Global Livestock CRSP

HANDBOOK
of
POULTRY DISEASES IMPORTANT IN AFRICA

Clinical Signs and Lesions
Differential Diagnosis
Cause and Transmission
Diagnosis, Treatment,
Prevention and Recovery

THIS HANDBOOK IS DESIGNED TO BE USED AS A REFERENCE FOR THE POULTRY HEALTH FOR DEVELOPMENT COURSE AND AS A REFERENCE FOR FIELD VETERINARIANS IN AFRICA

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INTRODUCTION

Purpose of this Handbook:

This handbook is designed as a reference of poultry diseases in Africa for the Poultry Health for Development course. The handbook is referred to in the course curriculum. This handbook will also serve as a stand-alone disease diagnostics, prevention and recovery reference for veterinarians working in the field.

In addition to general information on diseases, an attempt has been made to highlight issues of particular relevance to Africa.

Coverage for each disease generally includes these subtopics:

Name and (common names)
Clinical signs and lesions
Differential diagnoses
Cause, transmission, epidemiology
Diagnoses
Prevention
Treatment
Recovery

Some poultry health problems, such as stress, have different subtopics.

How to use this handbook:

For looking up information about poultry diseases, you will find the diseases listed alphabetically. In the first section of the handbook you will find information regarding causes of disease, categories of disease, clinical signs and necropsy.

For diagnosing diseases in the field:
1. Use the Poultry Disease Diagnosis Decision–Tree in Appendix A of this Handbook to narrow down the possible diseases based on clinical signs and lesions.

2. Look up brief descriptions of the suspected poultry diseases in the Categories of Disease charts on pages 13-16.

3. For more detailed discussion of signs, cause, transmission, differential diagnosis, and guidance on prevention, treatment, and recovery, look up the suspected diseases in the alphabetized section of this Handbook, pages 17-77..

CAUSES OF DISEASE

Important Point: If a bird is showing signs of disease, then there is a reason.

Diseases can be caused by things you can see and things that you can't, including bacteria, viruses, fungi, parasites, and poisons. An incomplete diet may also cause disease.

1. ***Bacterial*** diseases can be treated with antibiotics.

2. ***Viral*** diseases cannot be treated with antibiotics. Vaccines can be effective for preventing some viral diseases.

3. ***Fungi*** may cause illness either by growing in the birds or by producing poisons. There are no treatments for fungal diseases but they can be treated by cleaning the environment.

4. ***Parasites*** can irritate and annoy birds, and some can transmit bacteria and viruses. Parasites are categorized as either internal or external, depending on where they live in or on the bird.

   External parasites generally bite and irritate birds but can also cause blood loss and transmit diseases. *Mites, lice,* and *ticks* are all external parasites. *Flies, fleas, beetles,* and *mosquitoes*, although they live both on and off the bird, can transmit diseases like fowl pox between birds and, they can concentrate poisons.

   Internal parasites can be very small (like coccidia) or very large (like most worms). There are treatments and vaccines for some internal parasites.

5. ***Poisons*** like botulinum and aflatoxin are produced by living organisms (fungi and bacteria). Poisons that are made by humans, like pesticides or disinfectants, can also cause clinical signs in poultry if they eat or drink them.

6. ***Nutritional deficiencies*** can result in signs of illness and death, especially in young birds. Once the deficit has been identified and corrected, the birds will often make a rapid recovery.

7. ***Environmental conditions***, especially heat, can kill large numbers of birds and are among the key causes that should be considered when there is high mortality. Heat loss is more common in confined birds than those that are free-ranging.

8. ***Predation*** usually results in the loss of a few birds rather than whole flocks.

CATEGORIES OF DISEASE IN POULTRY

Bird diseases and conditions can be divided into three categories.

Category 1 Diseases:

1. Death in the flock is very high – often up to 100%.
2. Multiple organ systems (respiratory, digestive, nervous, reproductive, etc.) are affected by these diseases.
3. Trade restrictions may be associated with these diseases; quarantines and notification of animal health authorities may be required.
4. Prevention through vaccination and biosecurity are the only options. Treatment of active disease is ineffective. Stamping out flocks may be the only option for controlling the disease once birds are infected.

Category 2 Diseases:

1. Mortality is lower than in Category 1 disease and/or treatment is possible.
2. Only one or a few organ systems are involved.
3. These diseases limit how much income a community can earn from poultry flocks; they result in the death of some birds, decrease egg production, and/or lower feed conversion rates.
4. There are medications, vaccinations, and other treatments available for these diseases.

Category 3 Diseases:

1. These are conditions rather than diseases, and are not caused by organisms that are spread between birds.
2. Depending on the cause, they may affect multiple organ systems.
3. They are environmental in origin and control is mostly through providing adequate housing and sanitation.
4. Medication may be available for some conditions in this category.

CLINICAL SIGNS

Sick birds show clinical signs.

Clinical signs are caused when a disease or condition affects all or part of a bird's body.

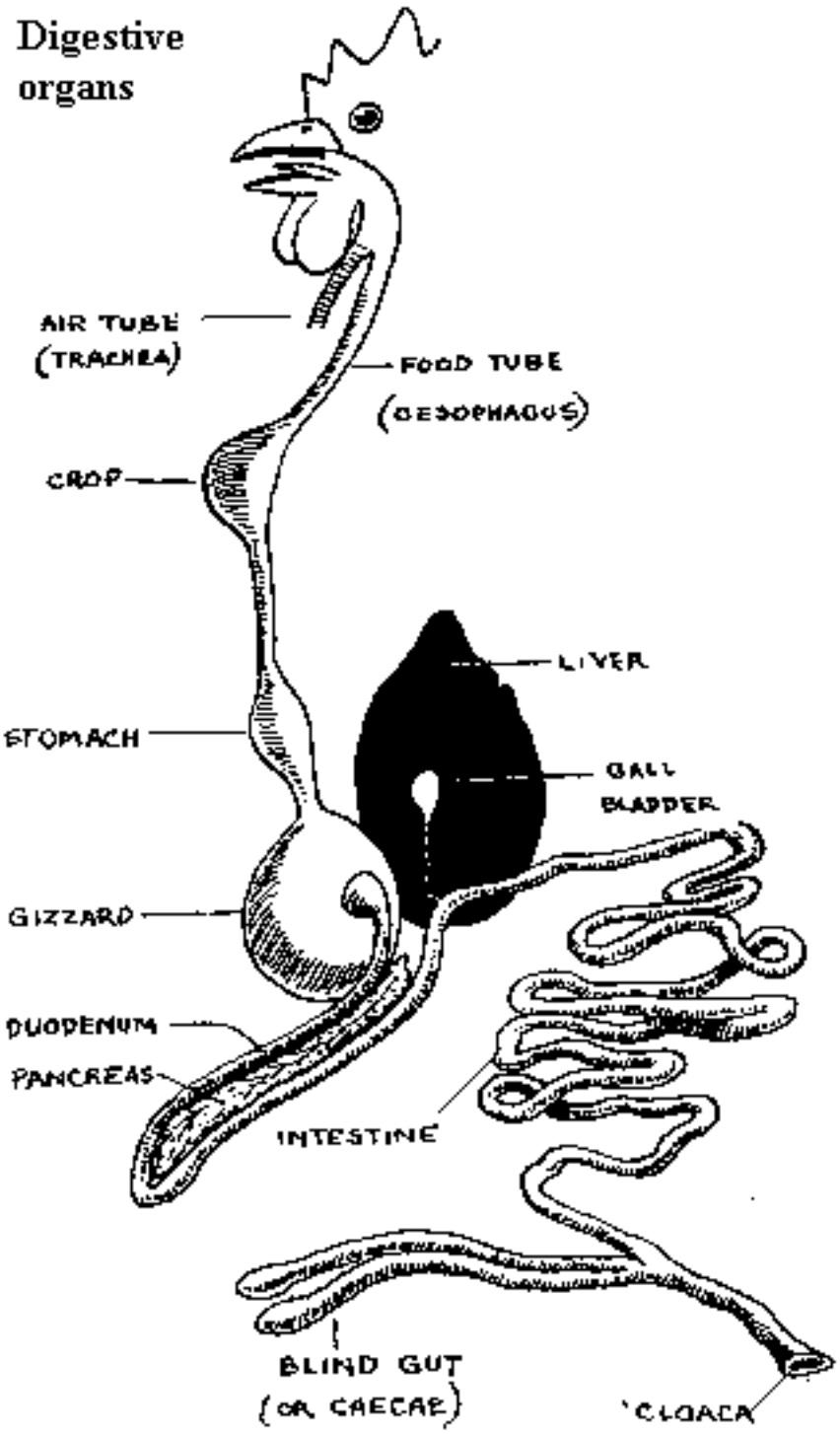
Important Point: **The part of the bird that is affected on the inside will**

determine the clinical signs the bird shows on the outside.

Many clinical signs are specific to the organ system that is affected on the inside of the bird. The parts of the bird that work together so that the bird can function comprise an organ system. For example, the lungs and nose and trachea are part of the respiratory system that keeps the bird breathing.

Table 1. Organ systems, their functions and what clinical signs appear.

Organ system	Function	Examples of clinical signs
Respiratory	Breathing	Gasping, coughing
Digestive	Eating, defecating, weight gain	Thin birds, abnormal feces
Skin & feathers	Protection from the environment	Sores, feather loss
Nervous	Coordination, standing, walking	Twisted neck, rolling, can't hold head up
Reproductive	Laying eggs, producing chicks	Decreased egg numbers, chicks don't hatch
Muscles & skeleton	Walking, flapping wings	Cannot stand, swollen joints
Immune	Protection from disease, response to vaccination	Frequent infections



Example of an organ system: The digestive system includes all of the parts of the bird that are involved in eating, drinking and digesting. The function of the digestive system is to provide fuel for all of the other systems.

Necropsy of Birds

1. Review the clinical history and consider what the likely diagnosis is.
2. Examine the outside of the bird. Observe how the bird acts if the bird is still alive. Check for external parasites.
3. Humanely euthanize the bird (See Handout Y: “Humane Euthanasia of Individual Birds”).
4. Moisten the feathers with water that contains a small amount of soap.
5. With scissors, cut through one corner of the mouth so that the oral cavity can be examined.
6. Continue the cut down the neck of the bird from the mouth to the chest, through the skin only. Examine the thymus, if present.
7. Make an incision down the esophagus from the mouth to the crop. Examine.
8. Make an incision down the trachea from the mouth to the chest. Examine.
9. With heavy scissors, cut across the beak just in front of the eyes. Examine the nasal cavities.
10. Using a scalpel or one side of a small scissors, cut into each infraorbital sinus, just below the eye. Examine the color and look for any extensive mucous or other material.
11. Pull the leg bone out of the hip joint. Bend the legs backwards, towards the back of the bird.
12. Cut the skin on the inside of each thigh from the hip to the stifle joint. Pull the skin back so you can see the muscles.
13. Make another cut through the skin of the abdomen that connects the two cuts on the thighs. Pull the breast skin up and the abdominal skin down so that the midsection of the bird is exposed.
14. Using scissors, make a cut in the abdominal body wall that follows the bottom edge of the rib cage. Be careful not to puncture the intestines.
15. Continue this cut up through the ribs on either side. Cut through the bones without damaging the organs underneath. Be sure to cut through the strong coracoid bones at the top of the rib cage.
16. Now that the ribs have been cut through, remove the rib cage and breast muscles as one piece. Observe the air sacs as you do this, because they will be disrupted as the rib cage comes off.
17. All the organs should now be exposed. Look at them without moving them

first.

18. Examine the pancreas.
19. Cut across the esophagus just above the proventriculus. Pull downward so that the digestive tract comes away from the bird and can be examined in detail. If desired, remove the digestive tract from the bird entirely by cutting down near the cloaca.
20. Using scissors, cut length-wise down the digestive tract to examine the inside. You may need heavy scissors or a scalpel to get through the tough muscle of the ventriculus. Examine for wounds or parasites.
21. Remove and examine the liver and spleen.
22. Examine the reproductive organs. In the female, remove the ovary and oviduct. Cut the oviduct length-wise to look at the inside.
23. Remove and examine the heart.
24. Examine the lungs. Remove them by freeing them from their attachment to the ribs.
25. Look at the sciatic nerves in each thigh. You may need to move the leg muscles to find it.
26. With a sharp blade, open each knee (tibiotarsal) joint and examine.
27. With a sharp blade, split one leg bone length-wise to expose the bone marrow.
28. To examine the brain, remove the head from the body. Skin the head. With strong scissors, carefully chip and peel off the top of the skull to expose the brain. Be careful not damage the brain.

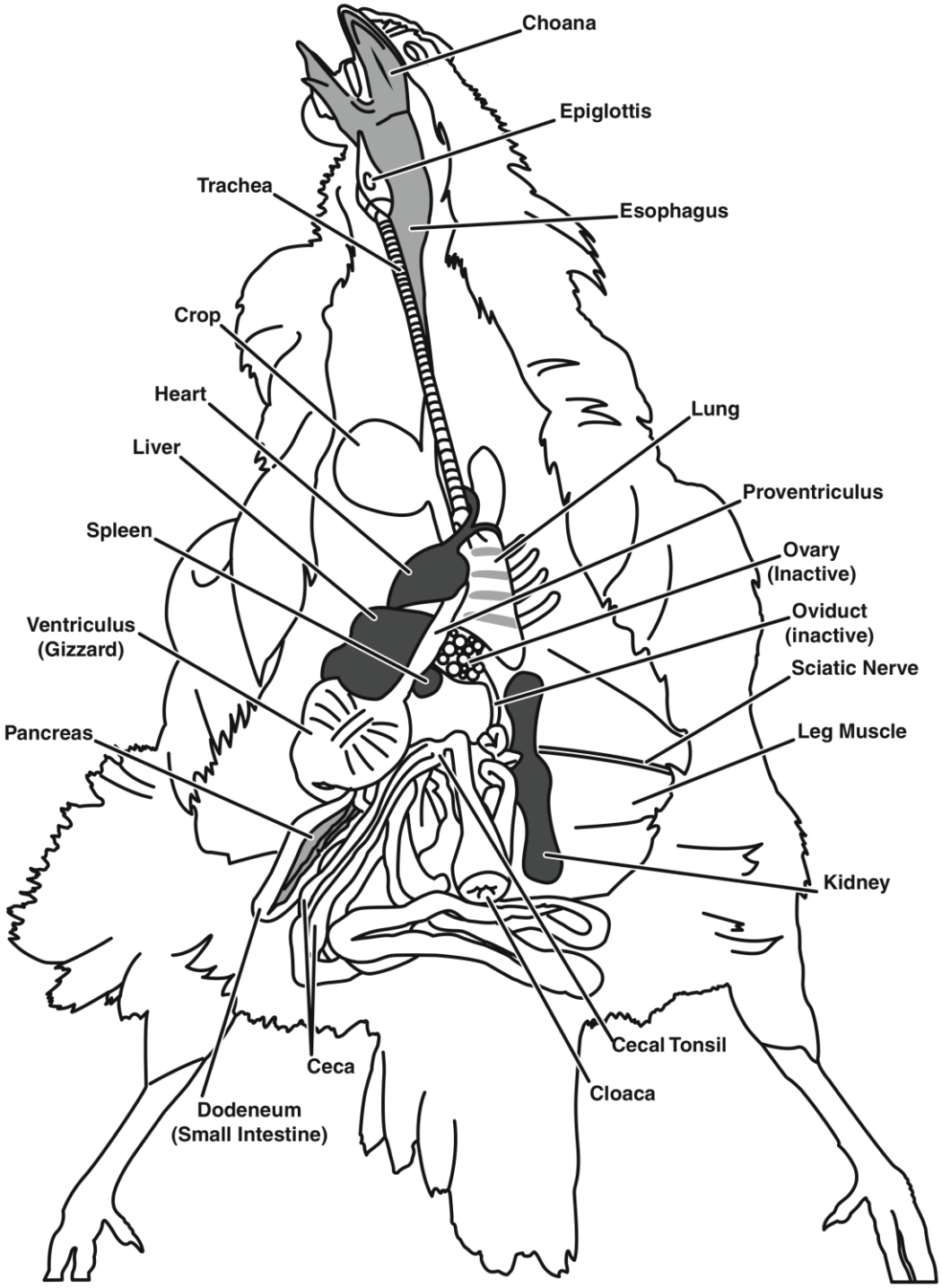


ILLUSTRATION: Anatomy of the chicken

Category 1 Diseases

Disease	Cause	Ages affected	Species	Mortality	Signs and Symptoms	Control
1. Velogenic viscerotropic Newcastle Disease (VVND)	Virus	Any	Most domestic birds	Mortality may reach 100% but often lower in ducks	Sudden mortality, often with few or minimal signs Nervous: Balance & walking problems, twisted necks Respiratory: gasping, difficulty breathing, swelling of the head Digestive: diarrhea Reproductive: decreased egg numbers	Vaccination, biosecurity
2. Highly Pathogenic Avian Influenza (HPAI)	Virus	Any	Most domestic birds	Mortality may reach 100% but often lower in ducks and pigeons	Sudden mortality, often with few or no signs Respiratory: gasping, swelling of wattles & combs Nervous: tremors of the head and neck Digestive: diarrhea, thirst Reproductive: soft-shelled or shell-less eggs, decreased egg numbers	Biosecurity, depopulation, (vaccination)
3. Duck viral enteritis (duck plague)	Virus	Any, although adults more severely affected	Wild and domestic ducks and geese	5-100% with the highest mortality in older birds	Sudden mortality, often with few or no signs Digestive: watery diarrhea, decreased appetite, thirst Reproductive: decreased egg numbers Nervous: difficulty walking, tremors Respiratory: pasted eyelids, nasal discharge	Biosecurity, (vaccination)
4. Pigeon Paramyxovirus (PPMV)	Virus	Any	Pigeons	Mortality may reach 100%	Adults neglect squab, resulting in their deaths. Nervous: Balance & walking problems, twisted necks, head tremors, inability to fly Digestive: diarrhea, thirst	Vaccination, biosecurity

Category 2 Diseases

Disease	Cause	Ages affected	Species	Mortality	Signs and Symptoms	Control
5. Newcastle Disease (lentogenic or mesogenic)	Virus	Any	Most domestic birds	Low, except in very young birds where mortality may reach 20%	Signs may vary by species. There may be no signs in waterfowl. Respiratory: sneezing, coughing, difficulty breathing Nervous: twisted necks Reproductive: decreased egg numbers	Vaccination, biosecurity
6. Low Pathogenicity Avian Influenza (LPAI)	Virus	Any	Most domestic birds	Usually <5% but may be up to 60% in turkeys.	Signs vary by species and infecting virus. There may be no signs in waterfowl. Respiratory: sneezing, coughing, wheezing Reproductive: decreased egg numbers	Biosecurity, depopulation, (vaccination)
7. Infectious Laryngotracheitis (ILTV)	Virus	Any but mostly in adults	Chickens	Usually 10-20% but may be very mild <2% mortality or very severe >50% mortality	Respiratory: nasal discharge, difficulty breathing, coughing, bloody mucus Reproductive: decreased egg numbers	Vaccination, biosecurity
8. Infectious bronchitis virus	Virus	Any but most severe in chicks	Chickens	0-25%	Birds may be depressed with ruffled feathers. Respiratory: gasping, coughing, sneezing, wet eyes Reproductive: thin-shelled, rough and misshapen eggs, decreased egg numbers	Vaccination, medication for secondary bacteria
9. Marek's Disease	Virus	Usually 3-30 weeks of age	Chickens	0-30% in unvaccinated flocks	Unthriftiness, failure to gain weight. Nervous: paralyzed in one or both legs or wings, difficulty standing. Pale eyes.	Vaccination
10. Avian Leukosis	Virus	>30 weeks of age	Chickens	Usually <3% although it may exceed 20% in some cases	Unthriftiness, weight loss, enlarged abdomen Reproductive: decreased egg numbers	Chicks from clean flocks, biosecurity
11. Duck virus hepatitis	Virus	Young, <6 weeks	Ducks	Close to 100% in ducklings <1 wk old. 50% in 1-3 wk old, very low in >4 wk old	Death may be the first sign of disease. Nervous: birds fall on their sides, kicking	Isolation of young ducks, (vaccination)
12. Infectious bursal disease	Virus	Mostly 3-6 weeks	Chickens	Usually 0-25% but in some cases, may be up to 100%	Prostration and death Digestive: vent picking, soiled vent feathers, whitish or watery diarrhea	Vaccination, biosecurity
13. Avian Encephalomye-litis	Virus	Mostly 1-3 weeks	Chickens	May reach 50% in young birds	Nervous: difficulty walking, paralysis, tremors	Vaccination
14. Fowl Pox	Virus	Any, except newly-hatched	Chickens, turkeys	<5% in skin form; 10-50% in respiratory form.	Poor weight gain. Skin: scabby, raised pocks on the face Digestive: Loss of appetite Respiratory: nasal discharge, difficulty breathing	Vaccination, biosecurity

