#### **Backyard Poultry Medicine**

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Backyard poultry represents a growing segment of the companion animal veterinary market. This lecture will cover the basics of backyard poultry husbandry and medicine.

Keywords: poultry, FARAD, chicken, zoonotic, salmonella, E. coli

#### Antibiotic use in poultry

Antibiotics used in the treatment of bacterial diseases in poultry that produce eggs or meat for human consumption needs careful consideration. Residues in human and animal food products has been a long-debated issue in the veterinary profession. These residues can potentially result in antibiotic resistant strains of bacteria that cause human disease.

Regulations concerning the use of antibiotics in food animals are becoming increasingly restrictive. Always ensure that you are aware of the latest regulations prior to issuing a prescription. For more information on drugs approved for poultry, and withdrawal times go to the FARAD website at <a href="http://www.farad.org/">http://www.farad.org/</a>

## FARAD Food Animal Residue Avoidance Databank 1-888-US-FARAD 1-888-873-2723 www.farad.org http://www.farad.org/vetgram/Sp/SpeciesList.asp

Reportable disease USDA Avian influenza (LPAI and HPAI) Newcastle disease (VVND) FLDAR AI and ND Infectious laryngotracheitis Infectious bronchitis Salmonella pullorum Mycoplasmosis Tuberculosis

**Zoonotic disease** Salmonellosis Colibacillosis (E. coli) Avian tuberculosis Avian influenza Newcastle disease Staph, Strep, Enterococcus, C. perfringens A,C Campylobacteriosis Listeriosis Strongyloides avium Cryptococcus, histoplasma Cryptosporidium spp Toxoplasma gondii Microsporum gallinae EEE, WEE, WNV Erysipelas Fowl mites Chlamydia Pasteurellosis Allergic alveolitis

# Mail-order hatcheries Salmonellosis in humans

2004-2011 Salmonella enterica serotype Montevideo 316 cases 43 states Median age of patient = 4 years old 50 million live poultry sold annually through mail-order hatcheries NEJM 366; 22 May 31, 2012

Chickens as pets?? **Growing trend** Particularly in suburban areas Dependent on city zoning/ordinances Roosters sometimes banned - crowing Entertainment/education/exhibition Fancy breeds Eggs/meat Natural pest control Alarm system Chickens are cool! Housing requirements **Predator proof!!!** Chicken coop Nest boxes Perches, ramps Run Waterers, feeders Hanging

Cups

Dust bath

#### Manufactured coops popular

Housing requirements 2-3.5 square feet per chicken 10 square inches per chick up to 2 wks At least 1' square by 4-8 wks

#### Chicks initially need 95F

Decrease by 5F weekly (raise heat lamp 3"/wk) until room temperature

# Nutrition

#### **Commercial diets**

20-25% protein during breeding Maintenance diet less than 20% protein Layer ration can be fed to juveniles/adults Pellet or crumble version Make sure non-medicated Provide additional calcium for laying hens Starter ration for young chicks

## Add fresh greens and allow foraging

Grass or corn silage/grain in small quantities **Fresh water always** 

+/- grit, can crush oyster shell

## **Diet for layers**

0-6 wks feed starter (18-20% protein) 6-14 wks feed grower (16-18% protein) 14-20 wks feed developer (14-16% protein) 20-24 wks start layer ration (16-18% protein)

#### Factors affecting egg production Noninfectious causes include:

Aging hens -> 2-3 years Improper nutrition Toxicoses Management mistakes Inadequate day length (> 14 hours) High ambient temperatures and other causes of stress Predators

#### Infectious factors affecting egg production in backyard chicken flocks Infectious causes include:

Infectious bronchitis Egg drop syndrome Fowl pox Coccidiosis Newcastle disease Avian influenza

#### Mycoplasma gallisepticum

#### Additives

#### Coccidiostatic agents in commercial diets

Halofuginone toxic to common pheasant, guinea fowl, and common partridge Monensin toxic to guineafowl

## **Antimicrobial agents**

Can be dangerous for species that depend on cecal flora for digestion Grouse

#### NPIP

## National Poultry Improvement Plan

Established in the early 1930s Improvement of poultry and poultry products throughout the country Initiated to eliminate Pullorum disease Salmonella pullorum 80% mortality in baby poultry Later addressed Salmonella typhoid, Salmonella enteritidis, Mycoplasma gallisepticum, Mycoplasma synoviae, Mycoplasma meleagridis, and Avian Influenza

#### Small backyard flock biosecurity http://youtu.be/HLF fx13qgQ

10-minute video on sanitation and biosecurity

#### http://www.mass.gov/agr/animalhealth/poultry/diagnostic\_testing.htm

MA NPIP info and contacts

#### **Common chicken breeds**

#### Anatomy and physiology

#### Vascularized thickening of skin on ventral thoracic region

Brooding spot

Feathers temporarily lost to allow heat transfer to eggs

#### Preen gland

Not present in tail-less breeds

#### Comb and wattles

Size influenced by hormones (more prominent in rooster) Paired cheek or earlobes Color of earlobe related to color of eggshell

#### Anatomy and physiology

#### Spurs

Osseous eminence from tarsometatarsus Covered by keratinized epidermis Poorly developed in hens In roosters can cause injury to rivals, hens, clients

#### Cockfighting

#### First documented in 1646

Book on subject from early 1600s

#### Specially bred birds

Conditioned for stamina/strength Comb and wattles cut off Allowed to fight after 2 years of age Birds equipped with metal spurs or knives at location of partially removed spur Illegal in the US

#### Plumage

Most gallinaceous birds molt naturally once a year After breeding Maintain ability to fly Commercial US chickens molted on scheduled basis Improve level of egg production and quality of eggshell Withdrawal of feed for 7-14 days Not practiced in the UK or Canada

#### 'Free range' vs humane

## 'Certified humane raised and handled'

http://www.certifiedhumane.org/index.php?page=fact-sheets

# **Integumentary diseases**

#### Cannibalism

Vent picking, feather pulling, toe picking, head picking, egg eating Overcrowding, incorrect feeding, poor housing

