming mite of chickens, pheasants and pigeons (*Knemidokoptes gallinae*) burrows into the basal shafts of the feathers producing intense irritation that will cause the host to pull out its body feathers. Loss of feathers can lead to inability to control body temperature and may increase susceptibility to other diseases. These mites are difficult to treat as the feather shaft protects them from chemical agents. Isolate affected birds. Treat as for *Dermanyssus gallinae* (red mites).

D. SCALY LEG MITE. (*Knemidokoptes mutans*)

This mite represents a group of closely related mites that live primarily within unfeathered skin, often on the shanks and feet. It cannot be seen without magnification. The affected skin becomes thickened and hyperkeratotic with white, powdery, exfoliating crusts [Fig. 1; Scaly leg mites; Univ Montreal]. Some mites in this group attack the beak of birds. Without treatment, the affected bird will eventually become crippled. Long term treatment of individual birds is necessary for recovery. Spread through a flock is usually slow.

CONTROL

Treatment must be tailored to the species of mite affecting the bird, i.e., accurate identification is important. In many cases, the bird and the environment must be treated simultaneously. Insecticides for mites can be applied as powders, dusts or sprays. It is important that the insecticide penetrates to the skin.

For mites that live off the host (e.g. *Dermanyssus gallinae*), facilities can be treated by spraying a wet-able powder or liquid spray that will penetrate small cracks and crevices. Floors and bedding should also be treated. Pesticides are highly regulated chemicals and have hazardous potential for animal and human health and environmental contamination. A current listing of approved pesticides should be obtained from local agricultural authorities or university specialists. A notable exception is the Scaly leg mite (*Knemidocoptes mutans*) which can only be treated by direct application of an oil based product, such as petroleum jelly or cooking oil and kerosene (50:50) on a daily basis for at least 2 weeks or until the appearance of the legs returns to normal. Gentle washing of affected areas with soapy water to loosen and mildly debride hyperkeratinized areas is also helpful.

III. MISCELLANEOUS PESTS

A. BEDUGS (*Cimex lectularius*)

Bedbugs attack mammals and birds, including poultry and pigeons. Adult parasites are up to 5.0 mm long and have 8 abdominal segments. Bedbugs usually feed at night and may be observed on parasitized birds. Bedbugs can survive in houses for a year in the absence of poultry. Birds parasitized by bedbugs soon become unthrifty and anemic.

B. CHIGGERS (*Neoschongastia americana*)

Larval mites of *Neoschongastia americana* are a serious pest of turkeys and birds in southern states. In turkeys the larvae attach to the skin and cause localized skin lesions that lead to market downgrading. The lesions resemble pimples and may be very numerous.

C. DARKLING BEETLE AND LESSER MEALWORM. (*Alphatobius diaperinus*)

This beetle and its larvae often are present in poultry house litter. They may act as disease vectors for Marek's disease virus and Infectious Bursal disease virus. Botulism toxin has been found in these beetles. They probably serve as vectors for tapeworms. Their primary importance to the commercial poultry industry is economic due to the severe damage they can cause to the insulation and wall materials of modern poultry houses.

D. STICKTIGHT FLEAS (*Echidnophaga gallinaceae*)

In poultry these parasites usually are tightly attached in clusters to the skin of the head. They irritate the skin, cause anemia, lower egg production and may kill young birds. The adult fleas are about 1.5 mm long and reddish brown.

E. PIGEON FLIES (*Pseudolynchia canariensis*)

Dark brown, bloodsucking flies, about 6.0 mm long, that often parasitize pigeons, especially nestlings. The flies cause anemia and dermatitis. These flies also transmit *Hemoproteus columbae*, the cause of a malaria-like disease of pigeons.

F. BLACKFLIES (*Simuliidae*)

Grey-black, thick, humpbacked insects up to 5.0 mm long. Swarms of the flies attack mammals and birds, including poultry. Blackflies are the vector s for the protozoan, *Ornithofilaria fallisensis* that causes leukocytozoonosis in ducks. Bloodsucking blackflies can produce severe anemia and may kill young birds.

G. MOSQUITOS

Multiple genera and species of mosquitoes feed on birds, including poultry. Mosquitos are most important as vectors of viral diseases such as Pox and Equine Encephalitis. They also transmit protozoa that cause avian malaria-like syndromes. Mosquitos' bites cause irritation which may affect performance.

H. FOWL TICKS (*Argas persicus*)

Soft ticks including the "fowl tick" parasitize a wide range of poultry, wild birds and, occasionally, mammals. Some of the ticks not only cause anemia, skin blemishes or tick paralysis but also transmit *Borrelia anserina*, the agent that causes spirochetosis. Fowl ticks occur more frequently in the southwestern states, including California. The ticks spend relatively little time on their hosts and are easily overlooked.

INTERNAL PARASITES

I. NEMATODES, CESTODES AND TREMATODES ("WORMS" AND FLUKES)

DEFINITION

The most important internal parasites of poultry belong to the taxonomic group Nematodes. They have spindle shaped bodies with tapered ends and are also known as roundworms [Fig. 1; Ascaridia; NCSU]. Eggs are shed in the host droppings. Infection is established when a bird ingests an embryonated egg from the environment (direct life-cycle) or when a bird consumes an intermediate invertebrate host that is infected with the parasite (indirect life-cycle). Other internal parasites of diagnostic significance are Cestodes (tapeworms) and Trematodes (flukes).

EPIDEMIOLOGY

Clinical disease in all-in-all-out production systems for commercial broilers and turkeys is rare. The short life span of the commercial broiler or turkey also circumvents severe parasitism. In meat birds, ascaridiasis is the most common parasitism observed. Commercial caged layers rarely have parasites without intermediate hosts (e.g. tapeworms) because of their lack of contact with soil. Commercial turkeys and broilers reared on built-up litter, breeder flocks, non-caged commercial laying hen flocks, backyard flocks, gamebirds and pet or zoologic specimens are likely to exhibit higher rates of parasitism.

CONTROL

Control measures that interrupt the life-cycle are effective for most nematodes with direct cycles of infection. For parasites with indirect life-cycles (some nematodes, cestodes and trematodes), control is often aimed at elimination of the intermediate host such as beetles or other insects, snails or slugs, or preventing access of poultry to the intermediate host.

Piperazine is the only FDA approved treatment for internal parasites in meat and egg producing fowl. It is effective against ascarids in areas where resistance has not developed. It is applied in the drinking water. FDA regulations now allow the off-label use of drugs approved for other food animals for treatment of poultry. Fenbendazole has been used as a feed or water additive has been successfully used against *Capillaria,* and *Heterakis* infections. Thiabendazole, mebendazole, cambendazole, levamisole and tetramisole have been used against Syngamus and other nematodes such as *Trichostronglylus*. Pyrantel tartrate and citarin have also been effective against some nematode infections. Butynorate is approved for treatment of some cestodes of chickens.

SEE TABLE OF COMMON WORMS AND FLUKES ON PAGE 160

II. BLOOD-BORNE PROTOZOAL PARASITES

INTRODUCTION

Avian species act as host for a number of blood-borne protozoal parasites. Although these infections are for the most part inapparent and undiagnosed, they can offer a diagnostic challenge to the uniformed. Diagnosis is generally by microscopic evaluation of blood smears or histologic sections. The parasites can be differentiated by their tissue distribution, size and physical characteristics.

PREVENTION AND TREATMENT

These diseases are rarely treated. Few effective treatments are known. Clopidol (Coyden) has been administered in the feed to turkeys for treatment of Leukocytozoonosis at 0.0125 to 0.025% for 14-16 weeks with apparent success. Prevention is attempted by vector control. In enzootic areas tight screening of housed flocks and programs to control black flies and midges have failed to control the disease. Most avoid raising poultry where conditions are ideal for propagation of vector species. Elimination of carriers by yearly disposal of old poultry may be helpful if contact with local, wild birds that are carriers is avoided.

A. ATOXOPLASMA

A two-host coccidial protozoan also known as Toxoplasma, Lankesterella, Isospora, Encephalitozoon, or Haemogregarina. The parasite is spread by oocysts in droppings and/or arthropod vectors such as the red mite. Acute deaths may occur in young birds, older birds may be chronically infected. Hepatosplenomegaly is common. Tumor-like lesions may be present in the liver and spleen of chronically infected birds. The parasite can be visualized microscopically as a clear notch in the nucleus of mononuclear cells and sometimes in erythrocytes.

B. HAEMOPROTEUS

DESCRIPTION

Haemoproteus belongs to the family Plasmodiidae and shares similarities with Plasmodium and Leucocytozoon. Over 120 species have been reported from birds, mostly in wild waterfowl, raptors, passerines, and others. Infection is host-specific. Species occurring in domestic poultry and pet birds include *Haemoproteus meleagridis* which has been diagnosed in domestic and wild turkeys, *H. columbae* and *H. saccharovi* in pigeons and doves and *H. nettionis* in waterfowl.

EPIZOOTIOLOGY

Biting flies and midges act as vectors. Sporogony occurs in the insect host and enters the avian host through fly bites. Schizonts (meronts) commonly infect pulmonary vascular endothelium or other visceral endothelial cells. Merozoites invade erythrocytes and mature. In some Haemoproteus species a second cycle of schizogony occurs involving the development of megaloschizonts in cardiac and skeletal muscles before merozoites invade red blood cells.

CLINICAL SIGNS, LESIONS & DIAGNOSIS.

Most infections are inapparent and remain undiagnosed. Clinical signs have been reported in quail, turkeys, pigeons, and some psittacines. Generally, only a small proportion of birds infected exhibit clinical symptoms. Turkeys experimentally infected with *H. meleagridis* have developed severe lameness, diarrhea, depression, emaciation and anorexia. Anemia and hepatomegaly have also been reported. Myopathy associated with megaloschizonts has been reported in wild turkeys where skeletal muscles contained fusiform cysts oriented parallel to the muscle fibers. Enlarged gizzards have been rarely reported in pigeons. Lameness, dyspnea, sudden death associated with edematous lungs and visceral organ enlargement has been rarely reported in Muscovy ducks infected with *H. nettionis*. Gametocytes and pigment granules can be visualized adjacent to the nuclei of red blood cells in blood smears stained with Giemsa or Wright's stain. Schizonts can be seen in vascular endothelial cells of the lung and visceral organs.

C. LEUKOCYTOZOA

DESCRIPTION

Leucocytozoonosis is an acute or chronic protozoal disease of birds, including turkeys, ducks, geese, guinea fowl and chickens. The disease was first observed in wild bird species and reported in turkeys in 1895. Relatively few reports of acute outbreaks of leucocytozoonosis in domestic poultry have been made during the last decade. The disease is generally clinically inapparent. Most acute outbreaks are in young

poultry whereas the chronic form of the disease usually occurs in older birds (breeding stock). Black flies (*Simuliidae*) and culicoid midges serve as intermediate hosts. The disease is most common in poultry housed near slow streams, shallow lakes or near marshy areas. Most outbreaks occur during the warmer seasons of the year when black flies are numerous. In the United States leucocytozoonosis occurs more frequently in southeastern states and in the upper Midwest.

EPIDEMIOLOGY

Birds appear to be the only hosts of most *Leucocytozoons*. The etiologic agents are *Leucocytozoons* but their classification may be inaccurate. Commonly applied names include: *L. smithi* (turkeys), *L. simondi* (ducks), *L. neavei* (guinea fowl) and *L. andrewsi* (chickens). Wild or domestic birds that survive the disease are inapparent carriers of leucocytozoons during the winter. During the warm seasons black flies (*Simuliidae*) and midges (*Culicoides*) feed on the carriers and become infected by the leucocytozoons. The parasites undergo sporogeny in the insects and pass to glands in their oral cavity. The insects then act as vectors and transmit leucocytozoons to young susceptible birds on which they feed. Birds that survive the disease become carriers in turn.

CLINICAL SIGNS, LESIONS & DIAGNOSIS

There is usually a sudden onset and numerous birds soon show signs. There is depression, anorexia, thirst, loss of equilibrium, weakness, and anemia. There may be rapid labored breathing. The course often is short and affected birds die or improve within a few days. Mortality varies but often is high. In birds that live a few days there may be splenomegaly, hepatomegaly and evidence of anemia. The most pronounced lesion often is splenic enlargement. Microscopically, gametes usually are seen within enlarged, distorted erythrocytes, leucocytes or both types of cells on blood smears stained with Wright or Giemsa stains. Histologic sections of the liver and brain often reveal megaloschizonts or schizonts.

D. PLASMODIUM

Plasmodium infections have been rarely reported in domestic pigeons, canaries, turkeys, penguins, falcons, bald eagles and cliff swallows. Plasmodia species are often not host-specific. The parasite causes a disease similar to malaria in man and is spread through the injection of infected blood by mosquito vectors. Parasites are found in red blood cells.

E. TRYPANOSOMES

Motile protozoa found in the plasma of numerous species of wild and domestic birds. Pathogenic significance in domestic poultry appears to be minimal or nil. A wide range of insect vectors includes mosquitoes, Culicoides (midges), Hippoboscids (black flies), mites and Simulids.

III. PROTOZOAL INFECTIONS OF THE DIGESTIVE TRACT

A. COCCIDIOSIS

DEFINITION

Avian coccidiosis is common protozoal disease of poultry and many other birds characterized by diarrhea and enteritis. Coccidiosis in poultry affects the intestinal tract, except for renal coccidiosis in geese.

OCCURRENCE

Coccidiosis is found in all segments of the poultry industry and has a world-wide distribution. The development of intensive confinement production systems has increased the economic significance of this disease. Subclinical disease has been recognized as having important impact on feed conversion in commercial meat-bird production and negative impacts on growth in layer pullets and breeders. The development of effective anticoccidial drugs and vaccines has made prevention of clinical disease and negative effects on performance possible. Where uncontrolled, coccidiosis remains an important cause of loss of production and mortality. Coccidiosis is a predisposing factor for necrotic enteritis caused by *Clostridia* perfringens. In gamebird production, coccidia control is important in preventing outbreaks of Ulcerative enteritis or "quail disease", caused by *Clostridia colinum*.

ETIOLOGY

1. Coccidiosis in chickens and turkeys is caused by the protozoal species of *Eimeria*. There are many species of *Eimeria* in chickens and turkeys, but not all are severe pathogens. Coccidia are host specific; hence do not pass among the various classes of poultry.

2. Coccidia have a direct but complex life cycle. Infection is by the fecal-oral route. Ingestion of infected feed, water, litter and soil results in infection. Briefly, when a sporulated (infective) coccidial oocyst is ingested, sporozoites are released to initiate asexual and sexual cycles that lead to development of thousands of new oocysts in the intestine. Oocysts are shed in the feces. These oocysts soon sporulate and then are infectious for other chickens. A single oocyst may give rise to as many as 100,000 progeny.

3. Coccidia produce lesions in the gut by destruction of the epithelial cells in which they develop and multiply, and by trauma to the intestinal mucosa and submucosa. Intestinal damage is directly proportional to the number of sporulated oocysts are ingested by a susceptible host.

4. Speciation of coccidia is by microscopic features of oocysts (size, shape, color, length, width), the preferred location of coccidia in the gut, the nature of lesions produced, pre-patent periods, sporulation times, etc. Identification frequently can be made with reasonable accuracy on only one or two features. Precise identification is of some value in selecting the most effective anticoccidial(s) for control.

5. If exposure is moderate, the birds develop short term immunity, often without clinical signs of infection. Current vaccines depend upon controlled exposure to *Eimeria* to stimulate immunity without clinical disease. Poultry maintain their immunity to a species of coccidia by repeated re-exposure. The host remains susceptible to coccidial species not yet encountered. Birds may be infected simultaneously with more than one species.

4. Oocysts are maintained in the litter of the poultry house or can be easily transported to poultry farms in blowing dust or on boots, shoes, clothing, crates, vehicle wheels, by other animals and insects. People are important vectors of coccidia. Wet litter and warm temperatures facilitate sporulation and precipitate outbreaks of coccidiosis.

COCCIDIA OF CHICKENS

Nine species of Eimeria have been described in chickens: *E. acervulina*, *E. necatrix*, *E. maxima*, *E. brunetti*, *E. tenella*. *E. mitis*, *E. mivati*, *E. praecox* and *E. hagani*. Clinical disease is determined by the species of the infecting coccidia. Less pathogenic species produce few or no signs. The more pathogenic species often cause diarrhea which may be mucoid or bloody. Dehydration often accompanies the diarrhea. Diarrhea and dehydration are soon followed by ruffled feathers, anemia, listlessness, weakness, retraction of the head and neck and somnolence. Growth rate is often adversely affected. In laying hens coccidiosis is usually manifested by a drop in egg production. Depigmentation of the skin may be apparent in well established cases. Morbidity and mortality within a flock may vary greatly, but both can be very high.

A. *Eimeria acervulina*.

E. acervulina is a moderately severe pathogen causing enteritis in the anterior one third of the intestinal tract [Fig. 1; Coccidiosis; NCSU]. In severe cases lesions may extend further down the tract. The enteritis can be mild to severe and cause thickening of the mucosa. May affect skin pigmentation due to malabsorption of carotenoids and reduce feed conversion. Transverse white to gray striations are visible in the mucosa [Fig 2; Coccidiosis; Univ Montreal]. Oocysts in mucosal scrapings are moderate in size and egg shaped. This type of coccidiosis occurs rather frequently in older birds. This location is also favored by 4 less pathogenic species i.e. frequently multiple species will be present, obscuring the diagnosis.

B. *Eimeria necatrix.*

Eimeria necatrix causes severe enteritis characterized by congestion, hemorrhage, necrosis and bloody feces in the middle one third of the intestine [Fig. 3; Coccidiosis; AAAP]. The intestine often is markedly dilated, inflamed and thickened. White to yellow foci (very large schizonts) and petechial hemorrhages may be seen through the serosa of the unopened gut [Fig. 4; Coccidiosis; NCSU]. Oocysts develop only in the ceca, and these may not be numerous; moreover, mortality may precede the appearance of oocysts in the feces. Often causes high mortality. Often causes disease in commercial broilers or layer pullets.

C. *Eimeria maxima*.

Eimeria maxima is moderately pathogenic. It causes mild to severe enteritis sometimes with thickening of the intestinal wall and marked dilatation (note the resemblance to the lesions of E. necatrix above) in the middle one third of the intestine [Fig. 5; Coccidiosis; AAAP]. Intestinal content may be bloody. Very large oocysts, often with a golden color. Subclinical infections may impede absorption and result in poor skin pigmentation.

D. *Eimeria brunetti.*

Eimeria brunetti causes enteritis in the lower small intestine, rectum and proximal cecum. In severe cases, a fibrinous or fibrinonecrotic mass of debris may cover the affected mucosa or produce caseous cores in the cecum or rectum. Large oocysts, each with a polar granule. *E. brunetti* is a moderately severe pathogen that can produce moderate mortality, loss of weight gain, and poor feed conversion.

E. *Eimeria tenella*.

Eimeria tenella is one of the most pathogenic coccidia of chickens. It causes a marked typhlitis [Fig. 6; Coccidiosis; AAAP] with occasional involvement of the adjacent areas of the intestine. Blood is often apparent in the ceca and feces in early cases; later, cheesy cecal cores may be found. Large clusters of schizonts may be seen in the ceca. *E. tenella* can cause high morbidity, mortality and reduced weight gain in commercial broilers or layer pullets.

F. *E. mitis*.

Affects the lower small intestine which may appear pale and flaccid. Pathogenic effects on weight gain and morbidity have been demonstrated.

G. E. mivati.

Early lesions appear in the duodenum and later the infection extends to the ceca. Causes reduced weight gain and morbidity.

H. E. praecox.

Causes watery intestinal contents with mucus and mucoid casts in the duodenum. There may be reduced weight gain, loss of pigmentation, dehydration and poor feed conversion.

I. E. hagani.

Reportedly causes watery intestinal contents and catarrhal inflammation. The taxonomic status of this species is in doubt.

COCCIDIA OF TURKEYS

Seven species of *Eimeria* have been described in turkeys in the USA. The 4 pathogenic species of *Eimeria* in turkeys are: *E. adenoeides*, *E. meleagrimitis*, *E. gallapovonis*, and *E. dispersa*. Nonpathogenic

species include: *E. innocua, E. mileagridis*, and *E. subrotunda*. Coccidiosis in turkeys resembles the disease in chickens but the diarrhea is seldom bloody and poults over 8-weeks-old are seldom severely affected.

A. *Eimeria meleagrimitis.*

Eimeria meleagrimitis causes spotty congestion and petechiae from duodenum to ileum, dilation of jejunum, and mucosal casts [Fig. 7; Coccidiosis; NCSU] in the anterior two thirds of the intestine. Lesions are most severe in the jejunum and from there extend anteriorly and posteriorly. Oocysts in mucosal scrapings are small in size (19.2 x 16.3 microns) and ovoid. This species is considered a moderate to severe pathogen. Mortality, morbidity, weight loss, dehydration and general unthriftiness may occur in young poults as a result of infection. Nonpathogens *E. innocua* and *E. subrotunda* may also be found in this region.

B. *Eimeria dispersa*.

Eimeria dispersa produces a cream-colored serosal surface, dilation of intestine and yellowish mucoid feces in the middle one third of the intestine. Lesions are principally located in the midgut region, but some infection may extend from the duodenum to the cecal necks. Oocysts are large (26.1 x 21.0 microns) and broadly ovoid. The oocyst wall is distinctively contoured and lacks the double wall common to other species. The prepatent period is the longest of the turkey cocci, 35-120 hours. This species is considered mildly pathogenic but can cause reduction of weight gain and diarrhea in young poults.

C. *Eimeria gallapovonis.*

Eimeria gallapovonis causes edema, ulceration of mucosal ileum, yellow exudate, and flecks of blood in feces. Lesions are principally located in the posterior one third of the intestine (ileum and large intestine) but may extend into the posterior jejunum and the ceca. Oocysts are small (17.1 x 17.2 microns), elongated and ellipsoidal. The prepatent period is 105 hours. This species can cause high mortality in young (2 to 6-week-old) poults.

D. *Eimeria adenoides*.

Eimeria adenoides affects the posterior one third of the intestine and is responsible for liquid feces with mucus and flecks of blood. There is edema and swelling of the cecal and/or intestinal wall. Cecal contents often contain hardened mucosal debris appearing as loose whitish cecal cores [Fig. 8; Coccidiosis; NCSU]. Lesions are principally located in the ceca but generally extend to the lower small intestine and cloaca. Oocysts are ellipsoidal and very elongated (25.6 x 16.6 microns). This species is one of the most pathogenic of the turkey coccidia. Infections in young poults may produce high mortality and infection in older turkeys can cause considerable weight loss. *E. meleagridis* may also be found in this area.

DIAGNOSIS

1. Gross necropsy should be performed on fresh (< 1 hour) dead birds typical of the flock. Post mortem changes can quickly obscure gross lesions.

2. Diagnosis is made primarily on the basis of clinical signs and the appearance and location of gross intestinal lesions. Large numbers of oocysts may be present in mucosal scrapings from affected birds. Histologic examination of the intestine can confirm the presence of asexual or sexual stages of coccidia (sporozoites, merozoites, schizonts). A history indicating recent flock exposure to a large source of sporulated oocysts may be helpful.

3. Subclinical infection (coccidiosis) is common. The presence of oocysts in the feces does not justify a diagnosis of clinical disease.

4. Coccidiosis frequently occurs in association with other avian diseases, such as necrotic enteritis, ulcerative enteritis, salmonellosis and histomoniasis. Immunosuppressive diseases may increase the severity and incidence of clinical disease

CONTROL

1. Anticoccidial Compounds in Feed. The most common method of control. Coccidia eventually become resistant to most anticoccidials, therefore rotation of types may be used to maintain efficacy. No anticoccidial is highly effective against all species of coccidia although some are effective against multiple species. Mild exposure of birds while being fed some anticoccidials stimulates an immune response ("leakage"). A wide range of anticoccidials are approved for prevention of coccidiosis, although not all are commercially available. Examples include: amprolium, monensin, clopidol, buquinolate, robenidine, lasalocid, halofuginone, salinomycin, and narasin.

2. Immunization. Commercial coccidiosis vaccines are available. Planned exposures of young chicks or poults to small numbers of oocysts by coarse spray at the hatchery or in feed, water or gel blocks have been used successfully. The number of oocysts of each *Eimeria* species provided in the vaccine is critical to initiating immunity without causing clinical disease. Some vaccines contain drug-sensitive strains of *Eimeria,* facilitating the establishment of drug-sensitive populations and extending the usefulness of chemical anticoccidials. Other vaccines contain attenuated lines of *Eimeria.* Products are also being developed for *in ovo* administration. These products consist of oocysts, sporozoites, merozoites or antigenic proteins produced by recombinant DNA technology.

3. Natural Exposure. If chickens are exposed to modest numbers of oocysts in their environment, they develop immunity to the species of coccidia represented. Exposure must be moderate or clinical signs will appear. Exposure can be limited if dry litter conditions (unfavorable for rapid sporulation of oocysts) are maintained. Wet litter (including wet areas around waterers) is especially to be avoided. This practice is rarely used in large commercial operations.

TREATMENT

Prevention is emphasized. However, chemical agents widely used for treatment include amprolium, sulfadimethoxine, sulfaquinoxaline, sulfamethazine. Sulfas should not be used in layers. Required withdrawal times are usually required prior to marketing. Increasing vitamins A and K in feed or water may reduce mortality and hasten recovery, respectively.

B. HEXAMITIASIS

DEFINITION

Hexamitiasis is a protozoal disease characterized by catarrhal enteritis and by foamy or watery diarrhea.

The etiologic agent in turkey poults and most other susceptible birds is *Hexamita meleagridis*. The etiologic agent in pigeons is *Hexamita columbae*.

HISTORICAL INFORMATION

1. For many years hexamitiasis was confused with trichomoniasis. In 1938 Hinshaw and others first clearly identified hexamitiasis and its etiologic agent. Rather extensive losses were attributed to hexamitiasis during the early development of the turkey industry, in range flocks.

EPIDEMIOLOGY

1. In turkeys, Hexamitiasis usually is seen in 1 to 9-week-old poults. Hexamitiasis also occurs in gamebirds (pheasants, quail, chukar partridge, etc.), peafowl and ducks. Pigeons have their own distinct form of hexamitiasis.

2. The parasite is shed in feces which contaminate feed, water and range. Recovered birds are often inapparent carriers. Susceptible birds get the organism by ingestion. Interspecies transmission, e.g., from game birds to turkey poults, occurs readily. This method of transmission is more likely in poults on range.

3. When consecutive broods of poults are raised in the same facilities, especially if sanitation is poor, there appears to be an increase in the number of Hexamita in each brood and a corresponding increase in signs of the disease in the later broods.

4. The disease has been reported in many different countries and states. It usually occurs during the warmer months of the year and on facilities that maintain a poor standard of sanitation. Heximitiasis is currently rare in commercial production, but may be seen in backyard or ornamental flocks.