15. Fowl cholera	Bacteria	Birds > 4 weeks are most susceptible	Chickens, ducks, geese, most birds	10-90% mortality. Mortality is highest in turkeys, ducks.	Death may be the first sign of disease. Respiratory: Gasping, difficulty breathing Digestive: diarrhea, especially in ducks	Medication, remove reservoirs, vaccination
16. Septicemia, omphalitis	Bacteria	Birds less than 2 weeks old	Any	Variable. May reach 100% or it may be small..	Late incubation mortality. Navel is inflamed and the abdomen is distended.	Medication, hatchery sanitation
17. Mycoplasmosis	Bacteria	Any	Chickens, pigeons, turkeys	Very low.	Respiratory: Facial swelling, nasal discharge, coughing, foamy eyes Reproductive: Decreased egg numbers, decreased hatchability Skeletal: joint swelling	Vaccination, biosecurity
18. Chlamydiosis	Bacteria	Any	Ducks, pigeons, turkeys	Variable, but severe cases may have 5-30% morality	Depression, weakness. Nervous (young ducks): trembling, imbalanced gait Respiratory: swollen eyelids, nasal discharge, difficulty breathing Digestive (young ducks): yellow-green diarrhea.	Medication, biosecurity
19. Infectious Coryza	Bacteria	Any, disease most severe in mature birds	Chickens	Rapid onset and high mortality.	Respiratory: Facial swelling, especially around the eyes, nasal discharge, rales Digestive: Loss of appetite, sometimes diarrhea. Reproductive: decreased egg numbers	Vaccination, remove reservoirs, biosecurity.
20. Trichomoniasis	Protozoa	Young birds	Pigeons	Can be up to 50% without treatment	Young birds lose weight and may die. Digestive: thick, yellow areas inside the mouth, difficulty closing mouth	Sanitation, medication
21. Coccidiosis	Protozoa	Young birds, older birds become immune	Most, although the coccidia of one species do not infest other birds	Variable depending on how severe the case is and the type of coccidia.	Depression, weakness, decreased weight gain, dehydration. Digestive: may have mucoid or bloody diarrhea.	Self immunization, medication
22. Histomoniasis	Protozoa	Turkeys: 3-12 weeks. Chickens: 4-6 weeks	Turkeys, chickens	Mortality is generally low in chickens <30% but higher in turkeys (up to 70%)	Depression, weakness Digestive: loss of appetite, yellow feces in turkeys, blooding droppings from chickens.	Medication, put birds on wire or cement

Category 3 Diseases

Condition	Cause	Ages affected	Species	Mortality	Signs and Symptoms	Control
23. External parasites	Insects, arachnids	Any	Any	Usually low unless infestations are severe	Birds can become weak and unthrifty if heavily infested. Skin: Mites, ticks, fleas and lice can cause itching, loss of feathers. Reproductive: decreased egg numbers	Cleaning of environment between flocks, pesticides applied to the bird and the environment
24. Internal parasites	Various worms	Any but the most severe	Any	Mortality is variable depending on the age of	Depression, failure to gain weight, anemia. Digestive: Diarrhea	Medication. sanitation

		disease is in young birds		the birds, type and severity of infestation		
25. Aflatoxicosis	Toxin from fungus	Young birds more severely affected	Any although signs more severe in ducks	Variable	Nervous: difficulty walking, convulsions, feather picking Reproductive: Reduced fertility and hatch rates. Decreased egg numbers.	Remove contaminated food
26. Botulism	Toxin from fungus that grows in rotting material	Any	Any, although more severe in ducks and geese	It depends on how many birds consume the toxin	Nervous: paralysis, especially of the neck. Birds will be flaccid.	Remove source of toxin, pick up carcasses, control flies, fix leaking water
27. Chemical toxins	Pesticides, disinfectants and other	Any	Any	Depends on the toxin, the amount consumed or inhaled, and the number of birds exposed.	Signs vary depending on the toxin.	Remove source of toxin, may need to clean environment
28. Predators	Wild and domestic predators	Any	Any	Predators usually kill a few birds but do not cause the deaths of large flocks	Missing birds or eggs. Occasionally, injured birds may appear or body parts may be discovered after an attack.	Secure housing can reduce losses to flocks.
29. Vitamin deficiency	Lack of complete nutrition	Any	Any	Usually low in free-ranging birds. May be moderate to high in young, confined birds.	***Vitamin E:*** Death before 4 days of age Nervous: difficulty walking and standing, 15 - 30 days of age Reproductive: Decreased hatchability. ***Vitamin A:*** Slow growth, drowsiness, and mortality. Respiratory: Discharge from nose and eyes. Reproductive: decreased egg numbers and hatching, increased blood spots in eggs.	Supplement vitamins in the water or feed. Add antioxidants to feed. Rotate feed.

POULTRY DISEASES

Coverage for each disease generally includes these subtopics:

- Name and (common names)
- Clinical signs and lesions
- Differential diagnoses
- Cause, transmission, epidemiology
- Diagnoses
- Prevention
- Treatment
- Recovery

Listed Alphabetically:

Aspergillosis

Other names: brooder pneumonia, mycotic pneumonia, fungal pneumonia, *Aspergillus,* pneumomycosis, bronchomycosis, and colloquialisms ('asper' and 'airsac'). When the source of the disease is the hatchery, the disease is called brooder pneumonia. In older birds, the disease is called aspergillosis.

Clinical signs and lesions: Aspergillosis occurs as an acute disease of young birds and a chronic disease in mature birds. Young birds have trouble breathing and gasp for air. Characteristically, there are no rales or respiratory sounds associated with aspergillosis. Feed consumption decreases. Occasionally there is paralysis or convulsions caused by the fungal toxin. Mortality in young birds averages 5-20 percent, but may be as high as 50 percent. Mature birds also show respiratory distress, reduced feed consumption, and may have a bluish and dark color of the skin (cyanosis). Nervous disorders, such as twisted necks, may occur in a few birds. Mortality in mature birds is usually less than 5 percent.

Lesions typically consist of friable grey, yellow to greenish nodules or plaques, and fibrin deposition and pus in air sacs, lungs, and trachea. Similar lesions can occur in other organs such as the liver, peritoneal cavity and other sites. Lesions in the brain consist of solitary abscesses. Mycelial growth with sporulation may be apparent on air sacs, bronchi and in the fungal lesions.

Differential diagnosis: Avian aspergillosis signs are nonspecific and depend on the system involved. The disease may produce similar signs to Dactylariosis. Pulmonary aspergillosis is usually differentiated from other avian respiratory diseases by the granulomatous lesions at necropsy, but needs to be differentiated from other mycoses and mycobacteriosis. However, exudative fibrinous or purulent air-sacculitis and pneumonia need to be differentiated from mycoplasmosis, colibacillosis, fowl cholera, and chlamydiosis. Aspergillosis affecting the skin needs to be differentiated from ectoparasitic infestations, smothering, cannibalism, and molting (natural loss of feathers) in birds.

Cause, transmission, and epidemiology: Aspergillosis fungi are ubiquitous in the environment and grow well at room temperature and higher. All birds (domestic poultry, pigeons, canaries, and zoo bird species), animals, humans, and plants are susceptible. All litter and nest materials (peat moss, peanut hulls, sawdust, peat, bark, and straw) are known to have become contaminated with *Aspergillus*. Feed and water should be suspect when attempting to identify the source of contamination. The fungus can penetrate egg shells and infect embryos. Infection is usually caused by inhalation of large amounts of fungal spores, contaminated eggs during incubation and dust from the poultry shelters, coops, and areas where birds aggregate.

In smallholder farms in Africa, aspergillosis occurs in free-range poultry during the planting and the harvesting periods. The main sources of the fungi are contaminated poultry environments and moldy cereals that are given to the birds as supplementation. In planting season, farmers use most of their cereals to plant farms; the remnants are used for household use, and rejects are thrown to animals including poultry. It is a wet period when fungus can multiply well and produce abundant spores. In harvesting periods, there are plenty of cereals but poor storage under humid, warm conditions favors fungal growth that can lead to poultry infection when bad grains are used as animal feeds.

Diagnosis: Clinical signs and lesions of aspergillosis can indicate the disease; confirmation is by microscopic demonstration of the fungus in the lesions or histologic sections.

Fungal species that can cause aspergillosis are *Aspergillus fumigatus,* and to a lesser extent, *A. flavus* and *A. niger.* Culturing the fungus from the lesions allows its identification. On Sabouraud's dextrose agar (SDA) at 25˚C, *Aspergillus* grows rapidly (within 2-5 days). Early growth of ***A. fumigatus*** is white and velvety. Later, when the conidia (spores) develop, the growth becomes blue-green to gray. Old cultures become dark brownish-gray and the texture ranges from velvety to granular or powdery. Colonies may be flat or folded. Isolates grow better at 37-45˚C. Some strains grow at 65˚C. Most isolates are sensitive to cycloheximide. Colonies of ***A. niger*** are white initially; later, when spores are produced, they become charcoal black, and are flat with a granular texture. The reverse side is colorless to white. Colonies of ***A. flavus*** are rough and woolly, and their surface is bright yellow to deep yellowish-green. The reverse side ranges from colorless to deep reddish-brown. Many isolates grow better at 37˚C than at 25˚C.

Microscopically, *Aspergillus* species produce thin, septate hyphae. Reproductive hyphae are called conidiophores. They arise from the vegetative mycelium, are thin-walled and may branch. They terminate in an inverted flask-shaped vesicle, from which sterigmata or phialides arise on the top half of the vesicle. There may be one (uniseriate) or two (biseriate) rows of sterigmata. Each sterigma bears long chains of conidia. This makes the conidiophore head look radiate.

Treatment: Clean and disinfect the house and spray it with 1:2000 copper sulphate or other suitable fungicide. Few and expensive birds can be treated with Nystatin or Amphotericin-B or other anti-mycotic agents. These are given together with antibiotics to prevent secondary bacterial infections.

The spread can be controlled by improving ventilation, eliminating the source of the infection, and adding a fungistat (mycostatin, mold curb, sodium or calcium propionate, or

gentian violet) to the feed and/or copper sulfate or acidified copper in the drinking water for 3 days. The litter can be sprayed lightly with an oil-base germicide to control dust and air movement of fungal spores.

Prevention: It is important to thoroughly clean and disinfect the brooding area between broods. Use only clean litter, preferably soft wood shavings. Do not use sawdust, litter high in bark content, or shavings that have been wet. Move feeders and waterers periodically.

Recovery: Cases will re-occur if fungi can grow in feedstuffs or litter on the farm because it is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Avian Encephalomyelitis

Other names: epidemic tremor, AE.

Clinical signs and lesions: Signs commonly appear during the first week of life and between the second and third weeks. Affected chicks may first show a dull expression of the eyes, followed by progressive incoordination, sitting on hocks, tremors of the head and neck, and finally paralysis or prostration. Affected chicks are inactive. Some may refuse to walk or will walk on their hocks. In advanced cases, many chicks will lie with both feet out to one side (prostrate) and die. All stages (dullness, tremors, prostration) can usually be seen in an affected flock. Feed and water consumption decreases and the birds lose weight. In adult birds, a transitory drop (5-20 percent) in egg production may be the only clinical sign present. However, in breeding flocks, a corresponding decrease in hatchability is also noted, as the virus is egg-transmitted until hens develop immunity. Chickens which survive the clinical disease may develop cataracts later in life.

Differential diagnosis: Newcastle disease (neurologic form), equine encephalomyelitis, Marek's disease, and nutritional deficiencies may all resemble avian encephalomyelitis and should be excluded.

Cause, transmission, and epidemiology: The disease is most prevalent in chickens less than 6 weeks of age. Pheasants, corturnix quail, and turkeys are natural hosts as well, but less susceptible than chickens. Ducklings, young pigeons, and guinea fowl can be experimentally infected.

The virus can be transmitted through the egg from infected hen to chick, accounting for disease during the first week of life. The disease can also be spread through a flock by direct contact of susceptible hatchlings with infected birds, accounting for the disease at 2-3 weeks of age. Indirect spread can occur through fecal contamination of feed and water. Recovered birds are immune and do not spread the virus.

Diagnosis: Avian encephalomyelitis is definitively diagnosed by histopathology of the brain of infected and affected chicks. It can also be diagnosed by a correlation of typical clinical presentation and serology. Alternatively, yolk sac inoculation of embryonating chicken eggs with brain, pancreas and/or duodenum from infected birds will result in

typical muscular dystrophy. The virus can be identified with PCR or electron microscopy in allantoic fluid.

Treatment: There is no treatment for outbreaks. Infected birds should be removed, killed and incinerated. Recovered chicks are unthrifty.

Prevention: A vaccine is available.

Recovery: Once a flock has been infected with avian encephalomyelitis on a premises, future flocks are likely to become infected both through the presence of the virus in the environment and also because chicks can get the virus from their dams. Vaccination is the best method to prevent the recurrence of clinical disease in new birds.

Avian Influenza

Other names: influenza, bird flu, fowl plague, AI, HPAI, LPAI.

Clinical signs and lesions: Avian influenza is categorized as low (mild) or highly pathogenic. The low pathogenic form (LPAI) produces listlessness, loss of appetite, respiratory distress, diarrhea, transient drops in egg production, and low mortality. HPAI is seen mostly in chickens and turkeys, although it occurs in other birds, and is characterized by a sudden onset, massive morbidity and mortality over a short period of time. It causes facial swellings, blue combs and wattles, diarrhea, respiratory distress and sometimes nervous disorders. Dark red/white spots develop in the legs and combs of chickens. There can be blood-tinged discharge from the nostrils.

Mortality can range from low in the mild form to nearly 100 percent in the highly pathogenic form. Sudden exertion adds to the total mortality. Egg production and hatchability decreases. There can be an increase in production of soft-shelled and shell-less eggs.

Typical lesions of HPAI include swollen face, blue combs and wattles, red discoloration of the shanks and dead tissue on the lining of the proventriculus and gizzard.

Differential diagnosis: Avian influenza must be distinguished from Newcastle disease as clinical signs and lesions are very similar. It should also be differentiated from *Mycoplasma* infections (chronic respiratory disease) and fowl cholera.

Cause, transmission, and epidemiology: AI is caused by Type A Influenza viruses. LPAI is caused by low pathogenic strains and HPAI by the strains that are more deadly.

AI is transmitted by direct contract. Water bodies which have been contaminated with the virus from droppings of waterfowl and shorebirds, the natural carriers of the virus, are important sources of infection for domestic poultry. Direct contact of wild birds with free-range birds is another important factor in the spread of AI. It is also spread on the farm and between farms by contaminated farm equipment, feed bags, egg crates, vectors such as rodents and insects, and contaminated shoes and clothing. The avian influenza virus can remain viable for long periods of time at moderate temperatures, and can live indefinitely in frozen material. As a result, the disease can be spread through improper disposal of infected carcasses and manure.

Live or 'Wet' poultry markets where many species of birds are brought for sale in cities have the potential to be foci for the concentration and then spread the AI virus.

Diagnosis: In birds that have died from LPAI, mild to moderate inflammation of the respiratory tract and air sacs are seen. The ovaries are inactive or inflamed.

Confirmation of tentative diagnosis based on symptoms and lesions can be made through isolation of the virus in chicken embryonated eggs, testing the virus for hemagglutination, and serological tests.

Treatment: There is no effective treatment for avian influenza. With the mild form of the disease, good husbandry, proper nutrition, and broad spectrum antibiotics may reduce losses from secondary infections. Recovered flocks continue to shed the virus. With the more lethal forms, strict quarantine and rapid destruction of all infected flocks remains the only effective method of stopping an avian influenza outbreak. If you suspect you may have Avian Influenza in your flock, even the mild form, you must report it to the veterinarian's office. A proper diagnosis of avian influenza is essential. Aggressive action is recommended even for milder infections as this virus has the ability to readily mutate to a more pathogenic form.

Prevention: Strict biosecurity procedures should be practiced. Direct or indirect contact of susceptible birds with waterfowl, shorebirds and birds or poultry products from endemic AI areas must be avoided. A vaccination program used in conjunction with strict quarantine has been used to control mild forms of the disease, although this approach is still controversial. Vaccines may only be used with special permit.

Recovery: Recovery is accomplished through depopulation, disinfection of equipment and poultry houses, and resting of pens.

Botulism

Other names: Limberneck, Western duck sickness, bulbar paralysis, alkali disease.

Clinical signs and lesions: 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. Paresis soon progresses to paralysis, and the recumbent bird closes its eyes and appears to be in 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.

There are no gross lesions. A few birds might show mild enteritis. The crop may show putrid ingesta or maggots, but is usually empty.

Differential diagnosis: Botulism should be differentiated from psuedobotulism (transient paralysis), which is a transient manifestation of Marek's disease.

Cause, transmission, and epidemiology: Botulism is a type of poisoning caused by ingestion of toxins of the bacterium *Clostridium botulinum.* 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 growers or mature birds. In ducks and other waterfowl occurrence is related to shallow water conditions in lakes with alkaline water and much decaying vegetation.

Botulism is caused by ingestion of the preformed toxins of *C. botulinum* in feeds, foods, dead poultry or toxin-containing maggots. The toxin is extremely potent. The minimum lethal dose (MLD) for guinea pigs is 0.00012mg/kg subcutaneously (MLD for cobra venom is 0.002mg/kg). The toxin is relatively heat stable. Of the 8 types of *C. botulinum*, Type C is the most common in poultry outbreaks.

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 feed, and decaying poultry carcasses can contain enough exotoxin to be highly lethal, even taken in small amounts.

It is speculated that wild waterfowl contract botulism as follows: the toxin may be consumed in decaying vegetation or dead fish 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 the anaerobic conditions contain toxin from growth of *C. botulinum* within them and may be consumed by some waterfowl.

Ducks that die from various causes may be invaded after death by *C. botulinum* type C normally present in their intestines. Toxins are formed in the carcasses. Ducks that feed on bits of the carcasses or on maggots from the carcasses may be poisoned. There is no spread from bird to bird.

Diagnosis: 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.

Treatment: Remove spoiled feed or decaying matter. Flush the flock with Epsom salts (1 lb/1000 hens) in water or in wet mash. It has been reported that potassium permanganate (1:3000) in the drinking water is helpful. Affected birds can be treated with botulism antitoxin injections. Treatment of flocks with sodium selenite and vitamin A, D, and E or antibiotics has been reported to reduce mortality.

Prevention: The disease can be avoided by preventing access of poultry to any source of toxin. Sick and dead birds should be picked up and incinerated regularly and frequently because they are a common source of toxin. Control flies, and replace suspected feed.

Recovery: Cases will re-occur if the conditions under which botulinum toxin is formed remain on the farm. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk of botulism although the *C. botulinum* organism will remain.

Candidiasis

Other names: Thrush, crop mycosis, sour crop, muguet, soor, levurosis, oidomycosis, stomatitis oidica, moniliasis.

Clinical signs and lesions: This is a mycotic infection affecting a wide variety of birds, man and other animals. It affects the upper digestive tract usually as a secondary infection in birds, but also of the reproductive tract in other animals. It is generally not a major clinical problem. The clinical signs are non-specific and include listlessness, poor appetite, stunted growth, unthriftiness, and ruffled feathers. The crop may be filled with sour, fermentable odor.

Lesions occur in affected part of the body but more so in the crop, esophagus, mouth, pharynx, and sometimes in the proventriculus and intestines as thickening of mucosa, raised circular formations, or curdy pseudomembranous dead tissue that is peeled easily from the mucosa. The affected mucosa is often diffusely or focally thickened, raised, corrugated and white. Necrotic epithelium may slough into the lumen as masses of soft cheesy material. The infected proventriculus is swollen, and has hemorrhagic mucosa which may be covered by catarrhal or necrotic material. Systemic and skin candidiasis has been reported. Lesions of other predisposing conditions may be present.

Differential diagnosis: Vitamin A deficiency and other upper gastrointestinal tract infections that may produce diphtheroid types of inflammations.

Cause, transmission, and epidemiology: Candidiasis is caused by yeast-like fungus, *Candida albicans* (*C. albicans). C. albicans* is a ubiquitous organism and a commensal in the gastrointestinal tract, skin, respiratory and reproductive tracts of birds, humans and animals. Its overgrowth is controlled by normal bacterial microflora in the mucosal linings. Young birds are more susceptible than adults although all ages are affected. It is often associated with other disease problems, especially bacteria that produce endotoxins, physiological disorders, long-term oral antibiotic administration in drinking water, immunosuppression, lack of good sanitation, heavy parasitism, vitamin deficiency, high carbohydrate diets, and debilitating conditions that may alter the bacterial flora on the affected surface or in the whole bird.

Diagnosis: Lesions and presence of septate fungal mycelium may be adequate. The fungus can be cultured on Sabouraud's dextrose agar. At room temperature or 37˚C, growth occurs in 1-3 days. Colonies are smooth, white to creamy, and moist and pasty, resembling those of bacteria. On further incubation, colonies become furrowed and roughened. When observed with a dissecting microscope, filaments that are submerged into the agar may be seen. Microscopically, large, oval, budding cells, occurring singly or in clusters, are seen. Commonly buds do not detach, but elongate to form pseudohyphae, from which lateral or terminal chlamydospores are produced.

Treatment: Control primary disease causes or conditions. Use cooper sulphate or Nystatin for treatment.

Prevention: Practice good sanitation, hygiene, prevent overcrowding; and use phenolic and iodine preparations to sanitize equipments. Avoid unnecessary use of antibiotics, drugs and anticoccidiostats.