#### Pododermatitis

Obesity, abrasive flooring, poor diet predisposes

#### **Bacterial diseases**

#### Escherichia coli

#### **Colibacillosis**

Colibacillosis (Air sacculitis, Omphalitis, Salpingitis) in poultry is a disease which primarily affects chickens, turkeys and ducks. Most of the outbreaks are associate with poor sanitation, inadequate nutrition or primary infections with viral or mycoplasma agents. Several disease entities have been associated with coliform infections. These include: air sacculitis, omphalitis (yolk sac infection) in baby chicks, septicemia in ducks (new duck syndrome) and chickens, enteritis, salpingitis, granulomatous lesions (coligranulomas sometimes referred to as Hjarre's disease), arthritis and synovitis, panophthalmitis and pericarditis. The latter two conditions are complications associated with septicemia. Serotypes commonly associated with poultry disease are O1-K1, O2-K1 and O78-K80. Under some circumstances *E. coli* can be passed through the pore in the egg shell to infect the embryo during the incubation period Some of the more important of these conditions are discussed below.

#### Air sacculitis

This condition is most common in 5-6 week old broiler chickens and is usually the result of an underlying mycoplasma infection of the air sacs. Newcastle virus, infectious bronchitis virus and laryngotracheitis virus have also been linked to this condition. The abdominal air sacs are thickened with a loss of normal transparency with flecks of yellow material on the inner walls. Fibrinous exudate is usually also evident on the surface of the liver and pericardial sac. This is really the most common manifestation of the disease known as chronic respiratory disease or CRD.

#### <u>Omphalitis</u>

*E. coli* is often isolate in pure culture form the unabsorbed yolk sac of recently hatched chicks which clinically show depression, and variable mortality during the first few days of life. On gross examination the yolk sac contains a foul-smelling pasty, yellow/brown yolk material. In additions the naval is swollen with moist exudate on the surrounding skin surface. Other common bacterial organisms have been associated with omphalitis including Proteus, Aerobacter, Salmonella, Staphylococcus, Streptococcus, and Bacillus.

# **Salpingitis**

This is a relatively common condition in older laying hens. It is thought to result from and ascending coliform infection of the lower oviduct which subsequently prevents normal motility and resultant impaction with partially calcified yolks. The oviduct is markedly distended with yolk material which sometimes has a fetid odor if sepsis is extensive. Affected birds can live for long periods (6 months and longer).

## Pasteurellosis (fowl cholera)

(Fowl Cholera, Avian hemorrhagic septicemia)

Fowl cholera is an acute, septicemic disease caused by Pasteurella multocida that effects chickens, turkeys, ducks, geese and many species of waterfowl. The disease occurs worldwide and is relatively common. A similar disease has been described in domestic ducks and is known as New Duck Disease, the causative organism is P. anatipestifer.

Fowl cholera almost always seen as an acute, rapidly fatal disease in poultry with a mortality that approaches 100%. The disease is more common in the late summer and fall with mature birds more susceptible than chicks. Recovered birds harbor the organism in the upper respiratory tract and nasopharynx. Wild birds and many mammalian species (cats and cattle) harbor the organism in the nasopharyngeal region. These species are thought to spread the organism to susceptible birds. Birds that die in acute septicemia have the organism in all tissues and cannibalism is a common means of spreading an infection within the flock.

## Clinical signs

Acute fowl cholera is dramatic.

Sudden deaths: laying hens are often found dead in the nest box, wild birds in large numbers are found in a given area.

Toxicity is often suspected in outbreaks of acute fowl cholera.

Sick birds are anorexic and depressed.

Cyanosis of comb with respiratory rales and nasal and oral mucus discharge.

White, watery or green-mucoid diarrhea is sometimes evident.

Chronic fowl cholera results in birds with swollen joints, wattles, foot pad and tendon sheaths. Cheesy exudate may also be present in the conjunctival sac, and infraorbital sinuses.

## Diagnosis

Gross lesions may be absent in birds that die peracutely. The most common findings are petechia or ecchymoses on the pericardium and serosal surfaces of the gizzard, small intestine, and abdominal fat. Numerous pinpoint grey/white foci may be present on cut surfaces of the liver. The lungs may be dark red with wet cut surfaces. A gram stained blood smear or imprint of the liver usually reveals numerous, gramnegative, bipolar bacilli characteristic of *P. multocida*. The organism is easily grown on blood agar.

## Differential Diagnosis

Erysipelas and acute colibacillosis in turkeys are clinically similar to fowl cholera. <u>Prevention</u>

Bactrins are available but are not always effective. A live oral vaccine is available for chickens and turkeys using the CU strain of *P. multocida*. Many antibiotics and sulfa drugs are available to treat fowl cholera. Culture and sensitivity tests are important since widespread resistance is common with this organism.

# Mycoplasmosis

## Mycoplasmal Diseases of Poultry

Avian mycoplasmosis represents the most ubiquitous, economically devastating respiratory poultry disease in the United States. A conservative estimate of the annual loss to the commercial poultry industry would be well over 100 million dollars. Similar to mycoplasma infections in mammalian species, once established in a group of animals it is impossible to eradicate the infection unless the entire population is destroyed. Mycoplasma organisms are easily spread by aerosols and it is also possible to transmit the organism in fertilized egg. Although of high importance in poultry, mycoplasma infections in pet bird species are rarely diagnosed.

Respiratory mycoplasma infections in chickens and turkeys result in clinical signs and gross lesions that are remarkably similar to those caused by C. psittaci however avian mycoplasma infections are of no consequence to humans.

Mycoplasma Diseases of Poultry		
Mycoplasma gallisepticum	Chronic respiratory disease (chickens)	
	Infectious choryza (chickens)	
	Infectious sinusitis (turkeys)	
Mycoplasma meleagridis	CRD (turkeys only!)	
Mycoplasma synoviae	Synovitis (Enlarged hock disease)	
	Tenosynovitis?	