CLINICAL SIGNS

1. Initially the affected birds are nervous and very active. They chirp excessively, shiver, crowd around any heat source and have subnormal temperatures. There is watery or foamy diarrhea and the birds dehydrate rapidly.

2. Later the birds are more depressed, stand with their heads retracted, feathers ruffled and wings drooping. Terminally the birds go into a coma, struggle and die. Terminal signs appear to be related to hypoglycemia.

3. Morbidity is high. Mortality varies with age and the quality of husbandry provided. Mortality may be very high (75-90%) in young birds that are poorly housed and which receive no treatment.

DIAGNOSIS & LESIONS

1. The cadaver is dehydrated. The intestine is flabby, may have areas of bulbous dilatation and contains excessive mucus and gas. The proximal one-half of the intestine is inflamed. Cecal tonsils may be congested.

2. *Hexamita meleagridis* is found in the crypts of Lieberkuhn, especially in the duodenum and upper jejunum of infected birds, including older carriers. The protozoan is roughly 3 by 9 microns, has 8 flagella and two nuclei that resemble eyes. It moves with a rapid, darting motion.

3. Duodenal scrapings should be examined from a freshly killed, infected bird using reduced light or phase contrast microscopy. Many *Hexamita* in the upper intestine suggest hexamitiasis. If only recently dead birds are available, warm saline added to the scrapings may revive enough of the *Hexamita* for identification. Dead *Hexamita* are difficult to identify so it is preferable to submit live birds for necropsy.

4. The history, signs and lesions are suggestive of the disease but must be differentiated from coronaviral enteritis, paratyphoid infection, trichomoniasis and blackhead.

CONTROL

1. Hexamitiasis seldom is reported now. Improved sanitary practices and management on turkey farms may have reduced the incidence or antihistomonal chemicals routinely given for blackhead control may also be controlling hexamitiasis.

2. Short periods of depopulation combined with thorough cleaning and disinfection of buildings will greatly reduce the population of *Hexamita*.

3. Clean and disinfect feeders and waterers and keep them on large wire platforms so concentrations of droppings are not available to the birds.

4. Do not hold possible carriers from old flocks. Raise only birds of the same age group together.

5. Prevent contact of poults with captive or wild gamebirds. Avoid using gamebird ranges for poults.

TREATMENT

No current medication is approved for food animals in the United States. Antiprotozoal drugs such as Metronidazole may be considered in non-food animals. Supportive treatment such as increasing the brooding house temperature to a point where the birds appear comfortable may be beneficial for young birds.

C. HISTOMONIASIS (Blackhead; Enterohepatitis)

DEFINITION

Histomoniasis is a protozoal disease caused by *Histomonas meleagridis* affecting turkeys, chickens, peafowl, grouse, quail and possibly other gallinaceous birds characterized by necrotizing lesions involving the ceca and liver.

OCCURRENCE

Histomoniasis occurs most frequently in exposed, unmedicated turkeys, especially turkeys under 3 months of age. It also occurs in chickens and in many captive game birds. Young birds are more frequently and severely affected. The disease is infrequent in areas where there are no earthworms i.e., where there is dry, sandy soil and where there are no vectors for transmission of histomonads. Histomoniasis occurs infrequently where proper measures are taken for its prevention.

HISTORICAL INFORMATION

1. Histomoniasis once limited the expansion of the turkey industry. Prior to development of safe antihistomonal drugs, it could be controlled only by cumbersome and relatively ineffective measures designed to prevent exposure of turkeys to the embryonated ova of cecal worms (*Heterakis gallinarum*).

2. Significant growth of the turkey industry occurred after safe antihistomonal drugs were developed. The disease is now uncommon and these drugs are no longer used routinely. It occurs sporadically when turkeys are raised where chickens were previously located. The disease is still common in chickens but its effect on production is mild and rarely recognized. Histomoniasis remains an important cause of death among other galliformes including peafowl, pheasant and quail.

ETIOLOGY

1. The etiologic agent is the protozoan *Histomonas meleagridis*, assisted by secondary bacteria. In the experimental absence of bacteria, the histomonad appears not to be pathogenic. *H. meleagridis* is a flagellate in the lumen of the cecum but assumes an ameboid form in tissue.

2. A larger histomonad distinguished by its 4 flagella, *H. wenrichi*, also occurs in the cecum but is not pathogenic.

EPIDEMIOLOGY

Transmission of *H. meleagridis* to susceptible birds is possible via three routes:

1. Ingestion of fresh feces. This route probably is relatively unimportant except for spread within a flock.

2. Ingestion of embryonated cecal worm ova containing the protozoan. Within these resistant ova the histomonad can survive for years. *H. meleagridis* is liberated in the intestine when ingested ova hatch, then invades the cecal wall and initiates the disease.

3. Ingestion of earthworms containing cecal worm larvae within their tissues. Earthworms serve as transport hosts for the cecal worm and the cecal worm acts as a transport host for the histomonad. Infection results after the cecal worm larvae are liberated during digestion.

DIAGNOSIS

1. Diagnosis can be made on the basis of clinical signs and characteristic lesion. Typical well-developed lesions are pathognomonic [Fig. 1; Histomoniasis; NCSU].

2. In turkeys histomoniasis appears 7-12 days after exposure. Initially there is listlessness, moderate anorexia, drooping wings and yellow ("sulfur colored") feces. Head parts may be cyanotic ("blackhead") although they often are not. In chickens with histomoniasis there may be some blood in the feces.

3. Later the affected turkey is depressed and stands with its wings drooping, eyes closed, head drawn close to the body. Emaciation is common in chronic cases, usually in older birds. In young turkeys morbidity and mortality are high, up to 100%. Older birds tend to be more resistant.

4. Gross lesions. Bilateral enlargement of the ceca with thickening of the cecal walls [Fig. 2; Histomoniasis; UC Davis]. The mucosa usually is ulcerated. The ceca often contain caseous cores which are yellow, gray or green and may be laminated. In chronic cases the cores may have been expelled. Peritonitis occurs when the cecal wall becomes perforated.

5. Liver contains irregularly-round, depressed, target-like lesions that vary in color [Fig. 3; Histomoniasis; UC Davis]. They often are yellow to gray but may be green or red. They vary greatly in diameter but often are 1-2 cm and may coalesce to produce larger lesions.

6. Lesions may not be entirely typical in birds under treatment, less susceptible avian species or young turkeys in the early stages of the disease. In most infected flocks typical lesions usually can be found if an adequate number of birds are examined. In quail, cecal lesions may not occur even though mortality is high.

7. Microscopically, histomonads can be found in the inflamed cecal walls and necrotic foci which develop in the liver. In birds killed for necropsy the agent sometimes can be identified in smears from the ceca or in scrapings from the margin of hepatic lesions.

CONTROL

1. Histomoniasis usually can be prevented by adding antihistomonal drugs to the ration in proper dosage. No current preventive medication is approved in the U.S. Histostat (nitarsone) is still used in poultry outside the U.S. In quail, a cholinesterase inhibiting carbamate (Sevin) increases susceptibility to histomoniasis.

2. Control by the use of antihistomonal drugs may fail unless reasonably good sanitation is practiced.

3. Other measures that assist in control follow:

    A. Do not keep chickens and turkeys (or other susceptible birds) on the same farm.

    B. Do not use chicken ranges for turkeys or other susceptible birds unless those ranges have been free of chickens for at least 4 years.

    C. *H. meleagridis* is quickly destroyed by disinfectants and drying unless protected within earthworms or within the cecal worm ova. Avoid exposure to vectors. If possible, raise susceptible birds on sandy, dry, loose soil. Prevent access to earthworms after rains. In range birds, rotate ranges periodically if possible. Some operators with small lots replace the top few inches of soil every few years using power equipment or plow the lots to reduce the number of cecal worm ova and other pathogens.

    D. Reduce access of birds to their own droppings or to feed and water contaminated with droppings. Place feeders and waterers on large wire platforms or keep them outside of the lot but accessible through a wire fence.

TREATMENT

There is currently no approved medication for treatment of histomoniasis in food animals. Small groups of birds not being raised for consumption can be effectively treated individually with metronidazole at a dose of 30 mg/kg orally SID for 5 days. Antihelmentic treatment may help suppress the population of cecal worms.

D. TRICHOMONIASIS (Canker in pigeons and doves; Frounce in falcons)

DEFINITION

Trichomoniasis is caused by *Trichomonas gallinae*, a flagellated protozoan. Pathogenicity varies greatly by strain. The disease is characterized by raised caseous lesions in the upper digestive tract, but may extend to other tissues. Pigeons, doves, turkeys, chickens and raptors are commonly affected.

HISTORICAL INFORMATION

Trichomoniasis was once recognized as an important disease of turkeys and chickens, especially of ranged turkeys, but is seldom reported now. Conversely, trichomoniasis in pigeons and doves continues to be a common and significant disease. Trichomoniasis may have played a role in the extinction of the carrier pigeon. Trichomoniasis can be a consequential disease in raptors.

EPIDEMIOLOGY

1. The organism is fragile in the environment and transmission occurs only through contact with infected oral secretions or through water contaminated by oral secretions of carriers. Pigeons are believed to be the natural hosts and primary carriers. The prevalence of the infection is near 100% for adult pigeons. Carrier birds show no signs or lesions.

2. Pigeons and doves transmit trichomonads to their young during feeding of regurgitated partially digested crop content (pigeon milk). Transmission in raptors (hawks, owls, eagles, etc.) And their young, is through ingestion of infected prey. Turkeys and chickens probably contract the disease after consuming stagnant surface water containing *T. gallinae*. Other disease may predispose turkeys to clinical trichomoniasis.

3. In established flocks or lofts, trichomoniasis may only be noted as a clinical disease after introduction of a more virulent strain of *T. gallinae* by new birds or exposure to wildlife carriers.

DIAGNOSIS

1. Typical signs and lesions are very suggestive of the diagnosis. In pigeons, doves and raptors, yellow plaques or raised cheesy masses involve the upper digestive tract. Masses often are large and conical or pyramidal and can be surprisingly invasive in soft tissue. Lesions are usually most extensive in the mouth, pharynx or esophagus but may occur at other sites including the crop, proventriculus or sinuses. In raptors, lesions may also occur in the liver and are accompanied by peritonitis.
2. Infected squab (baby pigeons) become depressed and die at 7-10 days of age. Lesions of the oral cavity are most common but may also occur in the nasal turbinates and brain. The infection may become systemic, with lesions in the liver and other visceral organs.

3. Adults pigeons, doves and raptors often have difficulty in closing their mouth because of lesions in the oral cavity. They drool and make repeated swallowing movements. Watery eyes may be apparent in occasional birds with lesions in the sinuses or periorbital area. Rare cases with penetrating cranial lesions may show signs of central nervous disturbances, including loss of balance.

4. Lesions are similar in turkeys but frequently are found only in the crop and upper or lower esophagus. Occasionally the proventriculus contains lesions. Infected turkeys often have a gaunt appearance with a hollowed area over the crop. Swallowing movements often are apparent and infected birds may have an unpleasant odor (“sour crop”).

5. Morbidity and mortality in affected birds varies but can be quite high. Demonstration of trichomonads in the oral fluids may not be significant in the absence of lesions since many normal birds have some trichomonads. Small plaques in the mucosa should not be confused with pox or candidiasis.

CONTROL

1. Eliminate any known infected birds and all suspected carriers. If possible, depopulate at regular intervals and thoroughly clean and disinfect the premises. Add no birds to an established flock since they may be carriers of a more virulent strain. Permit no contact among pigeons, doves and susceptible poultry.

2. Provide a source of clean, fresh water, preferably running water being replaced constantly. Eliminate all sources of stagnant water. Disinfect watering containers and water lines regularly (e.g. chlorine).

3. Avoid feeding infected pigeons and doves to captive raptors.

TREATMENT

There is currently no approved medication for treatment of trichomoniasis in food animals. Birds not being raised for food can be effectively treated individually with metronidazole (Flagyl) at a dose of 30 mg/kg orally SID for 5 days, or with Enheptin.

IV. OTHER PROTOZOAL INFECTIONS

A. CRYPTOSPORIDIOSIS

DEFINITION

Cryptosporidiosis is a protozoal disease, characterized in avians by acute or chronic disease of the respiratory or digestive tracts. Two avian species of *Cryptosporidium* are recognized, based upon tissue (site) specificity and organism morphology. *Cryptosporidium meleagridis* infects only the small intestine, while Cryptosporidium *baileyi* infects the digestive tract (especially the bursa of Fabricius and cloaca) and can also infect the respiratory tract. The organism has a coccidian life cycle that is completed in an intracellular, but extracytoplasmic location on the microvillous border of epithelial cells.

HISTORICAL INFORMATION

1. Dr. Earnest Tyzzer first described infections by *Cryptosporidium muris* (gastric mucosa) and *C. parvum* (small intestine) in laboratory mice (1907).

2. Tyzzer also was the first to describe and report (1929) infections by *Cryptosporidium* in the cecum of chickens, but no signs of disease were noted. The first report of avian morbidity and mortality caused by *Cryptosporidium* (1955) involved the distal small intestine of turkeys. The first report of respiratory cryptosporidiosis in any species also involved turkeys (1978).

3. Cryptosporidiosis was recognized in humans in 1976, and was soon identified as a life-threatening enteric disease associated with acquired immune deficiency syndrome (AIDS) and other immunosuppressive disorders.

EPIDEMIOLOGY

The disease is spread through ingestion or inhalation of oocysts shed in feces or in respiratory secretions. Oocyst will remain viable in the environment for long periods. Infections by *C. meleagridis* and *C. baileyi* have been reported in chickens, turkeys, quail, pheasants, peafowl, psittacines, finches, and waterfowl. Avian species of *Cryptosporidium* spp. do not appear to infect mammals, including man, i.e. no known public health threat exists. Other species of *Cryptosporidia* have documented zoonotic potential, passing between man and other mammalian species, reptiles, amphibians, and fish. Cryptosporidiosis can

be a severe and life-threatening disease in an immunosuppressed host, but immunosuppression is not a requirement for either infection or disease. Some infections are asymptomatic.

CLINICAL SIGNS

1. Clinical signs of cryptosporidiosis are not specific for the disease and other pathogens are frequently involved. Involvement of the small intestine causes diarrhea that may be fatal in young turkeys, quail, and psittacines. Mortality in young quail may exceed 95%. Digestive tract infections in broilers may cause reduced carcass pigmentation.

2. Respiratory cryptosporidiosis produces signs related to the site of infection: naso-ocular discharges, swollen sinuses, coughing, sneezing, dyspnea, and rales. Fatalities may occur with deep lung and air sac infections. Concurrent infections by other respiratory pathogens are the rule rather than the exception.

LESIONS

1. The small intestine becomes dilated with fluid. Cecae may be distended with foamy, fluid contents. Histopathology includes shortening of villi with necrosis and loss of enterocytes from the villous tips, and the presence of numerous organisms in the brush border of the mucosal epithelium. Organisms may also be found in the cecal tonsil mucosa, and in the mucosa of the bursa of Fabricius and cloaca. The bursal mucosal epithelium may be hyperplastic with heterophilic infiltration.

2. Respiratory cryptosporidiosis causes gray or white mucoid exudate on the mucosal surface infected: conjunctiva, sinuses or nasal turbinates, trachea, bronchi, or air sacs. Marked enlargement of the infraorbital sinuses may occur in turkeys. Infected lungs may appear gray and firm lungs.

DIAGNOSIS

1. Most cases of cryptosporidiosis are recognized during histologic examination of either biopsy specimens or tissues collected at necropsy. The organisms are basophilic with H&E stain, are 2 to 4 microns in diameter, and are intimately associated with mucosal brush borders.

2. Cytology can be used to confirm the diagnosis. Touch impressions of mucosal surfaces can be examined by phase-contrast or interference phase-contrast microscopy. They can also be air dried and stained with red carbon fuschin or with Giemsa stain. Differentiation from yeasts may be necessary with Giemsa stain.

3. Flotation techniques for oocyst identification from feces include Sheather's sucrose solution, zinc sulfate (33% to saturated), and sodium chloride (36% to saturated). Oocysts are 4 microns in diameter, with a thick cell wall, prominent residual body, smaller globular dense bodies, and curved sporozoites.

4. Concurrent respiratory tract pathogens identified with respiratory cryptosporidiosis include *Escherichia coli*, *Pasteurella multocida*, Newcastle disease virus, adenovirus, infectious bronchitis virus, and reovirus.

5. Most broiler chickens with bursal cryptosporidiosis have had infectious bursal disease, but it is not a pathogenic requirement. Among other avian hosts, diseases that occur with cryptosporidiosis include salmonellosis, candidiasis, turkey viral hepatitis, intestinal reovirus infections, and other parasites.

CONTROL

1. Reduction or elimination of oocysts from the environment is the primary means of controlling the spread of *Cryptosporidium*.

2. Stringent cleaning followed by disinfection with either steam or autoclaving is necessary for oocyst destruction to break the cycle of infection. Most disinfectants will not inactivate oocysts. Either formol saline (10%) or 5% ammonia will inactivate oocysts after a minimum of 18 hours contact. Undiluted commercial bleach is also effective.

TREATMENT

1. Drugs used for coccidiosis and other protozoan diseases have been ineffective for cryptosporidiosis in animals. Therapeutic agents are minimally effective for reducing the severity of clinical disease and for interfering with replication of the organism.

2. Supportive therapy for individual animals is a consideration, but with multiple animals, continued shedding of infective organisms may infect others in a flock, cage, or aviary. Treatment and control of concurrent or secondary infections reduces mortality.

B. SARCOSPORIDIA

DEFINITION

A parasitic infection caused by species of the genus Sarcocystis first described by Lankester in 1882. Sarcocystis has a world-wide distribution but is rarely reported in domestic fowl. The incidence is high in wild waterfowl and some passerines, such as grackles. Not an economically important disease in domestic poultry, but does occur extensively in wild ducks and gamebirds. Although not a public health hazard, the parasite is killed by cooking or freezing. Most affected carcasses are discarded for esthetic reasons.

ETIOLOGY

1. At least five species of Sarcocystis have been identified. *S. horwathi* is regarded as the etiologic agent in chickens (also call *S. gallinarum*). *S. anatina* and *S. rileyi* (*Balbiani rileyi*) are found in ducks.

2. All species of Sarcocystis have similar developmental stages and an obligatory two host life cycle. Sarcocysts in cardiac, smooth, or skeletal muscle tissues are eaten by a definitive host, releasing cystozoites which penetrate the intestinal wall and develop in the subepithelial tissue. Oocysts are produced and shed in the feces fully sporulated. When sporocysts are ingested by the intermediate host they go through a series of asexual reproductive cycles in endothelial cells of various organs. Merozoites are eventually released, invade muscle tissue and eventually develop into the macroscopically visible sarcocysts (1.0-6.5 x 0.48-1.0 mm), each containing numerous banana-shaped cystozoites (bradyzoites (2-3 x 8-15 microns).

DIAGNOSIS

Usually based on gross lesions, i.e. the presence of large, pale sarcocysts in the muscle tissue [Fig. 1; Sarcosporidia; Cornell U], arranged in parallel with the muscle fibers. Diagnosis can be confirmed histologically.

COMMON INTERNAL PARASITES OF POULTRY

Parasite	Common name	Description	Lifecycle	Site of Infection	Lesions/Clinical signs	Comments
Oxyspirura sp.	yeworm	Nematode up to 2.0 cm	Indirect or direct. Cockroaches are intermediate hosts for some species.	Conjunctival sac, often beneath the nictating membrane, or in nasolacrimal duct	conjuncitivis, opthalmitits, protrusion of nictitating membrane, adherence of eyelids	Occurs in all types of poultry and wild birds
Syngamus trachea	Gapeworms	Red nematodes up to 2.0cm long, forked appearance, male and female in permanent copulation	Indirect or direct earthworms, slugs, snails are intermediate hosts for many species	Trachea and possibly large bronchi.	Gasping, gaping, dyspnea and head shaking	Occurs in all species of poultry. Generally difficult to treat. *Cyathostoma bronchialis* may cause gaping in domestic geese.
Capillaria sp. *C. annulata* *C. contorta* *or* *Gongylonema ingluvicola*	Cropworms	Thin, threadlike nematodes up to 60 mm long	Direct	Crop or crop and esophagus	Infected tissues become thickened and inflamed. Causes malnutrition, emaciation and severe anemia	Occurs in all species of poultry. Best identified in mucosal scrapings, difficult to see grossly
Ascaridia galli	Roundworms	Large, thick, yellow-white nematodes	Direct Eggs ingested by insects remain infective	Lumen of small intestine	Weight loss, potential intestinal blockage and blood loss	Affects chicken, turkey, dove, duck and goose. Fowl > 3 mo. develop immune resistance. Similar to *A. dissimilis* reported in turkeys and *A. columbae* in doves, pigeons
Capillaria sp.	Intestinal worms	Hairlike nematodes 6 to 25 mm long	Direct or indirect with earthworm intermediate host	Mucosa of small intestine and ceca	Huddling, emaciation, diarrhea, hemorrhagic enteritis, or death. Thickened upper intestine with catarrhal exudate	Occur in many poultry species

COMMON INTERNAL PARASITES OF POULTRY

Parasite	Common name	Description	Lifecycle	Site of Infection	Lesions/Clinical signs	Comments
Dispharynx sp. *Tetrameres sp.* *Cyrnea sp.*	Proventricular worms	Grossly visible, 3 to 18 mm long nematodes	Indirect. Grasshoppers, cockroaches, sowbugs and pillbugs are intermediate hosts	Mucosa and glands of proventriculus	Diarrhea, emaciation and anemia. Mucosal ulceration, necrosis, hemorrhage and swelling	Occur in poultry and other birds
Cheilospirura sp. *Amidostomum sp.*	Gizzard worms	Small, broad nematodes up to 25 mm long	Indirect. Grasshoppers, beetles, weevils and sandhoppers are intermediate hosts	Under the gizzard lining	Muscular wall of gizzard may be sacculated or ruptured. Mucosa ulcerated, necrotic or sloughed.	Numerous poultry species.
Heterakis gallinarum	Cecal worms	Small white nematode up to 15 mm long	Direct. Eggs may be ingested by earthworms where they hatch and live for months	Most numerous in the tips of the ceca	Marked inflammation and thickening, nodules in cecal walls. May see hepatic granulomas	Affects numerous poultry species. Acts as carrier for *Histomonas meliagridis* (blackhead)
Cestodes *Raillietina sp.* *Choantaenia sp.,* *Davainea sp.,* *Hymenolepis sp.* *Amoebotaenia sp.*	Tapeworms	Flattened, ribbon-shaped, segmented. Usually grossly visible	Indirect. Many invertebrate intermediate hosts; flies, snails, beetles, earthworms, crustaceans	Intestine	Small lesions at point of attachment. Enteritis proportional to degree of infection	Usually infect a definitive host, but are not host specific.
Trematodes	Skin fluke	Hemispherical, flattened shape up to 5.5 mm long	Requires a molluscan intermediate host and may use a second intermediate host	Skin, usually near vent	Forms cutaneous cysts, usually with 2 flukes in vent area	Not host specific. Affect poultry and wild birds. Other flukes may invade oviduct, digestive organs, kidney, circulatory organs and eye

VITAMIN DEFICIENCIES

Chickens are particularly susceptible to vitamin deficiencies because they get little or no benefit from microbial synthesis of vitamins in their digestive tract. These birds also have a fast growth rate and are raised in modern management conditions hence submitted to various stresses. Their feed can be shipped or stored for various periods of time, at different temperatures, submitted to oxidative stress, these accounting for possible potency loss. For the above reasons, increased dietary vitamin levels are required in commercial diets in order to optimize growth and performances, when compared to the minimal requirements of the NRC established to prevent clinical signs in ideal conditions.

If birds received vitamins below requirement levels for any length of time, classical deficiency pathologies will develop at various speeds, depending on the age, the quantity of vitamin passed on from the breeder hen, and the vitamin storage level. For example, clinical signs will appear rapidly in young chicks, with the young embryo being the most sensitive model. Problems with water soluble vitamins such as B are soon observed because they are not stored to any extent, even excreted via the urine if in excess, while fat soluble vitamin deficiencies can take longer to develop because of adipose tissue and liver storage in older birds.

Since it is today a common practice to include vitamins and minerals in feed composite premixes, we are less likely to observe individual classical vitamin deficiencies, such as the ones described below, unless one has been omitted from a premix. It is therefore not unusual to see a situation where the entire premix has been inadvertently excluded with as a consequence, the development of a complex array of clinical signs.

BIOTIN DEFICIENCY

DEFINITION

Biotin is common in poultry feedstuffs, yet recent evidence concludes some of this biotin may be biologically unavailable. Turkeys seem more sensitive to biotin deficiency than chickens.

ASSOCIATED DISORDERS

Biotin acts as a coenzyme in carboxylases which are involved in lipid and carbohydrate metabolism. First signs of biotin deficiency are related to reduced cell proliferation.

1. Dermatitis. Biotin deficiency causes an exudative dermatitis around the mouth and eyes [Fig. 1; Biotin deficiency; NCSU], and on the feet and legs [Fig. 2; Biotin deficiency; NCSU], which must be differentiated from pantothenic acid deficiency. In turkeys dermatitis and cracking of foot pads occurs (foot pad dermatitis), soon followed by chondrodystrophy.

2. Chondrodystrophy (old name = perosis). A growth-plate disorder, where long bones are shorter than normal, is a sign of biotin deficiency in growing chickens and turkeys. Biotin may also play a role in varus leg deformities because it affects bone remodeling.

3. Fatty liver and kidney. Fatty liver and kidney syndrome in broiler chickens is a biotin-responsive disease. Chicks exhibit depressed growth, hypoglycemia, increased plasma-free fatty acids, and increased ratio of C 16:1 to C 18:0 fatty acids, in liver and adipose tissue. Necropsy reveals pale liver and kidney with accumulation of fat It has been suggested that biotin deficiency may impair gluconeogenesis by decreasing the biotin-containing enzyme pyruvic carboxylase and, therefore, increasing the conversion of pyruvate to fatty acids.

4. Fatty liver syndrome. Biotin may also be a complicating factor in fatty liver syndrome in laying hens.

5. Embryonic abnormalities and reduced hatchability. Biotin is essential for embryonic development. Embryos from deficient hens display parrot beak, chondrodystrophy, micromelia and syndactyly. Two peaks of embryonic mortality occur: one during the 1st week and another during the last 3 days of incubation.

TREATMENT

Water-soluble vitamins are readily available and may be administered as needed. To avoid problems, most starter and grower rations are supplemented with 0.1 to 0.3 mg/kg of biotin.

RIBOFLAVIN DEFICIENCY

(Vitamin B2 Deficiency; Curled Toe Paralysis)

DEFINITION

Riboflavin supplementation is generally required in rations for growing chicks, poults, and ducklings because few poultry feedstuffs contain large quantities.