Recovery: Cases will re-occur if the precipitating causes are still present. If, however, the cause of the disease is corrected, there is no residual risk to future flocks on the farm.

Cannibalism

Clinical signs and lesions: A vice of chickens, turkeys and other birds reared in captivity, characterized by loss of tissues from pecking and plucking of feathers (wings, tail, and other parts of the body), pecking of vent, toes and head region.

Feather Picking is a behavior expressed by dominant birds at subordinates. It varies from pecking to plucking the feathers of subordinate birds. Light chicken breeds (brown hybrids) are more prone than heavier breeds (white leghorns). **Vent picking** occurs in laying birds. It is common when birds raised in floor systems lay their eggs on the floor in crowded areas. This occurs after oviposition, when exposed mucus membranes stimulate pecking by other birds. **Toe picking** occurs commonly in young birds, while **head picking** is more common in older birds, especially in cages.

The severely damaged and lost feathers, hemorrhages on the head, wings, tail, vent and other body areas are characteristic signs. Feather picking, if severe, will cause fatal hemorrhage in any age of bird. Vent pecking may result in birds eating off portions of the intestines, resulting in death of the affected bird. Birds dying of cannibalism may show anemia, blood stained feathers and affected body areas, and may be missing visceral organs. Affected birds have poor thermoregulation, greater energy demands than others, and affected hens drop their egg production.
Differential diagnosis: Traumatic body injuries, and diseases causing hemorrhagic diarrhea and diathesis.

Cause, transmission, and epidemiology: The behavior is learned between birds. The cause is not clearly known. However, it could be related or stimulated or predisposed by overcrowding, bright light, temperature, pelleted feed or compressed feed, high-density rearing systems, mineral and nutritional deficiency, irritation by external parasites, injuries, foraging behavior or dust bathing, fearfulness, accelerated sexual maturity and increased egg production.

Diagnosis: Not applicable.

Treatment: See Prevention.

Prevention: Through proper husbandry:

- Providing adequate diet and supplying mash diets rather than pelleted feed
- Rearing birds on floor litter rather than on slats
- Reducing light intensity
- Providing perches as a refuge for pecked birds
- Avoiding overcrowding
- Environmental enrichment with pecking devices
- Beak trimming before 5 weeks of age (drawback – chronic pains and neuromas if not done correctly)

Recovery: Cull birds with severe injuries. Remedy contributing factors as listed in prevention.

Chlamydiosis

Other names: ornithosis, psittacosis, parrot fever.

Clinical signs and lesions: Clinical signs in most birds include nasal-ocular discharge, conjunctivitis, sinusitis, diarrhea, weakness, loss of body weight, and a reduction in feed consumption. In turkeys there is also respiratory distress and loose yellow to greenish-yellow colored droppings. Chylamydiosis runs rather slowly through turkey flocks, with a maximum incidence of around 50 percent.

Differential diagnosis: Chlamydiosis can clinically present similarly to fowl cholera, especially in turkeys. Viral infections (Newcastle disease, avian influenza, infectious bronchitis, swollen head syndrome) or Mycoplasmosis with colibacilosis may present similarly.

Cause, transmission, and epidemiology: Chlamydiosis is caused by the bacterium *Chlamydia psittaci.* The disease was formerly called psittacosis or parrot fever when diagnosed in psittacine (curve-beaked) birds, and ornithosis when diagnosed in all other birds or in humans. Currently, the term chlamydiosis is used to describe infections in any animal. Affected species include turkeys, pigeons, ducks, psittacine (curve-beaked) birds, captive and aviary birds, many other bird species, and other animals. Chickens are not commonly affected. Humans are susceptible, especially older and immunosuppressed individuals who are at a higher risk. Chlamydiosis in humans is an occupational disease of turkey growers, haulers, and processing workers in the live-bird areas and of workers in pet-bird aviaries although the incidence is rare.
The primary means of transmission is through inhalation of fecal dust and respiratory tract secretions. It can also be transmitted on contaminated clothing and equipment. Recovered birds remain carriers and will continue to intermittently shed the infective agent for long periods after clinical signs have subsided. Environmental stress may provoke a reccurrence of the disease.

Diagnosis: Chlamydiosis is definitively diagnosed by isolation and identification of *Chlamydia psittaci* in cell cultures or embryonating chicken eggs. However, this can be time consuming and the typical appearance of this organism with Giemsa or Jimenez staining make the staining of tissue imprints a rapid and accurate method of making a diagnosis.

Treatment: Chlortetracycline can be given in the feed (200-400 g/ton) for 3 weeks. Other antibiotics are usually ineffective. Recovered birds are safe for processing. Permanent lesions on the heart and liver are not infectious. Withdrawal periods for medications used must be strictly observed to avoid residual chemicals in the tissues.

Prevention: There is no vaccine. Have a good biosecurity program, excluding wild birds as much as possible.

Recovery: Because recovered birds may remain carriers, new birds should only be introduced to the premises after all of the birds from previous flock have been removed and the environment fully cleaned and sanitized.

Chronic Respiratory Disease (*Mycoplama gallisepticum)*

Other names: CRD, *Mycoplasma gallisepticum,* MG, infectious sinusitis, mycoplasmosis.

Clinical signs and lesions: Chickens, turkeys, pigeons, ducks, peafowl and passerine birds are affected by this bacterial disease. Clinical symptoms vary slightly between species. Infected adult chickens may show no outward signs if infection is uncomplicated. However, sticky, serous exudate from nostrils, foamy exudate in eyes, and swollen sinuses can occur, especially in broilers. The air sacs may become infected. Infected birds can develop respiratory rales and sneeze. Affected birds are often stunted and unthrifty.

There are two forms of this disease in turkeys. In the "upper form" the birds have watery eyes and nostrils, the infraorbitals (just below the eye) become swollen, and the exudate becomes caseous and firm. The birds have respiratory rales and show unthriftiness. In the "lower form" infected turkeys develop airsacculitis. As with chickens, birds can show no outward signs if the infection is uncomplicated. Thus, the condition may go unnoticed until the birds are slaughtered and the typical lesions are seen. Birds with airsacculitis are condemned.

CRD in chicken embryos can cause dwarfing, airsacculitis, and death.

Differential diagnosis: Clinically, the disease may be indistinguishable from infectious synovitis, caused by *Mycoplasma synoviae.* The swollen sinuses caused by MG may also look like fowl cholera or swollen head syndrome.

Cause, transmission, and epidemiology: *Mycoplasma gallisepticum* can be spread to offspring through the egg. Most commercial breeding flocks, however, are MG-free. Introduction of infected replacement birds can introduce the disease to MG-negative flocks. MG can also be spread by using MG-contaminated equipment.

Diagnosis: Clinical disease and typical lesions can lead to a presumptive diagnosis of MG but a definitive diagnosis is only possible through the isolation of the causative organism and its identification with specific antisera. Alternatively, the testing of paired sera (pre and post exposure) can also support a diagnosis.

Treatment: Outbreaks of MG can be controlled with the use of antibiotics. Erythromycin, tylosin, spectinomycin, and lincomycin all exhibit anti-mycoplasma activity, and have given good results. Administration of most of these antibiotics can be by feed, water or injection. These are effective in reducing clinical disease. However, birds remain carriers for life.

Prevention: Eradication is the best control of mycoplasma disease. The National Poultry Improvement Plan in the United States monitors all participating chicken and turkey breeder flocks for mycoplasmosis.

Recovery: Generally, once a farm has had a flock with mycoplasma, subsequent flocks will be infected. Premises can get rid of MG by hatching MG-free eggs (heating method or from breeders free of MG) and placing them in a clean environment and with very strict biosecurity in place. If biosecurity cannot be achieved then recovery is probably not possible and vaccination may be a better option.

Cryptococcosis

Other names: Torulosis, torula, yeast meningitis, European blastomycosis.

Clinical signs and lesions: Crytococcosis is a disease of humans and animals. It has been reported mostly in zoo and pet birds (pheasants, fowl, house martins, jackdaws, chaffinches, canaries, pigeons and psittacines), but not in other birds. It is of public health importance and occurs in poultry environments. This is a systemic fungal disease that may affect the lungs, central nervous system, and the skin.

It has been reported in pheasants with enterohepatitis. Chickens experimentally infected have granulomas and necrotic processes in liver, intestines, lungs, and spleen. Prognosis is grave in cases of cryptococcal meningoencephalitis.

Differential diagnosis: Other lung infections and systemic fungal infections such as aspergillosis, histoplasmosis and dactylariosis.

Cause, transmission, and epidemiology: The causative agent is *Cryptococcus neoformans*, an imperfect yeast that reproduces by budding to give spherical cells surrounded by a thick mucilaginous capsule. The cell diameter is 4-6 µm, and the capsule is 1-2 µm thick. It is commonly isolated from pigeon nest and dropping sites.

Diagnosis: Culturing of the organism, histopathology, and demonstration of its thick capsule and deep red budding spore using mucicarmine stain. The fungus grows well within 48 hr at 30 ^{o} C on glucose agar.

Treatment: Treatment is of little value.

Prevention: Because this disease occurs sporadically and specific risk factors have not been identified, there are no preventative measures that can be recommended. However, the isolation of infected individuals from a group may prevent spread to more individuals.

Recovery: There is no residual risk to birds placed in an environment that has previously had a case of cryptococcosis.

Dactylariosis

Clinical signs and lesions: This is a neurotrophic mycotic disease of turkey poults, quail chicks and young chickens, with many of the clinical and pathologic features of aspergillosis. Signs of dactylariosis are incoordination, tremors, torticollis, circling, recumbency due to mycotic lesions in the brain, and death.

Lesions sometimes occur in the air sacs, lungs, liver and eyes (globe) as granulomas. In the brain lesions involve the cerebellum and cerebrum as large, hardened, grayish, and circular or as focal areas of red colors.

Differential diagnosis: Aspergillosis.

Cause, transmission, and epidemiology: The etiological agent is *Dactylaria gallopava;* it grows naturally in old sawdust, which often is used as chicken litter.

Occurrence of this disease is associated with contaminated litter (wood chip and sawdust) and egg incubators. The organism grows well in acidic environments with moderately high temperatures.

Diagnosis: Culture the fungus on Sabouraud's dextrose agar at 25 ^{o} C and 37 ^{o} C or at 45 ^{o} C. Growth is inhibited by cycloheximide. Colonies are velvety, gray-brown with a flat or wrinkled surface, and the reverse side of the colony is a deep purple-red. Light tan to brown septate mycelium and oval, two-celled brownish conodia (3.2 x 9.0 µm) on unbranched conidiophores are seen under the microscope.

Treatment: Remove contaminated litter and decontaminate incubators by fumigation.

Prevention: Good sanitation.

Recovery: Cases will re-occur if fungi can grow in incubators or litter on the farm because it is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk to new birds.

Duck Enteritis

Other names: Duck plague.

Clinical signs and lesions: This 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.

Ducklings show diarrhea, dehydration, a blood-stained vent, and cyanotic bill. Death usually occurs in 1-5 days.

In adult ducks there is sudden, high, persistent rate of death and a marked drop in egg production. Sick birds show inappetence, weakness, ataxia, photophobia, adhered eyelids, nasal discharge, extreme thirst, prolapsed penis, and watery diarrhea. They are unable to stand, have drooping wings, and hang their heads down. Tremors may be apparent. Morbidity and mortality are usually high, but vary from 5 to 100%. Most birds that develop clinical signs die.

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

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 off.

Hemorrhage and/ or necrosis in the annular bands discs of lymphoid tissue along the intestines is present. The spleen is usually of normal or reduced size. 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. Microscopically there may be intranuclear inclusion bodies in degenerating hepatocytes, epithelial cells of the digestive tract, and in reticuloendothelial cells.

Differential diagnosis: Duck enteritis must be differentiated from duck viral hepatitis, pasteurellosis (fowl cholera), Newcastle disease, avian influenza, coccidiosis, and other causes of enteritis.

Cause, transmission, and epidemiology: The etiologic agents are variable strains of herpesviruses that are immunologically similar and non-hemagglutinating. The virus grows well on chorioallantoic membrane of 9-14 day-old embryonating duck eggs or on duck embryo fibroblasts. It can also be isolated in ducklings. The virus produces intranuclear inclusion bodies in a variety of cells of infected waterfowl.

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. A carrier state for as long 1 year has been demonstrated in wild ducks. Perhaps carrier birds under stress shed virus intermittently, thus exposing susceptible birds. It is suspected that viremic birds may transmit infection through feeding arthropods. Vertical transmission has been reported experimentally.

Wild and domestic ducks are affected. All age groups and many varieties are susceptible; however, mostly adult ducks are affected. It has been suspected in Kenya and other African countries.

Diagnosis: Typical clinical signs and lesions (especially demonstration of intranuclear inclusion and the virus in tissues using fluorescent antibody technique) are diagnostic. Isolate and identify the virus for confirmation using virus neutralization test.
Acute and convalescent sera can be used to demonstrate an increasing antibody titer to duck virus enteritis.

Treatment: There is no effective treatment.

Prevention: Prevent cohabitation or contact of domestic ducks with wild waterfowl. All appropriate quarantine and sanitary practices should be followed to prevent the introduction of this disease. Autogenous vaccines have been used effectively to prevent outbreaks.

Recovery: Most premises on which there have been outbreaks of DVE are likely to have continuing outbreaks because 1) there is a risk of re-introduction from wild birds if management is not changed, and 2) previously infected birds may be carriers. Recovery on most farms is only possible with vaccination.

Duck Viral Hepatitis

Clinical signs and lesions: This is a peracute, rapidly spreading viral infection of young ducklings characterized by short course, high mortality and by punctuate or ecchymotic hemorrhages in the liver. The disease occurs in ducklings less than 5 weeks of age. It is probably present in all major duck-raising areas of the world. In Africa it has been suspected in Kenya.

Three different viruses are known to cause disease:

DVH type 1: Has a short incubation of around 24 hr in experimental birds, and morbidity close to 100%. Onset and spread within a flock are very rapid and most mortality occurs within 1 week of onset. 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 opisthotonous position. Death often occurs within 1 hr of the appearance of signs.

Mortality is age related and occurs as follows: ducklings less than 1 week old – up to 90%; ducklings 1-3 weeks old – up to 50%; ducklings over 4 weeks and older ducks – negligible mortality. 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 post-infection. Signs include convulsions and opisthotonus position. Mortality ranges from 10-50% 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.

The lesions observed with all three viruses are similar. The carcass may be in opisthotonous position. The liver is swollen and contains punctuate or diffuse hemorrhages. 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.

Differential diagnosis: The disease must be differentiated from duck viral enteritis, Newcastle disease, avian influenza through susceptibility to chloroform and haemagglutination of erythrocytes.

Cause, transmission, and epidemiology: The etiologic agent of duck viral hepatitis (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. 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.

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 sera harvested. Antibodies for prophylactic use may also be obtained from the yolk of eggs produced by immune breeders, or from the eggs of chicken hyperimmunized with the virus.

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 post-infection. DVH type 3 is similar to but less severe than DVH type 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.

Diagnosis: The sudden onset, rapid spread, short course, and focal, hemorrhagic hepatitis in young ducklings suggest a diagnosis of DVH. DVH type 1 can be isolated in embryonating chick or duck embryos or 1-day old susceptible ducklings and identified by serum neutralization test. 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.

Treatment: Treatment is of no value.

Prevention: DVH type 1: In the initial stages of the outbreak, all susceptible ducklings should be inoculated intramuscularly with duck hepatitis viral antiserum. One inoculation should be adequate if the antiserum is potent. Unexposed ducklings can be actively immunized using a chicken embryo-adapted apathogenic vaccine. However, young ducklings with parental immunity may not respond to vaccination. DVH type 2 and 3: Strict biosecurity procedures should be employed.

Recovery: Eradication of DVH from a premises has been demonstrated with extensive cleaning and sanitation, implementation of strict biosecurity to prevent new introductions and the vaccination of breeders. However, vaccination may be more practical to limit the impacts of the disease.

Ectoparasites

These are external parasites of poultry. Ectoparasites found on poultry are in the phylum *Arthropoda*, which is characterized by segmented bodies, jointed appendages and chitinous exoskeleton. The phylum is divided into two classes: the ***Insecta*** that includes the orders *Phthiraptera* (lice), *Siphonaptera* (fleas) and *Diptera* (flies and mosquitoes); and the *Arachnida* with the order *Acarina* (ticks and mites).

Ectoparasites are very common in free-range systems, and usually controlled in commercial systems. These parasites may constitute a clinical problem; they can transmit a number of infectious diseases and can also act as transport/ intermediate hosts of a

range of helminth parasites. Although they are believed to occur in many family poultry, only a few African countries have published information on their prevalence or occurrence.

INSECT ECTOPARASITES: The insects that most often affect poultry are lice, fleas, mosquitoes, and other flies.

POULTRY LICE

All species of lice that affect chickens are in the suborder Mallophaga, and have mouthparts adapted for chewing. They feed on the epithelial debris of the skin of the host, or on feathers of birds. The meso- and metathorax are fused to form one piece behind the prothorax, which is a distinct and separate segment. Lice species affecting chickens are *Menacanthus stramineus, Menopon gallinae, Cuclotogaster heterographus, Lipeurus caponis, Goniodes gigas* and *Goniocoites gallinae.*

Occurrence:

1. *Menacanthus stramineus* (body louse): Nigeria, Zimbabwe, Kenya.
2. *Menopon gallinae* (shaft louse of poultry): Zambia, Zimbabwe, Nigeria, Kenya.
3. Cuclotogaster heterographus (head louse): Kenya, Nigeria.
4. *Lipeurus caponis* (wing louse): Nigeria.
5. *Goniodes gigas*: Nigeria, Zimbabwe.
6. *Goniocoites gallinae* (fluff louse): Zambia, Kenya.

Etiological characteristics:

1. *Menacanthus stramineus* is relatively large, with adults about 3.5 mm in length. It occurs on those parts of the body which are not densely feathered, like the breast, thigh, and around the vent. Its palpi and four-segmented antennae are distinct. The abdominal segments have each two dorsal rows of medium-length bristles. The eggs have filaments on the anterior half of the shell and on the operculum.

2. *Menopon gallinae* occurs largely on the body, thigh, and breast feathers. The adult is about 2 mm in length and pale yellow in color. It has small palpi and a pair of four-segmented antennae folded into grooves in the head. The abdomen has a sparse covering of small to medium-length setae.

3. *Cuclotogaster* heterographus occurs on the head. It has a rounded body with a large, round head. The adult is about 2.5 mm in length. Three long bristles project from each side of the dorsal surface of the head. The abdomen is barrel-shaped in the female and more elongate in the male.

4. *Lipeurus caponis* is an elongated, narrow species, about 2.2 mm in length and 0.3 mm in width. It occurs on the underside of the large wing feathers and moves about very little. The legs are narrow and, characteristically, the hind legs are about twice as long as the first the two pairs. There are characteristic small angular projections on the head in front of the antennae.

5. *Goniodes gigas* are large brown lice, about 3 mm in length, and occur on body feathers. The head is concave posteriorly, producing marked angular corners at the posterior margins, and carries two large bristles projecting from each side of its dorsal surface. Its antennae have five segments.

6. *Goniocoites gallinae* is the smallest lice found on poultry, with adults measuring 1 to 1.5 mm in length. The head is rounded, carrying two large bristles that project from each side of its dorsal surface. The antennae have five segments

Clinical signs and pathology: On the host, lice cause pruritus, scratching, excoriation, secondary feather damage (as birds pluck their feathers) and irritation, which lead to self-wounding and resultant formation of inflamed and scab covered skin. Chicken lice feed on dry scales, feathers, or scabs on the skin. As lice crawl over the bird, their mouthparts and sharp claws scratch the skin. This constant irritation causes the bird to become nervous and behave abnormally, causing a general unthriftness and unkempt appearance. Infestation in birds also leads to a drop in egg production, decreased hen weight, decreased clutch size, and death in young birds. *Menacanthus stramineus* can cause anemia by puncturing soft feather quills and feeding on the blood that oozes out.

Heavy infestations may cause feather damage and irritation but more importantly, are a sign of debility and poor husbandry. They can move directly between hosts or “hitch lifts” on hippoboscid flies.

Life cycle and epidemiology: Lice are permanent ectoparasites, spending their entire life cycle on their host. They tend to remain with single host bird throughout their lives; they are unable to survive for more than 1-2 days off their host. Eggs hatch in 5 to 7 days. Their life cycle from egg to adult is about 3 weeks. As many as 60 eggs are laid by adult female louse and are glued to the host feathers. A pair of lice may produce 120,000 descendants within a few months.

Diagnosis: This is based on clinical signs and identification of lice in plumage, and their eggs (nits) attached to feathers.

Treatment and control: Control of poultry lice requires treating the birds, since lice remain on the bird throughout its life. Birds may be sprayed, dusted or dipped with an appropriate insecticide like permethrin, carbaryl, malathion, or rotenone. Because lice live only a few days off the host, emptying a shed or yard for a week will clean it.

POULTRY FLEAS

Echidnophaga gallinacea (stick tight flea) is the only flea commonly affecting chickens. It may infest a wide variety of birds and mammals; poultry, rodents, rabbits, canids, felids, horses, and occasionally humans may all become infested. Poultry may develop clusters of the fleas around the eyes, comb, wattles, and other bare spots. *Echidnophaga gallinacea* are difficult to remove because their heads are embedded in the host's flesh and they cannot be brushed off.