## Mycoplasma gallisepticum

[MG, Chronic respiratory disease, CRD, Air sacculitis, Infectious sinusitis of turkeys, PPLO infection] This organism is widespread in the bird population. In addition to the chicken and turkey, natural infections have been documented in pheasants, peafowl, Coturnix and bob white quail, wild turkeys and housefinches. One source claims to have isolated the organism from a yellow-naped Amazon parrot.

## Clinical Signs

# Adult chickens

Signs are often non-specific consisting of decreased feed intake and a loss of normal growth rate. Laying chickens will show a variable drop in egg production. Signs of respiratory disease include: ocular and nasal discharge and mild dyspnea.

# **Turkeys**

Swelling of the infraorbital sinuses is a common finding in addition to the other signs usually seen in chickens. Nasal exudate is evident on the face, and wings. In general, *M. gallisepticum* produces a more severe disease in turkeys.

## Diagnosis

Air sacs are almost always affected but may vary in appearance from a mild cloudiness to severe thickening with floccular tan/yellow exudate on the inner lining. In severe cases there is extensive caseous material within the air sacs. Mucoid or mucopurulent exudate is often present in the nasal sinuses, choanae, and trachea. The pericardial sac is often thickened and cloudy. Turkeys may only show swelling

of the infraorbital sinuses. However, they may also show air sac and upper airway lesions similar to chickens. A positive diagnosis of MG is usually made with a plate or tube agglutination test. Since this disease is almost always chronic, serum samples from several birds in an involved flock are used for this test. Isolation and identification of the organism is also possible.

## **Differential Diagnosis**

Chlamydiosis, fowl cholera, colibacillosis, influenza and aspergillosis can resemble MG infections in the turkey and chicken.

## Prevention

The most practical means of controlling *M. gallisepticum* is through the depopulation of infected flocks with subsequent effort to use stock derived for mycoplasma free breeders. This is often economically unfeasible. A method of immunization by controlled exposure has been practiced in some farms. Vaccine strains of mycoplasma are administered in the drinking water. There are no antibiotics that can be administered to chickens producing meat or eggs for human consumption that will eliminate *M. gallisepticum* or prevent the transmission of this organism in fertilized eggs.

# Mycoplasma meleagridis (MM)

MM is a mycoplasma disease which only infects domestic turkeys. All age groups are affected. The pathogenesis and transmission are similar to that described above for *M. gallisepticum*. Although aerosols and egg transmission are the most common means of infection MM has also been shown to be transmissible to humans who handle poults during sex determination by the cloacal examination method. Semen has also been show to harbor the organism and MM can be venereally transmitted.

## Clinical Signs

Young poults will often show a mild sinusitis however respiratory signs are rarely observed.

## Diagnosis

Young poults show thickened air sacs, often with small amounts of flocculent, yellow material on the inner lining. Sinusitis and synovitis have also been seen in adults with MM infection. A plate agglutination test can be used to serologically confirm an outbreak.

## **Differential Diagnosis**

MG and chlamydial infection must be considered in a provisional diagnosis of MM

## Prevention

This disease is controlled in a similar manner as MG. However, the MM organism is somewhat more sensitive to antibiotic therapy and tylosin and tetracycline have been used to control and limit the signs of infection in poults. Lincomycin (spectinomysin) at the rate of 2gm/gal of drinking water has been used for the first 5-10 days of life to reduce the incidence of air sacculitis.

## Mycoplasma synoviae

[Synovitis, Enlarged hock disease, MS, Tenosynovitis?]

MS is not a respiratory mycoplasma infection but is included here for organizational purposes only. The disease is characterized by swollen hocks with exudates in the hock joints and Achilles tendon sheaths. Chickens and turkeys 4-12 weeks of age are most commonly affected. Guinea fowl are also susceptible. Infection is by aerosols and infected fertile eggs.

## **Clinical Signs**

Lameness is the usual sign. Birds will show swollen hocks and foot pads.

#### Diagnosis

The hock joints and tendon sheaths contain excessive, creamy to caseous, gray/yellow exudate in the joints and tendon sheaths. The sternal bursa may be filled with caseous material as a result of the birds resting on the keel and being unable to walk. This lesion is sometimes referred to as a "breast blister". Abscesses are sometime seen in the pads on the plantar surfaces of the feet. This lesion is sometime called "bumblefoot".

#### **Bordetellosis (turkey coryza)**

Occurs in backyard turkeys Gram-negative bacteria Bordetella avium

<u>Signs</u> Snicking (sneezing) Tracheal rales Labored breathing Foaming exudate in eye

<u>Diagnosis</u> Tracheal swab culture

Serology

# Staphylococcosis

*Staphylococcus aureus* Suboptimal environmental conditions

#### Sites of infection

Leg joints, foot pads (pododermatitis), tendon sheaths

#### Risk factors

Chickens and turkeys on wet litter Heavy manure buildup (ammonia burns) Wire cages with rough surfaces

#### **Fungal diseases**

#### Aspergillosis

Aspergillus fumigatus Rarely primary pathogen Can occur secondary to other respiratory disease Fowl cholera

#### **Candidiasis**

*Candida albicans* Secondary to prolonged antibiotic use Poultry raised in poor conditions

Viral disease Marek's disease/lymphoid leukosis Marek's Disease DNA - herpesvirus "T cell" lymphoma

Affects birds before the onset of sexual maturity

Virus can cause inflammatory as well as neoplastic lesions (gonadal lymphomas are common) Two types of lesions are seen in chickens with MD. Birds less than 6 months of age may develop visceral lymphomas. Birds of any age may also show non-suppurative encephalitis and neuritis with clinical signs of paralysis. Neurologic manifestation of MD in the absence of neoplasia is sometimes referred to as classical Marek's disease. Infectious virus is shed from feather follicle epithelial cells. Antigenically related, non-neoplastic herpes virus isolated from turkeys (HVT) has been used to protect susceptible birds from MD by vaccination at one day of age.