CLINICAL SIGNS

Riboflavin is essential for growth and tissue repair in all animals. Many tissues may then be affected by riboflavin deficiency, the most severely being the epithelium and myelin sheaths of various nerves.

Chicks

The characteristic clinical sign is "curled toe" paralysis caused by lesions to the sciatic nerve; however, if the deficiency is absolute or very severe, the chick will die before curled toe paralysis develops. In mild cases, chicks tend to rest on their hocks and the toes are only slightly curled. In moderate cases, there is marked weakness of the legs and a distinct curling of the toes on one or both feet. In severe cases, the toes are completely curled inward or under, the legs are extremely weak, and birds walk on their hocks with the aid of their wings ("wing-walking"). At rest, the wings may droop. Leg muscles are atrophied and the skin is dry and harsh. Other signs include stunting, diarrhea after 8-10 days, and high mortality at about 3 weeks. Feather growth in chicks is not generally impaired.

Poults

A dermatitis resulting in encrustations on eyelids and corners of the mouth will develop in approximately 8 days. The vent becomes encrusted, inflamed, and excoriated. Other clinical signs are similar to the chick. Growth is retarded or completely ceases by about day 17. Mortality occurs at about day 21.

Ducklings

Ducklings and goslings usually have diarrhea, stunting, and a bowing of the legs in conjunction with chondrodysplasia.

Laying hens

Riboflavin deficiency will cause decreased egg production and decreased hatchability that is roughly proportional to the degree of deficiency, with an increase in hepatic size and fat content.

Embryos

Embryonic mortality peaks at 4, 14, and 20 days of incubation are typical of riboflavin deficiency, with peaks more prominent early as deficiency becomes severe. In severe cases, embryonic death due to circulatory failure occurs at 4 days. Embryos with moderate inadequacies die at 14 days incubation, with the appearance of shortened limbs, mandible malformations, possible edema, and defective down. Clubbed down is caused by the failure of the down feathers to rupture the sheaths and may be seen in the neck and vent areas of late-stage embryos or hatched chicks. In marginal deficiencies mortality will be delayed until pipping with dwarfism and clubbed down the major signs.

LESIONS

In young poultry, riboflavin deficiency produces specific changes in the main peripheral nerve trunks. There may be marked swelling and softening of sciatic and brachial nerves. Myelin degeneration, Schwann cell proliferation, and axis cylinder fragmentation have been observed. Congestion and premature atrophy of the thymic lobes may also be observed.

TREATMENT

Marked improvement and alleviation of clinical signs can be expected if treatment occurs early in the course of the disease. Water-soluble vitamins are readily available and can be administered in the water if needed. Irreversible damage will occur over time.

VITAMIN A DEFICIENCY

OCCURRENCE

Most outbreaks of vitamin A deficiency occur in young birds, usually 1-7 week-old chicks or poults. Other outbreaks occur in pullets or hens. Because rations compounded by owners of small, backyard flocks are more likely to be deficient, most outbreaks are seen in those flocks. Vitamin A deficiency seldom occurs in commercially raised flocks.

ETIOLOGY

1. Most poultry rations contain some alfalfa meal or new yellow corn, both excellent sources of provitamin A carotenoids which are easily converted into vitamin A by enzymes in the intestinal mucosa. If rations do not contain alfalfa meal and if stored (depleted) corn is utilized, the ration may be low in vitamin A unless a vitamin A supplement is added. Vitamin A is naturally derived from fish oils.

2. Birds hatched from layers low in vitamin A have very low vitamin A reserves. If they are placed on deficient rations after hatching, they soon will be deficient in vitamin A.

3. Birds raised on range get large amounts of vitamin A from green plants. During confinement, this source of vitamin A is not available and deficiency may develop unless the formulated ration includes other sources.

CLINICAL SIGNS

Vitamin A is involved in numerous processes: it plays a role in vision, maintains mucous membrane integrity and cerebrospinal fluid pressure, is required for normal growth and reproduction

Recently hatched birds

1. Signs appear in 1-7 weeks according to the amount of vitamin A stored in the egg and present in the feed. First there is anorexia, growth retardation, then drowsiness, and mild ataxia. Combs and wattles may be pale.

2. Chicks usually die before the development of eye lesions. However, in birds surviving over 1 week, eyelids become inflamed and perhaps adhered with a cheesy like material present in nostrils and eyes.

Laying hens

1. Depending on liver storage levels, severe deficiency of vitamin A over a period of 2-5 months is necessary before signs develop. Unthriftiness, decreased egg production, decreased hatchability and embryonic mortality are observed.

2. Scattered birds in the flock have inflammation of the eyes or sinuses and the eyes and sinuses may be swollen. Mucoid or caseous exudate accumulates in the conjunctival sac and may be voluminous. There is nasal and ocular discharge. Owners often report "the birds have a cold".

LESIONS

1. In young birds the eyelids are inflamed, often adhered, by sticky exudate. There may be excessive urates in the ureters, in collecting tubules of the kidneys, and in the bursa of Fabricius.

2. In layers, 1-3-mm white pustule-like lesions are present in the mucosa of the mouth, pharynx, esophagus, and sometimes in the crop [Fig. 1; Vit A deficiency; UC Davis]. Mucoid exudate often is present in nasal passages. Conjunctival sacs or sinuses contain mucoid or caseous exudate and may be greatly distended. There may be a delicate pseudomembrane lining the trachea.

3. Microscopically there is squamous cell metaplasia of the secretory and glandular epithelium [Fig. 2; Vit A deficency; UC Davis] of the upper respiratory and digestive tracts, which blocks the mucous gland ducts resulting in glands becoming distended with necrotic material.

DIAGNOSIS

1. A careful study of the formula used in compounding the ration may reveal the likelihood of deficiency of vitamin A. One should consider not only the ingredients used but the quality of those ingredients. Analysis of the ration is expensive and time consuming and may be misleading unless the sample is truly representative.

2. Signs and lesions often are suggestive of vitamin A deficiency. Microscopic demonstration of squamous cell metaplasia is nasal passages may assist in diagnosis.

3. Low vitamin A levels in the liver are indicative of vitamin A deficiency.

4. Swollen infraorbital sinuses and exudate in the conjunctival sacs occur with other diseases of poultry. Differential diagnosis should consider infectious coryza of chickens, chronic fowl cholera, infectious sinusitis of turkeys, and influenza of turkeys, ducks, geese, and quail.

CONTROL

1. Prevention is easily accomplished by feeding a ration with adequate vitamin A (broiler chickens and turkeys: 7 000 to 12 500 U.I/kg, with the highest recommended levels in breeders: 10 000 to 14 000 U.I./kg).

2. Avoid long storage of prepared feeds or ingredients for those feeds. Buy or prepare feeds only in relatively small quantities.

3. Add chemical antioxidants to feeds at the time of preparation to protect vitamin A content or add stable forms of vitamin A.

TREATMENT

Treat affected flocks by adding a water-dispersible vitamin A supplement to the drinking water. Alternatively, add a stabilized vitamin A supplement to the ration at 2-4 times normal levels for about 2 weeks. Then feed a balanced ration at normal levels.

VITAMIN E DEFICIENCY

DEFINITION

Three distinct disorders (syndromes) related to or caused by vitamin E deficiency have been recognized in poultry. Each disorder usually occurs alone, although there are occasional overlaps. The three disorders are:

1. Encephalomalacia (crazy chick disease).
2. Exudative diathesis.
3. Muscular dystrophy.

Although each of these syndromes is associated to some degree with vitamin E deficiency, each can be prevented by dietary changes unrelated to the vitamin E content of the ration. There is some interaction with synthetic antioxidants, selenium and sulfur-containing amino acids, especially in preventing encephalomalacia, exudative diathesis and muscular dystrophy respectively.

OCCURRENCE

Vitamin E deficiencies usually are seen in young chicks or turkey poults but also occur in ducklings and, perhaps, in other poultry. Deficiencies usually occur in birds raised in confinement i.e., birds compelled to eat only what is offered to them. Most outbreaks occur in birds fed rations that are high in polyunsaturated fats (e.g., cod liver oil, soy bean oil), that oxidize and become rancid. Vitamin E is very unstable with oxidative destruction enhanced by minerals and polyunsaturated fats in diet.

ETIOLOGY

1. Vitamin E and the selenium-containing enzyme glutathione peroxidase prevent cell membrane destruction caused by peroxides and other powerful oxidants produced as metabolic by-products.
2. There is evidence that vitamin E, selenium, and sulfur-containing amino acids perform separate functions but still act together to prevent the accumulation of harmful peroxides in tissue. Peroxides are derived, in part, from polyunsaturated acids in feeds.
3. The following facts are of interest in considering etiology:
    A. Encephalomalacia can be prevented by adding synthetic antioxidants to the feed.
    B. Exudative diathesis can be prevented by adding selenium to the feed.
    C. Muscular dystrophy can be prevented by adding cysteine, a sulfur-containing amino acid, to the feed.

CLINICAL SIGNS

Vitamin E is involved in several metabolic functions but mostly play a role of natural antioxidant.

Encephalomalacia

Signs are those associated with lesions of the central nervous system and include ataxia, loss of balance, falling over backwards while flapping the wings, sudden prostration on the side with legs outstretched, toes flexed, and head retracted [Fig. 1; Vit E deficiency; NCSU]. Birds that show clinical signs often continue to eat.

The deficiency usually occurs between the 15th and 30th day of life; however, it may occur as early as the 7th and as late as the 56th day.

Exudative diathesis

There is a severe edema caused by increased capillary permeability. This edema is located along the ventrum of the thorax, the abdomen, and perhaps under the mandible. Birds with extensive edema may have difficulty in walking and may stand with their legs far apart because of accumulation of subcutaneous fluid ventral to the abdomen.

Muscular dystrophy

Signs are usually inapparent but there may be locomotor problems.

LESIONS

Encephalomalacia

The swollen cerebellum often contains congested, hemorrhagic, or necrotic areas visible on the surface [Fig. 2; Vit E deficiency; NCSU]. Lesions occur less frequently on the cerebrum. Lesions are accentuated by formalin fixation for a few hours. In turkeys, poliomalacia of the lumbar spinal cord is often found microscopically.

Exudative diathesis

There is green-blue blood-stained viscous edema in the skin and subcutis of the ventrum. Muscular dystrophy occasionally is apparent in breast or leg muscles of the same birds. Distention of the pericardium with fluid has been the cause of sudden deaths in birds.

Muscular dystrophy

In chicks white to yellow degenerative muscle fibers give a streaked appearance to skeletal muscles of the breast or legs. In poults the musculature of the gizzard may contain gray areas of muscle degeneration [Fig. 3; Vit E deficency; NCSU].

DIAGNOSIS

1. The diagnosis can usually be made on the basis of typical signs and gross lesions.

2. Examination and analysis of the ration may indicate rancidity or likelihood of deficiency of vitamin E and/or selenium. Feed analysis for vitamin E activity is time consuming and expensive, therefore care should be taken to submit truly representative samples. Storage temperature and duration are very important in evaluating the quality of the vitamin E ingredient.

3. Microscopic examination of typical lesions is of considerable value in confirming suspected vitamin E deficiency, especially with encephalomalacia or muscular dystrophy.

CONTROL

1. Mix new batches of feed at frequent intervals. Use only high quality ingredients. Avoid storage of mixed feeds for periods longer than 4 weeks. If prolonged storage is necessary, add chemical antioxidants.

2. Use only stabilized fats in the feed.

3. Store feeds in a cool, dry place to reduce vitamin and other quality losses.

4. Avoid improperly compounded do-it-yourself -type rations. Most well-known, commercially prepared feeds are superior in quality to unplanned, self-mixed feeds.

TREATMENT

1. Recommended vitamin E levels are 30 to 150 mg/kg in the diet. Be sure an antioxidant (0,25kg of BHT or santoquin per 1000kg of feed) is in the feed if storage is long or environmental temperatures high. However the newest forms of vitamins are enveloped hence more resistant to heat treatments, humidity and storage. A dose of 0.3 ppm of selenium is recommended in the broiler chicken and turkey diets. Zero to 3 week-old chicks and 0 to 6 week-old turkeys should receive half of this selenium in an organic form which is more readily available to the bird.

2. Oral administration of a single 300 IU of vitamin E per bird will often cure exudative diathesis or muscular dystrophy. Birds with encephalomalacia do not usually respond well to treatment.

RICKETS

DEFINITION

In poultry a deficiency of vitamin D_3, phosphorus or a wide imbalance in the calcium:phosphorus ratio of the diet can cause rickets.

OCCURRENCE

Deficiencies of vitamin D_3 and phosphorus are encountered most frequently in young chicks or poults a few weeks old. Calcium deficiencies usually affect birds of the same age or adult layers. Rickets occurs more frequently in small flocks of poultry raised on carelessly formulated rations. Most commercial feeds are carefully compounded and are adequate in required nutrients. The incidence of rickets is increased in chickens with infectious stunting or malabsorption syndrome.

ETIOLOGY

1. There are numerous forms of vitamin D, however only cholecalciferol or vitamin D_3 acts as the nutritional precursor of 1,25 dihydroxycholecalciferol, the hormone that stimulates active transport of calcium and phosphorus across the intestinal epithelium, bone and shell formation. Because of this role in calcium transport, as the ratio of calcium to phosphorus becomes wider or narrower, vitamin D_3 requirements increase.

2. Although rickets can occur because of phosphorus deficiency, most outbreaks are the result of inadequate vitamin D_3. Sometimes vitamin D_2 is erroneously fed to poultry instead of D_3.

3. Young, rapidly growing chickens or turkeys experiencing malabsorption or any intestinal condition impairing nutrient absorption, may develop rickets even with adequate dietary levels of phosphorus and vitamin D_3. Failure to absorb and/or utilize nutrients in the ration is considered to be secondary to other causes.

4. Calcium deficient laying hens may suffer from cage layer fatigue (up to 30 weeks of age) or bone breakage (old hens).

CLINICAL SIGNS

1. In young growing flocks, affected birds develop a lame, stiff-legged gait. There is retardation of growth. There may be enlargement of the ends of long bones, especially noticeable in the hocks. Birds often rest in a squatting position.

2. Laying hens suffering from cage layer fatigue lie on their side, with their legs extended or they crouched in the corner of their cage.

LESIONS

1. In young birds bones, beaks and claws are soft and rubbery and the epiphyses of long bones often are enlarged. There is a characteristic beading of the ribs, most noticeable at their junction with the spinal column. Ribs are thickened and tend to bend so that the thorax is flattened laterally. The beak becomes soft and rubbery and can be bent or flexed easily [Fig. 1; Rickets; Cornell U]. Parathyroids often are markedly enlarged.

2. Feathering is usually poor and an abnormal black banding of feathers has been observed in colored breeds such as red or buff chickens.

3. In laying hens bones are soft and easily broken, ribs may become beaded [Fig. 2: Rickets; NCSU] and parathyroids are enlarged.

DIAGNOSIS

1. In young birds, their age, signs, and lesions are all useful in diagnosis. Softening of the beak and beading of the ribs are almost pathognomonic.

2. Careful calculation of the calcium:phosphorus ratio, and vitamin D_3 levels of the ration may reveal that it is deficient or imbalanced. Chemical analysis of the ration for minerals and D_3 can be done but is expensive and time consuming. A single sample may not be typical of the ration. Extensive sampling may be advisable for forensic reasons.

CONTROL

1. Feed a balanced ration with adequate calcium, phosphorus, and vitamin D_3 levels. Rations should be carefully compounded to fit the age, purpose, and production of the flock. Note that poultry require vitamin D_3 a form differing from vitamin D_2, which often is fed to other types of livestock. Rovimix Hy-D, a commercial form of 1,25 dihydroxycholecalciferol can also be given to the birds alone or in combination with vitamin D_3.

2. The following calcium:phosphorus ratios are recommended:
   Broiler chickens : 1,35 to 1,5 :1,
   Turkeys (0 to 6 weeks) : 1,5 :1
   Turkeys (end of growth) : 1,35 to 1,5 :1
   Commercial layer: 5.8 to 7:1 (varies according the egg production period)
   Breeders : 4,5 to 5,5 :1

TREATMENT

1. Adjust the ration to fit the age and production level of the flock. If the ration has been deficient in vitamin D_3 give three times the usual amount for a period of 2-3 weeks. Then go back to a balanced ration with the usual recommended level. Liquid vitamin D_3 will also treat the birds.

2. Calcium-deficient, paralyzed, or down layers can be given 1g of calcium carbonate in a gelatin capsule daily for a few days. If the affected layers are caged layers, they should be removed from their cage and confined on the floor until fully recovered.

3. If housed birds are deficient in vitamin D_3, it may be useful to turn them out on range or otherwise expose them to sunlight.

4. Removing hens affected with cage layer fatigue from cages during the early stages of lameness may result in recovery.

FATTY LIVER-HEMORRHAGIC SYNDROME

(FLHS; Fatty Liver Syndrome)

OCCURRENCE

Fatty liver-hemorrhagic syndrome (FLHS) is a sporadic disease with worldwide distribution that occurs primarily in caged layers. Outbreaks are most common in high-producing flocks during hot weather.

HISTORICAL INFORMATION

Fatty liver syndrome was first reported in 1956 and was soon observed by many other diagnosticians. The appearance of the syndrome coincided with the practice of confining layers to cages. There has been much speculation as to the cause of the syndrome. In 1972, the syndrome was reproduced experimentally by force-feeding hens. The lesions closely resembled those of the natural disease. This appears to be a multifactorial problem.

ETIOLOGY

1. Excessive consumption of high-energy diets combined with restricted activity is believed to result in excessive fat deposition in the liver.

2. Contributing factors may include a genetic component.

3. The syndrome may be caused by a deficiency of lipotrophic agents, which are necessary for mobilization of fat from the liver.

4. Aflatoxin in laying hen diets has been shown to increase fat content (dry weight basis) approximately 20% over controls and may play a contributing role.

5. FLHS and caged layer fatigue are often diagnosed simultaneously.

CLINICAL SIGNS

Outbreaks of FLHS are often associated with a sudden drop in egg production (from 78-85% to 45-55%). The flock overall may be obese (body weights 25-30% above normal). Some birds may have pale combs and wattles covered with flaking epidermis. Mortality increases moderately with occasional hens in full production dying suddenly and unexpectedly. Often hens are found dead with pale heads. Mortality rarely exceeds 5%.

LESIONS

Dead birds have large blood clots in the abdomen, often enveloping the liver, as a result of subcapsular hepatic hemorrhage and rupture of the parenchyma. Subcapsular hematocysts may be visible within the parenchyma. Liver is generally enlarged, pale, and friable. Fat content in livers generally exceeds 40% dry weight and may reach 70%, hence the yellow coloration. Clinically healthy birds in the same flock may also have hematomas in the liver, either dark red (recent) or green to brown (older). Large amounts of fat are present within the abdominal cavity and surrounding the viscera.

TREATMENT

There has been no clear elucidation of dietary causes of FLHS other than excessive caloric intake. Reducing obesity of laying hens is the only successful preventive measure to date. However, further loss of production may result from diet changes during the laying cycle. Lipotropic agents such as vitamin E, vitamin B12, methionine and choline have been widely used with variable results. Management practices that reduce heat stress and minimize mold growth in feed may also be helpful. Results of feeding particular nutrients or formulations of nutrients to treat FLHS are inconsistent.

CARDIOVASCULAR DISEASES OF CHICKENS

I. ASCITES OR PULMONARY HYPERTENSION SYNDROME

DEFINITION

Ascites secondary to pulmonary hypertension syndrome (PHS) is one of the most important causes of mortality in broiler chicken flocks. It is associated with rapid growth and a high metabolic rate.

OCCURRENCE

Ascites occurs worldwide in rapidly growing broiler chicken flocks.

HISTORICAL INFORMATION

Ascites was first reported in 1968 in broiler chickens raised at a high altitude. However, the incidence of ascites caused by PHS, where broilers are grown at a low altitude, has increased over the past several years and coincides with genetic and nutritional improvements that resulted in better growth rate and feed conversion.

ETIOPATHOGENESIS

Four pathophysiological mechanisms are recognized to cause ascites: increased hydrostatic vascular pressure, decreased oncotic pressure, increased capillary permeability, and impaired lymphatic drainage. Although numerous chemical toxicities have been reported to cause ascites in broiler chickens through one of these mechanisms, the most common form of ascites in fast-growing broiler chickens is caused by increased hydrostatic vascular pressure.

Rapid growth, elevated metabolic rate, and therefore a high oxygen demand impose an increased workload on the heart. This, combined with the insufficient pulmonary capillary capacity of the modern broiler chicken, aggravates the pulmonary hypertension and further precipitates right ventricular hypertrophy.

Hypertrophy is soon followed by dilation, right ventricular failure, passive congestion, and then ascites. This process is accelerated in birds because of an anatomical particularity. The right atrioventricular valve is a muscular flap, an extension of the right ventricular wall. Any hypertrophy of the latter affects the valve and its apposition against the septum, facilitating venous regurgitation, passive congestion, and ascites.

CLINICAL SIGNS

Clinically affected broiler chickens are smaller than normal and depressed with ruffled feathers. Severely affected birds show abdominal distension with reluctance to move, respiratory distress, and cyanosis.

LESIONS

1. Hypertrophy and dilation of the right ventricle [Fig. 1; Ascites; AAAP] with or without accumulation of straw-colored ascitic fluid in the peritoneal cavities [Fig. 2; Ascites; NCSU], and a generalized passive congestion are characteristic of ascites secondary to PHS.

2. Hydropericardium, protein clots in the ascitic fluid, and a fibrotic liver [Fig. 3; Ascites; AAAP] may be present in chickens with chronic PHS.

3. Microscopic lesions show generalized passive congestion.

DIAGNOSIS

Macroscopic lesions are diagnostic.

If mortality in a flock is abnormally high, look for causes decreasing oxygen availability to the broiler chicken (poor ventilation, high altitude, concomitant respiratory pathology, etc.), or increasing oxygen needs (rapid growth, cold rearing temperature stimulating the metabolic rate).

Other pathological mechanisms can be involved in the development of ascites, and toxicities due to sodium, phenolic compounds, coal-tar derivatives, and dioxin, among others, might also be considered.

CONTROL

Lowering the oxygen requirement by slowing the metabolic rate will reduce, and if severe enough, prevent ascites. A variety of feed restriction and light programs have been used or recommended. The goal is to find a program that will maintain feed efficiency while reducing metabolic rate without increasing days to market.

TREATMENT

There is no treatment.

II. SUDDEN DEATH SYNDROME OF CHICKENS

DEFINITION

Apparently healthy fast-growing broiler chickens, mainly males, die suddenly after a short terminal wing-beating convulsion. Dead birds are found lying on their back. This is a common cause of "normal mortality" in a flock.

OCCURRENCE

This condition occurs from 1- 8 weeks of age in most intensive broiler-growing areas of the world. The incidence in a flock varies from 0.5% to more than 4% in some cases. Sixty to 80% of the affected birds are males.

HISTORICAL INFORMATION

This syndrome has been recognized for 30 years and has been described as acute death syndrome, heart attack, flip-over, dead in good body condition, and lung edema.

ETIOLOGY

The cause is unknown but this condition affects highly performing broiler chickens. It is suggested that death is the result of ventricular fibrillation secondary to a possible imbalance of metabolites or electrolytes. It is classified as a metabolic disease and the incidence appears to be affected by genetic, environmental, and nutritional factors.

CLINICAL SIGNS

There are no premonitory signs. Large healthy broiler chickens will start to convulse and wing flap, and rapidly die lying on their back.

LESIONS

Birds are in good body condition with a full digestive tract. There is red and white mottling of the breast muscle, the ventricles of the heart are contracted, and the auricles dilated with blood. Lungs might be congested secondary to postmortem blood pooling. There are no specific histopathologic lesions.

DIAGNOSIS

Dead birds appear healthy and there are no lesions except the findings described above.

CONTROL

Various feed and light regimens have been tried with little success in decreasing the incidence of sudden death without decreasing feed conversion.

TREATMENT

There is no treatment.

III. ROUND HEART DISEASE OF CHICKENS

This myocardial degeneration used to affect mature chickens (> 4 months of age) but has not been diagnosed in commercial poultry flocks for years. Birds die with a bilateral ventricular hypertrophy and dilation. Histopathology reveals myocardial fatty infiltration. The etiology is unknown.

CARDIOVASCULAR DISEASES OF TURKEYS

I. AORTIC RUPTURE OR DISSECTING ANEURYSM

Aortic rupture is an occasional cause of mortality in 12-16-week-old heavy turkeys, characterized by massive internal hemorrhage from a ruptured lower or, less commonly, upper aorta. The cause is unknown but some contributing factors such as the relatively high blood pressure of turkeys, their natural susceptibility to atherosclerosis, and the absence of an intramural *vasa vasorum* (intrinsic vascularization of the artery) in the lower aorta, might all play a role in the pathogenesis. The carcass is typically in good body condition but pale. Blood may be seen in the mouth or nostrils. A large amount of clotted blood is found in the body cavity, surrounding the kidneys [Fig. 1; Aortic rupture; NCSU] or filling the entire body cavity if rupture occurs in the lower aorta, or surrounding the heart if it occurs in the upper aorta. Careful examination will reveal a longitudinal tear in the wall of the aorta. Management procedures to decrease the incidence of this condition consist of avoiding excitement in the birds.

II. DILATED CARDIOMYOPATHY OF TURKEYS (ROUND HEART DISEASE)

This condition causes mortality in turkey poults between 1 and 4 weeks of age and is a common occasional finding in commercial turkey flock. Affected poults are found dead with a severe bilateral dilated cardiomyopathy [Fig. 1; Round heart disease; NCSU], [Fig. 2; Round heart disease; UC Davis] often accompanied by secondary ascites and hydropericardium [Fig. 3; Round heart disease; NCSU], and congestion of other organs. If the poult survives with this cardiac disorder, growth will stop and the bird will soon show ruffled feathers, unwillingness to move, respiratory distress, and death. Microscopic changes in the myocardium are nonspecific. The etiology is unknown, but several factors such as genetic factors, early viral myocarditis and hypoxic conditions during incubation have been suggested.

III. SUDDEN DEATH SYNDROME OF TURKEYS (PERIRENAL HEMORRHAGE)

DEFINITION

Sudden death syndrome of turkeys (SDS) or perirenal hemorrhage syndrome causes death in heavy- turkey flocks, particularly during grow-out period. Turkeys in good body condition, mainly males, die suddenly with postmortem lesions of acute generalized passive congestion.

OCCURRENCE

SDS is the main cause of mortality in fast-growing turkeys 8-15 weeks of age, but has been reported in turkeys older than 20 weeks of age. This syndrome is uncommon in female turkeys.

HISTORICAL INFORMATION

The condition was first reported in 1973 under the name sporadic renal hemorrhage. The disease has also been named perirenal hemorrhage syndrome, acute hypertensive angiopathy, or sudden death with perirenal hemorrhage. These confusing terms that describe lesions but give no indication of etiology and pathogenesis likely refer to the same condition.