Occurrence: *Echidnophaga gallinacea* is found in the tropics and subtropics. It has been reported in Nigeria, Kenya, and Tanzania.

Etiological Characteristics: Adults are approximately 1.5 to 4 mm in length, laterally flattened and are black to brown in color. Their head is sharply angled at the front. There are no genal or pronotal ctenidia. On the head behind the antennae are two setae, and in females, a well developed occipital lobe. Thoracic segments are narrowed dorsally. Mouthparts appear large, extending the length of the forecoxae and projecting from the head conspicuously. The maxillary laciniae are broad and coarse.

Clinical signs and pathology: The adult fleas attach to the skin around the face and head, causing severe irritation, nodular formation, and in some cases, blindness. They can cause blood loss, anemia, and even death. The skin over the nodules often becomes ulcerated, and young birds may be killed by heavy infestations.

Life cycle and epidemiology: On fertilization, the female fleas burrow into the skin of the fowl, mainly on the comb, wattles and around the eyes of the birds, resulting in the formation of nodules in which eggs are laid. Hatching occurs within the nodules. The female lays up to 20 eggs at a time and about 400-500 total during her lifetime. Larvae drop to the ground to develop in soil around chicken cages, pupating in two weeks. Adult fleas emerge from pupae and are free-living until breeding occurs. Female fleas then attach to the host and lay eggs to complete the cycle.

Diagnosis: This is based on history, clinical signs, and identification of fleas or flea feces on birds.

Treatment: Managing fleas on birds requires an integrated approach. Both the host bird and the environment must be treated at the same time to be effective. Birds and surroundings should be sprayed with insecticide. Control the fleas by treating the birds, removing them from the infested area for three weeks, and treating the area (removing fecal droppings and litter, and spraying a suitable insecticide) and birds (again) before returning them. Domestic animals having contact with poultry should be treated at the same time. A range of insecticides can be used, namely; organophosphates, carbamates, pyrethrins, and pyrethroids for flea control on the birds.

MOSQUITOES AND OTHER FLIES

The order *Diptera* includes, among others, the families Culicidae (mosquitoes), Simuliidae (black flies), Ceratopogonidae (midges), and Muscidae (house flies and stable flies). Most of these insects are found in poultry houses, where some feed on birds and other animals (including humans) especially at night. Others contaminate the feed and water in the poultry rearing areas.

Clinical signs, pathology and economic importance: The majority of these flies irritate the host by biting and sucking blood. They cause annoyance and physical damage to setting hens and young birds. Their greatest importance lies in their role as intermediate hosts or as mechanical vectors of disease.

Mosquitoes (*Aedes* spp., *Anopheles* spp., and *Culex* spp.), may act as intermediate hosts for *Plasmodium* spp., and mechanically transfer fowl pox virus.
Black flies and biting midges are intermediate hosts of the protozoa *Leucocytozoon* spp. Biting midges are vectors for fowl pox, avian infectious synovitis, and *Haemoproteus* spp.
Flies in the family Muscidae may transfer Newcastle disease virus, *Heterakis gallinarum, Pasteurella multocida,* and *Mycobacterium avium* to non-infected birds.

Diagnosis: Clinical signs and identification of feeding flies, especially at night. Traps can be used to collect and identify flying insects in the vicinity of poultry.

ARACHNID ECTOPARASITES: Arachnids include mites and ticks, some of which affect poultry.

POULTRY TICKS

1. ***Argas persicus*** (fowl tick) commonly affects chickens, turkeys, pigeons, ducks and geese in tropical and sub-tropical countries. In Africa they are found in Egypt, Kenya, Zimbabwe, and Nigeria. *Argas walkerae* and *A. reflexus hermanni* are reported from West Africa. They are found on the skin (especially nymphs and larval stages), but most of the time the ticks hide in cracks in chicken or human houses, market stalls and sheds, or under the tree bark, away from the host.

Etiological Characteristics: The unfed adult tick is pale yellow, turning reddish brown when fed. The female tick is about 8 mm in length, while males are 5 mm. The margin of the body appears to be composed of irregular quadrangular plates or cells, and the hypostome is notched at the tip.

Clinical signs and pathology: *Argas persicus* causes severe blood loss leading to progressive lowered production. The affected birds are ruffled, with poor appetite and diarrhea. This species produces tick paralysis in chickens.

2. ***Ornithodoros species*** (the eyeless tampan) affects poultry and other domestic and wild animals. This tick species occurs in tropical and subtropical habitats. They are found on the skin, but most of the time, the ticks hide in cracks or under the tree bark, away from the host. It is not well documented in Africa.

Etiological Characteristics: The integument has wrinkled patterns that run continuously over the dorsal and ventral surfaces. There are no distinct lateral margins of the body, which appears sac-like. These parasites are known to transmit *Borrelia anserina* and *Aegyptinella pullorum*.

Clinical signs and pathology:. *Ornithodoros* spp. cause anemia, emaciation, weakness and slow growth.

Life cycle and epidemiology of these tick species: Females lay eggs in the cracks and crevices they occupy, usually in batches of 30 to 100 or more; they lay several batches of eggs and produce an average of 700 to 800 eggs during their lifetime. A blood meal is needed to produce each batch of eggs. Eggs hatch in 2 to 4 weeks and 6-legged tick larvae appear. Larvae are active day or night and readily seek a host. After attaching to the host, larvae feed for 5-6 days. After this time, they drop from the host and molt to the nymphal stage. Nymphs have 8 legs, and feed only at night and for short periods. After two more nymphal molts, the ticks reach the adult stage. Under favorable conditions, the time from egg to adult is approximately 30 days. Adult ticks completely engorge on hosts in 30 to 45 minutes. Adults are extremely resistant to starvation, and can live more than a year without a blood meal.

Diagnosis: This is based on history, clinical signs, collection and identification of ticks from the skin. On examination of the skin, larvae are seen attached, especially on non- or less feathered areas of the skin (head, breast, abdomen, cloaca), but not on the leg

shanks. They are nocturnal feeders and examination of birds at night is necessary for diagnosis, especially in cases of low infestations.

Treatment: Treat birds and poultry shed(s) with insecticide, making sure that all cracks are penetrated. Nesting material should be removed and burnt or buried. Treatment must be repeated at seven days to break the life cycle. A follow-up treatment at one month is necessary. Spray captive birds in contact with family poultry to prevent re-infestation.

POULTRY MITES

In poultry, mites are found in different parts of the body, and most species are either microscopic or less than 1 mm in length. The common free-living ectoparasitic mites of poultry belong to the family Dermanyssidae, and include the chicken mite, northern fowl mite, and tropical fowl mite. These mites possess relatively well-sclerotized free dorsal and ventral plates, claws and caruncles on the tarsi, one lateroventral stigma near each third coxa, and small chelicerae on long-sheathed bases. Of lesser importance are members of many other mite families that bore into the skin or infect various internal passages and organs.

Dermanyssus gallinae (D. gallinae), Ornithonyssus sylviarum (O. sylviarum) and *Ornithonyssus bursa* (O. *bursa)* are the species found on the skin. They affect chickens, turkeys, ducks and other domestic and wild birds.

Etiological characteristics: *Dermanyssus gallinae* (chicken mites) are quite small, but they can be seen with the naked eye. They can be identified by the shape of the dorsal plate and by the long whip-like chelicerae that look like stylets. The adult measures about 0.7 x 0.4 mm, varying in color from gray to deep red, depending on its blood content. *Dermanyssus gallinae* is cosmopolitan in distribution and has been reported in Tanzania, Nigeria, Zambia and Kenya.

Ornithonyssus (Liponyssus) sylvarium, the northern fowl mite, is recognized as a serious pest in temperate countries and is also extremely common in almost all types of production facilities. It is easily distinguished from *Dermanyssus gallinae* by possession of easily visible chelicerae, and the shape of the dorsal and anal plates. It has not been reported in Africa.

Ornithonyssus bursa (tropical fowl mite) is distributed throughout the warmer regions of the world and possibly replaces *O. sylviarum* in these regions. The hosts include poultry, pigeons, sparrows, and humans. It closely resembles the Northern fowl mite, but can be distinguished by the shape of the dorsal plate and the pattern of the setae. *Ornithornyssus bursa* occurs in the tropics and subtropics and has been reported in Zambia and Nigeria, while O*. sylviarum* occurs in temperate regions.

Cnemidocoptes gallinae (feather mites or depluming mites) are the common mites observed in chickens ,but also found in pheasants, pigeons, and geese. Females are rounded and about 400 microns long. The legs are short and stubby, and the anus is terminal. The dorsal surface is covered by faint striation. However, mid-dorsally the striations are unbroken. The body has no spines or scales. Stalked pulvilli are present on all legs of larvae and males, but are absent in the nymphal stages and females. Copulatory suckers are absent in male.

Cnemidocoptes mutans (scaly-leg mite) lives under the scales on the feet and legs of birds, causing thickening that give the impression that the scales are protruding outwards. It also attaches to the comb, wattles and neck. *Cnemidocoptes mutans* is characterized by short stubby legs, the terminal anus, and the dorsal surface covered by faint striation. Mid-dorsally, the pattern of dorsal striations is broken in a plate- or scale-like pattern. The body has no scales or spines. *Cnemidocoptes mutans* has been reported in Zimbabwe, Tanzania, Zambia and Kenya.

Cytodites nudus (airsac mites) are found in air passages and lungs of wild birds and poultry. The mite is oval and about 500 microns long, with a smooth cuticle. The chelicerae are absent, and the palps are fused to form a soft, sucking organ, through which fluids are imbibed. This mite has been reported in Kenya.

Life cycle and epidemiology: Mites do not spend their entire life cycle on the host bird, except for *Cnemidocoptes mutans* and *Ornithonyssus* spp. Adult mites spend most of their lives on the host, but will wander from the birds into crevices and cracks. Adult female mites complete egg-laying in 2 days; the number of eggs laid averages 2 to 5 per female.

Dermanyssus gallinae (chicken mites) are gregarious and can be found in large numbers around poultry. The life cycle is fairly complicated, with a series of feeding and non-feeding immature stages. Eggs hatch in about 3 days, and the life cycle can be completed in 7 to 10 days under favorable conditions. Adults are resistant to starvation, and can live off the host for more than a month. This mite does not spend its entire life cycle on birds.

Ornithonyssus sylvarium breeds continuously on the host bird and is a particular problem for caged birds. The mites spend their entire life cycle on the host. After laying eggs, normally on feathers on the cooler regions of the bird, the mites migrate to the neck area. The eggs then hatch within a day, with both larval and nymphal stages completed in four days, and the entire life cycle within a week.

Ornithonyssus bursa can pass its entire life cycle on chickens. Its biology and habits are similar to those of *Ornithonyssus sylvarium,* although a greater proportion of its eggs are laid in the nests.

Clinical signs and pathology: *Dermanyssus gallinae* are bloodsuckers and are irritating to poultry. Anemia occurs in heavily parasitized birds, reducing feed efficiency, egg production, and the ability to withstand and overcome diseases. Birds infected with some mites will have a change of behavior due to itching effect of the mites.

On birds heavily infested with *Ornithonyssus sylvarium,* inspection can reveal heavy deposits of mite eggs and feces in the vent area. Parting of the feathers reveals the mites, eggs and excrement. The mites can also be seen crawling on eggs. The northern fowl mite is sometimes confused with the red mite, although unlike the red mite, it can be found easily on birds in the day as well as night. Heavy infestations result in blackened feathers and scabby and cracked skin, particularly around the vent, and infested male birds can be discouraged from breeding. *Ornithonyssus sylviarum* and *O. bursa* are associated with severe emaciation, droopiness, and reddened scabby skin in chicken.

Cnemidocoptes gallinae are also associated with severe emaciation, droopiness, and reddened scabby skin in chickens. These mites burrow into the epidermis at the base of

feather shafts, and cause intense irritation and feather pulling in chickens, pheasants, pigeons, and geese.

Cnemidocoptes mutans cause inflammation with exudates and subsequent keratinization of the legs. Pathological findings include small yellowish-grey or reddish-brown, wart-like skin proliferations that seem to begin on the soft parts of the planter side of the tarsus and spread along the digits and up the shanks to the hock. There is elevation of the scales and increased desquamation.

Small infestations with *Cytodites nudus* (air sac mites) may cause coughing and accumulation of mucus in trachea and bronchi. The affected air sacs are cloudy and may have some fibrin deposition. The bird's balance may be affected.

Diagnosis: This is made on basis of clinical features and finding mites on skin scrapings, acetate-tape strips and coated brushing.

Treatment: Mites can live for several months without food, so destocking a shed for a short time will not eliminate the problem. Spray birds and sheds with insecticide, making sure that all cracks are penetrated. Nesting material should be removed and burnt or buried. Treatment must be repeated at seven days to break the life cycle. Scaly leg of chickens can be treated by smearing Vaseline jelly with insecticide or dipping the leg in paraffin (kerosene) and then gently brushing the leg. Paraffin must not be allowed to touch the skin or feathers.

CAUTION ON THE USE OF INSECTICIDES

Birds, particularly ducks, are susceptible to poisoning by most insecticides if they are used too frequently or in high concentration. The directions on the container should be followed carefully. Do not use insecticides that are not intended for poultry. Some insecticides suitable for treating sheds and yards are not suitable for treating the birds themselves, and birds should not even be in the shed when they are being applied. Even if insecticides do not seem to affect the birds, they may be absorbed and appear in the eggs or meat, making these products unsuitable for human consumption. This can happen from treatment of the birds themselves or from exposure to a treated shed or yard. If exposure to insecticides is temporary the effect will wear off, so eggs and meat become safe again after a 'withholding period'. The withholding period for a particular insecticide is stated on the label and must be observed.

Egg Drop Syndrome

Other names: egg drop, egg drop syndrome 76, EDS-76.

Clinical signs and lesions: There are no reliable signs other than the effects on egg production and egg quality. Healthy-appearing hens start laying thin-shelled and shell-less eggs. Once established, the condition results in a failure to achieve egg production targets. Transient diarrhea and dullness occur prior to egg shell changes. Fertility and hatchability are not affected.

Differential diagnosis: Similar egg production impacts can be caused by failures in husbandry such as a lack of food or water or infectious diseases such as infectious bronchitis, avian influenza or Newcastle disease.

Cause, transmission, and epidemiology: Egg drop syndrome is caused by an adenovirus. It is believed that the syndrome was first introduced into chickens from contaminated vaccines. Vertical transmission occurs from infected breeders to chicks. Newly hatched chicks excrete the virus in the feces.

Diagnosis: EDS 76 is most definitively diagnosed by the isolation and identification of the causative adenovirus from birds with the typical clinical syndrome. Diagnosis can also be made serologically using a hemagglutination inhibition test.

Treatment: There is no successful treatment of infected birds. Induced molting will restore egg production.

Prevention: Prevention involves a good biosecurity program.

Recovery: The viral cause of EDS 76 is very stable in the environment and thus, it is difficult to eradicate once a premises has housed infected birds. Vaccination of subsequently placed birds will prevent the occurrence of disease.

Endoparasites: Acuaria hamulosa (nematode)

Other names: Gizzard worm.

Clinical signs and lesions: Clinical symptoms are mild and characterized by emaciation, weakness, droopiness and anemia. The worm lives underneath the horny lining of the gizzard where it produces soft nodules in the musculature and this weakens the organ. It is associated with glandular degeneration, epithelial necrosis, and inflammatory cell infiltration.

Cause, transmission, and epidemiology: This worm has been reported in Africa (Nigeria, Ethiopia, Tanzania, Kenya, Uganda, and Zimbabwe), the Americas, Europe, and Asia. It is a nematode in the family Acuariidae. The male worms are 10-14 mm and the females are 16-29 mm long. They have long cuticular cordons, which are irregular and wavy, running 2/3 of the way down the body. The adult worms are found embedded in nodules or abscesses under the keratinized layer of the gizzard.

This species utilizes the grasshoppers, beetles, sandhoppers and weevils as intermediate hosts.

Diagnosis: Presence of embryonated eggs in feces is indicative, but these needs to be differentiated from other spirurid eggs. Confirmatory diagnosis is at post mortem.

Treatment: There is no satisfactory treatment. The feasible control method is rearing the birds on wire.

Prevention: The feasible control method is rearing the birds on wire (off the ground).

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Amoabotaenia sphenoides* (cestode)

This worm occurs in the small intestine (duodenum) of the chicken. It has an indirect life cycle and uses the earthworms as the intermediate hosts. It is cosmopolitan in distribution.

Endoparasites: *Ascaridia galli* (nematode)

Clinical signs and lesions: Light to medium infestations may be tolerated without clinical signs; however, heavy infestations may cause diarrhea, intestinal occlusion, intussusceptions, emaciation, anemia and death. There is reduction in egg production, and birds appear unthrifty. Lesions caused by this worm are cattarhal or hemorrhagic enteritis. They have been implicated in some cases of egg peritonitis.

Differential diagnosis: *Hartertia gallinarum*, which has been reported from southern and western Africa, and Asia.

Cause, transmission, and epidemiology: *Ascaridia galli* is a nematode parasite that causes ascariasis in chickens, guinea fowl, turkeys, geese and other wild birds worldwide. It lives in the small intestine. Adult worms are semi-transparent; males measure 50-76 mm, while female worms are 72- 16 mm long. Their oral opening has 3 large lips and the esophagus has no posterior bulb.

This worm has been reported worldwide. It has a direct life cycle, and earthworms may act as transport hosts.

Diagnosis: Diagnosis is through finding eggs in feeces or worms during post mortem.

Treatment: Piperazines work well.

Prevention: Separate young birds from old birds when kept in enclosures. Moisture levels and ventilation should be monitored.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Capillaria* species (nematode)

Clinical signs and lesions: The birds appear weak and emaciated. *Capillaria contorta* and *C. annulata* cause catarrhal or croupous inflammation and thickening of the crop and esophagus. *C. caudinflata, C. bursata, C. obsignata and C. anatis* are associated with hemorrhagic enteritis and bloody diarrhea.

Cause, transmission, and epidemiology: These are small, hairlike worms found in the digestive tract. They include: *Capillaria annulata, C. contorta, C. caudinflata, C. bursata, C. obsignata, and C. anatis*. *C. annulata* and *C. contorta* are found in the crop and esophagus. *Capillaria caudinflata, C. obsignata, C. bursata* and *C. anatis* are found in the intestine. The *Capillaria* species are cosmopolitan.

Capillaria caudinflata and *C. annulata* utilize earthworms as the intermediate hosts. *Capillaria obsignata, C. anatis* have a direct life cycle. *Capillaria contorta* may have a direct or an indirect life cycle.

Diagnosis: Diagnosis is through detection of eggs in feces and worms during post mortem.

Treatment: Treatment can be achieved by use of Coumaphos and Febendazoles.

Prevention: Control and prevention is by separation of birds from possible transport and intermediate hosts, and effective cleaning of poultry houses and premises.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Choanotaenia infindibulum* (cestode)

This worm occurs in the upper small intestine of chickens and turkeys, and is distributed worldwide. It has an indirect life cycle, with houseflies and beetles acting as the intermediate hosts, and has been associated with weight loss in affected birds.

Endoparasites: *Davainea proglottina* (cestode)

Clinical signs and lesions: Clinical signs associated with this flatworm parasite are retarded growth, weakness, diarrhea, and nervous disorders characterized by partial or incomplete paralysis of the bird. It is the most pathogenic cestode in poultry, and is associated with nodules on the mucosa and haemorrhagic enteritis in heavy infestations.

Cause, transmission, and epidemiology: The worm belongs to the family Davaineidae. It occurs in the duodenal loop, and is found in domestic fowl and other gallinaceous birds

in most parts of the world. It has an indirect life cycle, with snails acting as the intermediate hosts.

Diagnosis: Diagnosis is made by accurate identification of the parasite during necropsy and demonstration of proglottides in feces of birds.

Treatment: Niclosamide is effective for treatment of this infestation.

Prevention: Improvement of sanitary practices and application of approved insecticides to the soil or litter in the premises which interrupts the parasite life cycle by destroying the intermediate host. Separate birds according to species and age groups where possible.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Gongylonema ingluvicola* (nematode)

Clinical signs and lesions: This nematode parasite creates convoluted tracts in the crop wall. It causes a mild chronic inflammatory reaction with flattening, compression and cornification of the epithelium.

Cause, transmission, and epidemiology: This nematode belongs to the family Gongylonematidae. It is a thread-like worm. Adults are normally found embedded in the epithelium of the crop, esophagus, and sometimes the proventriculus. Male worms are 17-20 mm while females are 32-55 mm long. It is found in chickens, turkeys, partridges and quail. It has been reported in many parts of the world and Africa (Central, Eastern and Northern).

The parasite has an indirect life cycle and utilizes beetles and cockroaches as the intermediate hosts.

Diagnosis: Definitive diagnosis can be arrived at during post mortem. The parasite has cuticular thickenings, which are oval to round on the anterior aspect. The tail of the male worm has a number of papillae and spicules. The left spicule is slender and longer than the right one.