Clinical signs

Neuritis and nonsuppurative encephalitis Transient paralysis Ocular lymphomatosis ("gray eye") Lymphomatosis (lymphosarcoma) Neurolymphomatosis (a combination of inflammatory and neoplastic forms)

# Infectious bronchitis

Coronavirus <u>Signs</u> Respiratory signs Gasping, coughing, tracheal rales, nasal discharge \*\*Egg shell quality issues Broken or misshapen eggs, thin shells, wrinkled, watery egg whites

Diagnoses Serology

Prevention Vaccination Serotype depends on area Serotypes: Connecticut, Massachusetts, Arkansas, etc.

<u>Treatment</u> Prevent secondary bacterial infections

## Infectious laryngotracheitis

Laryngotracheitis is an acute, highly contagious, respiratory disease of chickens caused by a herpes virus. The disease is principally seen in chickens and a few rare reports describe an LT-like disease in pheasants and peafowl. Amazon tracheitis virus is a related herpes virus that occasionally causes similar disease in psittacines.

## Clinical Signs

Nasal discharge, "coughing", gasping, dyspnea and expectoration of blood tinged mucus. Morbidity is high but mortality rarely exceeds 20%.

## Diagnosis

The diagnosis of LT usually requires laboratory assistance as other respiratory pathogens of poultry can cause similar clinical signs and lesions. A positive diagnosis can sometimes be made on the basis of gross and microscopic lesions in cases of severe acute LT with high mortality. Gross lesions are confined to the

tracheal mucosa and include mucoid and hemorrhagic exudate in addition to fibrinous and heterophil inflammation. Intranuclear, eosinophilic inclusion bodies are usually seen in sloughed tracheal epithelial cells. These inclusions may be difficult to identify forty-eight hours after first signs are observed since the damaged tracheal mucosa has been sloughed and is in are generating phase.

## **Differential Diagnosis**

LT is rarely confused with other common poultry diseases. However, remember other causes of mild upper respiratory disease: Infectious Bronchitis, mesogenic ND, low pathogenic avian influenza, mycoplasma and chlamydia.

#### Prevention

Many modified live vaccines are available. They are usually administered by eye drop, aerosol or in the drinking water

#### Poxvirus

Clinical Signs

On non-feathered skin

Slow spreading with indolent clinical course.

Birds with fowl pox behave normally other than showing cutaneous lesions.

Raised, proliferative nodular lesions typically appear on non-feathered parts of the body: comb, wattles, shanks and feet.

In wet pox, opaque plaques or nodules form on mucus membranes of the mouth, esophagus and trachea. This may result in death secondary to anorexia.

#### Diagnosis

Microscopic skin lesions are proliferative with swelling and hydropic changes of the epithelial layer. Intracytoplasmic, eosinophilic inclusions (Bollinger bodies) are apparent on microscopic examination. Fowl pox inclusions will stain positively with histologic stains used for lipids (Sudan IV, Oil red 0: this characteristic is only seen with avian poxviruses).

#### **Differential Diagnosis**

Vitamin A deficiency can resemble wet pox.

## Prevention

Vaccines are available for chickens, turkeys and pigeons. Fowlpox vaccine is commonly applied by the wing-web method to 4-week-old chickens and to pullets about 1—2 months before egg production is expected to start. It is also used to revaccinate chickens held for the second year of egg production. The vaccine is not to be used on hens while they are laying.

# **Parasitic diseases**

Lice

Common parasites in chickens/turkeys 1-6mm

Straw-colored, fast-moving

Rough feathering

Often found clustered at base of feather shaft

Sometimes felt on ventrum and near cloaca

## Treatment

Permectrin II, Prozap Garden and Poultry Dust

#### Mites

<u>Chicken mite</u> <u>Dermanyssus galinae</u> <u>Northern fowl mite</u> <u>Ornithonyssus sylviarum</u> <u>Tropical fowl mite</u> <u>Ornithonyssus bursa</u> Black, slow-moving, 0.5mm <u>Treatment</u> Similar to lice (ivermectin may be more effective), retreat in 2 weeks Treat housing area

#### Scaly-leg mite

Knemidocoptes mutans

Lesions on comb, wattles Upturned leg scales and crusts

#### Diagnosis

Scrapings of leg lesions

#### **Treatment**

Treat mites Petroleum jelly to soften leg scales

# Roundworms

## Ascaridia galli

Roundworm of backyard poultry and turkeys Weight loss when heavy worm burden Worms can be found in eggs

#### Diagnosis

Fecal floatation

## Treatment

Piperazine (not labeled for use in laying hens)

#### Cestodes

 Tapeworms

 Can be found in poultry

 Usually not associated with signs

 May be found on necropsy

 Occasionally detected on fecal floatation

#### Coccidiosis

#### *Eimeria* sp.

Coccidiosis is a common, protozoal disease of poultry and other avian species characterized by enteritis and diarrhea. Coccidiosis manifests an enteric disease in most instances, but it should be noted that renal coccidiosis in water fowl is caused by *Eimeria truncata*. Chickens are by far the most common poultry species affected with coccidiosis. Domestic turkeys are also susceptible to infection, but the disease is less severe in this species. Intestinal coccidiosis has been reported in geese, ducks, guinea fowl, pigeons, pheasants, quail, and chukar partridge.

Coccidiosis is usually a problem in young chickens, but clinical disease can occur in older birds, especially when virulent strains of coccidia are involved. Birds are infected through ingestion of sporulated oocysts in feed, water, litter and soil. Conditions of warmth and high humidity facilitate survival and transmission of the oocysts in litter. The various species of coccidia can be identified by microscopic features of oocysts in addition to the location of shizonts and gametocytes in the GI tract. Infection with one species of coccidia stimulates immunity to that species only and the host will remain susceptible to other species of coccidia. Birds are often simultaneously infected with more than one species. Clinical disease occurs when susceptible chickens ingest massive numbers of oocysts. This occurs when conditions for sporulation of oocysts are ideal, e.g., the litter is wet and temperatures are warm. If the number of ingested oocysts are low, birds will develop immunity without clinical signs of disease. Chickens maintain immunity to coccidia with repeated exposure. Immune chickens are asymptomatic and shed oocysts for long periods (coccidiasis).