ETIOLOGY

Through intense genetic selection and high-energy diets, the industry has developed a rapidly growing, heavily muscled turkey. SDS of turkey occurs during a period of fast growth and often follows exposure to stress or increased activity level in the flock.

Experimental studies have demonstrated the inability of the cardiovascular system of the domestic turkey to meet metabolic needs generated by exercise; within minutes, turkeys develop hypotension combined with severe lactic acidosis. Turkeys dying of SDS also show greater ventricular weights and cardiac changes described as concentric left ventricular hypertrophy.

It has been hypothesized that a certain percentage of the turkey population has concentric left ventricular hypertrophy which reduces myocardial blood flow and impairs coronary vascular reserve. Exercise or stress could therefore prompt acute myocardial ischemia triggering ventricular arrhythmias and terminal ventricular fibrillation. Ventricular arrhythmias could also be precipitated by the severe lactic acidosis developing during exercise subsequent to tissue hypoxia. Thus, an inadequate cardiovascular response of the turkey to stress or exercise may create hemodynamic instability leading to sudden death.

CLINICAL SIGNS

There are no clinical signs, except violent agonal wing flapping preceding death.

LESIONS

Turkeys dying of SDS are in good body condition with the digestive tract filled with ingesta, demonstrating the suddenness of death. Lesions are indicative of an acute generalized passive congestion with subcutaneous varicoses, pulmonary congestion and edema, perirenal hemorrhage [Fig. 1; Sudden Death Syndrome; NCSU], a swollen severely congested spleen, and congestion of other organs.

Perirenal hemorrhage has been reported to occur in other conditions and is not pathognomonic of the so-called SDS of turkeys. Birds possess a renal portal system with a superficial peritubular capillary plexus at the periphery of the renal lobule. Local passive congestion would therefore result in pooling of blood in the perirenal area and possible diapedesis, explaining the hemorrhages observed at the surface of the kidneys.

DIAGNOSIS

Lesions are diagnostic. Aortic aneurysm affects the same-age turkey, but in aortic aneurysm, birds are pale and free blood is present in the body cavity of the turkey.

CONTROL

Avoid excitement in the flock.

TREATMENT

There is no treatment.

DIGESTIVE DISORDERS

I. PENDULOUS CROP

Pendulous crop is a condition sporadically observed in broiler chicken and turkey flocks. Birds have a greatly distended crop filled with feed and malodorant material. Crop mucosa can be ulcerated or infected by *Candida albicans*. Affected birds keep eating but since feed transit is affected, they soon lose weight, eventually become emaciated and die. Affected carcasses reaching market age are condemned at slaughter. Increased water intake during sudden hot weather has been proposed as a possible cause since birds will over drink and eat at night when the temperature cools down. This appears to "over stretch" the muscular wall of crop and even sometimes the proventriculus, leading to permanent distention. Hereditary predisposition has also been suggested in turkeys.

II. PROVENTRICULAR DILATION

Proventricular dilation has been reported in birds fed a finely ground diet. The gizzard of birds fed such a diet does not need to contract much, hence its poor muscular development with secondary proventricular dilation. The proventriculus is enlarged with thin walls and no clear demarcation between the gizzard and proventriculus. Excessive histamine amounts will also cause proventricular dilation and flaccidity along with gizzard erosions.

DIGESTIVE DISORDERS OF CHICKENS

I. DYSBACTERIOSIS

DEFINITION

Terminology used in Europe to describe an intestinal microflora imbalance and overgrowth characterized by enteritis and mild diarrhea.

OCCURRENCE

Dysbacteriosis is commonly observed after 21 days of age in European commercial broiler chicken flocks but can occur as early as 15 days of age.

HISTORICAL INFORMATION

There has been an increase in the number of broiler chicken flocks affected with dysbacteriosis with the ban of growth promoters in Europe in 1999.

ETIOLOGY

Over growth of an abnormal bacterial duodenal population has been demonstrated in birds affected with dysbacteriosis. *Clostridium spp*. has been shown to contribute to this overgrowth. The absence of antimicrobial growth promotors, animal protein and animal fat appear to predispose farms to the disease. Other predisposing factors may include non-specific stress, mycotoxins and systemic disease.

CLINICAL SIGNS

Dysbacteriosis is characterized by normal water consumption, humid litter, poorly formed and wet feces and a reduction in feed intake.

LESIONS

Thinning and ballooning of the small intestines accompanied by viscous or watery intestinal contents.

DIAGNOSIS

History of diarrhea, wet droppings. Elimination of any other causes of diarrhea and wet litter. Empirical therapeutic response to antimicrobial effective against *Clostridium perfringens* or other enteric pathogens might be a diagnostic indicator for both necrotic enteritis and dysbacteriosis.

CONTROL AND TREATMENT

Monitoring litter quality with a litter box might help in assessing any changes in fecal water content and alert to early signs of diarrhea. Antibiotics might be required if there is associated mortality or subsequent necrotic enteritis. Competitive exclusion products might help.

II. POLYCYSTIC ENTERITIS OF BROILER CHICKENS or RUNTING-STUNTING SYNDROME OF BROILER CHICKENS

DEFINITION

Polycystic enteritis (PE) has recently appeared in the Southeastern United States. It is characterized by large numbers of chicks with marked growth depression, watery diarrhea, and cystic enteritis.

OCCURRENCE

PE may appear in chicks as early as 6-7 days of age, but the usual peak of the problem occurs at around 10-12 days of age, mostly during winter and spring. Farms that have short down times between flocks appear to be at higher risk for the disease. Turkeys are not known to be affected.

HISTORICAL INFORMATION

Runting Stunting Syndrome (RSS) has been recognized in chickens since the late 1970s. This condition occurs sporadically, usually with increasing severity over a year period within a given complex, then declines afterwards. During 2003-2005 a new clinical and pathological presentation appeared and caused economically significant problems in the Southeastern United States, and some countries in Asia, Middle East, and Latin America. In contrast to RSS, persistent problems with PE have been noted on specific "problem" farms as successive flocks are affected. Many research institutions are actively studying this condition, further characterizing enteric viruses, developing diagnostic tests and searching for potential vaccine candidates

ETIOLOGY

The disease has been reproduced by placing broiler chicks on contaminated litter obtained from previously affected farms, and by gavaging birds with intestinal contents from affected chickens. These resulted in severe weight depression. Multiple viruses have been isolated with two groups commonly present; reoviruses and astroviruses. Bacteria do not appear to be involved in the disease as primary agents. Vertical transmission is considered a possibility and is being investigated.

CLINICAL SIGNS

Affected flocks show large numbers of depressed chicks huddling around feeders and drinkers within hours after placement. Litter quickly becomes damp. Feed consumption decreases, there is loss of flock uniformity and many chicks will show severe growth depression (5 up to 20%). If allowed to remain in the flock, stunted chickens do not recover. This will translate in increased need for culling, reduced livability, increased feed conversion, and days to market.

LESIONS

At necropsy, affected chicks have small livers with enlarged gallbladder, pale, dilated thin-walled intestines with watery contents and undigested food. Histologically, intestinal lesions consist of numerous large cysts involving intestinal crypts with degenerating or necrotic cells and mucin inside the lumen of these cysts. As the condition progresses, intestinal villi become shortened and clubbed.

DIAGNOSIS

History, clinical signs, and microscopic intestinal lesions are suggestive of the disease.

CONTROL

Built-up litter and short downtime may contribute to PE. Proper brooding temperature minimizes early poor uniformity and delayed growth. Heat treatment of affected houses during downtime is likely to mitigate the condition.

TREATMENT

There is no specific treatment. Good husbandry and symptomatic support of an affected flock will lessen economic losses. Severely stunted chicks will not recover and should be culled.

III. TRANSMISSIBLE VIRAL PROVENTRICULITIS

DEFINITION

Transmissible viral proventriculitis (TVP) is a transmissible proventricular inflammation of viral etiology found in commercially raised broiler chickens and associated with increased proventricular fragility, impaired feed digestion, poor growth performances, and increased contamination and decreased efficiency at processing.

HISTORICAL INFORMATION

Within the past 15 years, commercial broiler chickens from Southeastern United States have sporadically been affected with this disease.

ETIOLOGY

TVP has experimentally been reproduced with homogenates from proventricular tissue of affected birds and a virus that is consistent with a new type of adenovirus. Presence of the virus in proventricular lesions in natural and experimentally infected birds indicate it is the cause of the disease. Chicks can be experimentally infected by oral or intracoelomic inoculation, but the natural route of infection is unknown.

CLINICAL SIGNS

Affected birds are pale and significantly smaller than uninfected flock mates. They show poor growth rate, increased feed conversion, and the passage of undigested or poorly digested feed in the feces.

LESIONS

At necropsy affected broilers show proventricular enlargement, especially the isthmus between the proventriculus and ventriculus, with mottled thickened, firm walls. Attenuation of mucosal papilla where ducts from the glands open into the lumen may be seen. The mucosa appears roughened. Dilated, cystic glands are not indicative of TVP. They are a postmortem changes that occurs rapidly following death. Four lesions characterize the microscopic changes in the proventriculi: 1) necrosis of the glandular epithelium, 2) lymphocytic infiltration in the interstitium of proventricular glands and mucosa, 3) hyperplasia of ductal epithelium, and 4) replacement of lost glandular epithelium by ductal epithelium. Epithelial cell nuclei are swollen, pale, and often have prominent nucleoli. Inclusion bodies are rarely seen and cannot be relied on to provide a specific diagnosis. Lesions do not occur in other tissues.

DIAGNOSIS

TVP is difficult to identify on the basis of gross lesions. In contrast, microscopic lesions are sufficiently characteristic of provide a diagnosis. Confirmation requires demonstration of the adeno-like virus by electron microscopy or a fluorescent antibody (FA) test. The FA test only works on fresh frozen tissue. Correlation between histopathology and virus presence is very high.

CONTROL, PREVENTION AND TREATMENT

There are no specific treatment, prevention, or control measures for TVP other than biosecurity measures effective against infectious agents.

IV. NECROTIC HEMORRHAGIC HEPATITIS

Although vibrionic hepatitis accurately describes a disease still seen sporadically in chickens, another hepatic disease has emerged in layers (both egg and meat types) with no regular relationship to C*ampylobacter* infection. This disease, termed necrotic hemorrhagic hepatitis, occurs in 40-60-week-old layers and is

associated with increasing mortality over a period of several weeks (up to 0.3% per week) and a parallel decrease in egg production (up to 20%). Affected hens are usually found dead without premonitory signs and the major gross liver lesions include marked hepatomegaly with diffuse pallor, variable stippling/mottling with red and yellow foci, friable consistency, and intralobar and subcapsular hematomas. The spleen is often large, pale, and friable and the ovaries are inactive. The hepatic destruction suggests a chronic progressive disease and there are histological changes pointing to a primary inflammatory process involving segments of portal veins. No microbiological agent has been consistently demonstrated using aerobic, anaerobic, and microaerophilic culture techniques, various special stains, direct electron microscopy, or chicken inoculation.

DIGESTIVE DISORDERS OF TURKEYS

I. POULT ENTERITIS COMPLEX
(PEC)

Poult enteritis complex is a terminology used to describe various infectious intestinal diseases of young turkeys. It includes numerous conditions such as turkey coronavirus (TCV), poult malabsorption or runting-stunting syndrome, and poult enteritis mortality syndrome (PEMS). These diseases all have the following common features: less than six-week-old turkeys develop diarrhea, soon followed by growth retardation and secondary nutritional deficiencies. However, while TCV is well characterized, poult malabsorption or runting-stunting syndrome and PEMS remain poorly defined in terms of etiology. Basic pathogenesis involves intestinal mucosal injury by one or more viruses, and possible secondary opportunistic infection by bacteria.

A. TURKEY CORONAVIRUS (TCV). See page 32.

B. POULT MALABSORPTION / RUNTING-STUNTING SYNDROME

DEFINITION

Poult malabsorption / runting-stunting syndrome is an intestinal disease condition of young turkeys characterized by malabsorption/maldigestion of nutrients that may result in stunting, secondary nutritional diseases such as rickets or encephalomalacia, and secondary infections such as cryptosporidia or bacterial enteritis.

OCCURRENCE

Poult malabsorption / runting-stunting syndrome generally occurs in turkeys between 7 and 28 days of age. Nutrient absorption and/or digestion are inhibited, causing decreased growth rate, stunting, poor feathering, skeletal problems, and an uneven flock. Lack of uniformity and skeletal lesions may persist throughout the grow-out period.

ETIOLOGY

Poult malabsorption/runting-stunting syndrome is a multifactorial disease of unclear etiology. Viruses regularly isolated from intestinal tracts of affected poults include astrovirus, enterovirus, parvovirus and rotavirus. However, the detection of a viral agent from a diseased host does not in itself constitute a cause and effect relationship for that disease. In addition, *Cryptosporidia*, *Cochlosoma,* and coccidia have been identified and will increase severity and disease duration. *Salmonella* species and Gram-positive filamentous bacteria are also commonly isolated from affected birds. Dietary factors such as high protein levels in starter feeds, poor quality fats and fish meal, and mycotoxins have also been implicated in increasing the severity of the disease.

C. POULT ENTERITIS MORTALITY SYNDROME (PEMS)

DEFINITION

Two clinical forms of PEMS have been identified; an acute form with a sharp peak of mortality (mortality is greater or equal to 9% between 7 and 28 days of age, and daily mortality on three consecutive days is greater than 1%), and a less severe form (mortality exceeds 2% between 7 and 28 days but daily mortality does not reach 1% during three consecutive days), which as been referred to as Excess Mortality of Turkeys (EMT).
Sick poults show diarrhea, dehydration, anorexia, growth depression, immunosuppression, and mortality, but also a variety of physiological abnormalities, including reduced body temperature, reduced energy metabolism and hypothyroidism.

OCCURRENCE

The disease occurs only in turkeys, when they are 7 to 28 days of age. There appears to be an age susceptibility; the younger the flock, the more severe the clinical expression. In the field, hens economically are

more affected than toms. The acute form of PEMS has mostly been observed in the southeastern United States and presents a seasonal pattern i.e., from late spring to early fall. While this form was prevalent in the late 90's, it is now an uncommon occurrence. However, EMT is still regularly reported.

ETIOLOGY

The etiology of PEMS is still unknown. The disease can be experimentally reproduced by either contact exposure or oral inoculation of healthy poults with intestinal contents of infected poults, and it is believed that transmission is strictly horizontal. Several agents, including turkey coronavirus, rotavirus, astrovirus, Group 1 avian adenoviruses, torovirus and unidentified small round viruses, have been isolated from PEMS cases. However, none has been found capable of reproducing the disease alone or has been consistently associated with the disease. In addition to viruses, certain atypical *Escherichia coli* strains as well as other bacteria, and protozoa have also been associated with PEMS.

CLINICAL SIGNS

Affected poults are initially hyperactive and vocal but within twenty-four hours they become depressed, anorexic, and huddle together near heat sources. Feed and water consumption drop, while diarrhea develops. Litter quality rapidly deteriorates from abundant and watery droppings. Marked lack of uniformity can be observed few days after the onset of the disease. Clinical signs will wane within seven to ten days, but unevenness will worsen and remain for the duration of the life of the flock.

LESIONS

Lesions are characteristics of an acute severe diarrheal disease. Carcasses are dirty and exhibit signs of dehydration and emaciation. The digestive tract is empty with occasional presence of some litter material while intestines are thin-walled and dilated with fluid and gas. Lymphoid organs are atrophied in more severely affected birds.

DIAGNOSIS

Diagnosis of PEC requires flock records comparison for analysis of growth and brooding performance, clinical evaluation, collection of diagnostic samples such as sera, fecal droppings, water and feed samples, necropsy, and isolation and identification of enteric pathogens.

CONTROL

Biosecurity is of primary importance to control PEC. Biosecurity procedures include management of dead bird disposal, litter management, movement of used litter, controlling traffic patterns of people and vehicles, rodent control and water sanitation. Affected farms should be placed under quarantine and premises should be thoroughly cleaned, disinfected and fumigated. All-in/all-out production or separate brooding and finishing units are helpful. No vaccines are available.

TREATMENT

Supportive care for affected flocks includes raising house temperatures slowly until poults appear comfortable. Water-soluble vitamins and/or electrolytes should be added to the drinking water. Vitamin E added to the feed at twice the recommended level has been shown to be helpful. Antibiotics have been used with mixed success. They should be directed toward Gram-positive bacteria since those with Gram-negative activity may further upset normal intestinal flora.

Any action that will increase feed intake, such as walking frequently through the flock, remixing feed, top dressing feed with rolled oats, whole grains, etc., should have a positive effect of PEC. On farms considered at high risk of experiencing PEC, it is recommended to avoid placing birds from young breeders because their progeny is smaller and would be more susceptible to the disease.

MUSCULOSKELETAL DISORDERS

It is generally agreed that skeletal disorders are a major source of economic loss in poultry. The specific cause of most leg problems is difficult to determine; they are often considered to have a genetic basis but may be influenced by or due to environmental or nutritional factors. With the advance of nutrition and the use of computer-generated diets, deficiencies responsible for arthroskeletal conditions are quite uncommon. However, most of the actual problems are associated with rapid growth and the incidence can be reduced by restricting growth rate.

I. ANGULAR BONE DEFORMITY
(Valgus-Varus Deformity of the Intertarsal Joint)

Angular bone deformity or valgus and varus deformity of the intertarsal joint is the most common form of long bone distortion found in broiler chickens and turkeys. There is medial or lateral angulation of the shaft of the distal tibiotarsal bone resulting in deviation of the lower part of the leg and frequent bending of the proximal shaft of the tarsometatarsus. Flattening of the tibial condyles and displacement of the gastrocnemius tendon may also occur. With severe angulation, the birds will walk on the hock joint with bruising of the area, ulceration of the overlying skin, and sometimes secondary infection. This condition results in significant trimming at processing.

II. CHONDRODYSTROPHY

Chondrodystrophy is a generalized disorder of the growth plate of long bones that impairs growth, while mineralization and appositional growth remain normal. It occurs in young growing poultry. In the past this condition was often described as perosis. Any condition, whether of genetic, nutritional, or environmental origin, resulting in a failure of physeal chondrocytes to proliferate can be called a chondrodystrophy. Chondrodystrophy results in shortened long bones, enlargement of hock joints, and often secondary valgus or varus deformity and subluxation of the gastrocnemius tendon.

III. CONTACT DERMATITIS OF FOOT PADS
(PODODERMATITIS OR BUMBLEFOOT)

This condition is characterized by a local injury to integument of the avian foot, usually the digital or plantar metatarsal pads, which lead to scab formation and inflammation of the subcutaneous tissues. Common sequelae include tendonitis, septic arthritis, and osteomyelitis. Trauma, poor litter condition, and devitalization of the weight-bearing plantar structures are generally suggested to initiate the disease. Severe foot lesions result in lameness, reluctance to move, body weight depression and might lead to sternal bursitis (breast burn or breast blister), and a cause of carcass downgrading at slaughter.

IV. DEEP PECTORAL MYOPATHY

This condition, also named green muscle disease, is an exertional myopathy involving the supracoracoideus (deep pectoral) muscle [Fig. 1; Deep pectoral myopathy; UC Davis] of heavy meat-type birds. Vigorous wing beating increases subfascial pressure in this muscle and results in ischemic necrosis. The lesion is unilateral or

bilateral and the macroscopic appearance varies according to the age of the condition. Earlier lesions consist of edema followed by hemorrhage and necrosis. In chronic cases, the necrotic muscle has contracted due to fibrosis and is uniformly green [Fig. 2; Deep pectoral myopathy; NCSU]. The defect is then visibly apparent at the abattoir as a depression in the breast over the affected muscle and cause downgrading. Turkey leg edema is another condition occurring secondary to exertional myopathy, for example during transportation to slaughter.

V. FEMORAL HEAD NECROSIS
(FHN)

Femoral head necrosis (FHN) is a poorly defined and often inappropriately used term for numerous lesions of meat-type birds. It has been used for and/or confused with the following conditions.

VI. IATROGENIC TRAUMA TO THE FEMORAL HEAD

During growth the femoral head is cartilaginous, and separation of the proximal femoral epiphysis from the femur on disarticulation of the coxofemoral joint is common during routine necropsy. The *teres* ligament and joint capsule frequently pull the articular cartilage and, occasionally, the femoral epiphysis detaches from the femoral shaft and remains in the acetabulum. This epiphyseal separation exposes dark, rough and pitted physes. This is not a lesion but an artifact. Epiphyseal separation may also occur spontaneously in live birds during rough handling and has been referred to as traumatic epiphyseolysis.

VII. OSTEOPOROSIS

With this condition, long bones are fragile and the growth plate is irregular; thus the femur is more susceptible to breakage during necropsy. No necrosis in the femoral head is present. Otherwise, any condition causing increased bone fragility, such as rickets, or the so-called malabsorption maldigestion syndrome, might result in shattering of fragile femoral necks during necropsy.

VIII. OSTEOMYELITIS

The physis of the proximal femur affected by bacterial osteomyelitis is fragile and disarticulation during routine necropsy may break the femoral neck. Small foci of osteomyelitis [Fig. 1; Osteomyelitis; NCSU] will be observed in the physes and metaphyses.

IX. OSTEOMYELITIS / SYNOVITIS

Osteomyelitis is usually one manifestation of a systemic disease. It occurs when, following a bacteremic episode, there is formation of an infective focus in the bone. *Escherichia coli* and *Staphylococcus aureus* are most commonly isolated; less commonly, *Salmonella*, *Yersinia*, *Streptococcus*, Pasteurella, and *Arizona* are cultured. The epiphyses of long bones, vertebral bodies, and associated joints are usually affected. Lesions in long bones consist of focal yellow areas of caseous exudate or lytic areas. Spondylitis can result in pressure on the spinal cord and paresis. Affected joints are swollen and filled with purulent exudate. Treatment is rarely effective. Prevention is based on adequate treatment of septicemic diseases.

X. OSTEOPOROSIS
(Cage Layer Fatigue)

Osteoporosis refers to a decrease in bone volume but no loss in density and affects laying hens reared in cages and is most common at the end of a laying cycle. Clinical signs are variable and include posterior paralysis or acute death with or without changes in egg production. Paralyzed hens are initially alert and may be laying on their sides. On postmortem examination birds have brittle, fragile bones, thin cortices, and sometimes fractures. Sternae are often deformed and there is a characteristic infolding of the ribs at the costochondral junctions. In the acute death form, an egg is present in the shell gland, with the shell partially or totally calcified and no macroscopic lesion. It is hypothesized that this acute form is due to acute hypocalcemia. Osteoporosis can be caused by a vitamin D_3, calcium, or phosphorus deficiency. Adding extra vitamin D and calcium to the diet may be of some benefit. Lack of activity, strain of birds, and type of housing are considered to be important risk factors. Cage layer fatigue can be a problem in a flock, but bone breakage at processing due to osteoporosis may be a more significant problem in terms of economic losses and in respect to animal welfare.

XI. PEROSIS
(Slipped Tendon)

The term perosis describes the subluxated gastrocnemius tendon, which is secondary to long bone shortening caused by growth plate damage (chondrodystrophy). Nutritional deficiencies affect the development of the growth plate, the classical example being manganese deficiency, but, less frequently, biotin, folic acid, niacin, or pyridoxine may be involved. The deformity involves the hocks of young birds and occurs more frequently in heavier birds. Marked malposition of one or both legs is observed from the hock(s) distally. In early cases, the hock is flattened, widened, and slightly enlarged. In advanced cases, the leg from the hock distally deviates sharply from its normal position, usually laterally. Dissection usually reveals that the gastrocnemius tendon at the hock has slipped from its trochlea.

XII. RICKETS

See section on Nutritional Diseases

XIII. SHAKY LEG

This is a severe lameness of 8-18-week-old turkeys. The etiology is unknown and specific lesions are absent. Affected birds spend most time sitting and if stimulated will walk with great difficulty, on "shaky legs". It is believed that lameness is triggered by soft tissue (muscle or tendon) pain. Most turkeys recover as growth slows, but other lesions, such as breast blister, might have developed secondary to the prolonged sitting. Shaky-leg as a flock problem secondary to inactivity caused by pododermatitis from wet litter has been reported.

XIV. SPLAY LEG

This condition occurs in young birds from hatching to 2 weeks of age. There is lateral deviation of the leg [Fig. 1; Splay leg; Univ Montreal], usually at the knee but occasionally at the hip. Splay leg may be unilateral or bilateral. The condition results from birds being on slippery surfaces. A high incidence has been seen in poults brooded on brown paper.

XV. SPONDYLOLISTHESIS

Spondylolisthesis affects broiler chickens and is characterized by posterior paresis and paralysis due to deformation and displacement of the fourth thoracic vertebra resulting in a pinched spinal cord [Fig. 1; Spondylolisthesis; NCSU]. It is considered to be a developmental problem influenced by conformation and

growth rate. Affected birds are ataxic or may assume a hock-sitting posture with their feet slightly raised off the ground, and use their wings to move. Severely affected birds often become laterally recumbent and die from dehydration if not culled. This condition has been referred to in the past as "kinky back".

XVI. TENDON AVULSION AND RUPTURE

Normal or excessive physical stress on the intertarsal joint of growing heavy meat-type birds frequently can causes rupture of the gastrocnemius or peroneus tendons. Swelling and red to green discoloration can be noted in the affected region. It has been suggested that viral-induced tenosynovitis may predispose birds to a ruptured gastrocnemius but this condition has been observed without any evidence of prior tenosynovitis.

XVII. TIBIAL DYSCHONDROPLASIA

Dyschondroplasia is a very common growth-plate cartilage abnormality of fast-growing meat-type birds characterized by persistence of the cartilage with failure of removal of avascular prehypertrophying chondrocytes. The cause is multifactorial, but rapid growth and electrolyte imbalance leading to metabolic acidosis are considered primary risk factors. Common dietary causes of dyschondroplasia include excessive chloride and excessive phosphorus with respect to calcium levels. Certain *Fusarium* spp. mycotoxins also produce this lesion. The pathogenesis is poorly understood, but three mechanisms have been suggested: rapidly produced prehypertrophying chondrocytes do not hypertrophy as quickly as they are produced to allow penetration by metaphyseal vessels, an inadequate vascular invasion of the cartilage cannot initiate hypertrophy, or chondrolysis is defective. Without hypertrophy and vascular penetration, degeneration and calcification do not occur and a mass of prehypertrophying cartilage remains in the metaphysis. The lesion is characterized by an abnormal mass of cartilage below the growth plate [Fig. 1; Tibial dyschondroplasia; NCSU]. If the mass is small, the condition will be subclinical. However, lameness will be observed if the mass of remaining cartilage is very large, the weakened bone may eventually bow or fracture. The lesion is most commonly observed in the proximal tibiotarsus [Fig. 2; Tibial dyschondroplasia; UC Davis], probably because this bone is routinely sectioned during necropsy.