Treatment: There is no treatment for this infection.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Heterakis* species (nematode)

Clinical signs and lesions: *Heterakis gallinarum* occurs in the caeca of chickens, guinea fowl, turkeys, ducks, and geese. These are small white worms with 3 lips in the mouth and the eosophageal bulb with a valvular apparatus. Its clinical effects are minimal, but heavy infections do cause thickening of caecal mucosa, petechial haemorrhages, and hepatic granulomas. This parasite is also the carrier of *Histomonas melegridis,* the causal agent for black head.

Heterakis isolonche occurs in the caecum of chickens, quail, and pheasants.
It causes diarrhea, wasting, emaciation and death. Pathological lesions include confluent nodular (warty) thickening of the caecal wall.

Differential diagnosis: *Ascaridia galli* and other nematode worms. *Subulura* species (*S. brumpti, S. minetti, S. differens, S. strongylina,* and *S. suctoria)* are important as a differential diagnosis, but are not associated with any pathology.

Cause, transmission, and epidemiology: This genus belongs to the family Heterakidae. *Heterakis gallinarum* and *Heterakis isolonche* are important parasites of poultry in Africa. Other species in the genus are *Heterakis dispar,* which has been reported in ducks and geese, and *Heterakis brevispiculum,* found in chickens and guinea fowl.

Heterakis isolonche has a direct life cycle, but earthworms may serve as a transport host.

Diagnosis: Diagnosis is made by finding eggs in feces. These eggs must be differentiated with those of *A. galli* and other related worms. Definitive diagnosis is arrived at post mortem by the presence of the worms.

Treatment: Treatment is by use of Phenothiazine.

Prevention: This infection can be minimized by strict sanitation in poultry houses.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. So, after a flock has been infested, new birds cannot be raised on the ground without the risk of infestation. A wire or cement flooring must be introduced to remove or reduce risk to new flocks.

Endoparasites: *Hymenolepis* species (cestode)

This genus contains *H. carioca, H. cantaniana*, *H. lanceleolata* and *H. nyrocae*. These tapeworms are threadlike and inhabit the small intestine.

Hymenolepis species have been reported in the chickens in Africa and the United States. They have an indirect life cycle with dung beetles acting as the intermediate host, and are associated with catarrhal enteritis and diarrhea in birds.

Endoparasites: Protozoans

Clinical signs and lesions: Some protozoans produce coccidiosis associated with decreased growth, diarrhea and high mortality in poultry. Coagulative necrosis and mucoid and hemorrhagic enteritis also occur.

Differential diagnosis: Necrotic enteritis.

Cause, transmission, and epidemiology: The main protozoan parasites in poultry are in the genera *Eimeria, Cryptosporidia, Histomonas, Trichomonas, Tetratrichomonas, Entamoeba,* and *Endolimax* species. There are also tissue protozoan parasites in the genera *Sarcocystis, Toxoplasma,* and *Neospora*. Of all these, coccidian and *Histomonas* parasites are the most important, especially in commercial birds. The most pathogenic *Eimeria* species are *E. tenella, E. necatrix,* and *E. brunnetti. E. cervulina* and *E. maxima* are less pathogenic.

These parasites are cosmopolitan, and all are of little importance in free-ranging family poultry.

Diagnosis: Diagnosis is by clinical signs and pathological lesions, and demonstration of various coccodian stages and oocysts in fecal materials.

Treatment: Several anticoccidial compounds are effective: amprolium, sulfadimethoxine, sulfaquinoxaline and sulfamethazine.

Endoparasites: *Raillietina* species (cestode)

Clinical signs and lesions: *Raillietina tetragona* is usually buried in the intestinal mucosa and is associated with weight loss and decreased egg production. *R. echinobothrida* is associated with catarrhal enteritis, nodular formation, and granulomas, and causes nodular disease. *R. cesticillus* causes emaciation and degeneration of intestinal villi.

Cause, transmission, and epidemiology: Species in this genus that have been reported in free ranging poultry include *R. tetragonal, R. echinobothrida* and *R. cesticillus*. These worms are cosmopolitan.

R. tetragona occurs in the posterior half of the small intestine and has been reported in chickens, pigeons and pea fowl. *R. echinobothrida* occurs in the small intestine of chickens and turkeys. *R. cesticillus* occurs in the small intestine of chickens, guinea fowl, and turkeys. All have an indirect life cycle that utilizes ants, beetles and flies as the intermediate hosts.

Diagnosis: Diagnosis is made by accurate identification of the parasite during necropsy and by identifying proglottides in feces.

Treatment: Broad spectrum benzimidazoles are effective for treatment of infestations of this tapeworm.

Prevention: Improve sanitary practices and apply approved insecticides to the soil or litter on the premises. This interrupts the parasite's life cycle by destroying the intermediate hosts. Separate birds according to species and age groups.

Endoparasites: *Strongyloides avium* (nematode)

Cause, transmission, and epidemiology: This genus belongs to the family Strongyloididae. It inhabits the small intestine and the caeca of fowl, turkeys, and wild birds. It is reported in most parts of the world including Africa.
Strongyloides avium has a direct life cycle. Only the female worms are parasitic. They are 2.2 mm long, and associated with thickening of the caecal wall.

Endoparasites:*Syngamus trachea* (nematode)

Other names: Gapeworm, red worm, forked worm.

Clinical signs and lesions: *S. trachea* causes gapeworm disease, which is characterized by dyspnoea and asphyxia, anemia, mucus production in the airways, and progressive emaciation. The bird may cough and perform gaping movements when the worms clog and obstruct the airways. Death may occur due to asphyxiation.

Worms may be seen in the posterior trachea.

Cause, transmission, and epidemiology: *S. trachea* is a nematode in the family Syngamidae. It is bright red in color, and both sexes are in permanent procreative conjunction. When the female gapeworm lays her eggs in the trachea of an infected bird, the eggs are coughed up, swallowed, and then defecated. When birds consume the eggs found in the feces or an intermediate host such earthworms, slugs and snails, they become infected with the parasite.

This worm is cosmopolitan and has been reported in Africa. It occurs in chickens, turkeys, geese, and various wild birds.

Cyathostoma bronchialis is a gapeworm of geese and turkeys.

Diagnosis: Diagnosis is based on clinical signs, characteristic eggs in the feces, and finding the worms in the airways at post-mortem.

Treatment: Albendazoles can be used to cure infection. Use appropriate disinfectant to treat infected pens.

Prevention: Keep pens dry. Turkeys should not be kept in the same pens with chickens.

Endoparasites: *Tetrameres* species (nematode)

Other names: Globular stomach worms.

Clinical signs and lesions: *Tetrameres* worms are associated with edema, thickening and obstruction of the proventriculus. Microscopically, they cause dilatation of proventricular glands, and compression and necrosis of glandular epithelial cells.

Cause, transmission, and epidemiology: This nematode worm has been reported in North America and Africa. Two species reported in Africa are *T*. *americana* (Botswana, Ethiopia, Kenya, Nigeria, Tanzania and Zimbabwe) occurring in chickens and turkeys, and *T. fissispina* (Tanzania, Ghana and Kenya) occurring in chickens, ducks, turkeys and pigeons.

The female worm is spherical in shape, blood red in color, measuring 3.5-4.5 mm by 3 mm long, and lies embedded in proventriculus gland of birds. The male is slender, measuring 5-5.5 mm long and 16-33µm wide, armed with four rows of cuticular spines, and is found free in the lumen of the proventriculus but may follow females into the gland temporarily for copulation.

These parasites have an indirect lifecycle. *T. americanas* uses grasshoppers and cockroaches as the intermediate hosts, and *T. Fissispina* uses earthworms, grasshoppers, amphipods and cockroaches.

Diagnosis: Definitive diagnosis is reached at post mortem through identification of the worm.

Endoparasites: Trematodes

There are many trematodes reported in birds, represented in the genera *Brachylaemus, Echinostoma, Echinoparyphlum, Hypoderaeum, Notocotylus, Cattropis,* and *Postharmostomum.*. *Prosthogonimus pellucidus* has been reported in Kenya and *Brachylaemus commutatus* from Uganda. *Prosthogonimus pellucidus* occurs worldwide in fowls and ducks. *Prosthogonimus macrorchis* is reported in poultry in North America. *Prosthogonimus ovatus* has been found in geese in Africa, Europe, Asia, and North and South America.

These flukes occur in the bursa of Fabricius, oviduct, cloaca and rectum. Adult worms measure 8-9 mm long and 4-5 mm wide, and are broader at the posterior end than the anterior end.

Birds infected by *Prosthogonimus* species have a tendency to sit on the nest, have a milky discharge from the cloaca, and lay eggs with soft or no shells. In chronic infestations the birds have peritonitis.

Endoparasites: *Trichostrongylus tenuis* (nematode)

Clinical signs and lesions: This species is associated with severe weight loss and anemia, and the caecal wall is congested and thickened.

Cause, transmission, and epidemiology: This parasite is worldwide distribution and occurs in the caecum and intestines of chickens, turkeys, ducks, and game birds. Male worms are 5.5–9 mm while females are 6.5–11 mm long. They have a direct life cycle.

Equine Encephalitis Viral Infection

Other names: Alphavirus infection.

Clinical signs and lesions: Many infected wild birds and poultry flocks have transient alphaviral infections and show no clinical signs, but antibodies can be demonstrated in their sera.

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

There are no significant gross lesions. Microscopic lesions occur in the brain of most clinically ill birds, but are not specific or definitive indicators of the disease.

Differential diagnosis: Equine encephalitis must be differentiated from diseases that cause neurologic symptoms in poultry including Newcastle disease (neurologic form), Marek's disease, avian encephalomyelitis, vitamin E deficiency, avian influenza and botulism.

Cause, transmission, and epidemiology: Equine encephalitis viral infection is an acute disease of pheasants, chukar partridges, turkeys, ducks, pigeons, or wild birds caused by one of a number of alphaviruses.

Eastern equine encephalitis (EEE) and Western equine encephalitis (WEE) are the causative agents, and are classified as alphaviruses in the family Togaviridae. These viruses naturally infect arthropods that ingest infected vertebrate blood. The viruses multiply in the arthropod and are transmitted when they bite susceptible vertebrates. Most outbreaks occur in captive birds and poultry less than 6 months old. Outbreaks usually occur during the mosquito season. The viruses of EEE and WEE readily infect humans with severe or fatal infection.

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. Infected mosquitoes then transmit the infection to other susceptible birds while feeding on them. Birds are the major source of infection for the 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. Certain biting insects (gnats, deerflies, horseflies, etc) may transmit the virus mechanically.

Diagnosis: In flocks with clinically ill birds, the signs are suggestive of central nervous system disease. 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.

Treatment: Treatment of sick birds is of no value.

Prevention: Protect birds against mosquitoes by the use of screens, sprays, or other mosquito control methods. In family poultry the traditional way is the use of fire and smoke.

Practice known methods of preventing cannibalism, including avoiding overcrowding and maintaining temperature controls. Prevent or treat ectoparasite infestations.

Recovery: There is no residual risk of infection after a case has occurred based on environmental contamination. However, if the conditions which resulted in the initial infection (presence of mosquitoes, presence of a reservoir host) are not corrected, then new birds may be infected.

Favus

Other names: Dermatophytosis, dermatomycosis, ringworm.

Clinical signs and lesions: Favus is a skin mycotic infection found primarily in gallinaceous birds. Favus is rare in commercial poultry, but occasionally found in hobby and backyard flocks, and game birds.

Lesions include white, scaly crusting on the comb and wattles that can extend to the feathered portion of the skin of the head and neck, with loss of feathers. It can form scutula around the bases of the feather follicles. Other than skin lesions, affected birds are typically healthy. The fungus infects the epidermis, which is thickened with degeneration, and neutrophils mixed with fungal mycelia.

Differential diagnosis: Other skin infections and causes of feather loss (ectoparasites and trauma).

Cause, transmission, and epidemiology: *Microsporium gallinae* is the agent most often isolated, although *M. gypseum* and *Trichophyton simii* have also been isolated. The infection is contagious and is transmissible to man, as are the majority of dermatophytes that form ringworm. It is reported in chickens, turkeys, ducks, quail, and canaries, and is a primary pathogen. Favus spreads slowly through a flock by direct contact or contact by contaminated cages or transport coops.

Diagnosis: Skin scrapings placed on 10% KOH on a glass slide with a cover lip that is gently heated will show the lesion mycelium or hyphae. They can also be grown on SDA. Histological examination of Gridley stained tissue will show characteristic hyphae.

Treatment: There is no approved treatment, but topical treatment with Nystatin has been effective on individual bird. Application of a 2% quaternary ammonium disinfectant, 1% tincture of iodine, or 5% formalin will eliminate the infection.

Prevention: Biosecurity precautions should be implemented to avoid introducing infected birds to the flock. Transport crates and other equipment should be thoroughly decontaminated and disinfected to prevent lateral transmission of the agent.

Recovery: Complete recovery requires that all individuals in a flock are cured of infection and that none of the causative agents remain in the environment. Recovery may be prolonged if treatment is incomplete or if the environment is not decontaminated.

Fowl Cholera

Other names: avian pasteurellosis, cholera, avian hemorrhagic septicemia.

Clinical signs and lesions: Fowl cholera usually strikes birds older than 6 weeks of age. In acute outbreaks, dead birds may be the first sign. Fever, reduced feed consumption, mucoid discharge from the mouth, ruffled feathers, diarrhea, and labored breathing may be seen. As the disease progresses birds lose weight, become lame from joint infections, and develop rattling noises from exudate in air passages. As fowl cholera becomes chronic, chickens develop abscessed wattles and swollen joints and foot pads. Caseous exudate may form in the sinuses around the eyes. Turkeys may have twisted necks.

Differential diagnosis: Fowl cholera must be differentiated from swollen head syndrome, Mycoplasmosis, avian influenza and Newcastle disease.

Cause, transmission, and epidemiology: Multiple means of transmission have been demonstrated. Flock additions, free-flying birds, infected premises, predators, and rodents are all possible sources of infection.

Diagnosis: A presumptive diagnosis can be made based on the clinical signs of the disease, the gross lesions evident on necropsy. A definitive diagnosis will be based on direct identification in tissue imprints or the isolation and identification of *P. multocida* from affected birds.

Treatment: A flock can be medicated with a sulfa drug (sulfonamides, especially sulfadimethoxine, sulfaquinonxalene, sulfamethazine, and sulfaquinoxalene) or vaccinated, or both, to stop mortality associated with an outbreak. In the United States, sulfa drugs are not FDA approved for use in pullets older than 14 weeks or for commercial laying hens because of their potential toxicity to humans. Sulfa drugs leave residues in meat and eggs. Other antibiotics can be used, but require higher levels and long term medication to stop the outbreak.

Prevention: On fowl cholera endemic farms, vaccination is advisable. Do not vaccinate for fowl cholera unless you have a problem on the farm. Rodent control is essential to prevent future outbreaks.

Recovery: Most flocks will recover from outbreaks of fowl cholera although if it is not detected and treated correctly, its economic impact may be devastating. After the flock has been treated, every effort should be made to ascertain the source of the bacterial disease agent and prevent future infections. Recovery is only possible if the environment is fully depopulated and cleaned and rodents on the premises removed before susceptible birds are introduced to the premises.

Fowl Pox

Other names: chicken pox (not to be confused with chicken pox in humans; the human disease does not affect poultry and vice versa), sore head, avian diphtheria, bird pox.

Clinical signs and lesions: There are two forms of fowl pox. The dry form is characterized by raised, wart-like lesions on unfeathered areas (head, legs, vent, etc.). The lesions heal in about 2 weeks. If the scab is removed before healing is complete, the surface beneath is raw and bleeding.

Unthriftiness and retarded growth are typical symptoms of fowl pox. In laying hens, infection results in a transient decline in egg production.

In the wet form there are canker-like lesions in the mouth, pharynx, larynx, and trachea. The wet form may cause respiratory distress by obstructing the upper air passages. Chickens may be affected with either or both forms of fowl pox at one time.

Differential diagnosis: Vitamin A deficiency, favus, sticktight fleas

Cause, transmission, and epidemiology: Fowl pox is a viral disease affecting most species of poultry. Chickens, turkeys, pheasants, quail, ducks, psittacine, and ratites of all ages (except newly-hatched chicks) are susceptible. It is transmitted by direct contact between infected and susceptible birds or by mosquitoes. Virus-containing scabs also can be sloughed from affected birds and serve as a source of infection. The virus can enter the blood stream through the eye, skin wounds, or respiratory tract. Mosquitoes become infected from feeding on birds with fowl pox in their blood stream. There is some evidence that the mosquito remains infective for life. Mosquitoes are the primary reservoir and spreaders of fowl pox on poultry ranges. Several species of mosquito can transmit fowl pox. Often mosquitoes over-winter in poultry houses, so outbreaks can occur during winter and early spring.

Diagnosis: Usually diagnosed by clinical signs, but lesion material containing fowl pox particles will produce pocks on the dropped chorioallantoic membrane of embryonated chicken eggs. Intracytoplasmic inclusion bodies may be seen in stained sections or in scrapings of the lesions in chickens, but may not be visible in turkeys.

Treatment: No treatment is available. However, fowl pox is relatively slow-spreading. Thus, it is possible to vaccinate to stop an outbreak.

Prevention: Fowl pox outbreaks in poultry confined to houses can be controlled by spraying to kill mosquitoes. However, if fowl pox is endemic in the area, vaccination is recommended. Do not vaccinate unless the disease becomes a problem on a farm or in the area. Chickens may be vaccinated at 4-6 weeks of age using the wing web-stick method, and turkeys older than 8 weeks by the thigh-stick method.

Recovery: Most individuals with fowl pox will recover and will clear the virus. Recovered birds are not reservoirs of infection. Flocks can recover from infection but the environment must be cleaned before new birds are introduced.

Gout

Uric acid is produced in the liver and is the end product of nitrogen metabolism in birds. Consequently, birds can develop gout secondary to an accumulation of urates. Gout is not a disease entity but a clinical sign of severe renal dysfunction that causes hyperuricemia and accumulation of urates in tissues of birds. There are two different syndromes that differ in etiology, morphology and pathogenesis. The two forms are visceral gout and articular gout.

A. Visceral Gout:

Clinical signs and lesions: The urate deposits appear as white chalky coating. Within the viscera they are observed microscopically as blue or pink amorphous material or as feathery crystals or basophilic spherical masses in tissues.

Cause, transmission, and epidemiology: The condition is due to a failure of urinary excretion, which can be as a result of:

- Obstruction of ureters
- Dehydration – most common cause
- Renal damage – kidney necrosis
- Vitamin A deficiency
- Secondary to urolithiasis
- Treatment with sodium bicarbonate

Except for the first cause, all occur due to turnover of nucleoprotein to cause hyperuricemia and precipitation and crystallization of urates. Other causes are mycotoxins such as oosporein, and renal cryptosporidiosis. Mechanisms of crystal precipitation in certain sites are unknown

Diagnosis: This form is characterized by precipitation of urates in the kidneys, and serous surfaces of the liver, heart, mesenteries, air sacs or peritoneum. In severe cases surfaces of muscles and synovial sheaths of tendons and joints are affected. Precipitation of urates can occur within the liver, spleen and other organs.

Treatment: Correcting the cause of the renal damage will prevent other birds from developing gout.

Prevention: Visceral gout is prevented by feeding a correctly balanced diet and providing adequate water for chickens.

Recovery: Individual affected birds will not recover, but once the cause of the condition is corrected, there will not be an increased residual risk to new birds on the premises.

B. Articular Gout

Clinical signs and lesions: Affected birds have shifting lameness and an inability to bend the toes. The disease is characterized by tophi - deposits of urates around joints, especially the feet (confused with bumble foot). Joints are enlarged and feet are deformed. In chronic cases, urate precipitation is found on the comb, wattles, trachea and other areas.

Differential diagnosis: This form of gout has to be differentiated from other diseases causing joint and skin swellings and abscesses, such as bumble foot, lymphoid leukosis (osteopetrosis), infectious tenosynovitis, *Mycoplasma synoviae*, Staphylococcosus.
Cause, transmission, and epidemiology: This form is generally restricted to individual birds and may be due to genetic defects in metabolism of uric acid. It may be a result of feeding high protein diets, which result in excess uric acid production. Affected chickens have a defect in kidney tubular secretion of uric acid.

Diagnosis: On opening the joints, periarticular tissue is white due to urate deposition, and semifluid deposits of urates are seen.

Treatment: There is no treatment for this condition although providing a lower protein diet may be helpful.

Prevention: Provide a lower protein diet, especially for males.

Recovery: This is a sporadic problem of little economic importance in poultry. Individual birds will not recover.

Hemoparasites

GENERAL INFORMATION

Clinical signs: A heavy infestation may contribute to anemia. Hemoparasites cause anemia and death by invading blood cells especially erythrocytes, which consequently are destroyed by the bird's immune system.