# Clinical Signs of coccidiosis

Signs in chickens vary depending on the species of coccidia; less pathogenic species produce few or no signs. Pathogenic species cause diarrhea which may be mucoid or bloody. Dehydration often accompanies severe diarrhea. Anemia, listlessness, weakness, retraction of the head and neck and somnolence follow.

Laying hens will have a drop in egg production with blanching of the yellow skin pigment. Growing birds, especially broilers, cease to grow satisfactorily.

Morbidity and mortality vary - both may be very high.

Coccidiosis in turkeys resembles the disease in chickens but diarrhea is seldom bloody and poults over 8 weeks old are seldom affected.

# Diagnosis

The diagnosis of coccidiosis is made on the basis of clinical signs and gross lesions. Knowing the location in the digestive where oocysts, sporozoites, merozoites and schizonts are found is useful in determining the species of coccidia involved in the outbreak. Coccidiosis must be differentiated from coccidiasis (subclinical infection in an immune bird). Additionally, oocysts in the mucosal scrapings may be from non-pathogenic or mildly pathogenic species of coccidia which are of no diagnostic significance. It is also important to remember that *E. necatrix* does not form oocysts at the site where the most severe lesions occur. The pathogenic stage of coccidiosis precedes oocyst formation and schizonts may be overlooked in the mucosal scrapings from birds with severe gross lesions.

The most important species of coccidia produce lesions in specific regions of the GI tract.

Proximal third of the small intestine - Eimeria acervulina

Gray/white striations are often visible on the mucosal surface but may go unrecognized if they coalesce. Oocysts in mucosal scrapings are moderate in size and ovoid shaped. This type of coccidiosis occurs rather frequently in older birds. *E. acervulina* is a moderately severe pathogen. In severe cases the lesions can extend into more distal portions of the GI tract. Enteritis can be mild to severe and may lead to a thickening of the intestinal mucosa. Frequently, other species of coccidia will be present, complicating the diagnosis.

This location is also favored by 4 non-pathogenic species.

Middle third of the small intestine - Eimeria necatrix

The middle one-third of the intestine is often markedly dilated and thickened. Yellow/white foci and petechia are visible through the serosa of the unopened gut. Lesions are seen in the middle third of the intestine but extend throughout the intestinal tract in severe cases. Enteritis is characterized by congestion, hemorrhage, necrosis and bloody feces. Oocysts develop in the ceca, and these may be minimal. Mortality may precede the appearance of oocysts in the feces. *E. necatrix* is a severe pathogen and often causes high mortality

Distal third of the small intestine - Eimeria tenella

Typhlitis with occasional involvement of the adjacent areas of the intestine is the principle lesion. In early cases blood is apparent in the feces and cecal lumena. Later, cheesy cecal cores are found. Large schizonts are found in cecal mucosal scrapings. E. tenella is a severe pathogen producing high mortality in young chickens.

## **Differential Diagnosis**

Other intestinal parasites including Capillaria sp. produce enteric signs that resemble coccidiosis. Ulcerative enteritis and salmonellosis also mimic coccidiosis.

# Prevention

A wide range of anti-coccidial drugs are available. Some of the more popular products include Amprolium, Monensin, Clopidol, Buquinolate and Robonidine.

## Histomoniasis

# Histomonas meleagridis

## (Blackhead, Enterohepatitis)

Histomoniasis is a protozoal disease of turkeys, pheasants, peafowl, grouse, quail and chickens caused by Histomonas meleagridis. Turkey poults three month of age and younger are the most susceptible. In young poults morbidity and mortality is high and often approaches100%; older birds are more resistant. Birds are infected through ingestion of protozoa contaminated feces. Infected ova of the cecal worm Heterakis gallinarum and earthworms containing larva of infected cecal worms containing the protozoal organisms can also serve as a less common source of infection. Lesions of histomoniasis are prominent in the liver and cecum. Chickens have low susceptibility to histomoniasis.

## **Clinical Signs**

Signs appears 7-12 days after exposure. Initially there is listlessness, moderate anorexia, drooping wings and sulfur colored feces. Comb, wattles and snood may be cyanotic.

Mortality in poults can approach 100%.

Emaciation is common in chronic cases; this is more likely to occur in older birds.

Chickens with histomoniasis may have hematochezia.

## Diagnosis

Gross lesions consist of bilateral enlargement of the ceca with thickened cecal walls and ulceration of cecal mucosa. The cecal lumen contains yellow/gray/green, necrotic, laminated cores. Peritonitis may be present if the cecal wall perforates. In quail, the cecal lesions may not be present, even though mortality is high. The natural surfaces of the liver have 1-2 cm diameter, round, depressed, yellow/gray to red, targetlike, multifocal and coalescing lesions. Protozoal organisms can sometimes be found in cecal mucosal scrapings or from liver tissue imprints made from the margin of hepatic lesions. Histologic sections of the liver lesion reveal numerous histomonads.

## **Differential Diagnosis**

Clinical signs and some of the gross lesions of histomoniasis are similar to those of cecal coccidiosis.

## Prevention

Histomoniasis can be prevented by adding antihistomonal drugs to the ration. Only Ipropan (irpronidazole) and Histostat (nitarsone) are approved for use in poultry. Directions for use provided with for each product should be followed carefully.

\*\*\*House chickens separate from turkeys\*\*\*

## **VVND and HPAI**

## Clinical presentation very similar High mortality – up to 100% in susceptible flocks Sudden death with no clinical signs Diarrhea (viscerotropic form) Neurologic signs (neurotropic form) If in doubt report the case immediately to your State Vet Do NOT perform a necropsy without appropriate facilities and equipment!

#### Paramyxovirus, PMV-1, or Newcastle disease

ND virus (NDV) is a ubiquitous virus that can be isolated from many species of wild and domesticated birds. In addition to chickens - turkeys, peafowl, guinea fowl, pheasants, quail and pigeons are naturally susceptible. A wide range of virulence is encountered in different strains of virus. Identification is based on the antigenic make up of neuraminidase and hemagglutinin receptors on the envelope. NDV cause a wide spectrum of disease in domestic fowl and pet and wild birds.