XVIII. TIBIAL ROTATION
(Twisted Leg)

This condition affects young broiler chickens and turkeys. There is lateral rotation of the distal tibia on its long axis, which results in lateral deviation of the lower leg [Fig. 1; Tibial Rotation NCSU]. Rotation is usually unilateral and can approach 90 degrees [Fig. 2; Tibial Rotation NCSU]. Morbidity is low (less than 1% in broiler chickens, but occasionally up to 5% in turkeys), and the cause is unknown. Tibial rotation must be differentiated from slipped tendon, because the tendon remains in place in tibial rotation.

XIX. TWISTED TOES

Twisted toes are common in heavier breeds. Most digits or a single digit may be bent laterally or medially. There is no known economic significance, but abnormal weight bearing on the toes may cause ulceration followed by pododermatitis. This is different from the ventral curling of toe due to paralysis and secondary to riboflavin deficiency.

REPRODUCTIVE DISORDERS

I. OVARIAN LESIONS

1. Atrophy and inactivity of the ovary is perhaps the most disturbing lesion in a hen of productive age. In the absence of other diseases, this finding is indicative of severe stress such as lack of feed and water and is often accompanied by neck or body molt and other evidence of difficulties including emaciation, dehydration, and so on.

2. Neoplasms affecting the ovary are fairly frequent and include involvement by Marek's disease, lymphoid leukosis, and myelocytomatosis. Adenocarcinomas, granulosa cell tumors, and arrhenoblastomas are also seen in hens. Adenocarcinomas are particularly common and are striking in their presentation because of numerous trancoelomic implants on the surfaces of abdominal organs. Ovarian neoplasms are generally readily identified by histopathological evaluation.

3. Oophoritis or follicular regression is associated with a variety of infectious diseases. The normally turgid yellow ovules become wrinkled, hemorrhagic, or discolored (green, gray-yellow, beige, etc.) and many times there is evidence of premature rupture with spillage of yolk material into the abdominal cavity. Follicular regression and rupture with abdominal yolk material is also a frequent finding in extreme dehydration. Among the infectious diseases notorious for this ovarian effect are highly-virulent Newcastle disease, fowl cholera, pullorum disease, and avian influenza.

4. Ovarian cysts are occasionally encountered in laying hens, sometimes in active functional ovaries. Usually these cysts are very thin walled and contain clear fluids. It is frequently difficult histologically to identify the origin of ovarian cysts but it is presumed that most are of follicular origin.

II. OVIDUCT LESIONS

1. Atresia, hypoplasia, and atrophy of the oviduct have been documented in hens of laying age. Simple atrophy associated with severe stress, chronic infection, certain intoxications, etc., is most common. Hypoplasia as a result of early infectious bronchitis in sexually immature pullets may lead to "false layer" status in hens with fully developed ovaries and partially developed oviducts. Many of these have the external appearance of active layers but yolk material is deposited in the abdominal cavity leading to the classical "yolk peritonitis". Hereditary atresia of the oviduct has been reported and the condition may affect segments or the entire oviduct. Again yolks and any surrounding albumin deposits cannot pass and are refluxed back into the abdominal cavity.

2. Neoplasms of the oviduct *per se* are rare or are rarely recognized. Adenocarcinomas of the oviduct arise predominantly in the upper magnum and tend to be highly invasive, frequently resulting in widespread peritoneal implantation. The much more common tumor occurs in the mesosalpinx arising from smooth muscle in the center of the ligament. These leiomyomas are always firm and encapsulated and vary in size from barely detectable to several centimeters in diameter. Large leiomyomas may occasionally interfere with oviduct function.

3. Salpingitis is usually seen as a sporadic individual bird problem, although, in flocks with *Mycoplasma* infection, *Salmonella* infections, and some outbreaks of pasteurellosis and colibacillosis, this lesion may occur with substantial incidence. Infections affecting the left greater abdominal air sac or the peritoneal cavity have the potential for extension into the oviduct as a descending inflammatory process in this tubular organ. In many cases, however, oviductal infections appear to originate in the distal extremity, suggesting that infection ascends from the cloacal orifice. In the early stages of salpingitis the only apparent changes may be irregularities in the mucosal surfaces including erosions or small ulcerations, edema of the mucosal folds, and accumulation of adherent fibrinopurulent exudate. As the lesion progresses the amount of exudate in the lumen increases rapidly and, ultimately, the oviduct becomes an irregular thin-walled sac filled with laminated masses of yellow cheesy exudate. The oviduct becomes nonfunctional early in the infection and the ovaries of affected hens are usually atrophied. It is however possible, that some of the

oviductal content in salpingitis represents impacted egg components. Apart from specific bacterial infections noted above, which might be present in the early stages of salpingitis, the luminal exudate in terminal stages may yield a wide array of bacteria and even fungi.

4. Impacted or egg-bound oviducts are seen sporadically in cull hens. This condition may be more prevalent in pullets that were brought into production too early (prior to full body development) or hens that are extremely obese. Whether oviduct obstruction results from a lodged egg or an intermingled mass of broken shells, shell membranes, or aggregates of coagulated albumin and yolk, the result is the same. When impaction occurs in the uterus or vagina (which is usually the case) eggs enclosed by shell membranes may be found in the abdominal cavity. This indicates that eggs continued to form but were refluxed back into the peritoneal cavity. Hens with numerous abdominal eggs may assume a penguinlike posture.

5. Cystic oviducts are seldom clinically significant but cystic remnants of the right oviduct are very prevalent. In the chicken only the left components of a paired embryonal reproductive system develop after hatching. Segments of the right oviduct may develop to varying degrees. These segments are closed (no anterior or posterior drainage) and, if the wall contains significant glandular tissue, a fluid secretion will accumulate resulting in production of cysts. These cysts usually occur on the right side adjacent to the cloaca and range from barely perceptible in size to massive cysts occupying most of the abdominal cavity. Affected hens appear to have ascites (water belly) but if the abdomen is opened carefully the fluid is found to be contained within a cyst. Cysts have also been described in the left oviduct but they are much less common. Left oviductal cysts may form in segments of the oviduct proper, in the wall of the oviduct or in the mesosalpinx adjacent to the oviduct. Oviductal cysts may be intriguing postmortem findings but they rarely, if ever, have any significant impact on flock performance.

6. Prolapse or eversion of the terminal oviduct can occur with alarming prevalence in some layer flocks and ranges from barely perceptible everted vaginal mucosa protruding from the vent to elongate exteriorized segments of prolapsed oviduct. Circumstances in which this problem is most severe include young poorly developed pullets just coming into egg production, flocks with higher than recommended cage densities, increased levels of cannibalism associated with hyperactive flocks (hysteria) or flocks exposed to a sudden surge of increased light intensity (as in open houses in the first bright days of spring), increasing levels of obesity, and flocks that were poorly beak trimmed with substantial regrowth of beak tips. When oviposition occurs there is a normal eversion of the uterus or vaginal mucosa surrounding the egg as it is delivered. In poorly developed or obese birds this everted mucosa may be slow to retract afterwards. If there is any increased tendency toward cannibalism in the flock, cage- or pen-mates will peck at the everted mucosa causing trauma and edema, which will further slow or prevent retraction. Continuing irritation of the exposed mucosa may promote straining and overt prolapse of the oviduct. An association has also been observed between a tendency for eversion and prolapse and an increased incidence of salpingitis. Also losses from cannibalism and culling are increased in flocks with a high rate of uterine/vaginal prolapse. Control of this condition can be achieved to some degree by allowing full development of pullets before bringing them into lay, maintaining proper stocking density of cage or floor houses, careful control of lighting intensity, proper beak trimming, and maintaining feed formulations and consumption levels to avoid obesity.

URINARY DISORDERS

UROLITHIASIS

(Nephrosis, Renal Gout, Caged Layer Nephritis)

DEFINITION

Urolithiasis is an etiologically undefined condition seen particularly in caged laying hens and characterized by blockage of one or both ureters by urate concretions with attendant atrophy of one or more lobes of the kidney drained by the obstructed ureter.

OCCURRENCE

This condition has been recognized for years as a sporadic individual bird problem in laying flocks. More recently urolithiasis has been described as a flock problem accounting for substantial mortality in caged layers in England, the United States, and other countries throughout the world.

ETIOLOGY

A number of causative factors have been implicated in precipitating gouty deposits in kidneys, joints, or in serosal membranes throughout the body. These include excessive dietary protein (30-40%), dietary calcium excess (3% or greater), sodium bicarbonate toxicity, mycotoxins (oosporin, ochratoxin), vitamin A deficiency, and nephrotropic strains of infectious bronchitis virus. However, the recently described urolithiasis in caged layers appears to be associated with feeding relatively high calcium levels (3% or greater) during the pullet grow-out period. Available phosphorus in the grower ration appears to be contributory in that urolithiasis is enhanced when levels are below 0.6%. Many investigators feel that infectious bronchitis viruses are involved in the process and there also is evidence that dietary electrolyte imbalances (low sodium and potassium, high chlorides) may play a role. Finally, there are many diagnosticians who consider all current etiologic explanations of this condition to be unsubstantiated, or at best, poorly supported hypotheses.

CLINICAL SIGNS

In many cases of urolithiasis there are no consistent clinical signs other than increasing mortality. Among signs associated with the condition are depression, weight loss, and an inclination of affected birds to hide. Roughened or thin eggshells may increase slightly in affected flocks and total egg production will decrease in parallel to increasing mortality. Mortality may be gradual and persistent (2-4% per month) throughout the productive lifetime of the hens or it may be more precipitous. Total mortality has approached 50% in severely affected flocks.

LESIONS

The affected ureter is usually markedly distended by cylindrical concretions surrounded by thick mucus. Although usually unilateral, both ureters may be involved. One or more lobes of the kidney drained by the obstructed ureter often are severely atrophied. The opposite functional kidney may be hypertrophied. Many affected hens will have white chalky material (urate deposits) on serosal membranes of various visceral organs.

DIAGNOSIS

Diagnosis is based on classical ureteral and renal lesions in most of dead birds necropsied. Observation of urolithiasis in an occasional dead bird is indicative of a sporadic individual bird problem and is of little consequence. Confirmation of etiologic factors noted above is usually difficult unless feed samples have been retained for analysis.

CONTROL

Until etiologic factors are better defined it is difficult to make specific recommendations. Of course it is advisable to observe reasonable limits of calcium and available phosphorus in rations during grow-out and to avoid electrolyte imbalance, mycotoxins, water deprivation, and so on.

INTEGUMENT DISORDERS

I. KERATOCONJUNCTIVITIS
(Ammonia Burn)

Keratoconjunctivitis is an inflammation caused by excessive levels of ammonia in poorly ventilated poultry houses. Lesions include keratitis, conjunctivitis, and a corneal opacity with a possible ulceration. Birds may become blind, but recovery is possible depending on the severity of damage to the cornea. Because ammonia is produced by the degradation of uric acid by bacteria in the litter, control of litter moisture and proper ventilation will prevent this problem.

II. SCABBY HIP SYNDROME

Scabby hip syndrome is a lesion observed at the slaughter plant in broiler chickens and is characterized by superficial ulceration and scabbing of skin on the thighs. This is a multifactorial problem; poor feathering, high stocking density, and poor litter conditions have been incriminated. Affected carcasses are downgraded. In recent years, improvements in litter management and use of nipple drinkers have contributed to the reduction in incidence of this condition.

III. STERNAL BURSITIS
BREAST BLISTER or BREAST BURN or BREAST BUTTON

Sternal bursitis is a fluid-filled lesion located on the ventral aspect of the keel bone of poultry. Chickens and turkeys have a synovial bursa, the sternal bursa, which under repeated trauma increases in size and may become secondarily infected. This lesion is closely associated with locomotor problems in heavy birds and increased contact time with litter. Such blisters, if not too large, are trimmed from the carcass at processing resulting in downgrading. The terms breast blister, breast button, breast burn are also used for this condition.

IV. XANTHOMATOSIS

Xanthomatosis is an unusual condition characterized by the abnormal subcutaneous intracellular accumulation of cholesterol in chickens. Skin lesions are initially soft with fluctuating honey-colored fluid, and later become firm with marked thickening and irregularity of the surface [Fig. 1; Xanthomatosis; Cornell U]. This condition is rare and in the past was probably due to contamination of feed fat with hydrocarbons.

BEHAVIOR DISORDERS

I. CANNIBALISM

This vice can cause severe losses in some poultry flocks. Many forms are described, the most common ones being feather pulling, and vent, head [Fig. 1; Cannibalism; Univ Montreal], and toe picking. Feather pulling occurs in any age of bird. In severe cases birds will die of hemorrhage and carcasses will be picked and eaten by pen-mates. Vent picking in cage-reared laying hens is most common in overweight birds. There is a normal eversion and prolapse of the vagina at lay. If the hen is obese, the vaginal mucosa will be exposed for a prolonged time and cage-mates will be attracted by this shiny red mucosa. The assaulted hen will bleed to death and dried blood will be present on the feathers of the pericloacal area and on the back of the legs. A pecking order has often to be established in a poultry flock and head lesions are common in turkeys, even though birds have trimmed beaks. Toe picking occurs in young chicks started on paper or in batteries, and is often initiated by hunger. Several predisposing factors such as light intensity, dense stocking, reduced animal protein feed content, lack of vitamins, amino acids, or salt in feed, sodium imbalance due to heat stress, being without feed for too long, and irritation from external parasites have all been mentioned. Recent work has shown that feather pecking may be related to low levels of insoluble fiber in the diet. Once a bird develops this habit, it will continue. Outbreaks can be sometimes controlled by using red light or reducing light intensity. In laying stock and commercial turkey flocks, trimming a third of the upper beak with an electrocautery or laser is a widely used preventive measure.

II. HYSTERIA

Sporadic cases of broiler chicken and replacement pullet flocks with extremely high activity levels have been reported. The cause is unknown but tryptophan supplementation appears to alleviate the problem.

MANAGEMENT-RELATED DISORDERS

I. DEHYDRATION/STARVATION OF CHICKS/POULTS

Losses due to so-called "normal mortality" should not be more than 1% in the first 10 days of the growout period. Any mortality higher than this should be investigated

DEFINITION

These are the most common causes of mortality in chicks and poults during the first week. Young birds which cannot find water nor feed will eventually die of starvation and inanition, once the yolk has been absorbed i.e., before the fifth day.

ETIOLOGY

Failure to eat and/or drink can be related to farm management conditions. Since recently hatched chicks/poults are poikilothermic, optimal environmental temperatures are a must for the brooding period. A comfort zone i.e., an area where environmental temperature is ideal for the chick/poult must be established inside the barn. Check temperature charts since this temperature varies according to age and species. Feed and water must be located in the bird's comfort zone in a brightly lit area, in order for the young bird to access them.

CLINICAL SIGNS

Birds that die of dehydration or starvation do not usually show other signs of illness than weakness before death. Bear in mind that uncomfortable chicks/poults will be noisy. Affected individuals are also smaller.

LESIONS

Dehydrated carcasses are light with darker feet and beak. Legs appear thinner with a prominent metatarsal vein. The skin adheres tightly to dark pectoral muscles. Upon opening the coelomic cavity, white chalky material (urates deposits) can be observed on various serosal surfaces. Ureters are often dilated with urates and there is none or very little feed in the gizzard.

DIAGNOSIS

Based on history and lesions.

TREATMENT AND CONTROL

Check temperature, luminosity and bird's distribution in the brooding area as well as water and feed availability. Cold birds will huddle together while hot birds will be panting and lying on their belly, often too weak to be interested in finding the water.

II. HYPOGLYCEMIA-SPIKING MORTALITY SYNDROME IN BROILER CHICKENS

DEFINITION

Hypoglycemia-spiking mortality syndrome (HSMS) is characterized by a sudden increase in mortality in a previously healthy, normal appearing broiler chicken flock between 7 and 18 days of age. Two clinical forms have been described; type A more severe but of short duration and type B, a milder form occurring over a longer period.

ETIOLOGY

Although the disease has been reproduced with tissue homogenates and viral particles have been identified in affected birds, the etiology is still unknown and the causal agent remains to be identified. Clinical signs and death are caused by hypoglycemia. Hypoglycemia could either be explained by a virus blocking pancreatic glucagon production or hypothetically related to melatonin deficiency and associated glycogenolysis. Melatonin deficiency could be caused by a lack of a long dark period. Stress and/or acute fasting could trigger HMSM in either situation.

CLINICAL SIGNS

Flock experiences a rapid, unexplained increase in mortality, which will decrease as quickly in a matter of a few days. Live chicks are found recumbent and uncoordinated, frequently lying on their breasts with legs extended. Evidence of blindness and hyperexcitability can be seen. Death occurs rapidly, often within a few hours. Blood glucose levels are lower than <150mg/dL. Birds with very low levels from undetectable to less than 60 mg/dL frequently occur.

LESIONS

There are no specific gross or microscopic lesions. Birds appear normal and typically have food in the crop. Infrequently sinusoidal congestion or small hemorrhages are seen in the liver.

DIAGNOSIS

Mortality pattern (a high spike in a mortality curve at 7-18 days of age) and low blood glucose levels are diagnostic. TheraSense FreeStyle glucose meter (Abbott Labs) works with avian blood (Note: other glucometers also may work, but have not been tried or reported).

CONTROL AND TREATMENT

Although it is important to give a 24 hour period of full light to day-old chicks, a progressively decreases day length resulting in a long daily dark period will usually prevent this problem.

III. HEAT STRESS and HYPERTHERMIA

High temperatures are stressful for poultry and frequently cause death from hyperthermia. Millions of birds die each year from hyperthermia usually because of high environmental temperature, but also because of electric power failure in closed buildings. Birds do not have sweat glands and thermoregulate via non-evaporative cooling (radiation, conduction and convection). Effect of ambient temperature on body temperature varies with body heat production which is directly related to body mass and feed intake (metabolism). If panting fails to prevent increase in body temperature birds will become depressed, then comatose, and soon die. Lethal internal high body temperature is 116^{o}F for chicks and 117^{o}F for adult birds. Dead birds are usually found on their breast, in good body condition. Breast muscles may have a cooked, pale appearance. Prevention of hyperthermia is based mainly on proper building insulation, optimal ventilation and evaporation techniques, feed removal early in the day to reduce metabolic heat production and adequate drinking water availability.

Panting and increased respiratory rates affect acid-base balance and cause respiratory acidosis. Higher blood pH will reduce plasma ionized calcium, which is needed for eggshell formation, hence the risk for increased thin-shelled eggs in summertime laying flocks.

IV. VACCINE REACTION (ROLLING REACTION)

DEFINITION

A normal respiratory (Newcastle disease or infectious bronchitis) vaccine reaction occurs within the week after hatchery vaccination. However, if environmental conditions are poor, or the flock is infected with vertically transmitted *Mycoplasma spp.*, this reaction might aggravate with possible secondary *Escherichia coli* or *Mycoplasma spp.* infection.

CLINICAL SIGNS AND LESIONS

Affected chicks will show head shaking, wet eyes with nasal discharge and mild coughing or sneezing. Chicks will appear depressed and will huddle together or under a heat source. There will be increased mortality, growth retardation and loss of flock uniformity. At necropsy there will be serous to caseous exudates in the upper respiratory tract with airsacculitis in the case of secondary bacterial infection.

DIAGNOSIS

Based on poor environmental conditions, clinical signs and lesions. ELISA antibody titers to infectious bronchitis are within normal.

TREATMENT AND PREVENTION

Chicks must be provided with optimal environmental temperature and rearing conditions. Antibiotics can be administered if there is secondary bacterial infection.

DISEASES OF CHICKENS AND TURKEYS CORRELATED WITH AGE

By knowing the species affected, salient clinical feature, and age of the flock, it is often possible to make a list of potential differential diagnoses. In the following table, some of the more common diseases are presented by age and clinical problem. Of course, this will not be absolute but can be used as a guide.

BROILERS, PULLETS, LAYERS

Typical losses to 7 weeks of age are 4-5%. Losses in the first 2 weeks account for 30-50% of total mortality.

A. BROODING PERIOD (0-2 weeks)

1. Mortality

    A. Mismanagement
    B. Starveout/dehydration— floor temperature, water management
    C. Navel and yolk sac infection: *Salmonella*, *Escherichia coli*, *Staphylococcus*, *Proteus*, etc.
    D. Vaccine contamination
    E. Improper incubation conditions: small, weak hatchlings or increased susceptibility to infection

2. Respiratory Disease

    A. Aspergillosis (Brooder Pneumonia)
    B. Vaccine Problems—Respiratory reaction

3. CNS Disease

    A. Avian Encephalomyelitis
    B. Encephalomalacia
    C. Poor vaccine placement (pox, MDV)
    D. Spiking Mortality

4. Nutritional Deficiencies

    A. Rickets
    B. Other

5. Eye Diseases

    A. Ammonia Burns
    B. Mycotic Keratoconjunctivitis

B. GROWING PERIOD (2-8 weeks)

1. Mortality

    A. Coccidiosis
    B. Aspergillosis
    C. Ascites
    D. Marek's Disease
    E. Clinical Infectious Bursal Disease
    F. Inclusion Body Hepatitis/Aplastic Anemia
    G. Ulcerative Enteritis
    H. Necrotic Enteritis
    I. Chicken Infectious Anemia Virus
    J. Gangrenous Dermatitis
    K. Blackhead

2. Respiratory Disease

    A. Mycoplasmosis
    B. Newcastle Disease
    C. Infectious Bronchitis
    D. Infectious Laryngotracheitis
    E. Colisepticemia
    F. Avian Influenza

3. Lameness

    A. Tibial Dyschondroplasia
    B. Long Bone Distortion (Valgus-Varus Deformities)
    C. Infectious Synovitis
    D. Viral Arthritis
    E. Bumblefoot
    F. Osteomyelitis
    G. Staphylococcosis/Other septic arthritides
    H. Spondylolisthesis
    I. Rickets
    J. Ionophore/3-Nitro Toxicity

4. Skin Disease

    A. Gangrenous Dermatitis
    B. Fowl Pox
    C. Exudative Diathesis
    D. Skin Leukosis

5. CNS Disease

    A. Avian Encephalomyelitis
    B. Nutritional Encephalomalacia
    C. Newcastle Disease
    D. Marek's Disease

6. Other

    A. Roundworms
    B. Toxicities—Mycotoxin, Botulism, Ionophore, 3-Nitro, etc.
    C. Crop Mycosis
    D. Cellulitis
    E. Swollen Head Syndrome
    F. Inflammatory Process
    G. Immunosuppression - IBD, CIA

C. PULLET PERIOD (8-20 weeks)

1. Neoplastic Diseases

    A. Marek's Disease
    B. Avian Leukosis (subgroup J)

2. Respiratory Diseases

    A. Infectious Coryza
    B. Infectious Laryngotracheitis

C. Mycoplasma
D. Infectious Bronchitis
E. Newcastle Disease
F. Avian Influenza

3. Systemic Diseases

A. Fowl Cholera

D. LAYERS (>20 weeks)

1. Neoplasia
   A. Lymphoid Leukosis
   B. Carcinoma
   C. Sarcoma
   D. Marek's Disease

2. Respiratory Diseases

   A. Newcastle Disease
   B. Avian Influenza
   C. Infectious Bronchitis
   D. *Mycoplasma*
   E. Infectious Coryza
   F. Laryngotracheitis

3. Egg Production Drops

   A. Newcastle Disease
   B. Avian Influenza
   C. Avian Encephalomyelitis
   D. Infectious Bronchitis
   E. *Mycoplasma gallisepticum*
   F. Infectious Coryza
   G. Nutrition/Management

4. Salpingitis/Peritonitis

5. Cage Layer Fatigue

6. Fowl Mites

7. Fatty Liver Hemorrhagic Syndrome

8. Parasitism: Capillariasis, Heterakis, Roundworms, etc.

9. Uterovaginal Prolapse

10. Fowl Cholera

E. SPORADIC DISEASES

1. Tuberculosis

2. Botulism

3. Streptococcosis
4. Arbovirus Infection
5. Pullorum/Typhoid
6. Other Parasitic Diseases

TURKEYS

A. EARLY BROODING PERIOD (0-3 weeks)

1. Mortality/Poor Growth

    A. Mismanagement
    B. Omphalitis: *Salmonella*, *S. arizonae, E. coli*, *Proteus*, etc.
    C. Starveout
    D. Cannibalism
    E. Candidiasis
    F. Poult Enteritis
    G. Turkey Viral Hepatitis
    H. Coccidiosis/Cryptosporidiosis
    I. Poor Beak Trimming

2. Respiratory Disease

    A. Aspergillosis (Brooder Pneumonia)
    B. Turkey Coryza (Bordetellosis)

3. Lameness

    A. Splay Leg, Tibial Rotation
    B. Rickets
    C. Staphylococcosis

4. Nervous Signs

    A. Avian Encephalomyelitis
    B. Arizonosis
    C. Encephalomalacia—Vitamin E Deficiency
    D. Mycotic *Encephalitis—Aspergillus*, *Dactylaria*

5. Eye Diseases

    A. Ammonia Burns
    B. Mycotic Keratoconjunctivitis —*Aspergillus*
    C. Arizonosis
    D. Injuries

B. LATE BROODING/EARLY GROWING PERIOD (3-12 weeks)

1. Mortality

    A. Round Heart Disease
    B. Hemorrhagic Enteritis
    C. Aortic Rupture/Hypertensive Angiopathy

D. Histomoniasis
E. Leucocytozoonosis
F. Ulcerative/Necrotic Enteritis

2. Respiratory Disease

A. Mycoplasmosis—MM, MS, MG
B. Turkey Coryza (Bordetellosis)
C. Newcastle Disease
D. Colisepticemia
E. Fowl Cholera
F. Avian Influenza

3. Lameness

A. Spondylolisthesis ("Kinky Back")
B. Bacterial Arthritidis—Staphylococcus, E. coli

4. Other

A. Roundworms
B. Mycotoxins

C. FINISHING PERIOD (>12 weeks-market)

1. Mortality

A. Cannibalism
B. Erysipelas
C. Aortic Rupture

2. Respiratory Diseases

A. Fowl Cholera
B. Aspergillosis
C. Chlamydiosis
D. Newcastle Disease
E. Avian Influenza

3. Lameness

A. Long Bone Distortion
B. Tibial Dyschondroplasia
C. Osteomyelitis
D. Bacterial Arthritidis—Staphylococcus, E. coli, Erysipelas, Pasteurella
E. Scoliosis

4. Other

A. Internal Parasites: Round Worms, Cecal Worms
B. External Parasites: Mites, Lice
C. Pendulous Crop
D. Umbilical Hernias
E. Breast Buttons/Blisters
F. Turkey Pox

D. BREEDERS (>30 weeks). Diseases of the finishing period can also occur during the laying period.

1. Mortality
    A. Fowl Cholera
    B. Aspergillosis
    C. Salpingitis/Peritonitis

2. Neoplasia
    A. Reticuloendotheliosis ("Turkey Leukosis")
    B. Carcinomas

3. Egg Production Drops
    A. Newcastle Disease
    B. Avian Influenza
    C. Other Paramyxoviruses
    D. Mycoplasmosis
    E. Nutrition/Management

DISEASES WITH LESIONS IN THE CARDIOVASCULAR SYSTEMS

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Ascites	Metabolic; related to rapid growth rate and high yield in broilers.	Young, fast- growing chickens (males > females).	Enlarged heart. Ascites. Enlarged or cirrhotic liver. Fibrin exudation in severe cases.	Exacerbated by low oxygen conditions at hatch or brooding, high altitude, heavy dust, lung pathology. Controlled via lighting programs to slow growth.
Anatipestifer infection	*Pasteurella anatipestifer*	Ducks, turkeys, other waterfowl.	Pericarditis, often adhesive.	Often with airsacculitis, fibrinous perihepatitis.
Chlamydiosis	*Chlamydia psittaci*	Turkeys, pigeons, ducks, cage and wild birds.	Pericarditis, often adhesive.	Often with airsacculitis, fibrinous perihepatitis, splenomegaly, and hepatomegaly.
Colibacillosis	*Escherichia coli* septicemia.	Turkeys, chickens, commercial ducks.	Pericarditis, often adhesive.	Often with airsacculitis, fibrinous perihepatitis.
Dissecting aneurysm (aortic rupture)	Unknown. Non-infectious. A strong nutritional influence, especially copper metabolism.	Turkeys. Occasionally, chickens.	Ruptured artery, usually abdominal aorta. Rarely, aortic arch. Extensive internal hemorrhage.	Sudden deaths in rapidly growing, highly conditioned birds. Losses can be extensive. Usually in males.
Endocarditis	Various bacteria: Erysipelas, Pasteurella, Staphylococcus, Streptococcus.	Chickens and turkeys.	Yellow, irregular masses on the heart valves.	Low incidence.
Listeriosis	*Listeria monocytogenes*	All poultry and many wild birds.	Focal myocarditis, pericarditis. Focal hepatic necrosis. Encephalitis.	Uncommon. May be secondary. Usually sporadic losses. Septicemia or encephalitis may be only manifestation.
Marek's disease	Herpesvirus	Chickens	Focal or multifocal tumors in the myocardium.	May be associated with tumors in other organ systems.
Mycoplasma gallisepticum infection	*M. gallisepticum*	Turkeys, chickens, other poultry, birds.	Pericarditis, often adhesive.	Often with airsacculitis, fibrinous perihepatitis.
Pullorum disease, fowl typhoid, possibly paratyphoid	*Salmonella pullorum, S. gallinarum,* other salmonellae.	Chickens, turkeys, and geese.	Nodules in myocardium. Adhesive pericarditis.	Oophoritis or orchitis may occur in some adult birds. Enteric or septicemic diseases with diarrhea in young birds.
Round heart disease	Possibly toxic agents in turkeys (antitrypsin and furazolidone implicated).	Chickens and turkeys.	A greatly enlarged, round heart. Ascites. Fibrin exudation in severe cases.	Uncommon in chickens. Most outbreaks associated with built-up litter. Variable mortality. Potentiated by furazolidone treatment in turkeys.