Cause, transmission, and epidemiology: Hemoparasites, or blood parasites, are fairly common in many birds, especially wild birds. Hemoparasites are mainly found in poultry in tropical areas and belong to the following genera: *Plasmodium* spp., *Leucocytozoon* spp., *Haemoproteus* spp., *Aegyptinella* spp., *Eperythrozoon* spp., *Trypanosoma* spp., and microfilaria of nematodes belonging to the suborder Filariata. Over 10 species of these are of pathogenic and economic importance. Attempts at treatment are usually unsuccessful, since it is difficult to control insect vectors involved in transmission of these parasites. These vectors include the mosquitoes, other flies, and the poultry soft tick (*Argas persicus*). Unlike semi-wild and wild birds whose blood parasites have been investigated, information on haemoparasitic infections in domestic family chickens in Africa is limited.

Diagnosis: *Preparation of blood smears* - Thin blood films are prepared from fresh blood, EDTA blood or a blood buffy coat layer. Blood films are air-dried within 5–10 sec after preparation, fixed in methanol for 5 minutes, and then stained with Giemsa (10%) for 10 to 15 minutes, washed with tap water, blotted and examined under light microscope for

hemoparasites. *Examination of blood smears* - Blood films are examined for 10–15 minutes at low magnification (X10), (X40) and then at least 20 fields are studied at high magnification (X100).

SPECIES INFORMATION

1. Plasmodium species

Avian malaria has a worldwide distribution and is of great economic significance for the poultry industry. *Plasmodium gallinaceum* and *P. juxtanucleare* are the two main species of malaria parasites found in chickens and turkeys. *Plasmodium gallinaceum* is found in Asia and Africa, whereas *P. juxtanucleare* occurs in South America, Africa and Asia.

Clinical signs and lesions: *Plasmodium gallinaceum, P. juxtanucleare* and *P. durae* may cause up to 90% mortality in poultry. They cause progressive emaciation, anemia and enlargement of the spleen and liver in affected birds. Paralysis may be observed where there are massive numbers of erythrocytic forms in endothelial cells of the brain capillaries, and death occurs in untreated cases.

Gross lesions include hepatomegally and splenomegally with subcutaneous, pulmonary and epicardial edema. Erythrocytic schizogony is the most striking microscopic lesion. This lesion is most evident in the lungs, spleen and liver. Acute interstitial pneumonia and diffuse reticulo-endothelial hyperplasia in spleen and other organs are present.

Cause, life cycle, transmission, and epidemiology: Birds are infected with *Plasmodium* sporozoites, which are transferred from the mosquito salivary glands to the bloodstream. The parasites undergo schizogony in macrophages and fibroblasts and then liver cells, producing merozoites. These merozoites enter erythrocytes, multiply by schizogony and finally form gametes, which are picked up by mosquitoes during feeding. Both gametocytes and schizonts of *P. gallinaceum* can be round, oval, irregular, or slightly elongated in shape. The nucleus of host cells is displaced by the parasite and host cells are distorted during infection. *Plasmodium* spp. have been reported in Nigeria and Kenya.

In *Plasmodium* spp. infections (avian malaria), merozoites or ring forms of the organism are usually apparent within erythrocytes. The rings may be single or multiple. Microrgametocytes and macrogametocytes also form within erythrocytes but are observed infrequently. *Plasmodium* species produce an insoluble pigment called hemozoin. This pigment is derived from the digestion of hemoglobin found within the host's erythrocytes and appears as refractile, yellow to brown granules within the host's erythrocytes.

Diagnosis: This is based on pathological lesions due to plasmodium infection. Evaluation of blood smear and monitoring the white blood cell counts for a lymphocytic leucocytosis are considered to be a reliable method of ante mortem diagnosis.

2. Leucocytozoon species

Clinical signs: *Leucocytozoon caulleryi* is the most virulent. Infected chickens frequently show signs of anorexia, thickened oral discharge, ataxia, anaemia and have difficulty breathing. They frequently die because of hemorrhages as a result of rapture of

megalomeronts that may develop in all organs and tissues. In addition, birds may be susceptible to secondary infection that may increase mortality.

Splenomegally, hepatomegally and anaemia are the most prominent gross lesions. Histologic sections show megaloschizonts in vascular endothelium of the brain. Smaller schizonts are seen in hepatic kupffer cells.

Cause, life cycle, transmission, and epidemiology: There are two main species of *Leucocytozoon* commonly found in chickens: *L. caulleryi* and *L. sabrazesi.* Other frequently encountered species include *L. smithi* (turkeys), *L. anatis* (ducks), *L. anseris* (geese), *L. neavei* (guinea fowl), and *L. andrewsi* (chickens). *Leucocytozoon* species are most easily distinguished because of their large size and football-like distortion of infected cells, with pointed ends. These parasites may infect erythrocytes or leukocyte cells. *Leucocytozoon sabrazesi* has been reported in Zimbabwe, while *L. caulleryi* has been reported in Nigeria and Kenya. *Leucocytozoon schoutedeni* (a new species) has been reported in Uganda and Cameroon.

Leucocytozoon gametocytes are found in erythroblasts and mononuclear leucocytes as ovoid (10 by 15 microns) or elongated (24 by 4 microns) forms. The host cells with elongated gametocytes become spindle shaped, with nuclei appearing as thin bands beside the parasite.

The vectors of *Leucocytozoon* spp. are biting midges, *Culicoide*s spp. and black flies, *Simuliidae* spp. After transmission to the birds, the sporozoites develop into schizonts. The schizogony takes place in the brain, liver, spleen, lungs and many other organs. Merozoites are then released and may enter a new cycle, or may enter erythrocytes or erythroblasts to develop into gametocytes. The gametocytes are only found in erythrocytes. The parasitic organism lies next to the host cell nucleus and is round in form. In the vector the zygote elongates into an ookinete that passes through the intestinal wall to form oocysts under the basal lamina. In the oocysts, sporozoites are formed and introduced into new hosts. Infections occur frequently when numbers of *Culicoides* spp. are abundant.

Diagnosis: Pathological lesions and evaluation of blood smear to demonstration the parasite in infected cells.

Control and treatment: Prevention of Leucocytozoonosis is usually directed towards control of black flies or midge populations. Screening of susceptible birds has been carried out in some instances. No effective treatment is known.

***3. Haemoproteus* species**

Clinical signs: Infections with most *Haemoproteus* spp. do not result in significant clinical signs. Experimental infection of turkeys with *Haemoproteus meleagridis* (*H. meleagridis*) resulted in lameness, diarrhea, depression, emaciation, anorexia and occasional anemia. Muscovy ducks infected with *H. nettionis* suffered lameness, dyspnea and sudden death. Pigeons infected with *H. columbae* had enlarged gizzards. In other avian species, anemia, anorexia, and depression have been reported occasionally, but *Haemoproteus* generally is considered non-pathogenic in most avian species. Post-mortem findings of infected birds include enlargement of the spleen, liver, and kidneys. These organs also may

appear chocolate-brown due to hemozoin deposition. Cytologic imprints of these organs may reveal schizont-laden endothelial cells. Some species of *Haemoproteus* will also form large, cyst-like bodies within skeletal muscles that resemble those seen with *Sarcocystis* spp. infections. However, these parasites are of little importance in domestic chicken.

Cause, life cycle, transmission, and epidemiology: *Haemoproteus* spp. are intracellular, protozoan, hemotropic parasites that infect red blood cells of birds (chickens, ducks and geese), turtles, and lizards. They are found worldwide and are capable of infecting a variety of birds including gamebirds (Galliformes), waterfowl (Anseriformes), raptors (Accipitriformes, Falconiformes, Strigiformes), pigeons, doves (Columbiformes), and perching birds or songbirds (Passeriformes). These parasites have been reported in Nigeria and Kenya.

The gametocytes partially encircle the erythrocyte nucleus forming a "halter-shaped" appearance. *Haemoproteus* gametocytes often occupy over one-half of the erythrocyte cytoplasm with little displacement of the host cell nucleus. Like *Plasmodium, Haemoproteus* produce an insoluble pigment called hemozoin. This pigment is derived from the digestion of hemoglobin found within the host's erythrocytes and appears as refractile, yellow to brown granules within the host's erythrocytes.

Haemoproteus is transmitted by blood-sucking insects including mosquitoes, hippoboscid flies (louse flies), and *Culicoides* species (biting midges). Successful transmission depends on the presence of the vector, and therefore infections occur more often in the warmer months of the year. The infective stage is the sporozoite, which is present in the salivary glands of the insect vector. Once the vector bites a new host, the sporozoites enter the bloodstream and invade endothelial cells of blood vessels within various tissues including the lung, liver, and spleen. Within the endothelial cells, the sporozoites go through asexual reproduction to become schizonts, which then produce numerous merozoites. These merozoites penetrate the erythrocytes and mature into either female gametocytes (macrogametocytes) or male gametocytes (microgametocytes). Gametocytes can then be ingested by another blood-sucking insect where they undergo sexual reproduction in the midgut of the insect to produce oocysts. The oocysts rupture and release numerous sporozoites that invade the salivary gland and serve as a focus of subsequent infection for another host once the insect takes its next blood meal.

4. *Aegyptinella* species

Clinical signs: *Aegyptinella pullorum* causes fever, diarrhea, anorexia and jaundice. At post-mortem there is anaemia, enlargement of the spleen and degeneration of the kidneys.

Cause, life cycle, transmission, and epidemiology: The two species of *Aegyptinella, A. pullorum* and *A. mushkovskii*, occur in chickens, turkey, ducks, geese, and other birds in Africa, Asia and Southern Europe. They are transmitted by the poultry tick *Argas persicus*, and appear as small (0.5 to 1.0 microns), round to oval bodies within the erythrocytes. *Aegyptinella pullorum* has been reported in Zimbabwe and Ghana.

The developmental cycle of *Aegyptinella* in the avian host consists of the formation of initial bodies, developmental forms, and marginal bodies. Following feeding by an adult

tick on an infected fowl, 25 days or more is required before the organism is transmissible to another bird.

5. *Trypanosoma avium*

Trypanosoma avium occurs in a wide range of birds. The most common vectors of avian trypanosomes are arthropods belonging to flies in the families Hippoboscidae, Culicudae, Ceratopogonidae and Simuliidae. In addition, dermanyssid mites have been identified as avian trypanosome vectors. *Trypanosoma avium* has been reported in Zimbabwe, Uganda and Cameroon.

6 *Borrelia anserina*

Borrelia anserina are transmitted by the poultry tick, *Argas persicus*, *Culex* mosquitoes, and red mites. Within 24-72 hours post-infection, *Borrelia anserina* spirochaetes appear in the bloodstream and there is marked temperature elevation. Affected birds are listless, have urates around the vent, and manifests leg weakness. This parasite has been reported in Zimbabwe.

Histoplasmosis

Clinical signs and lesions: The signs are varied and nonspecific. Histoplasmosis is a systemic fungal infection. There may be weight loss leading to emaciation with chronic respiratory disease. It causes disseminated granulomatous lesions in affected animals. Yellow-white variable-sized nodules may be found in the lungs, air sacs, liver and lymphoid tissue.

Differential diagnosis: Other lung infections and systemic fungal infections such as aspergillosis, cryptococcosis, and dactylariosis.

Cause, transmission, and epidemiology: This is a chronic infectious mycotic disease of man and animals (especially dog and cat). It has been reported in zoo birds, chickens and turkeys.

The causative agent is a dimorphic fungus, *Histoplasma capsulatum*, which is commonly found in soil containing bird and bat manure. Infection is through aerosol inhalation of conidia produced by the mold, which contaminate the respiratory system. Lungs and lymphoid tissue are the sites of primary infection. It enters the bloodstream at the primary site to cause widespread disease in the body.

Diagnosis: Clinical signs, culture of the organism, and tissue granulomatous lesions with intracytoplasmic narrow-based budding yeasts measuring 2-4 µm in diameter in the macrophages.

The fungus grows readily in culture media and soil as a white to brown mold that produces microconidia (3-4 µm, spiny and spherical) and macroconidia (8-12 µm, spherical).

Treatment: Treatment is symptomatic, or use amphotericin B.

Prevention: Disinfect equipment with phenol-based disinfectants.

Recovery: Once infested birds have shed parasite eggs into the environment, viable eggs will remain until they are removed, which is not feasible. A wire or cement flooring must be introduced to remove or reduce risk to new flocks. However, over time, risk will decrease as the eggs are dispersed more widely and new birds are exposed to lower egg doses.

Infectious Bronchitis

Other names: IB, bronchitis, cold.

Clinical signs and lesions: Typical respiratory signs such as coughing, sneezing, rales, wheezing, and/or gasping are present. Watery discharge from the eyes and/or nostrils, swelling of the head, and head shaking may be observed. Feed and water consumption declines. Affected chickens will often be chirping. Breathing noises are more noticeable at night while the birds rest. Baby chicks show weakness, loss of appetite, reduced water intake, and depression, and huddle around the source of heat. The infectious bronchitis virus infects many tissues of the body, including the reproductive tract. Layer birds show severe and prolonged reduction in egg laying; the few eggs laid have soft or rough shells, and the egg white becomes watery. Production recovers in 5 or 6 weeks, but at a lower rate than before infection.

Lesions outside the respiratory system include swollen kidneys, with ureters and tubules distended and containing whitish uric acid crystals. Broken eggs in the abdomen may cause inflammation of the abdominal lining. The ovaries are soft.

The severity of infectious bronchitis infection is influenced by the age and immune status of the flock, by environmental conditions, and by the presence of other diseases.

Differential diagnosis: Newcastle disease.

Cause, transmission, and epidemiology: Infectious bronchitis occurs in chickens only, but affects all ages and is common on highly intensive farms. The causative virus belongs to the family Cornaviridae, and is easily destroyed by most disinfectants. The virus is transmitted through inhalation of droplets expelled by infected birds showing clinical signs of the disease. The disease spreads rapidly through a flock in this way, but may also spread through dead birds, contaminated feed bags, and through mechanical transmission by rodents. The virus can also be transmitted through eggs; however, affected embryos usually will not hatch.

A similar disease, quail bronchitis, is caused by a different virus and affects only quail.

Diagnosis: Tentative diagnosis based on clinical signs and lesions is confirmed by isolation of the virus in embryonated chicken eggs, and differentiation from ND (which shows similar signs and lesions). IBV does not agglutinate red blood cells.

Treatment: There is no specific treatment for infectious bronchitis. Antibiotics given for 3-5 days may aid in combating secondary bacterial infections. Raise the room temperature 5°F for brooding-age chickens until symptoms subside. Baby chicks can be encouraged to eat by using a warm, moist mash.

Prevention: Establish and enforce a biosecurity program. Vaccination is practiced in young chickens, but vaccine should be of the prevailing serotype on the farm and must be strictly preserved in a cold-chain.

Recovery: As IB infection tends to persist on a farm, depopulation is strongly recommended, followed by disinfection and resting of the pens. An effective vaccination program should be introduced with the arrival of the next batch of chicks.

Infectious Bursal Disease

Other names: Gumboro, IBD, infectious bursitis, infectious avian nephrosis.

Clinical signs and lesions: In affected chickens greater than 3 weeks of age, there is usually a rapid onset of the disease with a sudden drop in feed and water consumption, watery droppings leading to soiling of feathers around the vent, and vent picking. Feathers appear ruffled. Chicks are listless and sit in a hunched position. Chickens infected when less than 3 weeks of age do not develop clinical disease, but become severely and permanently immunosuppressed.

Differential diagnosis: Acute cases of IBD should be differentiated from coccidiosis while bursal atrophy should be differentiated from Marek's disease.

Cause, transmission, and epidemiology: The virus is spread by bird-to-bird contact, as well as by contact with contaminated people and equipment. The virus is shed in bird droppings and can be spread through the air on dust particles. Dead birds are also a source of the virus and should be incinerated.

Diagnosis: A strong presumptive diagnosis can be made based on the rapid onset of morbidity and mortality and a rapid recovery in 5-7 days, which are characteristic of this disease. Gross lesions at necropsy and histopathology can confirm the etiology as can the isolation of the causative virus.

Treatment: There is no specific treatment. Antibiotics, sulfonamides, and nitrofurans have little or no effect. Vitamin-electrolyte therapy is helpful. High levels of tetracyclines are contraindicated because they tie up calcium, thereby producing rickets. Surviving chicks remain unthrifty and more susceptible to secondary infections because of immunosuppression.

Prevention: A vaccine is commercially available. Practice good sanitation and biosecurity.

Recovery: The virus is very stable in the environment so, it is usually not possible to prevent the spread of cases from previous flocks to new flocks. Vaccination is the key to recovery.

Infectious Coryza

Other names: Croup, cold, coryza.

Clinical signs and lesions: Swelling around the face, foul smelling, thick, sticky discharge from the nostrils and eyes, labored breathing, and rales are common clinical signs. The eyelids are irritated and may stick together. The birds may have diarrhea, and growing birds may become stunted.

Mortality from coryza is usually low, but infections can decrease egg production and increase the incidence and/or severity of other diseases. Mortality can be as high as 50 percent, but is usually no more than 20 percent. The clinical disease can last from a few days to 2-3 months, depending on the virulence of the pathogen and the existence of other infections such as mycoplasmosis.

Differential diagnosis: Clinical cases of infectious coryza can resemble swollen head syndrome, fowl pox (wet form), chlamydiosis, vitamin A deficiency, mycoplasmosis or chronic fowl cholera.

Cause, transmission, and epidemiology: Infectious coryza is a bacterial disease of chickens, pheasants, and guinea fowl. It is common in game chicken flocks. The causal agent is *Hemophilus paragallinarum*, which is primarily transmitted by direct bird-to-bird contact. This can be from infected birds brought into the flock as well as from birds that recover from the disease but remain carriers of the organism, which they may shed intermittently throughout their lives. Birds risk exposure at poultry shows, bird swaps, and live-bird sales. Inapparently infected adult birds added into a flock are a common source for outbreaks. Within a flock, inhalation of airborne respiratory droplets and contamination of feed and/or water are common modes of spread.

Diagnosis: A presumptive diagnosis can be made based on the epidemiology of the outbreak, clinical signs of disease and lesions at necropsy. However, a definitive diagnosis will require isolation and identification of the bacterial disease agent.

Treatment: Water soluble antibiotics or antibacterials can be used. Sulfadimethoxine (Albon®, Di-Methox™) is the preferred treatment. If it is not available, or not effective, sulfamethazine (Sulfa-Max®, SulfaSure™), erythromycin (gallimycin®), or tetracycline (Aureomycin®) can be used as alternative treatments. Sulfa drugs are not approved for pullets older than 14 weeks of age or for commercial layer hens. While antibiotics can be effective in reducing clinical disease, they do not eliminate carrier birds.

Prevention: Good management and sanitation are the best ways to avoid infectious coryza. Most outbreaks occur as a result of mixing flocks. All replacement birds on "coryza-endemic" farms should be vaccinated. The vaccine (Coryza-Vac) is administered subcutaneously on the back of the neck. Each chicken should be vaccinated four times, starting at 5 weeks of age with at least 4 weeks between injections. Vaccinate again at 10 months of age and twice yearly thereafter.

Recovery: *Hemphilus paragallinarum* is easily removed from a contaminated environment with cleaning and disinfection. However, any birds on the premises must be depopulated to eliminate the disease since carrier birds are the major source of infection. If depopulation is not practical, then vaccination should be considered.

Infectious Laryngotracheitis

Other names: Often abbreviated as ILT or LT.

Clinical signs and lesions: Chickens and pheasants are affected by LT. Older chickens (14 weeks and above) are more susceptible. The clinical sign usually first noticed is watery eyes. Affected birds remain quiet because breathing is difficult. Coughing, sneezing, and shaking of the head to dislodge exudate plugs in the windpipe follow. Birds extend their head and neck to facilitate breathing (commonly referred to as "pump handle respiration"). Inhalation produces a wheezing and gurgling sound. Blood-tinged exudates and serum clots are expelled from the trachea of affected birds. Many birds die from asphyxiation due to a blockage of the trachea when the tracheal plug is freed. Severely affected chickens extend their head and neck to facilitate breathing. In addition to coughing, gasping with loud sounds and wheezing they shake their heads to try to expel bloody mucus exudates from their trachea (windpipe) and nostrils. Egg production is reduced. Although morbidity is high, mortality rates are low.

Lesions are similar to those for other viral respiratory diseases: tracheae are inflamed, showing excessive mucus content or blood and congestion of the tracheal lining; lungs may contain excess blood or fluid; air sacs are often opaque and thickened. Inflammation of the mucous layer of the respiratory tract may progress to a false membrane made of dead tissue, with caseous material in the trachea which may block the passage and cause suffocation.

Differential diagnosis: ILT must be differentiated from other viral respiratory infections such as Newcastle disease or avian influenza. Mild cases of ILT may also be confused with wet pox, infectious bronchitis or Mycoplasmosis.

Cause, transmission, and epidemiology: The agent of this acute disease of chickens is a member of the group Herpesviridae. It is easily destroyed by most disinfectants. The many strains of this virus are immunologically similar, although they vary in their ability to cause disease.

The virus spreads by inhalation of droplets within a flock, but does so less rapidly than other viruses. It can also be spread through fomites – contaminated objects found in the environment of the birds. Birds that have recovered from ILT or that have been vaccinated may be carriers for long periods of time. LT may be harbored in specialty poultry such as exhibition birds and game fowl.

Diagnosis: The signs and lesions of ILT are strongly suggestive. Tentative diagnosis can be confirmed by examining sections of the trachea microscopically for bodies within the nucleus of the cells of tracheal lining. Other more sophisticated techniques are available to confirm suspicion, such as the fluorescent antibody technique.