Lentogenic, mesogenic and velogenic strains of NDV are described in the literature. These terms indicate differences in viral pathogenicity using the chicken as the index species. Lentogenic NDV causes clinically non-apparent disease in chickens but this same virus may kill canaries. Conversely, velogenic NDV causes relatively mild disease in Amazon parrots but this same virus would produce lethal disease in a flock of egg laying hens with 100% mortality. Mesogenic NDV is the most common form of virus isolated from commercial poultry flocks. Many outbreaks present with mild or atypical lesions. This is the result of the common practice of vaccinating commercial poultry with modified live NDV vaccine. The vaccine virus sometimes reverts to mildly virulent virus that may produce clinical disease. Commercial NDV vaccines are usually administered in the drinking water or by aerosol.

Velogenic NDV (VVND) is the most virulent form of the virus. It has been documented to be introduced onto the U.S. through importation of wild psittacine species subsequently sold as pets. This disease is still considered exotic to the US and is REPORTABLE to the USDA <u>APHIS</u>.

Pigeon paramyxovirus (PMV-1 pigeon, PPV) was first seen in Europe and Great Britain in the early 1980s. It has been observed in pigeons in the U.S. since 1984. Paramyxovirus is very common in pigeons, both wild and domestic. Show and racing pigeons are the most common species infected with pigeon paramyxovirus. This form of the virus is also infectious to chickens, Common Blackbirds and House Sparrows. Serologic testing can be performed to confirm the pigeon paramyxovirus. Several vaccines are available to assist in controlling the pigeon disease in captive flocks.

Clinical signs Mesogenic virus in chickens Signs and gross/microscopic lesions non-diagnostic Catarrhal nasal exudate Chicks (1-6 weeks) : anorexia, depression, profound respiratory signs, CNS signs common, paralysis, dehydration ; MORTALITY USUALLY >50% Adults (6 mos.+): mild anorexia, +/- depression, laying hens stop laying Definitive diagnosis requires virus isolation or serology. Velogenic virus in chickens Morbidity/Mortality approaching 100% Mucosal hemorrhages in GI tract, especially proventriculus and cecal tonsils Rapid onset of dyspnea and diarrhea Severe mortality (often approaching 100%) Neurologic signs can be seen birds that survive a few days after the onset of GI disease, but such signs are infrequent because of the high mortality.

# Paramyxovirus in pigeons

Anorexia, weight loss, diarrhea and dehydration.

CNS signs include leg paralysis and paresis, drooped wings, ataxia, incoordination, and muscle tremors.

#### Diagnosis

A suspicion of ND can often be made on the basis of history, clinical signs and gross and microscopic lesions which include: cloudy air sacs with frothy, yellow air sac contents, chronic infection may result in dry caseous exudate in the air sacs. Non-suppurative encephalitis is one of the most consistent microscopic lesions.

VVND usually shows hemorrhagic, ulcerative and necrotizing lesions in gastrointestinal epithelial and lymphoid tissues. It is impossible to confirm a diagnosis of ND without serologic (i.e. rising titre in paired sera), virologic or molecular (e.g. Reverse-transcription polymerase chain reaction (RT-PCR), gene sequencing, and restriction enzyme analysis) confirmation. These include: HA and HAI tests, virus neutralization, FA tests using conjugated anti-NDV serum and demonstration of rising anti-NDV activity in pooled sera.

#### **Differential Diagnosis**

Laryngotracheitis and Infectious Bronchitis may be difficult to differentiate from mesogenic ND in chickens. Also remember other causes of mild respiratory signs such as low pathogenic avian influenza, mycoplasma and chlamydia. The differential diagnosis for velogenic ND includes Highly Pathogenic Avian Influenza, Laryngotracheitis, Acute Fowl Cholera, Infectious Bursal Disease, Duck Plague (Duck Viral Enteritis) and acute poisonings.

#### Prevention and Control

Many ND vaccines are available. The commercial vaccines are usually modified live type that are administered by aerosol to large numbers of birds. Other vaccines are administered by eye drop or drops in the nares. Killed, oil-emulsified vaccines are also available for parenteral administration mainly in point of lay pullets. Note that vaccinated birds can still be infected with and replicate NDV, however, the amount of virus shed and the duration of shedding is significantly reduced.

Other components of a ND prevention program are: active and passive surveillance; good biosecurity; and good food safety practices. Components of a control program include: quarantine and movement controls; stamping out; carcase disposal; and decontamination.

## Highly pathogenic avian influenza (fowl plague)

This disease is caused by more virulent strains of AI virus. In October of 1983 several point mutations of avian influenza virus occurred (antigenic drift) resulting in a severe epidemic in domestic poultry in the eastern U.S. The economic significance of this seemingly trivial biologic event was emphasized by a cost of more than sixty million dollars paid in indemnities for millions of chickens slaughtered in an effort to control the disease. In February of 1993 H5N2 avian influenza virus was identified in a commercial egg laying flocks in New York, New Jersey and Pennsylvania. This disease is REPORTABLE to the USDA <u>APHIS</u>. Some strains are ZOONOTIC (H5N1).

## Clinical Signs

## Low to moderately virulent virus in chickens

Coughing, "sneezing", respiratory rales and lacrimation, anorexia, lethargy and emaciation. Egg laying hens may show a drop in egg production.

Sinusitis with infraorbital swelling have been reported in some outbreaks however, this finding my be due to secondary infection with *Hemophilus paraganinarum* (coryza). Sinusitis is a common finding in ducks with avian influenza. Diarrhea, edema of the head and nervous signs are occasionally observed.

# Highly pathogenic influenza virus in chickens

MORBIDITY > 90%, MORTALITY >90%

Facial edema with focal cyanosis and hemorrhage of the comb, wattles and non-feathered skin (probably the result of virus induced DIC)

Focal mucosal hemorrhage of the proventriculus occurs similar to lesions seen in VVND but in fowl plague this lesion is not associated with submucosal lymphoid tissue necrosis.

## Diagnosis

Confirmation of AI must be made based on virus isolation and identification or molecular techniques (e.g. Reverse-transcription polymerase chain reaction; RT-PCR). Serologic information can be useful but seropositive birds may be the result of a previous, sub-clinical infection with a low virulence AI virus.