DISEASES WITH SIGNS SUGGESTIVE OF CNS DISEASE[A]

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Aspergillosis	Usually *Aspergillus fumigatus*.	Usually young chicks and poults or captive game birds.	Various CNS signs. Respiratory signs usually precede CNS signs and predominate. A minority develop CNS involvement.	Yellow mycotic nodules often grossly visible in brain. Lesions of aspergillosis in lungs or air sacs, perhaps in conjunctival sac or globes of eyes.
Avian encephalomyelitis (epidemic tremor)	Picornavirus	Chicks, young poults, and pheasants.	Tremors of head and neck and legs. Paresis progressing to paralysis and prostration.	Microscopic lesions in the CNS. Survivors often develop cataracts. Invert the birds to accent the tremors for diagnosis.
Bacterial encephalitis	*Salmonella, S. arizonae,* paratyphoid species, *Escherichia coli*.	Turkey poults, chicks.	Various CNS signs. Ophthalmitis and omphalitis often present in some of the poults.	Exudate grossly visible in meninges and ventricles. Confirm by culture.
Botulism	Preformed toxin of *Clostridium botulinum*.	Usually chickens	Paresis progressing to paralysis of legs, neck, wings, nictitating membrane. Loose feathers.	No gross or microscopic lesions of value. Perhaps putrid feed or maggots in crop.
Crazy chick disease, encephalomalacia	Vitamin E deficiency	Chicks (usually less than 8 weeks old), turkeys (usually 2-4 weeks old).	Ataxia, falling and flying over backwards, loss of balance; prostration with legs outstretched, toes flexed, and head and neck back.	Hemorrhage and malacia of cerebellum often grossly visible. Confirm by microscopy. Perhaps exudative diathesis along ventrum or muscle necrosis.
Dactylariosis	*Dactylaria gallopava* (fungus)	Turkey poults, chicks.	Incoordination, tremors, torticollis, paralysis, perhaps ocular opacities.	Focal gross brain lesions, often pulmonary nodules or airsacculitis. Fungus often in sawdust litter.
Equine encephalomyelitis	Alphaviruses	Pheasants, partridges, turkeys, Pekin ducks, quail.	Circling or staggering followed by paralysis. Perhaps blindness in recovered birds. Often high morbidity and mortality.	Microscopic lesions only. Transmitted by mosquitoes.
Fowl cholera	*Pasteurella multocida*	Turkeys, chickens, perhaps other species.	Abnormal positions of head and neck. Ataxia, loss of equilibrium.	A localized form of chronic fowl cholera. May/may not accompany acute outbreak. Lesions may be present in cranial bones or inner ear.
Marek's disease	Herpesvirus	Chickens usually 6–20 weeks old.	Paresis progressing to paralysis of a leg or wing. Often one leg is held forward and one leg is held backward in the recumbent bird.	Microscopically there are infiltrating neoplastic cells in affected nerve trunks and the CNS. Grossly, lesions may be visible in affected nerve trunks.

DISEASES WITH SIGNS SUGGESTIVE OF CNS DISEASE[A]

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Newcastle disease	Paramyxovirus	Usually chickens but most birds susceptible.	In chicks CNS signs, usually proceeded by respiratory signs. Progressive paresis followed by paralysis and death.	Microscopic lesions are present and helpful in diagnosis. Only a small part of birds with respiratory signs have CNS signs.
Pox vaccination reaction	Fowl pox vaccine	Chickens < 2 weeks. Low incidence for ~7 days.	Ataxia, mild extensor rigidity. Head drawn back, wings drawn up, tiptoe gait.	In-ovo administration of pox vaccine; errors in subcutaneous injection. May relate to vaccine titer.
Spiking mortality	Unknown. Arenavirus, rotavirus, thiamine deficiency, mycotoxins have been implicated.	Young chicks and poults (< 3 weeks).	Ataxia and death. Dead often found ventrally recumbent with head and neck outstretched.	Sudden, high mortality that may last ~12 days. Often associated with a stress event such as movement from brood chamber to whole house. Capsular or parenchymal liver hemorrhage may be present. Blood sugar levels are very low in affected compared to normal birds.

[A]Signs suggestive of CNS disease in birds include ataxia, paralysis, circling, trembling, twisting of the head and neck, falling over backward, and loss of balance. Any combination of CNS signs may occur.

DISEASE WITH LESIONS IN THE MOUTH, PHARYNX, ESOPHAGUS, CROP, PROVENTRICULUS, GIZZARD

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Candidiasis (crop mycosis)	*Candida albicans*	Poultry, game birds, perhaps other birds.	Gray, thin, pseudomembranous patches on the mucosa. Little inflammation.	Often secondary to parasitism, malnutrition, poor sanitation, impaction, antibiotic usage, other disease. Affects any or all organs listed in title.
Capillariasis	*Capillaria contorta, C. annulata*	Chickens, turkeys, game birds.	Worms sewn into inflamed, thickened mucosa.	In the esophagus and crop. Common in game birds. Scrapings usually necessary for identification.
Duck plague (duck virus enteritis)	Herpesvirus	Ducks, geese, swans.	Hemorrhage and necrosis of the esophageal and cloacal tissue. Liver has petechial hemorrhages.	Intranuclear inclusions produced in infected tissue.
Mycotoxicosis	Trichothecenes	All poultry	Oral ulcerations	Produced by *Fusarium* species of mold.
Pendulous crop	If epizootic, influenced by coarse roughage; or by genetics in turkeys.	Turkeys, chickens, perhaps others.	Crop and esophagus enlarged, perhaps impacted.	Secondary mycosis often present in atonic crop or esophagus. Sporadic cases sometimes from vagal paralysis.
Trichomoniasis (canker in pigeons; frounce in falcons)	*Trichomonas gallinae*	Raptors, doves, pigeons, turkeys, chickens.	Raised conical masses in mucosa of mouth, pharynx, esophagus, crop.	Many trichomonads in oral fluids. Lesions sometimes in proventriculus. Also in the liver of pigeons and some raptors. Lesions often invasive.
Vitamin A deficiency	Inadequate vitamin A	Chickens, turkeys.	Pustule-like lesions in esophagus, perhaps mouth and pharynx. Variable rhinitis, sinusitis, conjunctivitis. Perhaps excessive urates in urinary tract or cloaca.	Sticky eyelids and ataxia often the only gross lesions and signs in young birds. Squamous metaplasia of columnar epithelium in esophageal mucous glands and nasal epithelium.
Wet pox	Poxvirus	Most birds, including poultry.	1–5 mm yellow-gray plaques in mucosa of mouth, pharynx, or esophagus. Less often in sinuses or conjunctiva.	Skin lesions often on face, wattles, eyelids, comb, feet, legs, ear lobes, caruncle, snood.

DISEASES WITH LESIONS IN THE INTESTINE

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Arizonosis	*Salmonella arizona*	Poults, chicks.	Enteritis, unabsorbed yolk. Large mottled liver, perhaps peritonitis or intraocular turbidity.	Poults often unthrifty and with excessive mortality. Perhaps diarrhea.
Coccidiosis	Many species of *Eimeria*	Chickens, turkeys, ducks, geese, perhaps in most birds.	Enteritis of variable severity and location. *E. acervulina* – upper small intestine. *E. necatrix* and *E. maxima* mid-small intestine. *E. tenella* ceca. *E. brunette* – posterior gut.	Five major pathogens for chickens are listed. For details see text. Coccidiosis often occurs concurrently with other diseases. Uncommon in ducks, geese.
Colibacillosis	*Escherichia coli*	Chickens, turkeys.	Enteritis, omphalitis, salpingitis, peritonitis, arthritis, and panophthalmitis are frequent lesions. In respiratory disease often associated with pericarditis, perihepatitis, and airsacculitis. See notes.	Many syndromes identified. Seen in very young and in adults. Three serotypes of the agent account for most outbreaks. Often a secondary infection.
Coligranuloma	A mucoid coliform	Chickens, turkeys.	Granulomas along cecum, duodenum, in mesentery and liver.	Resembles tuberculosis but no acid-fast bacteria in lesions. Must be differentiated from tuberculosis. An uncommon disease.
Coronaviral (transmissible) enteritis	Coronavirus	Turkeys, especially poults.	Marked mucoid enteritis, dehydration.	Diarrhea present. Mortality may be very high with young poults. Less severe in older turkeys.

DISEASES WITH LESIONS IN THE INTESTINE

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Duck plague (duck virus enteritis)	Herpesvirus	Wild or domestic ducks, geese, swans.	Widespread hemorrhages, severe enteritis. Perhaps elevated plaques in esophagus, ceca, cloaca or bursa. Hemorrhage and/or necrosis in lymphoid rings or discs of gut.	Epizootic losses in waterfowl are suggestive. Typical lesions and inclusion bodies helpful in diagnosis. An exotic, reportable disease.
Fowl typhoid	*Salmonella gallinarum*	Chickens, turkeys, occasionally other poultry.	In recently hatched, same as pullorum (above). In older birds, pale cadaver, marked enteritis, splenomegaly, gray foci in liver, bile-stained (bronze) liver.	Closely resembles pullorum in recently hatched chicks and poults but mortality persists into adulthood. Diarrhea and anemia in older birds.
Hemorrhagic enteritis (HE)	Adenovirus	Turkeys often 10-12 weeks old.	Severe enteritis in small intestine with much blood in the gut. Spleen enlarged and mottled early in the course.	Bloody feces often noted. Mortality may be high. A similar virus (perhaps the same) causes marble spleen disease in pheasants and splenomegaly in chickens. Subclinical HE may be associated with immunosuppression and secondary *E. coli* infection
Hexamitiasis	*Hexamita meleagridis.* In pigeons *H. columbae*.	Turkey, poults, game birds, pigeons	Catarrhal enteritis in upper half of small intestine. Local bulbous dilations in affected gut. *Hexamita* in crypts of Lieberkuhn.	*E. coli* infection birds have watery diarrhea. They often die in convulsions. Closely resembles paratyphoid or transmissible enteritis.
Histomoniasis (blackhead)	*Histomonas meleagridis*	Turkey poults, game birds, chickens.	Ceca swollen and usually with cecal cores. Circular or oval recessed lesions in liver.	Typical lesions in ceca and liver are pathognomonic.
Infectious bursal disease	Birnavirus	Chickens typically 3–6 weeks old.	Marked inflammation of the bursa of Fabricius, which is swollen early but atrophic later. Enlarged spleen. Hemorrhages common in heavy muscles.	Diarrhea, incoordination, dehydration, vent picking are usual signs. Course is about 1 week. Immune system damaged. May be followed by inclusion body hepatitis, gangrenous dermatitis, ulcerative or necrotic enteritis, etc.

DISEASES WITH LESIONS IN THE INTESTINE

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Internal parasitism	Ascarid tapeworms, cecal worms, *Capillaria* spp.	Many birds, including poultry.	Parasites usually visible in appropriate location. Variable degree of enteritis. Emaciation may be marked.	Microscopic exam of scrapings necessary to identify *Capillaria*.
Marek's disease	Herpesvirus	Chickens.	Diffuse neoplasia involving an area of gut.	Focal of diffuse neoplasia usually apparent in other visceral organs, e.g., liver, spleen, gonads, kidneys, lungs.
Nonspecific enteritis	Many infectious agents.	Poultry and other birds.	Enteritis accompanies many infectious diseases that have lesions of greater diagnostic value in other systems.	Other diseases that may have enteritis include: cholera, erysipelas, salmonellosis, vibrionic hepatitis, spirochetosis, botulism, aflatoxicosis, influenza, candidiasis, and others.
Paratyphoid	*Salmonella sp*. (about 20 major species).	Poults, chicks, other young birds. Sometimes in adult poultry.	Severe enteritis. Often mucosal plaques and/or cheesy cecal cores. Occasionally the other lesions described above for pullorum disease may occur.	Usually in birds less than 8 weeks old but occasionally in older birds. Occurs frequently in young turkey poults. Interspecies transmission possible.
Pullorum disease	*Salmonella pullorum*	Chickens, turkeys, occasionally other poultry.	Enteritis, dehydration, unabsorbed yolk. Nodules in lungs, heart, or gizzard. Perhaps mucosal plaques, cecal cores, and focal necrotic hepatitis. May present as synovitis in broilers.	Typically epizootic in birds up to 4 weeks old. Begins shortly after hatching. White adherent diarrhea common. Persists in some adults as oophoritis, orchitis, or myocarditis.
Tuberculosis	*Mycobacterium avium*	Chickens, most other poultry and birds.	Round nodules (granulomas) attached to serosa of gut. Focal granulomas in many other organs. Extreme emaciation in advanced cases.	Usually seen in old chickens kept beyond one laying season. Causative bacilli readily demonstrated in acid- fast-stained smears of lesions.
Ulcerative enteritis (quail disease)	*Clostridium colinum*	Captive game birds, turkeys, chickens.	Acute enteritis early. Usually many deep ulcers along the intestine. Enlarged spleen. Focal and/or diffuse yellow areas in liver.	A common disease of game birds. Resembles coccidiosis in chickens. Often secondary in chickens.

DISEASES WITH LESIONS IN LIVER

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Aflatoxicosis (mycotoxicosis)	Toxin usually from *Aspergillus flavus*.	Poults, pheasants, chicks, ducklings.	Liver pale and mottled and with bile duct hyperplasia. Catarrhal enteritis.	Toxin in feeds, especially peanut meal; in spilled feed in litter; in litter alone. Signs often include ataxia, convulsions, opisthotonos. The only signs may be unthriftiness, poor gains, low production.
Avian vibrionic hepatitis	*Campylobacter fetus* spp. *jejuni*	Chickens, usually well started or adults.	Asterisk or cauliflowerlike foci of hepatic necrosis. Sometimes hemorrhages, subcapsular hematocyst, ascites, or hydropericardium.	Only about 10% of affected birds have hepatic lesions. *Campylobacter* often cultured from bile.
Duck viral hepatitis	Picornavirus	Ducklings, typically less than 4 weeks old.	Liver swollen and with many hemorrhages	Signs: acute onset, short course, high morbidity and mortality. Typical lesions in young ducklings almost pathognomonic.
Fowl cholera	*Pasteurella multocida*	Poultry, wild birds, especially waterfowl.	Diffuse streaking of the liver in acute cases. Later there may be 1-3-mm focal areas of hepatic necrosis.	Focal hepatic lesions closely resemble those of salmonellosis, tuberculosis, and listeriosis. Fowl cholera often septicemic.
Histomoniasis (Blackhead)	*Histomonas meleagridis*	Turkeys, game birds, chickens.	Recessed round to oval focal hepatic lesions up to 2.0 cm. Typhlitis with/without cecal cores.	Classical hepatic and cecal lesions together are pathognomonic. Frequently occurs in turkeys raised with or after chickens. Agent transmitted in ova of cecal worms and in earthworms.
Inclusion body hepatitis	Adenovirus	Chickens typically 5-8 weeks old.	Yellow-tan hepatic areas with hemorrhages. Intranuclear inclusions. Perhaps icterus. Hemorrhages at many sites (skin, muscles, subserosa).	Often follows infectious bursal disease, which damages immune system.
Leukosis complex	Retroviruses	Chickens, perhaps other species.	Focal or diffuse neoplastic lesions in the liver. Other organs frequently affected with focal or diffuse neoplastic lesions.	If epizootic in chickens less than 5 months old the disease may be Marek's disease. In older birds, probably lymphoid leukosis. See section on viral tumors.

DISEASES WITH LESIONS IN LIVER

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Salmonellosis, pullorum diseases, fowl typhoid, paratyphoid	*Salmonella pullorum, S. gallinarum,* other *Salmonella*.	Chickens and turkeys. Many kinds of poultry, birds, and mammals have paratyphoid.	Sometimes 1-3 mm focal areas of hepatic necrosis. Often enlargement of the spleen. Enteritis, sometimes with raised plaques in the mucosa and with cheesy plugs in the gut or cecum. May be septicemic with few lesions, especially in the very young.	Salmonella infections predominate in the young. Many are transmitted through the egg. Interspecies transmission possible with paratyphoid. Salmonella infections often are septicemic.
Tuberculosis	*Mycobacterium avium*	Usually in chickens. Also, other poultry and wild birds.	Focal granulomas in the liver. Lesions often are numerous. Nodules (granulomas) along the periphery of the gut. Lesions in most organs and marrow in advanced cases. Emaciation.	Usually encountered in older chickens – those over 1 year. Extreme emaciation is the hallmark of tuberculosis.
Ulcerative enteritis	*Clostridium colinum*	Captive game birds, turkeys, chickens.	Focal and/or diffuse yellow areas in the liver. Deep ulcers scattered throughout the intestine.	A common disease of captive game birds. Increasing in poults and chickens.

DISEASES WITH LESIONS IN HEMOPOIETIC SYSTEM[A]

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Chicken infectious anemia (CIA)	Chicken infectious anemia virus	Chickens (2-4 weeks old).	Anemia, thymic atrophy. Pale pink to yellow bone marrow.	Often associated with gangrenous dermatitis. Particularly on wings (blue wing disease). Course is usually about 1 week. Affected broilers can be traced to a CIA-shedding breeder flock.
Duck plague (duck virus enteritis)	Herpesvirus	Wild or domestic ducks, geese, swans.	Widespread hemorrhages. Severe enteritis. Perhaps elevated plaques in esophagus, ceca, rectum, cloaca, or bursa. Hemorrhage and/or necrosis in lymphoid rings (discs) of gut.	Epizootic losses in waterfowl are suggestive. Typical lesions and inclusion bodies helpful in diagnosis. An important reportable disease.
Hemorrhagic syndrome	Marrow or bursal damage from: sulfa drugs, antibiotics, mycotoxins, viral infection. Possibly vitamin K deficiency.	Chickens.	Numerous hemorrhages at many sites. Pale fatty hypoplastic marrow.	May follow infectious bursal disease. Many outbreaks follow over-medication. Usually related to marrow damage. Some outbreaks may be due to inclusion body hepatitis (adenoviral infection).
Infectious bursal disease	Birnavirus	Chickens typically 3-6 weeks old.	Marked inflammation of the bursa of Fabricius, which is swollen early but atrophic later. Enlarged spleen. Hemorrhages common in heavy muscles.	Diarrhea, incoordination, dehydration, vent picking are usual signs. Course is about 1 week. Immune system damaged. May be followed by inclusion body hepatitis or gangrenous dermatitis.

DISEASES WITH LESIONS IN HEMOPOIETIC SYSTEM[A]

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Leukocytozoonosis	*Leukocytozoon* sp.	Turkeys, ducks, geese, guinea fowl, chickens.	Pallor, splenomegaly, liver degeneration and hypertrophy in some birds. Leukocytozoons visible in blood smears. Schizonts often in liver, spleen, brain.	Outbreaks correspond with hot months when simulid flies and culicoid midges are numerous; these flies breed in and along water courses. Surviving birds (wild or domesticated) often act as carriers. Signs in birds related to anemia.
Lymphoid leukosis	Retroviruses	Chickens, perhaps other species.	Internal neoplasms. Neoplastic lymphoblastic cells in bursa of Fabricius and many other organs.	May resemble Marek's disease. Usually observed in chickens over 4 months old. Neoplasia frequently in liver, spleen, kidneys; nodular tumors in bursa of Fabricius.
Marek's disease	Herpesvirus	Chickens, perhaps other species.	Internal neoplasms. Neoplastic pleomorphic lymphoid cells infiltrate, CNS, spleen, liver, kidney, nerve trunks, iris, ovary, and many other organs.	Often epizootic in chickens 6-20 weeks old. Persists in older birds. May closely resemble lymphoid leukosis. Bursa of Fabricius seldom neoplastic.

[A]This table contains only diseases that are more frequently encountered, are more significant economically, or have hepatic lesions clearly of diagnostic value. Many other avian diseases may have hepatic lesions, e.g., psittacosis, Arizona (paracolon) infection, turkey viral hepatitis, synovitis, *Pasteurella anatipestifer* infection, staphylococcosis, and listeriosis.

DISEASES WITH LESIONS IN THE MUSCULOSKELETAL SYSTEM

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Biotin deficiency	Biotin deficiency	Turkey poults.	Large twisted hocks and bowed shanks. Hyperkeratosis of skin on soles of feet and toes.	Lesions include hyperkeratotic skin at corners of mouth and on eyelids.
Bumblefoot	Trauma with secondary bacterial infection of feet.	Chickens, turkeys, falcons, other birds.	Swollen foot or toepads often with open lesions. May involve joints or toes or feet.	Usually sporadic. Concrete floors without litter may result in many cases. In falcons related to small, hard perches.
Cage layer fatigue (osteoporosis, adult rickets)	Controversial etiology. Possibly low phosphorus, low calcium, or imbalance. Caging a factor.	Chickens (caged layers).	Bones that easily break and splinter. Fractures, sometimes vertebral. Depletion of bone mineral.	Leg weakness or inability to stand. May recover if promptly removed and kept on floor.
Crooked (twisted) toes	Unknown	Chickens, turkeys.	Affected toes deviated laterally or medially.	Incidental finding. Don't identify as cause of lameness or confuse with riboflavin deficiency.
Deep pectoral myopathy (green muscle disease)	Ischemia following exertion.	Chickens, turkeys.	Unilateral or bilateral green areas of necrosis in deep pectoral muscles.	An aseptic necrosis of muscle that follows stress leading to muscle swelling.
Gout (articular gout)	A metabolic disease with deposition of uric acid crystals.	Chickens, turkeys, possibly other birds.	White to gray semisolid tophi deposited in and around joints. Affected joints enlarged and distorted. Most obvious on feet and legs. Emaciation.	Similar uric acid crystals may be deposited on viscera, especially the pericardium and capsule of liver. Perhaps in the ureters.
Infectious synovitis	*Mycoplasma synoviae*	Chickens, turkeys.	Joints and tendon sheaths swollen, most apparent on hocks, shanks, feet. Sticky synovial exudate. Sometimes the liver is green.	Many birds are lame and squat on floor. Breast blisters a common sequel. Other mycoplasmas may produce similar lesions

DISEASES WITH LESIONS IN THE MUSCULOSKELETAL SYSTEM

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Leg edema	Unknown	Turkeys.	Edema in subcutaneous tissues of leg, especially along medial thigh. Usually unilateral.	Perhaps related to degree of transport stress between farm and slaughter plant. Causes significant economic loss.
Nutritional myopathy	Deficiency of vitamin E, selenium, and sulfur-containing amino acids.	Chickens, turkeys, ducks.	In chickens and ducks necrotic muscle fibers in breast muscles or legs appear as white streaks or masses. In turkeys gray-white patches in gizzard musculature.	In chicks muscle lesions may be accompanied by encephalomalacia or exudative diathesis.
Osteomyelitis	Various bacteria metastasize to marrow.	Poultry and other birds.	Osteomyelitis at various sites. Commonly in femur, tibia, or vertebra.	Sporadic. Nonspecific.
Osteopetrosis	Associated with retrovirus infections.	Chickens.	Shanks and other long bones thickened, heavy, dense and with small marrow cavity.	Higher incidence in roosters. Bone lesions non-neoplastic.
Chondrodystrophy (perosis or slipped tendon)	Usually manganese or choline deficiency. Sometimes niacin, biotin.	Poultry and game birds in captivity.	Gastrocnemius tendon often slips off trochlea at hock and leg deviated (usually laterally) at hock. Bizarre position of leg distal to hock.	May be unilateral or bilateral. A disease of rapidly growing, young birds. Often in flocks fed mostly corn.
Rickets	Imbalance or deficiency of Ca/P/vitamin D_3.	Poultry and other birds raised in captivity.	In young birds - soft beaks and bones, beaded ribs, crooked keels, enlarged epiphyses, and enlarged paratyphoids.	Usually seen in birds a few weeks old; deficiency of vitamin D_3 frequently the cause.
Splay leg	Slippery surface under birds.	Young chickens, turkeys.	Lateral deviation of legs at hips.	High incidence in flocks brooded on slippery paper.