Treatment: Administer antibiotics to control secondary infection, and vaccinate the flock. Mass vaccination by the spray or drinking water method is not recommended for large commercial or caged flocks. Individual bird administration by the eye-drop route is suggested. Follow the manufacturer's instructions. In small poultry flocks, use a swab to remove any tracheal plugs from gasping birds, and vaccinate by the eye-drop method.

Prevention: It is advisable to vaccinate birds, using attenuated vaccine in endemic areas. Vaccination for LT is not as successful as for other diseases, but is an excellent preventive measure for use in outbreaks and in epidemic areas. Birds are vaccinated after 10 weeks of age by eye drop or aerosol spray. Birds that have recovered from ILT, or been vaccinated against it, should not be added to flocks of susceptible birds because of their carrier state.

Recovery: Dispose of dead birds properly by deep burial or incineration. Depopulation, thorough disinfection of the premises and equipment, and resting of pens for 4-6 weeks are all necessary before restocking in order for a complete recovery to occur.

Infectious Synovitis – See *Mycoplasma synoviae* infection

Infectious Tenosynovitis

Other names: viral arthritis, tenosynovitis, teno, reovirus enteritis, reovirus septicemia, malabsorption syndrome, helicopter disease.

Clinical signs and lesions: The principal sign of tenosynovitis is lameness with swelling of the tendon sheaths of the shank and the area extending above the hock. Affected birds are lame, sit on their hocks, and are reluctant to move. Rupture of the tendon can occur in older roaster birds, resulting in permanent lameness of the affected leg. If more than two joints are affected, the entire carcass will be condemned.

Infection can also play a part in broiler stunting, the result of malabsorption syndrome. In chicks, malabsorption due to viral enteritis is called "helicopter disease" because feathering is affected. Wing feathers protrude at various angles. A reovirus is believed to play only a secondary role in this syndrome.

In commercial layer flocks, increased mortality may be the first sign of the septicemia form. Egg production will decrease by about two to three times the mortality rate. For example, a mortality rate of 5 percent will be accompanied by a 10-15 percent drop in egg production. In the septicemic form, joint involvement is present but less pronounced. Affected birds become cyanotic and dehydrated. The tips of the comb turn purplish. The entire comb darkens as the disease progresses.

Differential diagnosis: For tenosynovitis, staphylococcosus, articular gout, and *Mycoplasma synoviae* infection should be considered.

Cause, transmission, and epidemiology: Several serotypes of the reovirus have been identified. Some localize in the joints (tenosynovitis) while others target respiratory or intestinal tissues (septicemic form). The virus is shed in the feces, and infection is by the respiratory and digestive tract routes. The infection spreads rapidly through broiler flocks, but less rapidly in caged layers.

Diagnosis: A presumptive diagnosis can be made based on the appearance of the joints at necropsy. The direct demonstration of reovirus in the tendons or the isolation and identification of the virus can be used to make a definitive diagnosis.

Treatment: There is no satisfactory treatment for the viral infection available. With hens, tetracycline to treat secondary bacterial infections, molasses, and oyster shell therapy may be helpful.

Prevention: A vaccine is available for use in endemic areas or on endemic farms.

Recovery: Once infection has occurred on a premises, it is not practical to eradicate it since it is very stable outside the bird. Vaccination will prevent disease and permit recovery of productivity.

Lymphoid Leukosis

Other names: visceral leukosis, leukosis, big liver, LL.

Clinical signs and lesions: Signs of lymphoid leukosis begin to show in birds only after the age of 16 weeks, because it has a long incubation period. Clinical signs are non-specific. The abdomen is distended as a result of an enlarged liver, the comb is reduced in size, and birds become emaciated and progressively weaker. If many birds in the flock are infected, egg production is reduced. Greenish diarrhea develops in terminal stages.

Lesions are restricted to internal organs. The liver, spleen, kidney, and ovary contain white or grey nodules which are cancerous lymphoid tissue that has spread from the bird's bursa.

Differential diagnosis: Lymphoid leukosis may be confused with Marek's disease, but the latter occurs in younger birds (from 3 weeks of age), and LL always involves the bursa of Fabricius.

Cause, transmission, and epidemiology: Although primarily a disease of chickens, lymphoid leukosis can infect turkeys, guinea fowl, pheasants, and doves, but not on a large scale. The cause of LL is a family of retroviruses, the avian leukosis viruses. Virus transmission occurs through the egg to chick; the rate of transmission is low, but the chick is a life-long carrier and lays fewer eggs as a hen. Some bird-to-bird transmission and through contact with contaminated environments also takes place.

Diagnosis: Tentative diagnosis is easily achieved by consideration of the age of affected birds, the lesions, and involvement of the bursa. For confirmation, the bursal tumors must be examined histologically. LL will result in tumors of the bursal follicles while a rare MD-caused tumor of the bursa will be interfollicular.

Treatment: There is no treatment for LL.

Prevention: Eradication is the most effective method of prevention. The virus is present in the yolk and egg white of eggs from infected hens. Most national and international layer breeders have eradicated lymphoid leukosis from their flocks. Most commercial chicks are

lymphoid-leukosis negative because they are hatched from LL-free breeders. The disease is still common in broiler breeder flocks.

Recovery: Because LL is primarily transmitted from breeders to progeny, recovery requires that replacement chicks are obtained from LL-free suppliers.

Marek's Disease

Other names: acute leukosis, neural leukosis, range paralysis, gray eye (when the eyes are affected).

Clinical signs and lesions: Marek's disease is a type of avian cancer. Chickens between 12 to 25 weeks of age are most commonly clinically affected. Occasionally pheasants, quail, game fowl and turkeys can be infected. Tumors in nerves cause lameness and paralysis. Tumors can occur in the eyes and cause irregularly shaped pupils and blindness. Tumors of the liver, kidney, spleen, gonads, pancreas, proventriculus, lungs, muscles, and skin can cause incoordination, unthriftiness, paleness, weak labored breathing, and enlarged feather follicles. In terminal stages, the birds are emaciated with pale, scaly combs and greenish diarrhea.

Differential diagnosis: Marek's disease is very similar to lymphoid leukosis, but Marek's usually occurs in chickens 12 to 25 weeks of age and lymphoid leukosis usually starts at 16 weeks of age.

Cause, transmission, and epidemiology: Marek's disease virus is transmitted by air within the poultry house. It is in the feather dander, chicken house dust, feces and saliva. Infected birds carry the virus in their blood for life and are a source of infection for susceptible birds.

Diagnosis: The classical appearance of swollen peripheral nerves at necropsy and the distribution of lymphocytes in nerves will confirm a diagnosis of Marek's disease. In cases where the nerves are not affected, the age distribution of affected birds will help to differentiate MD from LL.

Treatment: None.

Prevention: Chicks can be vaccinated at the hatchery. While the vaccination prevents tumor formation, it does not prevent infection by the virus.

Recovery: Marek's disease is very stable in feather dander and infectious virus can remain on infected premises for very long periods of time. Once infection has occurred, future infections will occur and disease can only be prevented with vaccination. Birds with clinical signs of disease will not recover and must be culled.

Mycoplasma meleagridis Infection

Other names: MM, N strain, H strain.

Clinical signs and lesions: A drop-off in production and hatchability is seen in breeder flocks. There can be very high mortality in young poults. Unthriftiness, respiratory distress, stunting, crooked neck with deformity of cervical vertebrae, and leg deformation are common in young birds.

Differential diagnosis: Although typical of mycoplasmosis, the gross lesions of MM (especially in the air sac) cannot be differentiated from those caused by other *Mycoplasma* sp.

Cause, transmission, and epidemiology: This bacterial disease affects turkeys of all ages, although poults are affected more severely than mature turkeys. Recently, MM has been shown to infect pigeons, quail and peafowl.

Egg transmission is low in the early breeding period, but rises as the age of the flock increases. Infections can be introduced into a flock by contaminated equipment, shoes, and clothing of workers and visitors.

Diagnosis: Clinical disease and typical lesions can lead to a presumptive diagnosis of MM but a definitive diagnosis is only possible through the isolation of the causative organism and its identification with specific antisera. Alternatively, the testing of paired sera (pre and post exposure) can also support a diagnosis.

Treatment: Several antibiotics have been effective including tylosin, erythromycin, spectinomycin, and lincomycin-spectinomycin.

Prevention: The best preventive measure is to keep MM-free breeders. The MM-free status of breeders can be confirmed by periodic blood tests as has been instituted through the National Poultry Improvement Plan in the United States.

Recovery: Generally, once a farm has had a flock with mycoplasma, subsequent flocks will be infected. Premises can get rid of MM by hatching MM-free eggs and placing them in a clean environment and with very strict biosecurity in place. If biosecurity cannot be achieved then recovery is probably not possible.

Mycoplasma synoviae Infection

Other names: MS, Infectious Synovitis, Tenovaginatis

Clinical signs and lesions: This disease occurs in chickens and turkeys. Early signs are respiratory difficulty, lameness and a tendency of birds to rest on the floor. It occurs in growing birds. Other signs are pale combs, swollen hock joints, greenish diarrhea, an unwillingness to move to feed and watering troughs, loss of weight, and breast blisters (a result of persistent floor rest). In layer flocks there is a transient drop in egg production.

Although infectious synovitis is predominantly a disease of the upper respiratory tract, spread of infection in the body occurs, resulting in the inflammation of the joints of many infected birds. Joints contain mucoid yellowish to grey fluid, which becomes more solid as

the disease progresses. In the respiratory tract mild inflammation may be seen in the trachea and in air sacs.

Differential diagnosis: Clinically, the disease is indistinguishable from chronic respiratory disease, caused by *Mycoplasma gallisepticum.* Diseases that may show similar signs and lesions are staphylococcal arthritis and infectious tenosynovitis (viral arthritis).

Cause, transmission, and epidemiology: Infectious synovitis is caused by *Mycoplasma synoviae* (MS). Egg (vertical) transmission is the more important form of spread of MS, but it is also transmitted by contact and air droplets (respiratory route) within the flock.

Diagnosis: Clinical signs and lesions provide information for a dependable tentative diagnosis. It can be confirmed by isolation of the causative organism on a MS media or in embryonating chicken eggs.

Treatment: Lame birds are not successfully treated, but several antibiotics (tetracycline, oxytetracycline, tylosin, erythromycin and others) have been used to reduce losses, especially when given by injection rather than in feed or water. Recovery is slow for both respiratory and synovitis forms. Several antibiotics are variably effective. The most effective are tylosin, erthromycin, spectinomycin, lincomycin, and chlorotectracycline.

Prevention: Use replacement chicks from certified MS-free breeders. The all-in, all-out management system helps to avoid a build-up of *Mycoplasma* on a poultry farm. Eradication is the best and only sure control.

Recovery: Generally, once a farm has had a flock with mycoplasma, subsequent flocks will be infected. Premises can get rid of MS by hatching MS-free eggs and placing them in a clean environment and with very strict biosecurity in place. If biosecurity cannot be achieved then recovery is probably not possible.

Mycotoxins: Aflatoxicosis

Other names: Turkey X disease.

Clinical signs and lesions: This is primarily a disease of the liver, but affects other body functions, causing production problems and death in affected birds. Turkey poults and ducklings are highly susceptible. The affected birds show depression, inappetence, reduced growth rate, loss of condition, bruising, decreased egg production, fertility and hatchability, and increased mortality. Some birds may show ataxia, convulsions, and opisthotonos. Spontaneous aflatoxicosis is associated with increased susceptibility to infectious disease due to its immunosuppression effects.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: Aflatoxin is produced by *Aspergillus flavus*, *A. parasiticus* and *Penicillium puberulum* growing on the feed. The mycotoxins of the aflatoxin group are identified as B1, B2, G1, and G2. Aflatoxin B1 is the most common in grains and is highly toxic. The production of the toxin occurs when substrate, temperature, and humidity are ideal. Grains damaged by insects and drought stress, and broken pieces

of grain are more likely to support fungal growth and toxin formation. Aflatoxin B1 is a potent, naturally occurring carcinogen hence public health consideration.

Diagnosis: On post mortem examination of birds with aflatoxicosis, there is jaundice, generalized edema, hemorrhages, tan or yellow liver, and swollen kidneys. There may be marked catarrhal inflammation of the duodenum and excessive bile production. Some degree of increased peritoneal fluid (ascites) and visceral edema is usually evident. Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water- based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Citrinin Mycotoxicosis

Clinical signs and lesions: Citrinin mycotoxicosis affects chickens, turkeys and ducklings. There are marked watery droppings due to increased water intake and urine output, as well as metabolic alterations of electrolytes and acid-base balance. Young birds have reduced weight gain.

Slight gross lesions are present on the kidneys, which may be swollen. Lymphoid depletion and liver necrosis may also be present.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: This toxin is produced by *Penicillium citrinum*, other species of *Penicillium* and some *Aspergillus* species.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water-based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Ergotism

Clinical signs and lesions: In chickens, ergotism causes reduction in growth and egg production; and nervous incoordination. Lesions include abnormal feather development, necrosis of the beak, comb, and toes, and enteritis.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: The common cause are parasitic plant fungi, *Claviceps purpurea* and other *Claviceps* species, that colonize wheat and rye to produce ergot alkaloids, compounds causing ergotism in man and animals. The alkaloid causes constriction of the blood vessels. It is transmitted through contaminated feed.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water- based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Ochratoxicosis

Clinical signs and lesions: Ochratoxins are among the most toxic mycotoxins (fungal toxins) to poultry. Clinical signs are reductions in feed intake and increased mortality. Weight loss and reduced egg production can occur due to Ochratoxin A.

Acute lethal Ochratoxin A causes pale liver, pancreas and kidneys; swollen kidneys; white urate deposits in the ureters; and visceral urate deposition. There is tubular nephrosis. Subacute Ochratoxin A causes increased kidney and liver weight, and decreased weight of lymphoid organs. There is reduction in plasma carotenoids, immune function, and certain blood coagulation factors.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: Ochratoxins A, B, C, and D, and their methyl ethyl esters are produced by *Penicillium viridicatum* and *Aspergillus ochraceous*, which grow on numerous grains and feedstuffs. Ochratoxin A is the most toxic and is the greatest threat to poultry production.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water- based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Oosporein Mycotoxicosis

Clinical signs and lesions: Oosporein is a red, toxic, dibenzoquinone. Oosporein mycotoxicosis causes a dose-related decrease in growth and an increase in water consumption. Chickens are more susceptible than turkeys.

It causes visceral and articular gout, swollen pale kidneys, due to nephrotoxicity and mortality. The liver is mottled with focal necrosis and gall bladder is distended

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: The toxin is produced by *Chaetomium* species and other fungi that are contaminants of cereal grains and feedstuff.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water-based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Trichothecene Mycotoxicosis

Other names: Fusariotoxicosis

Clinical signs and lesions: Chickens have reduced growth, abnormal feathering, severe depression, and bloody diarrhea. In chickens, pigeons, ducks, and geese, the caustic properties of trichothecenes are manifested as feed refusal, extensive necrosis of the oral mucosa and areas of skin in contact with the mold, and acute gastrointestinal disease.

Lesions observed are necrosis of the oral mucosa, reddening of the mucosa of the rest of the gastrointestinal tract, mottling of the liver, distension of the gall bladder, atrophy of spleen, and visceral hemorrhages.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: Trichothecenes are produced by soil and plant fungi including *Fusarium.* Over 40 trichothecenes are known to exist. T-2 toxin is the one most toxic to poultry.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water-based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Mycotoxins: Zearalenone Mycotoxicosis

Clinical signs and lesions: This fungal disease has been associated with high mortality in chickens. Affected birds have cyanotic combs and wattles and difficulty walking. Turkeys may develop swelling of the cloaca and reduced fertility. Male geese may have reduction in sperm quantity and viability

Lesions in affected birds are ascites, and cysts inside and outside the oviduct. Oviducts are swollen and inflamed, and are obstructed with fibrinous fluid. Some oviducts rupture.

Differential diagnosis: Other mycotoxicoses.

Cause, transmission, and epidemiology: Zearalenone is a mycotoxin produced by *Fusarium roseum* (*Gibberella zeae*) and other *Fusarium* species. A period of warm temperature and high humidity followed by low temperature is most conducive to toxin formation on grains.

Diagnosis: Definitive diagnosis of mycotoxins involves isolation, identification and quantification of the specific toxin(s).

Treatment: Reduce the toxic feed and replace it with good feed. Treat concurrent diseases (parasitic, bacterial) identified during diagnosis. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can be compensated for by feed formulation and water-based treatment.

Prevention: Prevention is based on detection of contaminated ingredients and their exclusion from rations if possible. The correct storage of ingredients is necessary. Feed additive mold inhibitors such as propionate and gentian violet will suppress proliferation of fungi and elaboration of toxins. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by good sanitation.

Recovery: Cases will re-occur if mycotoxins are formed on the farm because feed is not handled properly. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk.

Necrotic Enteritis

Other names: enterotoxemia, rot gut.

Clinical signs and lesions: Initially there is a reduction in feed consumption as well as dark, often blood-stained feces. Infected chickens will have diarrhea. Chronically affected birds become emaciated. The bird, intestines, and feces emit a fetid odor.

Differential diagnosis: Necrotic enteritis should be differentiated from ulcerative enteritis and coccidiosis. This can be complicated when necrotic enteritis and coccidiosis appear concurrently, as is often the case.

Cause, transmission, and epidemiology: The disease is caused by a *Clostridium* bacterium (*C. perfringens*). Rapidly growing young birds, especially chickens and turkeys 2-12 weeks of age, are most susceptible. Necrotic enteritis is a disease associated with domestication and is unlikely to threaten wild bird populations. It is primarily a disease of broilers, roasters and turkeys. Ulcerative enteritis, on the other hand, commonly affects pullets and quail.

Necrotic enteritis does not spread directly from bird to bird. Bacteria are ingested along with infected soil, feces, or other infected materials, then grow in the intestinal tract. Infection commonly occurs in crowded or immunosuppressed flocks, and flocks maintained in poor sanitary conditions.

Diagnosis: The diagnosis of necrotic enteritis (NE) can be made on the basis of typical gross and histologic lesions in the intestine. The causative agent can be isolated to confirm the specific diagnosis.

Treatment: The clostridia bacteria involved in necrotic enteritis are sensitive to the antibiotics bacitracin, neomycin, and tetracycline. However, antibiotics such as penicillin, streptomycin, and novobiocin are also effective. Bacitracin is the most commonly used drug for control of necrotic enteritis.

Prevention: Prevention consists of the use of preventative levels of medication, parasite control, good sanitation, husbandry, and management.

Recovery: *Clostridium perfringens* is a spore-forming organism and infectious spores will remain in the soil after an outbreak. Over time, the spores will be dispersed resulting in lowered exposure for naïve birds but risk will not disappear. Controlling predisposing factors, like immunosuppressive diseases and internal parasites will make birds more resistant. In highly susceptible species, like quail, preventative medication may be the only means of recovery.

Newcastle Disease

Other names: Sometimes called pneumoencephalitis, exotic Newcastle disease, Asian Newcastle disease or pigeon paramyxovirus. Newcastle disease is referred to by its three virulence types – mildly pathogenic, moderately pathogenic, and very pathogenic. It may also be classified according to the predilection site of the virus: pneumotropic (respiratory system), viscerotropic (gastrointestinal tract), or neurotropic (nervous system).

Clinical signs and lesions: Newcastle disease is characterized by the sudden onset of clinical signs, which include hoarse chirps (in chicks), watery discharge from nostrils, labored breathing (gasping), facial swelling, paralysis, trembling, and twisting of the neck. Mortality ranges from 10 to 80 percent, depending on the pathogenicity of the responsible strain.

The mildly pathogenic strains may cause reduced egg-laying only, apart from mild respiratory signs. Moderately pathogenic strains may show transient nervous signs and drastic reduction in egg-laying in addition to mild respiratory signs. Layer birds show

sudden, severe drop in egg production, and the eggs laid are soft-shelled. Lesions outside the respiratory system include hemorrhages in the ovaries and proventriculus, and sometimes plaques and hemorrhages in the gizzard, intestinal lining, and caecal tonsils. The latter lesions are considered characteristic of ND. The very virulent strains cause signs similar to those caused by the moderately pathogenic strains, but mortality rates are high and can wipe out the entire flock. Newcastle disease can cause a mild conjunctivitis in humans and other mammals.

Differential diagnosis: Avian influenza, infectious bronchitis, infectious laryngotracheitis.

Cause, transmission, and epidemiology: Newcastle disease virus (NDV) belongs to the Paramyxovirus Type 1 group. It is believed that local, free-roaming chickens serve as reservoirs of infection for newly-hatched local birds and commercial birds. However, serious seasonal (December – March) outbreaks of velogenic ND occur annually in village chickens in West Africa. Guinea fowl are known to harbor the virus without showing clinical signs. They may be the source of outbreaks in village chickens in certain parts of West Africa.

In live birds, the virus is shed in body fluids, secretions, excreta, and breath of birds incubating the disease, showing clinical signs, or recovering from ND. During an outbreak the virus is spread in exhaled air, respiratory discharges, feces and eggs. Healthy birds may be infected from these sources, and hatcheries from infected eggs.