## Gross lesions

These vary widely depending on the strain of the virus involved in the outbreak. Reddened tracheal mucosa, sinusitis, cloudy air sacs and conjunctivitis may be the only lesions seen in outbreaks of low virulence AI.

Highly pathogenic strains of AI will produce diffuse petechia and fibrinous exudate on abdominal serosal surfaces. Mucosal hemorrhages in addition to GI tract can also be seen. Microscopic lesions may be helpful in establishing a diagnosis. These consist of perivascular lymphocytic cuffing in myocardium, lungs, brain, liver and wattles. Additionally, necrosis of splenic lymphoid follicles and pancreatic acinar cells has been seen in highly pathogenic AI.

<u>Differential Diagnosis</u> Newcastle disease (mesogenic and velogenic) Mycoplasmosis Chlamydiosis Acute Fowl cholera Laryngotracheitis Infectious Bursal Disease <u>Prevention</u>

Use of vaccines is controversial and not usually endorsed EXCEPT during a crisis (e.g. H5N1). Note that vaccinated birds can still be infected with and replicate AIV, however, the amount of virus shed and the duration of shedding is significantly reduced if there is a good match between the vaccinal and field strains of virus.

Other components of an AI prevention program are: active and passive surveillance; good biosecurity; and good food safety practices. Components of a control program include: quarantine and movement controls; stamping out; carcase disposal; and decontamination.

## Avian tuberculosis

Tuberculosis of poultry, often termed avian mycobacteriosis, avian tuberculosis, avian TB or TB, is a contagious disease caused by Mycobacterium avium. Avian tuberculosis is a chronic infection. It is a slow spreading chronic granulomatous bacterial infection. Persistence in a flock once established, induces unthriftiness, decreased egg production, and finally causes death. Although tuberculosis in commercial poultry in the United States is rarely diagnosed, tuberculosis still occurs sporadically in

backyard poultry and game birds, and it remains an important problem in captive exotic birds. All poultry are susceptible and pheasants highly susceptible. It can also infect captive exotic birds as well as rabbits, pigs and minks. Transmission occurs via infective fecal material and respiratory tract secretions. It is a zoonotic disease that is very resistant and can survive in the environment for up to 4 years. M. avium infections have been common in patients with acquired immune deficiency syndrome (AIDS). In the United States, M. avium serovars 1, 4, and 8 are isolated most frequently from AIDS patients, and serovars 4, 8, 9, 16, and 19 are isolated most frequently from non-AIDS patients. M. avium serovar 1 commonly is isolated from wild birds as well as AIDS patients.

<u>Clinical signs of avian tuberculosis</u> Develop slowly

Gradual weight loss – thin, lethargic Lameness occasionally Feathers dull and ruffled

Diagnosis

Avian tuberculin test (intradermal injection in wattles) Necropsy lesions suggestive Large numbers of acid-fast bacteria in smears from lesions

## Differential diagnosis

Granulomas are rather characteristic, but other conditions must be differentiated. These include coligranulomas (Hjarre's disease), pullorum disease, other salmonella infections, enterohepatitis, fowl cholera, and neoplasia. Presence of numerous acid-fast bacilli in lesions is significant. When available, culture and identification of *M. avium* is helpful but not essential for a diagnosis.

## Necropsy

**Chickens** - pinpoint to several centimeter, irregular grayish yellow or grayish white nodules in spleen, liver and intestine; granuloma formation common in bone marrow

Turkeys, ducks, and pigeons - lesions predominate in the liver and spleen but occur also in many other organs

## Trauma

Predator trauma common Wound debridement +/- C/S Antibiotics Wound closure vs wound management

# Drug comments on layers

Ivermectin

8 wk withdrawal oral 3 mths if injected

5 muis n mj

Carbaryl

0 tolerance

Alternatives: Permectrin II, Prozap Garden and Poultry Dust

# Diatomaceous earth

No withdrawal

# Clavamox

14-day owner consumption

## 8 weeks if selling eggs Metronidazole Illegal Fluoroquinolones Illegal Cephalosporins Illegal Meloxicam 14 days owner consumption 6-8 weeks if selling eggs SMZ/TMP 21 days owner consumption 8 weeks if selling eggs or drug sensitive

# Poultry or "Flock" Diagnostic and Therapeutic Techniques

Although pet bird medicine often utilizes hematology and clinical chemistry to help establish a diagnosis, such tests are almost never employed with poultry.

#### Necropsy

A necropsy is the diagnostic procedure commonly used in establishing a diagnosis when a disease outbreak occurs in a poultry flock. The most import diagnostic material that one can submit to a diagnostic laboratory are one or two live birds showing representative clinical signs of disease in addition to a few recently dead birds that have also shown typical disease signs prior to death. Guidelines on necropsy of poultry may be found at:

http://www.partnersah.vet.cornell.edu/Avian-Necropsy-Examination http://aciar.gov.au/publication/MN139 (Appendix 4)

## Physical examination

Physical examination of a live bird is usually helpful in establishing a diagnosis. The feather cover should be examined and chickens should be assessed to determine if molting is in progress. The feathers should also be examined for presence of external parasites. Ataxia, tremors, paralysis, abnormal gait or leg weakness should be evaluated. Respiratory difficulty, depression or blindness should also be determined. It is sometimes helpful to examine poultry while they are confined to a cage and accustomed to their surroundings. Sometimes it is also important to observe birds 24 - 48 hours after they have been removed from the flock to determine if signs of recovery have occurred. This is especially helpful in the diagnosis of transient paralysis, respiratory infection, chemical toxicity, feed or water deprivation and overheating. Guidelines on the clinical examination of poultry may be found at: http://aciar.gov.au/publication/MN139 (pp. 97-101)

#### Collecting blood samples in poultry

Blood samples are sometimes collected for serologic tests in order to confirm the diagnosis of some of the common viral diseases. These samples are usually drawn from one of the following sites: Brachial (wing) vein - least traumatic (<u>http://aciar.gov.au/publication/MN139</u> [Appendix 3]) Tibiotarsal vein Jugular vein Heart bleed not acceptable in pet chickens

Since serum is used for most of these tests anticoagulants are not required. If unclotted blood is required the same anti-coagulants commonly used for mammalian blood can be employed for avian blood (EDTA, heparin, oxalate).