DISEASES WITH LESIONS IN THE MUSCULOSKELETAL SYSTEM

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Staphylococcal arthritis	*Staphylococcus aureus* infection with localization in various joints.	Turkeys, chickens.	Affected joints swollen, painful, usually with exudate. Lesions may be generalized but usually involve hocks and feet. May involve thoracolumbar junction.	Affected birds often severely crippled. Arthritis often preceded by septicemia. Common disease in turkeys.
Tibial dyschondroplasia	Possibly influenced by genotype. May be related to excess phosphorus or other dietary factors.	Broiler chicks, young turkeys.	Anterolateral bowing of tibias. Abnormal mass of cartilage in proximal tibia and/or metatsus. Perhaps fracture at site. Normal parathyroids. Osteomyelitis often occurs concurrently.	Squatting, reluctance to move, abnormal posture and gait. Retarded growth. Similar syndrome in ducks.
Viral arthritis (reovirus infection)	Reovirus	Chickens (meat type).	Swelling of tendons and tendon sheaths, mostly near hocks.	Sometimes leads to rupture of gastrocnemius tendon(s).

DISEASES WITH LESIONS IN THE REPRODUCTIVE TRACT

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Egg yolk peritonitis	Yolk ovulated into peritoneal cavity. Exact cause unknown.	Layers.	Yolk material among abdominal organs. Peritonitis of variable severity.	Rarely associated with abnormal ovary or oviduct. Occasionally accompanies many acute diseases.
Internal laying	Eggs regurgitated into peritoneal cavity. Cause unknown.	Layers.	Hard- or soft-shelled eggs in peritoneal cavity. Peritonitis.	Perhaps some cases are a sequel to oviduct damage from infectious bronchitis.
Lymphoid leukosis	Retrovirus	Chickens, perhaps other species.	Neoplasia of ovary. Focal or nodular neoplasia of the bursa of Fabricius.	Accompanied by many other neoplastic lesions. Usually in sexually mature birds. See notes on lymphoid leukosis.
Marek's disease	Herpesvirus	Chickens, perhaps other species.	Neoplasia of ovary. Atrophy of bursa of Fabricius.	Often accompanied by other neoplastic lesions. Usually in sexually immature birds. See notes on Marek's disease.
Prolapse of oviduct	Unknown. Often accompanies obesity.	Layers.	Oviduct prolapsed and often cannibalized.	Occurs frequently in young, fat pullets beginning to lay.
Pullorum disease fowl typhoid, paratyphoid	*Salmonella pullorum, S. gallinarum, Salmonella* sp.	Adult chickens and turkeys.	Oophoritis with bloody, cheesy or atrophic follicles. Orchitis.	Sometimes accompanied by focal myocarditis and pericarditis.
Salpingitis	May accompany mycoplasmosis or follow infectious bronchitis. Usually nonspecific. *E. coli* commonly cultured.	Layers.	Salpingitis of variable extent. Oviduct may be dilated with exudate.	A common cause of routine daily mortality in laying hens. May be an incidental finding in broilers with a history of airsacculitis.

DISEASES WITH LESIONS IN THE RESPIRATORY TRACT[A]

Name of Disease(s)	Etiology	Species Affected	Lesions[B]	Comments
Aspergillosis	*Aspergillus fumigatus*	Most poultry and birds susceptible. Commonly in chickens, turkeys, waterfowl, penguins, captive game birds.	Mycotic granulomas usually in lungs, perhaps along airways. Mycotic plaques or fuzzy mycelium often in air sacs. Lesions may transplant or metastasize to internal organs, brain, globes of eyes. Rarely in conjunctival sac.	Usually transmitted via moldy feed, litter, or hatchery contamination. *Dactylaria gallopava* and other fungi may produce similar signs and lesions.
Avian influenza (AI)	Orthomyxovirus (strains vary greatly in pathogenicity).	Turkeys, ducks, pheasants, quail, many wild birds, other poultry.	Highly variable. In mild form: often swollen sinuses, ocular or nasal discharge. In severe form: hemorrhages, exudation, focal necrosis in respiratory, digestive, urogenital, cardiovascular, or multiple systems. See AI section.	Enzootic forms in U.S. usually mild to moderate in severity and involve respiratory system. Egg production declines and shell abnormalities common in turkeys. Most outbreaks of AI in U.S. are in turkeys and ducks.
Bordetellosis rhinotracheitis (turkey coryza)	*Bordetella avium*	Turkeys, chickens.	Nasal, ocular, and sinus exudation. Tracheitis. Perhaps flattened trachea. Pneumonia uncommon in uncomplicated cases.	Causes deciliation of trachea leading to increased susceptibility to other respiratory diseases.
Chlamydiosis	*Chlamydia psittaci*	Occasionally turkeys, ducks. More often in wild exotic birds.	Variable, but often airsacculitis, pericarditis, fibrinous perihepatitis. Splenomegaly may be the only lesion in chronic cases.	Among poultry, most often seen in turkeys. May infect humans processing infected poultry, resulting in flu-like symptoms.

DISEASES WITH LESIONS IN THE RESPIRATORY TRACT[A]

Name of Disease(s)	Etiology	Species Affected	Lesions[B]	Comments
Infectious bronchitis (IB)	Coronavirus	Chickens.	Mild to moderate inflammation of respiratory tract. Occasional nephropathic outbreaks. When complicated by mycoplasmosis, severe airsacculitis, pericarditis, fibrinous perihepatitis.	Signs predominantly respiratory. Little mortality unless complicated and mostly in chicks less than 10 weeks old. In layers, egg production may drop as much as 50%.
Infectious laryngotracheitis	Herpesvirus	Chickens. Occasionally pheasants.	Usually severe hemorrhagic laryngotracheitis with bloody exudate and possibly fibrinous casts or cheesy plugs. Occasionally only mild laryngotracheitis with conjunctivitis.	Usually in semimature or mature chickens. Spreads more slowly than other viral diseases. May cause high mortality. Severe dyspnea with loud gasping and expectoration of bloody exudate. Mild form has few signs and lesions.
Mycoplasma gallisepticum infection (MG)	*M. gallisepticum*	Primarily chickens, turkeys, but also in many other poultry/birds.	Airsacculitis of variable severity. Usually pericarditis and, perhaps, fibrinous perihepatitis.	Often secondary to other diseases and vaccinations (especially Newcastle and infectious bronchitis) and stresses. Usually a chronic respiratory disease.
Newcastle disease (ND)	Paramyxovirus (strains vary greatly in pathogenicity).	Most poultry and birds susceptible. Usually seen in chickens.	Highly variable with various viral strains. Enzootic ND produces few/no gross lesions. Exotic ND produces many but variable lesions: swelling around eyes and on neck, hemorrhages at many sites including intestinal mucosa, severe tracheitis. See ND section.	Enzootic ND in U.S. usually manifested as a respiratory disease but a modest number of young birds may show concurrent or closely following CNS signs. Exotic ND rarely in the U.S. In layers with ND egg productions drops drastically or ceases.
Turkey Rhinotracheitis	Pneumovirus	Chickens and turkeys of any age, pheasants, guineas	Upper respiratory signs with nasal and ocular exudate, swollen heads and sinuses.	Variations in clinical presentation dependent on secondary infections. Egg production drops can reach 70%.

[A]Respiratory signs include one or more of the following: snicks, sneezes, dyspnea, gasping, rapid breathing, rales, ocular or nasal discharge, swollen sinuses. Signs in broilers may be severe, even with the milder strains. Secondary *Escherichia coli* infection is common.

[B]Unless stated otherwise lesions are those seen in turkeys or chickens.

DISEASES WITH LESIONS IN SKIN

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Biotin deficiency	Biotin deficiency	Chicks, turkey poults.	Dermatitis on feet and shanks. Crusty, scab like lesions at the commissures of the mouth, perhaps of the eyelids.	In turkeys enlarged hocks and bowing of the metatarsus usually are more obvious and may precede or accompany cutaneous lesions. Sometimes implicated in perosis, especially in poults.
Fowl pox , pigeon pox, canary pox, turkey pox	Poxvirus	Perhaps all birds.	Yellow pustules or dark brownish-red scabs on any unfeathered skin (face, wattles, eyelids of chickens; caruncle, snood of turkeys; feet and legs of cage and wild birds).	Yellow or gray plaques may occur in oral cavity, pharynx, conjunctiva, sinuses. Can be transmitted by mosquitoes. Intracytoplasmic inclusion bodies in epithelium of lesions.
Gangrenous dermatitis	Skin wounds with secondary *Staphylococcus*, *Clostridia*, etc.	Chickens.	Traumatized skin with an underlying cellulitis.	Severe losses have occurred in 4-16 week-old chickens and turkeys. Some outbreaks follow infectious bursal disease or adenoviral infection.
Inflammatory process, IP, cellulitis	*E. coli*	Young chickens.	Yellowish exudate under the skin may be edematous, jelly like to fibrinous and dry. With or without external dry, crusty skin.	Appears to be associated with scratches. Pathogenesis not completely understood.
Nonspecific dermatitis	External parasites, vitamin deficiencies, contact dermatitis.	Poultry and other birds.	Mild pityriasis. Ragged dull feathers. Mild dermatitis.	Ruffled, ragged feathers accompany many diseases and are nonspecific indicators of poor health. Check nutrition and for external parasites.

DISEASES WITH LESIONS IN THE SKIN

Name of Disease(s)	Etiology	Species Affected	Lesions	Comments
Pantothenic acid deficiency in chicks	Deficiency of pantothenic acid.	Chicks, perhaps other poultry.	Dermatitis on feet and shanks. Crusty scab like lesions at the commissures of the mouth, perhaps of the eyelids.	Ataxia followed by inability to stand. Myelin degeneration in much of cord.
Riboflavin deficiency	Riboflavin deficiency	Turkey poults and chicks.	Poults: Dermatitis on feet, shanks. Crusts at corners of mouth, on eyelids, vent. Chicks: long primary wing feathers. Both: hypertrophy and myelin degeneration of nerve trunks.	Poults and chicks may walk on hocks or sprawl with toes curled. A common deficiency.
Vesicular dermatitis (photosensitization)	Photosensitizing seeds, plants, feeds with/without fungus; phenothiazine. Sunlight required on skin.	Chickens, perhaps other birds.	Vesicles or scabs on unfeathered skin (comb wattles, face, legs caruncle, feet).	Affected skin wrinkled, ulcerated, and shrunken after vesicles rupture.
Xanthomatosis	Uncertain etiology. Possibly toxic materials in animal fats.	Chickens.	Thickened, roughened yellow areas of skin. Swelling of the wattle(s) of intermandibular area	Skin lesions are permanent. Affected birds condemned at slaughter. Cholesterol crystals and large foamy macrophages in affected skin.

DIFFERENTIAL DIAGNOSIS OF COMMON RESPIRATORY DISEASE OF CHICKENS

Diagnostic Aid	Mesogenic Newcastle Disease	Infectious Bronchitis	Infectious Laryngotracheitis	Chronic Respiratory Disease	Infectious Coryza
Speed of spread in the flock	Rapid	Rapid	Moderate	Slow; persistent	Rapid
Duration of flock signs	2 weeks	2 weeks	2-4 weeks	Weeks to months	Weeks to months
Egg production slump	Nearly complete cessation of lay.	Up to 50%	1-20%	1-20%	1-20%
Mortality in chicks less than 3 weeks old	25-90%	5-60%	Rarely occurs in chicks.	5-40% (seldom occurs in chicks)	Seldom occur in chicks.
Mortality in adults	0-5% (very high with exotic Newcastle).	Usually 0	Up to 50%	Low; many culls	Low; many culls.
Egg-borne transmission	No	No	No	Yes	No
Cause	Virus	Virus	Virus	*Mycoplasma gallisepticum*	*Hemophilus paragallinarum*
Vaccine available	Yes	Yes	Yes	Yes	Yes

DIFFERENTIAL DIAGNOSIS OF COMMON RESPIRATORY DISEASE OF CHICKENS

Diagnostic Aid	Mesogenic Newcastle Disease	Infectious Bronchitis	Infectious Laryngotracheitis	Chronic Respiratory Disease	Infectious Coryza
Natural carrier chickens	Rarely	Yes - up to 2 weeks (vaccinates also).	Yes – probably lifelong (vaccinates also).	Yes	Yes
Clinical diagnosis	Adults cease lay within 3 days. In chicks, acute respiratory disease with CNS signs in some and high mortality.	Acute epornitic respiratory disease without CNS signs but with sharp drop in egg production and shell quality.	Severe dyspnea with bloody mucus from the nares and high mortality in adult birds.	Chronic respiratory signs influenced by weather and season.	Respiratory signs with facial edema ocular and nasal discharge. Exudate has foul odor.
Unique features of agent	Produces hemorrhagic skin and death in chick embryos. Hemagglutinates.	Curling, stunting of embryos with passaging. Does not hemagglutinate.	Pocks on CAM, intranuclear inclusions in CAM and tracheal epithelium.	Growth in special broth; but not on blood agar initially. Hemagglutinates.	Tiny dewdrop colonies on blood agar in candle jar (needs nurse colony). Pleomorphic, Gram negative.
Serologic tests	HI test of value after 5 days; VN test of value after 10-21 days. ELISA test available.	VN test of value. ELISA test available.	ELISA test available. Seroconversion is slow.	Plate and tube agglutination tests. HI test. ELISA test available. Seroconversion is slow.	No test.
Gross lesions	Often none. Possibly mild airsacculitis and airway inflammation.	Possibly none. Often airsacculitis and tracheitis.	Bloody mucus on face, beak. Severe tracheitis with cheesy plugs in dead bird.	Marked airsacculitis. Fibrinous perihepatitis, adhesive pericarditis.	Facial edema. Eyelids adhered. Mucoid ocular and nasal discharge.

[A]Aspergillosis, avian influenza, and chlamydiosis have been omitted from this table although they may be classified as respiratory diseases. Occasional respiratory noises may be heard with certain other diseases if the bird is sick enough that mucus secretion accumulates in the respiratory tract.

POULTRY DRUG USE GUIDE

The following listing of approved poultry drugs for United States use is intended to provide a general guide of dose and preslaughter withdrawal time. When calculating withdrawal time, each day is a full 24 hours long, starting with the hour the bird last received the drug. The listing is neither inclusive nor exclusive and may change. Many of the listed drugs are approved for use in combination with other drugs. Drug approval does not indicate cross-clearance. For information regarding approved combinations and specific indications of use, the reader should consult one of the references listed at the end of this section. Italicized compounds are not for use in laying hens.

CHICKEN DRUG LIST

Active Ingredients	Route	Withdrawal Time (days)	Dose	Brand Name Examples
Amprolium	Water	0	0.006-0.024%	Amprol
Amprolium	Feed	0	0.0004-0.25%	Amprol
Arsanilic acid	Feed	5	90 g/ton	Pro-Gen 20%
Bacitracin methylene disalicyclate	Water	0	100-400 mg/gal	Solutracin
Bacitracin methylene disalicyclate	Feed	0	4-200 g/ton	BMD
Bacitracin zinc	Water	0	100-400 mg/gal	Baciferm Soluble
Bacitracin zinc	Feed	0	4-50 g/ton	Baciferm, Albac
*Bambermycin*s	Feed	0	1-2 g/ton	Flavomycin
Ceftiofur sodium[A]	Inject	0	0.08-0.20 mg/chick, 0.17 – 0.5 mg/poult	Naxcel
Chlortetracycline	Water	1	100-1,000 mg/gal	CTC Solable, Aureomycin Soluble Powder
Chlortetracycline	Feed	2	10500 g/ton	CLTC, ChlorMax, Aureomycin
Clopidol	Feed	5	0.0125-0.0250%	Coyden 25
Cyromazine[B]	Feed	3	1 lb/ton	Larvadex
Decoquinate	Feed	0	27.2 g/ton	Deccox
Diclazucil	Feed	0	1 ppm	Clinacox

Active Ingredients	Route	Withdrawal Time (days)	Dose	Brand Name Examples
Erythromycin[C]	Feed	1-2	92.5–185 g/ton	Erymycin
Gentamicin sulfate	Inject	35	0.2 mg	Garasol Injectable
Hygromycin B	Feed	3	8–12 g/ton	Hygromix
Lasalocid	Feed	0	68-113 g/ton	Avatec
Lincomycin	Feed	0	2 g/ton	Lincomix
Lincomycin HCl	Water	0	64 mg/gal	Lincomycin Soluble
Lincomycin/ spectinomycin[D]	Water	0	2 g antibacterial action/gal	LS 50 Water Soluble
Monensin	Feed	0	90-110 g/ton	Coban
Narasin	Feed	0	54-72 g/ton	Monteban
Narasin/nicarbazin	Feed	5	5490 g/ton of combination	Maxiban
Nicarbazin	Feed	4	0.0125%	Nicarb
Nitarsone	Feed	5	0.01875%	Histostat 50
Novobiocin	Feed	4	7-14 mg/lb BW/day	Albamix
Oxytetracycline hydrochloride	Water	5	200-800 mg/gal	Oxylet Soluble, Terramycin Powder
Oxytetracycline[E]	Feed	0-5	10–500 g/ton	Terramycin
Penicillin (from procaine penicillin)	Feed	0	2.4–0g/ton	Penicillin 100
Piperazine	Water or Feed	0	50 – 200 mg/bird	Wazine
Robenidine hydrochloride	Feed	5	30 g/ton	Robenz
Roxarsone	Water	5	0.002% (21.7g/oz)	3-Nitro
Roxarsone	Feed	5	22.7-45.4 g/ton	3-Nitro
Salinomycin	Feed	0	40-60 g/ton	Sacox, Bio-cox

Active Ingredients	Route	Withdrawal Time (days)	Dose	Brand Name Examples
Semduramicin	Feed	0	22.7 g/ton	Aviax
Spectinomycin dihydrochloride	Water	5	0.5-2 g/gal	Spectam Water Soluble
Spectinomycin dihydrochloride[A]	Inject	0	2.5-5 mg/bird	Spectam Injectable
Steptomycin Sulfate	Water	4	10-15 mg/lb	Streptomycin Oral Solution
Sulfadimethoxine	Water	5	0.05-0.25%	Albon, sulfadimethoxine soluble powder
Sulfadimethoxine/ ormetoprim	Feed	5	113.5 g/ 68.1 g/ton	Rofenaid
Sulfamethazine sodium	Water	10	50-124 mg/lb BW/day	Sulmet
Sulfaquinoxaline	Water	10	0.025-0.04%	Sulfaquinox-aline
Tetracycline hydrochloride	Water	4	200-800 mg/gal	Tetracycline, Solutet 324
Tylosin tartrate	Water	1 - 5	50 mg/lb BW/day	Tylan Soluble
Tylosin[F]	Feed	0□5	4-1,000 g/ton	Tylan
Virginiamycin	Feed	0	5-20 g/ton	Stafac
Zoalene	Feed	0	303 – 454 g/ton	Zoamix

[A] For use in 1 to 3-day-old chicks only.
[B] For us in layers or breeders only.
[C] Do not use high dose level (185 g/ton) in layers.
[D] Use only up to 7 days of age.
[F] For layers use 20-50 g/ton dose. Highest dose level (1,000 g/ton) requires 5-day withdrawal.

TURKEY DRUG LIST

Active Ingredients	Route	Withdrawal Time (days)	Dose	Brand Name Examples
Amprolium	Water	0	4-16fl oz/50 gal	Amprol
Amprolium	Feed	0	0.0125-0.025%	Amprol
Arsanilic Acid	Feed	5	90 g/ton	Pro-Gen 20%, 100%
Bacitracin methylene disalicyclate	Water	0	400 mg/gal	Solutracin
Bacitracin methylene disalicyclate	Feed	0	4-200 g/ton	BMD
Bacitracin zinc	Feed	0	4-50 g/ton	Baciferm, Albac
Bambermycins	Feed	0	1-2 g/ton	Flavomycin
Carbarsone	Feed	5	227-340.5 g/ton	Carb-O-Sep
Ceftiofur sodium [A]	Inject	0	0.17 – 0.5 mg/poult	Naxcel
Chlortetracycline	Water	1	100-400 mg/gal	Aureomycin Soluble
Chlortetracycline	Feed	1	10-400 g/ton	Aureomycin, Granular
Clopidol	Feed	5	0.0125-0.025%	Coyden 25
Diclazuril	Feed	0	1 ppm	Clinacox
Erythromycin	Feed	2	92.5-185 g/ton	Erymycin 100
Gentamicin	Inject	63	0.2 mg/bird	Garasol
Lasalocid	Feed	0	68-113 g/ton	Avatec
Monensin	Feed	0	54-90 g/ton	Coban
Neomycin sulfate	Water	0	10 mg/lb BW/day	Neomix
Nitrasone	Feed	5	0.01875%	Histostat
Novobiocin	Feed	4	4-8mg/lb BW/day	Albamix
Oxytetracycline hydrochloride	Water	4	200-400 mg/gal 25 mg/lb body wt	Terramycin
Oxytetracycline	Feed	0-5	10-200 g/ton	Terramycin
Penicillin (G potassium)	Water	1	1.5 million units/gal	Penicillin G Potassium
Penicillin (from procaine penicillin)	Feed	0	2.4 - 50 g/ton	Procaine Penicillin G for feed

Active Ingredients	Route	Withdrawal Time (days)	Dose	Brand Name Examples
Piperazine sulfate	Water	14	100-200 mg/bird	Wazine
Roxarsone	Feed	5	22.7-45.4 g/ton	3-Nitro
Spectinomycin dihydrochloride	Inject	0	2.5-5 mg/bird	Spectinomycin HCL Injectable
Sulfadimethoxine	Water	5	0.025%	Albon, sulfadimethoxine soluble powder
Sulfadimethoxine/ Ormetoprim	Feed	5	0.01%	Rofenaid
Sulfamethazine sodium	Water	10	53-130 mg/lb BW/day	Sulmet
Sulfaquinoxaline	Water	4	25 mg/lb BW/day	
Triple Sulfa (sulfamethazine, sulfamerazine, sulfaquinoxaline)	Water	14	0.025% solution	PoultrySulfa
Tylosin tartrate	Water	5	60 mg/lb BW/day	Tylan Soluble
Virginiamycin	Feed	0	10-20 g/ton	Stafac
Zoalene	Feed	0	151-454 g/ton	Zoamix

[A] For use in 1 to 3-day-old poults only.

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NECROPSY OF THE FOWL

1. Review the clinical history and consider all likely diagnoses.

2. Examine the externa and observe the clinical signs if the bird is alive. Pay particular attention to abnormalities of the hocks, tendon sheaths, eyes, and mouth. Check carefully for external parasites.

3. If alive, the fowl may be killed by any of three methods.

   A. Administration of CO2 gas in an appropriate closed container.

   B. Disarticulate the head at the atlantooccipital joint.

   C. Intravenous injection of barbiturate.

4. Moisten the feathers with water containing detergent. If ornithosis or psittacosis is suspected, the bird should be soaked in 5% Lysol solution and a laminar flow hood should be utilized for the necropsy.

5. With an enterotome or scissors, cut through one lateral commissure of the mouth and examine the oral cavity. [Fig. 1; Necropsy Technique #1]

6. Continue at the cut commissure and make a longitudinal incision through the skin of the neck to the thoracic inlet. Reflect the skin laterally and examine the paired vagus nerves and the thymus if present.

7. Make a longitudinal incision in the esophagus and crop. [Fig. 2; Necropsy Technique #2]
   Note the content and odor.

8. Make a longitudinal incision in the larynx and trachea and examine. [Fig. 3; Necropsy Technique #3]

9. With tin snips or heavy scissors, remove the upper beak by a transverse cut near the eyes. [Fig. 4; Necropsy Technique #4] This will allow inspection of the nasal cavity and will expose the open anterior end of the infraorbital sinuses.

10. Insert one blade of a sterile scissors into the infraorbital sinus. Make a longitudinal lateral incision through the wall of each sinus and examine them. [Fig. 5; Necropsy Technique #5] Culture the sinuses if indicated.

11. Incise the loose skin between the medial surface of each thigh and the abdomen. [Fig. 6; Necropsy Technique #6] Reflect the legs laterally and disarticulate the hip joints. [Fig. 7; Necropsy Technique #7] Incise the skin on the medial aspect of each leg and reflect it to expose the muscles and stifle joint. [Fig. 8; Necropsy Technique #8].

12. Connect the lateral skin incisions with a transverse skin incision across the middle of the abdomen. Reflect the skin of the breast anteriorly and, the skin of the abdomen posteriorly [Fig. 9; Necropsy Technique #9].

13. Make a longitudinal incision through the pectoral muscles on each side of the keel and over the costochondral junctions. The anterior end of each incision should intersect the thoracic inlet at the dorsoventral midpoint. With heavy scissors cut through the coracoid and clavicle bones.

14. With sterile scissors make a transverse incision through the posterior part of the abdominal muscles. On each side continue the incision anteriorly through the costochondral junctions. [Fig. 10; Necropsy Technique #10] Remove the ventral abdominal wall and breast as one piece, observing the air sacs as they are torn during removal.

15. Without touching them, examine the viscera and air sacs in situ. [Fig. 11; Necropsy Technique #11]

16. Using sterile instruments remove any organs and take any swabs desired for culturing. The spleen can be exposed aseptically by freeing the left margin of the gizzard and reflecting that organ to the bird's right side [Fig. 12; Necropsy Technique #12]. All unnecessary manipulations and delays prior to culture increase the probability of contamination. Take intestinal cultures last.

17. Examine the pancreas. Transect the esophagus at the anterior border of the proventriculus [Fig. 13; Necropsy Technique #13]. Reflect the entire gastrointestinal tract posteriorly [Fig. 14; Necropsy Technique #14] by cutting the mesenteric attachments and then remove it after transecting the rectum. Examine the bursa [Fig. 15; Necropsy Technique #15].

18. Remove and examine the liver and spleen.

19. Examine the genitalia. In the female, remove the ovary and oviduct and open the oviduct longitudinally.

20. Examine the ureters and kidneys in situ. If indicated you may remove them for closer examination.

21. Remove and examine the heart. [Fig. 16; Necropsy Technique #16] Examine the lungs [Fig. 17; Necropsy Technique #17] by reflecting them medially from their attachment to the rib cage. [Fig. 18; Necropsy Technique #18]

22. With an enterotome make a longitudinal incision through the proventriculus, ventriculus, small intestine, ceca, colon, and cloaca. [Fig. 19; Necropsy Technique #19] Examine for lesions and parasites.