NDV can also be transmitted by contaminated shoes, caretakers, feed deliverers, visitors, tires, dirty equipment, feed sacks, crates, and wild birds. The virus can be passed in the egg, but Newcastle-infected embryos die before hatching.

Diagnosis: Definitive diagnosis may be made by isolation of the virus in Newcastle antibody-free chicken embryonated eggs, and identification of the virus by the hemagglutination-inhibition test. Other serological tests are available.

Treatment: There is no specific treatment; antibiotics administered for 3-5 days to help prevent secondary bacterial infections (especially *E. coli*) in mild cases are useful. For chicks, increasing the brooding temperature 5°F may help reduce losses.

Prevention: Good sanitation and implementation of a comprehensive biosecurity program are necessary to prevent Newcastle and other avian diseases. Well-designed vaccination schedules, using low-virulence live vaccines, give very effective immunization results. Inactivated oil-emulsion vaccines administered to the parent flock before onset of egg dropping ensure passive immunity in day-old chicks. Active immunization is induced in the chicks just when their passively-acquired antibody levels are dropping. A second vaccination four weeks later gives life-long protection if proper vaccination procedures are applied.

Recovery: Depopulation and thorough disinfection of the premises are recommended.

Omphalitis

Other names: Navel-ill, yolk-sac disease, mushy chick disease.

Clinical signs and lesions: Affected chicks show dullness and lack of appetite. Morbidity and mortality rates are variable. Diarrhea, with a pasty vent area, is characteristic. The pasty vent area and cloaca are sometimes plugged with dry feces, unabsorbed yolk sac, containing abnormal yolk material (thin, thick, cheesy, or containing blood). External infection of the navel may be present. Edema of the skin of the ventral body area, septicemia, and dehydration may occur. There are often fibrin deposits on abdominal organs.

Differential diagnosis: Pullorum disease and other *Salmonella* infections in chicks.

Cause, transmission, and epidemiology: Several bacterial species can singly or in combinations cause this disease. However, the commonest cause is *E. coli*, in which case the disease is also called colibacillosis. Other causative organisms can be various species of *Staphylococcus* and *Proteus*.

The causative bacteria are widely distributed in nature and may contaminate water and the soil. *E. coli* in particular is a normal resident of the intestine, and may contaminate feed and water through fecal contamination. It may infect ovaries and through them, chicken embryos before they are hatched. However, contamination of hatching eggs is the common mode of transmission of omphalitis in chicks. Infection occurs at the time of hatching or shortly thereafter, before the navel is healed. Chicks from dirty hatching eggs or eggs with poor quality shells, or newly hatched chicks placed in dirty holding boxes, are most susceptible. Chicks removed prior to complete healing of the navel due to improper temperature and/or humidity are also more susceptible. Eggs that explode in the hatching tray contaminate other eggs in the tray and increase the incidence.

Diagnosis: Clinical signs and post-mortem lesions are strongly suggestive. Diagnosis is confirmed by isolation in profuse culture of the bacterial agent and identification.

Treatment: Most affected birds die in the first few days of life. Prompt treatment with suitable antibacterial agent may be successful for a flock although individual birds may not recover.

Prevention: Control is by prevention through effective hatchery sanitation, hatchery procedures, breeder flock surveillance, and proper preincubation handling of eggs. Mushy chicks should be culled from the hatch and destroyed. If chick mortality exceeds 3 percent, the breeder flocks and egg handling and hatching procedures should be reviewed.

In village chickens, eggs should be cleaned before they are given to hens for brooding.

Recovery: Stunted chicks should be removed and destroyed; surviving chicks should be moved into a cleaner pen and the contaminated one cleaned and disinfected.

Predators

Predation from mongoose, kites, hawks, wild and domestic dogs, and cats can be a large problem for poultry keepers.

Predators are controlled by housing/sheltering poultry at night, keeping a close watch on the birds while scavenging, killing stray dogs/cats/mongoose, chasing away and trapping predators (kites and hawks), and repairing housing, shelters, and coops regularly.

Pullorum

Other names: Bacillary White Diarrhea, BWD.

Clinical signs and lesions: Chicks are weak and may die, with morbidity and mortality rising 4-5 days after hatch. Signs include drowsiness, huddling, droopiness, and lack of appetite, but because of fever, birds tend to drink excessively. Respiratory distress may be seen, but characteristically diarrhea, showing as a white pasty vent area, is a major manifestation of pullorum disease. Sometimes the feces are stained with green bile. Survivors become asymptomatic carriers with localized infection in the ovary.

Post-mortem examination of chicks shows the following changes:

- Pasted white feces in the cloacal area.
- Grey patches (nodules) in the lungs, gizzard wall, heart and intestinal wall.
- Intestinal contents whitish and fluid or cheesy.
- Pin-point hemorrhage or grayish dead patches may be seen in the liver.
- Enlarged spleen.
- Ureters distended with whitish uric acid crystals.

Differential diagnosis: Other conditions that may present similar clinical signs are omphalitis, diseases caused by other *Salmonella* species, and chilling.

Cause, transmission, and epidemiology: *Salmonella pullorum* is pathogenic only to poultry. The organism is spread through infected eggs of layer carrier hens. From this vertical mode of infection hatched chicks can then transmit the infection horizontally to other chicks, through infected secretions or feces. It can spread further through contaminated incubators, hatchers, chick boxes, houses, equipment, poultry by-product feedstuffs, and carrier birds.

Diagnosis: Tentative diagnosis may be made based on history, clinical signs and post-mortem lesions. Definitive diagnosis is based on agglutination tests and isolation and identification of the causative agent, *Salmonella pullorum*.

Treatment: Results of treatment of pullorum disease are poor. Treatment is for flock salvage only. Several sulfonamides, antibiotics, and antibacterials are effective in reducing mortality, but none eradicates the disease from the flock. Survivors remain stunted and act as carriers. Eradication requires destroying the entire flock.

Prevention: In commercial poultry production prevention should be aimed at maintaining parent stock free from infection. These flocks are tested serologically, using the rapid plate agglutination test, confirmed with the tube agglutination test. Both tests employ stained *S. pullorum* antigen, which is commercially available.

Incubator and hatching units should be properly disinfected, and if possible, the hatching operation should be in a pullorum-free area and be quarantined. Poultry farmers should ensure they obtain chicks from pullorum-free hatcheries.

Recovery: The entire flock must be culled and the poultry house properly disinfected and left empty for about a month before re-stocking.

Salmonellosis and Colibacilliosis

Older birds suffer diseases caused by *Salmonellae* and *Escherichia coli*. These conditions are characterized by gastrointestinal disorders, but as the bacterial agents also invade the blood circulatory system, clinical signs consistent with septicemia are observed in affected birds.

Other names: Fowl typhoid and paratyphoid (for salmonellosis).

Clinical signs and lesions: These include diarrhea, inappetence, unthriftiness, respiratory difficulties, drowsiness, and reduced egg-laying. Mortality is usually high.

In colibacillosis in growing broilers, the intestine is bloated and contains watery and mucoid material tinged with blood. The air-sacs are thickened and may have caseous deposits. The heart covering is also thickened. Abdominal organs are swollen and may be covered with a layer of fibrin.

Lesions observed in birds that have died of salmonellosis (fowl typhoid and paratyphoid) are as in pullorum disease. The livers are enlarged and bile-stained, or may have pale, dead patches. The spleen and kidneys are also enlarged, and the blood is thin and watery. The intestinal lining may have raised plaques, and the caecal content is cheesy.

Differential diagnosis: Fowl cholera, Pullorum disease.

Cause, transmission, and epidemiology: *E. coli* disease in growing birds is caused by poultry–pathogenic *E. coli* serotypes, e.g. 078. Fowl typhoid is caused by *Salmonella gallinarum* and paratyphoid by over 200 other species of *Salmonella*. The most common of these are *S. typhimurium, S. enteritidis,* and *S. heidelberg.*

Egg-transmission occurs in fowl typhoid and paratyphoid, but transmission through contaminated water and feed is equally important. Infected birds remain carriers for a long time. The bacteria, having located in the gall bladder, are excreted in the feces regularly to act as source of contamination. Rodents and lizards are also known reservoirs for paratyphoid organisms.

Diagnosis: Bacterial isolation and identification of the organism(s) from the intestinal and visceral organs constitute definitive diagnosis. However, serological tests as for pullorum disease may be convincing evidence of *Salmonella* infection.

Treatment: For *E. coli* disease, antibacterial therapy is normally successful.

Prevention: Hatchery hygiene and farm biosecurity are important in controlling these diseases.

Recovery: Depopulation and disinfection followed by resting of the premises are recommended.

Staphlococcus Infection

Other names: staph septicemia, staph arthritis, bumblefoot.

Clinical signs and lesions: Staphylococcal infections appear in three forms -- septicemia (acute), arthritic (chronic), and bumblefoot. The septicemia form appears similar to fowl cholera in that the birds are listless, without appetite, feverish, and show pain during movement. Black rot may show up in eggs (the organism is passed in the egg). Infected birds pass fetid, watery diarrhea. Many will have swollen joints, and production drops. The arthritic form follows the acute form. Birds show symptoms of lameness and breast blisters, as well as painful movement. Birds are reluctant to walk, preferring to sit rather than stand. Bumblefoot is a localized chronic staph infection of the foot, thought to be caused by puncture injuries. The bird becomes lame from swollen foot pads.

Differential diagnosis: Fowl cholera for septicemic form, infectious tenosynovitis or Marek's disease for the arthritic form and traumatic injury for bumblefoot.

Cause, transmission, and epidemiology: The causative agent, *Staphylococcus aureus,* is soil-borne, and outbreaks in flocks often occur after storms when birds on range drink from stagnant rain pools.

Diagnosis: A definitive diagnosis is based on the clinical appearance of the disease and the recovery of the causative bacteria.

Treatment: Novobiocin can be given in the feed for 5-7 days. Erythromycin and penicillin can be administered in the water for 3-5 days or in the feed for 5 days. Other antibiotics and drugs are only occasionally effective.

Prevention: Remove objects that cause injury. Isolate chronically affected birds. Provide nutritionally balanced feed.

Recovery: After treatment, if the immediate environment of the birds is removed or cleaned, then there will be no residual risk of infection on the premises to new birds.

Stress

Physiological reaction to stress: Stress acts on the hypothalamus resulting in the release of corticotrophin releasing factor (CRF). CRF stimulates the anterior pituitary gland to release adrenocorticotrophin hormone (ACTH), which acts on the adrenal gland medulla to release epinephrine, norepinephrine and adrenorphines. These compounds have an effect on energy metabolism (an increase in lipolysis, glycogenolysis, and insulin

production, and a decrease in gluconeogenesis and glucose utilization), and blood flow (an increase in blood flow to skeletal muscles, heart muscles, brain, skin and gastrointestinal tract as a result of increased heart rate and contraction and increased blood volume). Energy that could have been used in the production of more meat and eggs in these birds ends up being used to respond to stress.

Causes of stress: A husbandry system can be stressful if it exerts abnormal or extreme demands on the bird. Stresses can be mental, physical or a combination of both.

Family poultry are subjected to **mental stress** (pain, fear, anxiety) during a variety of common activities including:

- Catching
- Being chased by predators
- Running away from vehicles
- Tying and wing restraint,
- Bundling in crates ("tenga")
- Being on vehicle roof tops for transportation
- Not having adequate food or water

The mental stress associated with these activities may be caused by visual, auditory, social, and/or physical factors.

Birds are subjected to **physical stress** (environmental: heat, cold, wind, strokes, wounds, etc.) through injuries and by actions such as:

- Being caught by the legs and tied in groups
- Holding upside down
- Loading and unloading
- During transport (vehicle movements, windy conditions, change of environment)

Mixed stressors can come from living conditions such as:

- Environmental changes (fluctuations in temperature, lighting schedules or intensity, rains, flooding),
- Changes in the social order in a flock (new entrants from the market or gifts)
- They can also be caused by disease, such as, parasitism (ecto-, haemo-, and endoparasitism, inapparent infections, and exposure to poisons (household insecticides and herbicides, poisonous plants and poisonous animals)
- Crowding

Consequences of stress:

Immunosuppression: All stimuli that provoke stress will cause immunosuppression. Indigenous poultry are particularly susceptible to stress and this may be reflected in their poor responses to vaccination (Newcastle disease, infectious bursal disease). This has been demonstrated by comparing the responses of commercial birds and indigenous chickens and ducks.

Immunosuppressed birds succumb more easily to both infectious conditions (viral diseases and bacterial diseases) and non-infectious conditions (such as worm infestations) than non-immunosuppressed birds. Some of these birds become carriers of various diseases for other healthy birds around them.

To be optimal, any disease control and prevention measures should consider the stressors in family poultry. The raising, transportation, marketing, and movement of birds to and from various sites need to be done in a way that minimizes stress for the birds. Vaccination programs need to be appropriately targeted for scavenging poultry, taking into account their interactions with wild birds, other livestock, and humans, but also considering the difficulty and in handling and vaccinating them.

Swollen Head Syndrome

Other names: Facial cellulitis, thick head, Dikkop, SHS, TRT, turkey rhinotracheitis

Clinical signs and lesions: In chicks and poults, there is initial sneezing, followed by reddening and swelling of conjunctiva with attendant foamy exudate. Facial swelling extends over the head and down the jaw and wattles. Adult chickens have mild respiratory disease followed by some birds having swollen heads. Other signs include disorientation, twisting of the neck, and a significant decrease in egg production (up to 70%).

Differential diagnosis: A disease closely mimicking SHS can be caused by a mixed infection of respiratory viruses (Newcastle disease virus, avian influenza virus, infectious bronchitis virus) and specific bacteria (*Hemophilus paragallinarum*, *Pasteurella multocida*, *Mycoplasma sp.*).

Cause, transmission, and epidemiology: The disease is caused by a virus classified as a pneumovirus. Chickens and turkeys are the known natural hosts. Experimentally, guinea fowl and pheasants are susceptible but pigeons, ducks, and geese are resistant to the infection. The infection spreads by direct contact with infected birds or indirectly by exposure to infectious material. SHS is present in most countries of the world. Humans can be infected and will develop flu like symptoms if exposed to sick or ill birds.

Diagnosis: A definitive diagnosis can be made through the direct identification or isolation and identification of the disease agent in embryonating chicken or turkey eggs or chicken organ culture from the nasal secretions of clinically ill birds. Serology may also be helpful in confirming exposure when birds have recovered. ***Treatment:*** There is no proven treatment for swollen head syndrome. Antibiotic therapy may be helpful against the bacterial component.

Prevention: A commercial vaccine is available but not widely distributed. Prevention is dependent on a comprehensive biosecurity program.

Recovery: A premises with infected birds can be rid of the organism through cleaning and sanitation of all facilities and equipment in contact with birds and the removal of infectious material on the farm like manure. New introductions must be prevented with strict biosecurity if recovery is to be complete.

Ulcerative Enteritis

Other names: Quail disease.

Clinical signs and lesions: In chickens, signs are less dramatic than in quail. Acute signs are extreme depression and reduction in feed consumption. Affected birds sit humped with eyes closed. Other signs included emaciation, watery droppings streaked with urates, and dull ruffled feathers. Accumulated mortality can reach 50 percent if the flock is not treated. At necropsy, punctate ulcers are typical and may be visible from the mucosal surface of the gut. Any section of the intestine may be involved. The liver will have circumscribed areas of necrosis and hemorrhage that with time, will become depressed. The spleen will be enlarged, congested and hemorrhagic.

Differential diagnosis: A differential diagnosis of ulcerative enteritis will include coccidiosis, histomoniasis and necrotic enteritis. Complicating the differentiation of ulcerative enteritis is the fact that coccidiosis and necrotic enteritis may be predisposing on concurrent infections in affected birds or flocks.

Cause, transmission, and epidemiology: The causative agent is the bacterium *Clostridium colinum.* Chickens, turkeys, quail and other species are occasionally clinically affected. Birds become infected by direct contact with carrier birds, infected droppings or contaminated pens, feed and water. Bacteria are passed in the droppings of sick and carrier birds. Infection can be spread mechanically on shoes, feed bags, equipment, and from contamination by rodents and pets.

Diagnosis: A diagnosis of ulcerative enteritis is indicated by the presence of typical button ulcers in the intestine with concurrent target lesions in the liver. Confirmation is based on the isolation of the causative agent, *C. colinum*. However, this can be very difficult because the organism is fastidious and difficult to grow in culture. In these cases, a diagnosis will be based on the presence of typical gross lesions.

Treatment: Bacitracin and Neomycin can be used singly or in combination. Other antibiotics and drugs such as tetracyclines, penicillin, Lincomycin, and Virginomycin are also effective. Consult a veterinarian for dose, route, and duration of treatment.

Prevention: Ulcerative enteritis is difficult to prevent in quail. When quail have access to their own droppings, this disease commonly occurs. To eradicate, depopulate stock, thoroughly clean and disinfect, and start over with young, clean stock.

Recovery: *Clostridium colinum* is a spore-forming organism and infectious spores will remain in the soil after an outbreak. Over time, the spores will be dispersed resulting in lowered exposure for naïve birds but risk will not disappear. Controlling predisposing factors, like immunosuppressive diseases and internal parasites will make birds more resistant. In highly susceptible species, like quail, preventative medication may be the only means of recovery.

Urolithiasis

Clinical signs and lesions: This is primarily observed in laying flocks and characterized by severe atrophy of one or both kidneys, distended ureters with uroliths, and varying degrees of renal and visceral gout. It is associated with increased mortality and decreased egg production. Clinical signs include:

- Mortality of up to 50%
- Laying chickens die suddenly although in good condition and in production
- Reduced muscle mass, small pale combs and white pasty exudate on the peri-cloacal feathers
- Atrophied kidneys and dilated ureters, often accompanied by diffuse visceral urate deposits
- Kidney atrophy is often more severe in the anterior lobes and is usually unilateral, but may be bilateral
- The surviving ipsilateral or contralateral lobes may be enlarged
- Ureters from atrophied lobes are dilated, full of clear mucus, and often contain white irregular concretions or uroliths
- Uroliths are compact masses of microcrystalline to fine pleomorphic crystals of calcium/sodium urates, with random substitution of magnesium and potassium for the calcium and sodium, respectively

Microscopic lesions include:

- Dilated ureter branches and tubules
- Tubular degeneration and loss of tubules
- Cellular casts, urate crystals and varying degrees of fibrosis
- Minor focal cortical tubular necrosis
- Some eosinophilic globules in glomeruli and interstitial infiltration of heterophils and lymphocytes

Differential diagnosis: Increased mortality and decreased egg production can be associated with any number of diseases. Urolithiasis will generally mimic the appearance of, and also can be caused by management problems like a lack of water or feed.

Cause, transmission, and epidemiology: Physiologic impact of urolithiasis on kidney damage is the result of reduced renal mass, rather than inappropriate renal handling of minerals and electrolytes. Uroliths may cause sudden death by plugging ureters, which is probably secondary to kidney damage. It can also be secondary to infectious bronchitis, or due to excess dietary calcium with low phosphorous levels, water deprivation, and some nephrotoxic mycotoxins.

Diagnosis: Diagnosis is made by gross necropsy. However, a diagnosis of the cause of the renal disease will depend on what that cause is. Feed analysis and testing for infectious bronchitis viruses should be standard in cases of urolithiasis.

Treatment: There is no treatment for this disease syndrome.

Prevention: Urolithiasis is prevented by feeding a correctly balanced diet, providing adequate water for chickens and preventing infectious bronchitis through a combination of vaccination and biosecurity.

Recovery: Affected individuals will not recover but once the cause of the kidney damage is corrected, then no additional individuals will develop the condition. There is no residual risk to new birds placed on the premises.

Zygomycosis

Other names: Mucormycosis and phycomycosis

Clinical signs and lesions: There are both local and systemic types of infections. The clinical syndrome depends on the organ or system infected. There may be incoordination, photophobia and paralysis. Nodular airsacculitis with involvement of intercostal muscles, multiple nodules in the lungs, ventriculitis, and proventriculitis (ostriches) may be seen. The lesions are pyogranulomatous (with necrotic center) with numerous multinucleated giant cells. Necrosis and angioinvasion are near-constant features.

Differential diagnosis: The symptoms of zygomycosis may be non-specific depending on the system affected. Lung infections and systemic fungal infections such as aspergillosis, cryptococcosis, histoplasmosis and dactylariosis; mycobacteriosis should be considered.

Cause, transmission, and epidemiology: The disease is caused by fungi belonging to genera *Mucor, Rhizopus, Absidia, Rhizomucor,* and *Mortierella*. They are acquired from environmental sources (soil, manure, and rotting vegetation), and are not contagious.

Diagnosis: Clinical signs of disease, lesions at necropsy and the epidemiology of the case can be indicative of zygomycosis or other fungal disease. Confirmation of the specific disease agent is by microscopic examination of histological lesions or culture.

Treatment: There is no satisfactory treatment.

Prevention: Good sanitation and hygiene.

Recovery: Cases will re-occur if fungi can grow in the birds' environment. If, however, the cause of the contamination is corrected and/or the source removed, there is no residual risk to flocks.

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APPENDIX A: Poultry Disease Diagnosis Decision-Tree