Serology is frequently used to establish a diagnosis for the following diseases: Pullorum disease Mycoplasmosis Newcastle disease Infectious bronchitis Avian influenza Infectious bursal disease

Collection of other avian diagnostic samples

Information on external and internal diagnostic sampling of birds and the packaging and shipping of diagnostic specimens may be found at: http://www.partnersah.vet.cornell.edu/Avian-Diagnostic-Sample-Collection

Antibiotic use in poultry \*\*\*It bears repeating\*\*\*

Antibiotics used in the treatment of bacterial diseases in poultry that produce eggs or meat for human consumption needs careful consideration. Residues in human and animal food products has been a long-debated issue in the veterinary profession. These residues can potentially result in antibiotic resistant strains of bacteria that cause human disease.

Regulations concerning the use of antibiotics in food animals are becoming increasingly restrictive. Always ensure that you are aware of the latest regulations prior to issuing a prescription. For more information on drugs approved for poultry, and withdrawal times go to the FARAD website at <a href="http://www.farad.org/">http://www.farad.org/</a>

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	Clinical signs	Possible reasons
General signs	Depression, listlessness,	Wide range of causes
	productivity,	
	Emaciation	Chronic, long-lasting disease
Respiratory signs:		
- Breathing	With open beak	ND, infectious laryngotracheitis, Other severe respiratory disease, Very hot weather
	Distressed, noisy, rattling	ND, HPAI, severe respiratory disease
	Coughing, sneezing, wheezing, gurgling	Infectious Coryza, IB, CRD, other respiratory disease, Fowl cholera
- Nostrils	Wet or crusted because of nasal discharge	Infectious coryza, CRD, other respiratory disease
- Eyes	Swelling around the eyes	Infectious coryza, CRD, other respiratory disease, ND, fowl cholera

	Discharge	Infectious Coryza, CRD, other respiratory
	-	disease,
		Fowl Cholera
	Cloudy pupils	Various reasons
Diarrhea	Dirty cloaca	Coccidia, worms, spirochaete infection
		ND, Gumboro,
		Fowl typhoid, pullorum disease, fowl
		cholera, colisepticaemia or other
		infectious diseases
Nervous signs	Twisted neck (torticollis)	ND, HPAI or any other disease affecting
0		the nervous system
	Lameness (paralysis)	ND or any other disease affecting the
		nervous system
	Trembling, shaking	ND or any other disease affecting the
		nervous system
Movement	Lameness	Crusted legs due to scaly leg mites
1010 venient	Lumeness	Marek's disease or
		Various other reasons
	Swollen joints	Fowl Cholera or
	Swohen joints	Various other reasons
	Abnormal position of legs	Marek's disease
	Achorman position of legs	Nutritional deficiencies
		Any accident or
		Various other diseases
	Swellings under the feet	Infected injuries
Feathers	Small spots, maybe moving	External parasites- lice or mites
		1
	Moulting	Usual moulting process (once a year),
		Under stress
	Ruffled	Indicates that a chicken is sick
	Lesions damage	Lice
	Lesions, damage	Eacther packing or connibalism
Slrin	Dala alrin	Cymboro (IDD) or
SKIII	Pale skin	Guillooro (IBD) or Second infectation with external neuroiter
		Severe intestation with external parasites,
Comb	Vallessiah ta daula huarren amatar	Especially the Red Fowl Mile
- Comb	spots	FowI pox
	Pale	Chronic diseases or
		Severe infestation with parasites
	Dark, bluish	Acute feverish diseases affecting the
		cırculatory system (e.g. Fowl Cholera,
		(HPAI)
- Wattle	Yellowish to dark brown lesions	Fowl pox
	Dark, bluish	Acute febrile diseases affecting the
		circulatory system (e.g. Fowl Cholera,
		HPAI)
	Swollen	Fowl Cholera, HPAI

Inside the beak	Yellow-white cheesy looking	Fowl pox
	lesions	

## **References and Resources**

#### Websites

Poultry Breeds <u>http://www.ansi.okstate.edu/poultry/</u> Poultry Examination and Diagnostics <u>http://www.partnersah.vet.cornell.edu/Avian-Diagnostic-Sample-Collection</u> <u>http://www.partnersah.vet.cornell.edu/Avian-Necropsy-Examination</u> <u>http://partnersah.vet.cornell.edu/avian-atlas/</u> <u>http://aciar.gov.au/publication/MN139</u> Poultry NetVet <u>http://netvet.wustl.edu/birds.htm</u> World Organization for Animal Health (OIE) http://www.oie.int/eng/en\_index.htm

## **Textbooks and Articles**

Ahlers, C., Alders, R.G., Bagnol, B., Cambaza, A.B., Harun, M., Mgomezulu, R., Msami, H., Pym, R., Wegener, P., Wethli, E. and Young, M. 2009. *Improving village chicken production: a manual for field workers and trainers*. ACIAR Monograph No. 139. Australian Centre for International Agricultural Research, 194 pp.

Capua, I. and Alexander, D.J. 2009. Avian Influenza and Newcastle Disease: A Field and Laboratory Manual. Springer-Verlag, Italy.

Damerow, G. 1994. The Chicken Health Handbook. Storey Books.

Gaber, L. *et al.* 2007. Non-commercial poultry industries: surveys of backyard and gamefowl breeder flocks in the United States. Preventive Veterinary Medicine **80**:120-128.

Jordan, F.T.W. and Pattison, M. (ed) 2002. Poultry diseases. London: Bailliere Tindall. 2002.

Morishita, T.Y. 1996. Common infectious diseases in backyard chickens and turkeys (from a private practice perspective). Journal of Avian Medicine and Surgery, 10(1):2-11.

Saif, Y.M. et al. (ed) 2008. Diseases of Poultry, 12th ed., Wiley-Blackwell.