23. Both brachial plexuses and sciatic nerves should be examined. The brachial plexus is most easily observed anterior to the first rib. The extrapelvic sciatic nerve [Fig. 20; Necropsy Technique #20] is exposed by careful separation of the adductor muscles. The intrapelvic portion is exposed by removal of the overlying portion of the kidneys by blunt dissection.

24. Using a sharp knife or scalpel, open each tibiotarsal joint and examine the joint fluid for signs of exudate.

25. Use the sharp knife or scalpel to make a longitudinal cut through the anterior or medial aspect of the head of the tibia to expose the growth plate of immature birds.

26. With an osteotome split one femur longitudinally and examine the bone marrow.

27. To examine the brain, disarticulate the head and skin it. Remove the calvarium with strong scissors using the same technique as for mammals. [Fig. 21; Necropsy Technique #21]

GLOSSARY

Airsacculitis	An inflammation of the air sacs.
All-in, All-out	Obtain all chicks or poults at one time, preferably from a single source and hatch. Start all the birds together and never add any birds to the flock.
Breeders	Poultry selected and managed specifically to produce eggs for hatching.
Broiler	A young chicken of a size and breed suitable for broiling, usually weighing 1-52 lb. Also called fryer.
Brooder	A shelter or hover, usually heated, for young fowl.
Broody	Ready to brood; said of hens showing a strong inclination to sit on a nest.
Bursa of Fabricius	An epithelial outgrowth of the cloaca in poultry that is the primary site of B-cell production. It regresses with age and is usually a vestigial organ in mature birds.
Candling	The process of examining an egg in front of a strong beam of light to determine quality, fertility, etc.
Capon	A castrated rooster.
Caponization	The castration of a rooster.
Caruncle	The fleshy, unfeathered area of skin on the upper neck of a turkey.
Cecum	Blind pouch. Poultry have paired ceca, which are attached to the large intestine near its junction with the small intestine.
Chick	A young chicken.
Cockerel	A young rooster (cock).
Comb	A red fleshy outgrowth on the top of the head of certain fowl. There are a number of types (e.g., single comb, rose comb).
Confinement Rearing	The practice of raising poultry indoors.
Cornish weight.	Meat-type chickens, often pullets, grown to no more than 2 pounds dressed
Debeaking	The practice of trimming the beak to prevent or reduce cannibalism. The term should be replaced by "beak trimming".
Drake	A male duck.
Ear lobe chickens.	A rounded, sometimes-pigmented area of skin near the external ear canal of
Egg dipping	The practice of submerging eggs, under certain conditions of time and temperature, in solution of antibiotics. The purpose is to reduce or eliminate certain micro organisms within the egg. For the same reason, fertile eggs may be heated or inoculated with antibiotics.

GLOSSARY

Gander	A male goose.
Gizzard	Ventriculus. A muscular enlargement of the alimentary canal in birds for grinding material entering from the proventriculus.
Hatchability	Refers to the number of eggs, in a fertile set, that hatch successfully. Also, may refer to the number of eggs that hatch of the total number of eggs set.
Hatcher	A piece of equipment for hatching eggs.
Hen	The female chicken and certain other female birds.
Incubation	The process of hatching chicks, by artificial means, usually in closely controlled sanitation and climatic conditions.
Incubation period	The time required for hatching of fertile eggs.
Layer	A common breed of chicken developed for egg laying. In the United States most layers are white leghorns.
Meat birds	See broiler, cornish, and roaster.
Molting	The process of shedding and replacing the feathers. Chickens can be "force molted" by manipulating the diet and light.
Parental immunity	Immunity obtained through antibodies passed to the chick through absorption of yolk material.
Potentiation	The process of increasing the activity of certain antibiotics. This is accomplished by combining two drugs that have synergistic action or by decreasing the calcium level in the diet.
Poult	A young turkey, pheasant, or similar fowl.
Proportioner	A mechanical device that dispenses a measured amount of medicinal or nutritional solutions into drinking water lines.
Proventriculus	Grandular stomach of the chicken and other avian species.
Pullet	A young hen, usually not more than 1 year old.
Ranged Poultry	Birds allowed access to a pasture or large lot.
Roaster	Meat-type chickens grown to live weights between 6 and 8 lb. Often these are cockerels because of their more rapid growth rates.
Rooster	A male chicken (cock) and certain other male birds.
Sentinel bird	Birds lacking immunity to specific diseases used for the detection of infectious agents by placement into commercial flocks. Seroconversion or pathogen isolation is determined in these previously naive birds.
Snood	A fleshy protuberance at the base of the beak of a turkey.
Spent fowl	Birds from layer or breeder flocks marketed for meat at the end of the laying cycle.

PHOTO INDEX

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DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
	Fig. 10; Marek's Disease; UC Davis	Kidney tumors	HL Shivaprasad; CAHFS, UC Davis
	Fig. 11; Marek's Disease; NCSU	Proventricular tumor	HJ Barns; NCSU
	Fig. 12; Marek's Disease; UC Davis	Nerve infiltration (HP)	HL Shivaprasad; CAHFS, UC Davis
II. AVIAN LEUKOSIS/ SARCOMA VIRUS			Cornell University
	Fig. 1; Leukosis; Cornell U	Osteopetrosis	
	Fig. 2; Leukosis; Cornell U	Visceral tumors	Cornell University
	Fig. 3; Leukosis; Cornell U	Bursal tumors	Cornell University
	Fig. 4; Leukosis; UC Davis	Myelocytomatosis	HL Shivaprasad; CAHFS, UC Davis
III. RETICULOENDOTHELIOSIS	**Fig. 1; Reticuloendotheliosis**	Liver tumor	HL Shivaprasad; CAHFS, UC Davis
IV. LYMPHOPROLIFERATIVE DISEASE			
CHICKEN INFECTIOUS ANEMIA	**Fig. 1; Chicken infectious anemia; Cornell U**	Fatty bone marrow	Cornell University
CORONAVIRAL ENTERITIS OF TURKEYS	**Fig.1; Coronaviral enteritis; Cornell U**	Gaseous Intestines	Cornell University
DUCK VIRUS ENTERITIS			
DUCK VIRUS HEPATITS	**Fig. 1; Duck viral hepatitis; Cornell U**	Duck with opisthotonus	Cornell University
	Fig. 2; Duck viral hepatitis; Cornell U	Liver with hemorrhages	Cornell University
EQUINE ENCEPHALITIS VIRAL INFECTION			
FOWL POX	**Fig. 1; Fowl Pox; UC Davis**	Pox scab	HL Shivaprasad; CAHFS, UC Davis
	Fig. 2; Fowl Pox; UC Davis	Skin pox	HL Shivaprasad; CAHFS, UC Davis
	Fig. 3; Fowl Pox; NCSU	Wet pox	HJ Barns; NCSU
	Fig. 4; Fowl Pox; UC Davis	Turkey pox	HL Shivaprasad; CAHFS, UC Davis
	Fig. 5; Fowl Pox; UC Davis	Cytoplasmic inclusion bodies (HP)	HL Shivaprasad; CAHFS, UC Davis
INFECTIOUS BRONCHITIS	**Fig. 1; Infectious bronchitis; Univ Montreal**	Chick with ocular discharge	M Boulianne; University of Montreal
	Fig. 2; Infectious bronchitis; Univ Montreal	Misshapened eggs	M Boulianne; University of Montreal
INFECTIOUS BURSALDISEASE	**Fig. 1; Infectious bursal disease; AAAP**	Edematous bursa	AAAP Slide Set # 14
	Fig. 2; Infectious bursal disease; Cornell U	Hemorrhagic bursa	Cornell University
INFECTIOUS LARYNGOTRACHEITIS	**Fig. 1; Infectious Laryngotracheitis; AAAP**	Chicken with dyspnea	AAAP Slide Set # 15
	Fig. 2; Infectious Laryngotracheitis; Cornell U	Expectoration of bloody mucus	Cornell University
	Fig. 3; Infectious Laryngotracheitis; AAAP	Conjunctivitis & nasal discharge	AAAP Slide Set # 15
	Fig. 4; Infectious Laryngotracheitis; AAAP	Bloody trachea	AAAP Slide Set # 15
	Fig. 5; Infectious Laryngotracheitis; AAAP	Intranuclear inclusion bodies	AAAP Slide Set # 15
			AAAP Slide Set # 15
NEWCASTLE DISEASE	**Fig. 1; Newcastle Disease; Univ Montreal**	Misshapened eggs	M Boulianne; University of Montreal
	Fig. 2; Newcastle Disease; UC Davis	Diphtheritic laryngotracheitis	R Crespo; CAHFS, UC Davis
	Fig. 3; Newcastle Disease; UC Davis	Diphtheritic oro-pharyngo-esophagitis	R Crespo; CAHFS, UC Davis

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
	Fig. 4; Newcastle Disease; UC Davis	Small intestine hemorrhage	R Crespo; CAHFS, UC Davis
	Fig. 5; Newcastle Disease; UC Davis	Cecal tonsil necrosis	R Crespo; CAHFS, UC Davis
	Fig. 6; Newcastle Disease; UC Davis	Proventricular hemorrhages	R Crespo; CAHFS, UC Davis
TURKEY VIRAL HEPATITIS	**Fig. 1; Turkey viral hepatitis; NCSU**	Hepatic necrosis	HJ Barns; NCSU
	Fig. 2; Turkey viral hepatitis; NCSU	Pancreatic necrosis	HJ Barns; NCSU
VIRAL ARTHRITIS	**Fig. 1; Viral arthritis; AAAP**	Bilateral rupture	AAAP Slide Set # 1
	Fig. 2; Viral arthritis; NC Dept of Ag	Ruptured gastrocnemius tendon	L Munger; RADDL; NC Dept of Ag
	Fig. 3; Viral arthritis; Cornell U	Ruptured gastrocnemius tendon	Cornell University
	Fig. 4; Viral arthritis; AAAP	Eroded cartilage	AAAP Slide Set # 1
BACTERIAL DISEASES			
AVIAN CHLAMYDIOSIS	**Fig. 1; Avian chlamydiosis; Cornell U**	Giemsa stain elementary bodies	Cornell University
	Fig. 2; Avian chlamydiosis; Cornell U	Pericarditis	Cornell University
	Fig. 3: Avian chlamydiosis; Cornell U	Giemsa stain elementary bodies	Cornell University
AVIAN TUBERCULOSIS	**Fig. 1; Avian tuberculosis; Cornell U**	Multifocal tubercles in intestines	Cornell University
BORDETELLOSIS	**Fig. 1; Bordetellosis; NCSU**	Conjunctivitis and bottle jaw	HJ Barns; NCSU
	Fig. 2; Bordetellosis; UC Davis	Collapsed Trachea	HL Shivaprasad; CAHFS, UC Davis
	Fig. 3; Bordetellosis; UC Davis	Trachea (HP)	HL Shivaprasad; CAHFS, UC Davis
BOTULISM	**Fig. 1; Botulism; Cornell U**	Limber neck	Cornell University
COLIBACILLOSIS	**Fig. 1; Colibacillosis; UC Davis**	Airsacculitis	B Charlton; CAHFS, UC Davis
	Fig. 2; Colibacillosis; UC Davis	Pericarditis & perihepatitis	B Charlton; CAHFS, UC Davis
	Fig. 3; Colibacillosis; NCSU	Omphalitis	HJ Barns; NCSU
	Fig. 4; Colibacillosis; UC Davis	Salpingitis	B Charlton; CAHFS, UC Davis
	Fig. 5; Colibacillosis; Cornell U	Coli-granulomas	Cornell University
	Fig. 6; Colibacillosis; UC Davis	Cellulitis	J Schrade; VMTRC, UC Davis
	Fig. 7; Colibacillosis; UC Davis	Cellulitis	J Schrade; VMTRC, UC Davis
ERYSIPELAS	**Fig. 1; Erysipelas; NCSU**	Swollen & necrotic snood & wattle	HJ Barns; NCSU
	Fig. 2; Erysipelas; Cornell U	Swollen hemorrhagic spleens	Cornell University
FOWL CHOLERA	**Fig. 1; Fowl Cholera; AAAP**	Swollen Wattles	AAAP Slide Set # 19
	Fig. 2; Fowl Cholera; AAAP	Torticollis	AAAP Slide Set # 19
	Fig. 3; Fowl Cholera; AAAP	Hepatic Necrosis	AAAP Slide Set # 19
	Fig. 4; Fowl Cholera; AAAP	Fibrinous Pneumonia	AAAP Slide Set # 19
	Fig. 5; Fowl Cholera; AAAP	Fibrinous Pneumonia (HP)	AAAP Slide Set # 19
	Fig. 6; Fowl Cholera; AAAP	Synovitis	AAAP Slide Set # 19
	Fig. 7: Fowl Cholera; AAAP	Cellulitis	AAAP Slide Set # 19
GANGRENOUS DERMATITIS			
INFECTIOUS CORYZA	**Fig. 1; Coryza; AAAP**	Swollen Sinus	AAAP Slide Set # 10
	Fig. 2; Coryza; AAAP	Swollen Wattles	AAAP Slide Set # 10
	Fig. 3; Coryza; AAAP	Nasal Exudate	AAAP Slide Set # 10
	Fig. 4; Coryza; AAAP	Satellitism	AAAP Slide Set # 10

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
MYCOPLASMOSIS			
I. MYCOPLASMA GALLISEPTICUM INFECTION	**Fig. 1; M. gallisepticum; AAAP**	Swollen infraorbital sinuses	AAAP Slide Set # 11
	Fig. 2; M. gallisepticum; AAAP	Chronic respiratory disease	AAAP Slide Set # 11
	Fig. 3; M. gallisepticum; AAAP	Airsacculitis	AAAP Slide Set # 11
II. MYCOPLASMA MELEAGRIDIS INFECTION	**Fig. 1; M. meleagridis; AAAP**	Healthy appearing MM infected breeder flock	AAAP Slide Set #13
	Fig. 2; M. meleagridis; AAAP	Mild airsacculitis	AAAP Slide Set #13
	Fig. 3; M. meleagridis; NCSU	Airsacculitis	HJ Barns; NCSU
	Fig. 4; M. meleagridis; AAAP	Bilateral varus deformity	AAAP Slide Set #13
III. MYCOPLASMA SYNOVIAE INFECTION	**Fig. 1: M. Synoviae; AAAP**	Synovial exudate	AAAP Slide Set # 12
	Fig. 2; M. Synoviae; AAAP	Synovial swelling	AAAP Slide Set # 12
NECROTIC ENTERITIS			
ORNITHOBACTERIUM RHINOTRACHEALEINFECTION			
SALMONELLOSIS			
I. PULLORUM DISEASE	**Fig. 1; Pullorum; Cornell U**	Atretic ovarian follicles	F. Williams, III; Cornell University
	Fig. 2; Pullorum; AAAP	Nodular myocarditis	AAAP Slide Set # 22
	Fig. 3; Pullorum; AAAP	Cecal cores	AAAP Slide Set # 22
		Bile-stained ("bronzed") liver	AAAP Slide Set # 22
II. FOWL TYPHOID	**Fig. 1; Fowl Typhoid; AAAP**	with necrotic foci	
III. ARIZONOSIS	**Fig. 1; Arizonosis; NCSU**	Torticollis	HJ Barns; NCSU
	Fig. 2; Arizonosis; NCSU	Cloudy eye	HJ Barns; NCSU
	Fig. 3; Arizonosis; NCSU	Encephalitis	HJ Barns; NCSU
IV. PARATHYPOID INFECTION			
SPIROCHETOSIS	**Fig. 1; Spirochetosis; Cornell U**	Blood smear showing spirochetes	Cornell University
STAPHYLOCOCCOSIS			
ULCERATIVE ENTERITIS	**Fig. 1; Ulcerative Enteritis; NCSU**	Intestinal ulcers	HJ Barns; NCSU
	Fig. 2; Ulcerative Enteritis; UC Davis	Intestinal ulcers	HL Shivaprasad; CAHFS, UC Davis
VIBRIONIC HEPATITIS			

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
FUNGAL DISEASE			
ASPERGILLOSIS	**Fig. 1; Aspergillosis; AAAP**	Gasping chicks	AAAP Slide Set #9
	Fig. 2; Aspergillosis; AAAP	Lung nodules	AAAP Slide Set #9
	Fig. 3; Aspergillosis; UC Davis	Tracheal plug	HL Shivaprasad; CAHFS, UC Davis
	Fig. 4; Aspergillosis; AAAP	Aspergillus fruiting body	AAAP Slide Set #9
	Fig. 5; Aspergillosis; AAAP	Aspergillus fumigatus on Sab Dex media	AAAP Slide Set #9
CANDIDIASIS	**Fig. 1; Candidiasis; UC Davis**	Crop mycosis	HL Shivaprasad; CAHFS, UC Davis
	Fig. 2; Candidiasis; UC Davis	Severe crop mycosis	HL Shivaprasad; CAHFS, UC Davis
DACTYLARIOSIS	**Fig. 1; Dactylariosis; NCSU**	Brain encephalitis	HJ Barns; NCSU
FAVUS	**Fig. 1; Favus; Cornell U**	White crusting of comb	Cornell University
MYCOTOXICOSIS			
I. AFLATOXICOSIS	**Fig. 1; Aflatoxicosis; Univ Missouri**	Tan liver (3ppm aflatoxin in feed) vs. normal liver	A Bermudez; VMDL, University of Missouri
	Fig. 2; Aflatoxicosis; Univ Missouri	Tan liver (3ppm aflatoxin in feed)	A Bermudez; VMDL, University of Missouri
	Fig. 3; Aflatoxicosis; Univ Missouri	Swollen kidneys (3ppm aflatoxin in feed) vs. normal	A Bermudez; VMDL, University of Missouri
II. CITRININ MYCOTOXICOSIS			
III. ERGOTISM			
IV. OCRATOXICOSIS			
V. OOSPOREIN MYCOTOXICOSIS			
VI. TRICHOTHECENE MYCOTOXICOSIS	**Fig. 1; Trichothecene; Auburn Univ**	Abnormal feathering	F Hoerr; Auburn University
	Fig. 2; Trichothecene; Auburn Univ	Oral ulceration & necrosis	F Hoerr; Auburn University
VII. ZEARALENONE MYCOTOXICOSIS			
PARASITIC DISEASES			
PARASITES AND PESTS			
LICE	**Fig. 1; Lice; Cornell U**	Lice eggs on feathers	Cornell University
MITES	**Fig. 1; Scaly leg mites; Univ Montreal**	Scaly leg mites	M Boulianne; University of Montreal
BLOOD-BORNE PARASITES			
COCCIDIOSIS	**Fig. 1; Coccidiosis; NCSU**	E. acervulina	HJ Barns; NCSU
	Fig. 2; Coccidiosis; Univ Montreal	E. acervulina	M Boulianne; University of Montreal
	Fig. 3; Coccidiosis; AAAP	E. necatrix	AAAP Slide Set #7
	Fig. 4; Coccidiosis; NCSU	E. necatrix	HJ Barns; NCSU
	Fig. 5; Coccidiosis; AAAP	E. maxima	AAAP Slide Set #7
	Fig. 6; Coccidiosis; AAAP	E. tenella	AAAP Slide Set #7
	Fig. 7; Coccidiosis; NCSU	E. meleagrimitis	HJ Barns; NCSU
	Fig. 8; Coccidiosis; NCSU	E. adenoeides	HJ Barns; NCSU

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
NEMATODES	**Fig. 1; Ascaridia; NCSU**	Intestinal ascarids	HJ Barns; NCSU
CRYPTOSPORIDIOSIS			
HEXAMITIASIS			
HISTOMONIASIS	**Fig. 1; Histomoniasis; NCSU**	Hepatitis & cecal cores	HJ Barns; NCSU
	Fig. 2; Histomoniasis; UC Davis	Cecal cores	HL Shivaprasad; CAHFS, UC Davis
	Fig. 3; Histomoniasis; UC Davis	Hepatitis	HL Shivaprasad; CAHFS, UC Davis
SARCOSPORIDIA	**Fig. 1; Sarcosporidia; Cornell U**	Sarcocysts in muscle	Cornell University
TOXOPLASMOSIS			
TRICHOMONIASIS			
NUTRITIONAL DISEASES			
BIOTIN DEFICIENCY	**Fig. 1; Biotin deficiency; NCSU**	Exudative dermatitis	HJ Barns; NCSU
	Fig. 2; Biotin deficiency; NCSU	Exudative dermatitis	HJ Barns; NCSU
FATTY LIVER-HEMORRHAGIC SYNDROME			
RIBOFLAVIN DEFICIENCY			
VITAMIN A DEFICIENCY	**Fig. 1; Vit A deficiency; UC Davis**	Pustules in crop	HL Shivaprasad; CAHFS, UC Davis
	Fig. 2; Vit A deficiency; UC Davis	Squamous metaplasia (HP)	HL Shivaprasad; CAHFS, UC Davis
VITAMIN E DEFICIENCY	**Fig. 1; Vit E deficiency; NCSU**	Chicks with paresis/paralysis	HJ Barns; NCSU
	Fig. 2; Vit E deficiency; NCSU	Hemorrhagic cerebellum	HJ Barns; NCSU
	Fig. 3; Vit E deficiency; NCSU	Gizzard muscle degeneration	HJ Barns; NCSU
RICKETS	**Fig. 1; Rickets; Cornell U**	Rubbery beak	Cornell University
	Fig. 2: Rickets; NCSU	Beaded ribs	HJ Barns; NCSU
MISCELLANOUS DISEASES			
CARDIOVASCULAR DISEASES			
I. ASCITES OR PULMONARY HYPERTENSION SYNDROME	**Fig. 1; Ascites; AAAP**	Ventricular hypertrophy	AAAP #23
	Fig. 2; Ascites; NCSU	Ascites	HJ Barns; NCSU
	Fig. 3; Ascites; AAAP	Fibrotic liver	AAAP #23
II. AORTIC RUPTURE OR DISSECTING ANEURYSM	**Fig. 1; Aortic Rupture; NCSU**	Aortic rupture	HJ Barns; NCSU
III. ROUND HEART DISEASE OF CHICKENS			

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
IV. ROUND HEART DISEASE OF TURKEYS (DILATED CARDIOMYOPATHY)	**Fig. 1; Round heart disease; NCSU**	Dilated heart	HJ Barns; NCSU
	Fig. 2; Round heart disease; UC Davis	Dilated heart	HL Shivaprasad; CAHFS, UC Davis
	Fig. 3; Round heart disease; NCSU	Ascites	HJ Barns; NCSU
V. SUDDEN DEATH SYNDROME OF CHICKENS			
VI. SUDDEN DEATH SYNDROME OF TURKEYS(PERIRENAL HEMORRHAGE)	**Fig. 1; Sudden Death Syndrome; NCSU**	Perirenal hemorrhage	HJ Barns; NCSU
DIGESTIVE DISORDERS			
I. NECROTIC HEMORRHAGIC HEPATITIS			
II. POULT ENTERITIS COMPLEX			
MUSCULOSKELETAL DISORDERS			
DEEP PECTORAL MYOPATHY	**Fig. 1; Deep pectoral myopathy; UC Davis**	Deep pectoral myopathy	HL Shivaprasad; CAHFS, UC Davis
	Fig. 2; Deep pectoral myopathy; NCSU	Deep pectoral myopathy	HJ Barns; NCSU
OSTEOMYELITIS	**Fig. 1; Osteomyelitis; NCSU**	Femoral osteomyelitis	HJ Barns; NCSU
SPLAY LEG	**Fig. 1; Splay leg; Univ Montreal**	Splay leg	M Boulianne; University of Montreal
SPONDYLOLISTHESIS	**Fig. 1; Spondylolisthesis; NCSU**	Spondylolisthesis	HJ Barns; NCSU
TIBIAL DYSCHONDROPLASIA	**Fig. 1; Tibial dyschondroplasia; NCSU**	Tibial dyschondroplasia	HJ Barns; NCSU
	Fig. 2; Tibial dyschondroplasia; UC Davis	Tibial dyschondroplasia	HL Shivaprasad; CAHFS, UC Davis
TIBIAL ROTATION	**Fig. 1; Tibial Rotation NCSU**	Tibial rotation	HJ Barns; NCSU
	Fig. 2; Tibial Rotation NCSU	Tibial rotation	HJ Barns; NCSU
REPRODUCTIVE DISORDERS			
URINARY DISORDERS			
INTEGUMENT DISORDERS			
XANTHOMATOSIS	**Fig. 1; Xanthomatosis; Cornell U**	Xanthomatosis	Cornell University

DISEASE	TEXT FIGURES	SUBJECT	CREDIT/AUTHOR
BEHAVIOR DISORDERS			
CANNIBAISM	**Fig. 1; Cannibalism; Univ Montreal**	Head pecking	M Boulianne; University of Montreal
APPENDIX	**Fig. 1; Necropsy Technique #1**	Scissors at commissure of the beak	M Boulianne; University of Montreal
NECROPSY OF THE FOWL	**Fig. 2; Necropsy Technique #2**	Esophagus being exposed	M Boulianne; University of Montreal
	Fig. 3; Necropsy Technique #3	Trachea being opened	M Boulianne; University of Montreal
	Fig. 4; Necropsy Technique #4	Beak being transected	M Boulianne; University of Montreal
	Fig. 5; Necropsy Technique #5	Sinus & turbinates exposed	M Boulianne; University of Montreal
	Fig. 6; Necropsy Technique #6	Skin being reflexed from the breast	M Boulianne; University of Montreal
	Fig. 7; Necropsy Technique #7	Reflextion of legs to disarticulate hip joint	M Boulianne; University of Montreal
	Fig. 8; Necropsy Technique #8	Tarsometatarsus being incised	M Boulianne; University of Montreal
	Fig. 9; Necropsy Technique #9	Abdominal wall exposed	M Boulianne; University of Montreal
	Fig. 10; Necropsy Technique #10	Incision of abdominal cavity	M Boulianne; University of Montreal
	Fig. 11; Necropsy Technique #11	Air Sacs exposed	M Boulianne; University of Montreal
	Fig. 12; Necropsy Technique #12	Spleen Exposed	M Boulianne; University of Montreal
	Fig. 13; Necropsy Technique #13	Transections of digestive tract	M Boulianne; University of Montreal
	Fig. 14; Necropsy Technique #14	Gastrointestinal (intestines laid out)	M Boulianne; University of Montreal
	Fig. 15; Necropsy Technique #15	Bursa exposed	M Boulianne; University of Montreal
	Fig. 16; Necropsy Technique #16	Heart exposed	M Boulianne; University of Montreal
	Fig. 17; Necropsy Technique #17	Lungs exposed	M Boulianne; University of Montreal
	Fig. 18; Necropsy Technique #18	Lungs being pulled from the rib cage	M Boulianne; University of Montreal
	Fig. 19; Necropsy Technique #19	Longitudinal opening of the GIT	M Boulianne; University of Montreal
	Fig. 20; Necropsy Technique #20	Sciatic nerve exposed	M Boulianne; University of Montreal
	Fig. 21; Necropsy Technique #21	Brain exposed	M Boulianne; University of Montreal