Small Scale Poultry Keeping

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Poultry includes domestic fowl, turkeys, domestic ducks, geese and guinea fowl. Game includes wild duck, partridges, grouse, pheasants and quail.

Origins of poultry

Domestic Fowl

The domestic chicken, *Gallus domesticus* belongs to the genus *Gallus* of the sub-family *Phasininae*. The wild ancestors of the domestic chicken probably originated in Southeast Asia. Four species of wild Jungle fowl are still found in this area:

Gallus gallus:	the wild red jungle fowl (northern India, Burma, Southern Vietnam, the Malay Archipelago and the neighbouring islands);
G. sonnerati:	the gray jungle fowl (west, central and southern India);
G. lafayetti:	the Ceylon jungle fowl (Sri Lanka); and
G. varius:	the green jungle fowl (Malay islands).

The Red Jungle Fowl, *Gallus gallus*, has the widest distribution of the wild species and may be the chief ancestor of the domestic fowl.

Geese

Geese may have been the first poultry domesticated. The Gray Lag Goose of Europe (*Anser anser*) is considered the progenitor of all the domesticated European breeds, while the wild China goose (*Anser cygnoides*) is considered the ancestor of the Asiatic breeds (e.g., the Chinas and the Africans).

Duck

With the exception of the Muscovy (*Cairina moschata*), all domestic breeds of ducks are lineal descendants of the wild common mallard (*Anas platyrhynchos*). The mallard is the only species of wild duck in which the males exhibit the so-called sex feathers at the base of the tail. These feathers tend to disappear, or become vestigial, when the mallard is crossed with other wild breeds. All drakes of domesticated true ducks have these sex feathers, so that the conclusion they are descended from the mallard is inescapable.

The Muscovy is a native to Southern middle America, Central America and Northern South America. During its early days of domestication the Muscovy was known variously as the Guinea Duck, Barbary Duck, Cairon Duck, Indian duck, turkey duck, and, recently, the Muscovy. The latter name has been corrupted into Musk duck, a name which carries a derogatory implication for a table duck. The wild Muscovy existed from Mexico to

Argentina, where it was known as the Pato (Spanish for duck). A member of the Order *Anseres*, Family *Anatidae*, Subfamily *Anatinae*, Tribe *Cairinae* ("perching ducks"). There are two other members in this Tribe, the African species *hartlaubi* and the South and South-East Asia species *scutulata*. Hybrids between this species and the common domestic duck *Anas platyrhynchos* are sterile.

Guinea Fowl

Guinea Fowl are African birds. There are 38 natural species and subspecies, divided between four genera (*Agelastes, Guttera, Numida, Acryllum*) and seven species. Domesticated guinea fowl (*Numidia meleagris*, family *Numididae*) come originally from open plains of Central Africa. The family has several sub-species, with mutated colours of pearl, white, lavender, buff blue and royal purple.

Pheasant

According to legend, the pheasant was introduced by the Argonauts into Europe and later introduced into England by the Romans. This bird, *Phasianus colchicus torquatus*, the Chinese ringneck, is the type of the Family. Ring-necked pheasants were introduced to America in 1881, and are now well established as wild populations in southern Canada and northern United States. The golden pheasant (*Chrysolophus pictus*), Amherst's pheasant (*C. amherstiae*), the silver pheasants (*Gennaeus* spp) and reeves' pheasant (*Syrmaticus reevesii*) are the most beautiful of poultry. The monals (*Lophophorus*) inhabit the Himalayas, and the peacock pheasants (*Chalcurus* and *Polyplectron*) inhabit Indochina and the Malayan Archipelago.

Management

Domestic Fowl

Domestic fowl generally need to be brooded for 4-6 weeks after hatching, depending on the environmental temperature. Chickens need supplementary heat when they hatch because they are unable to maintain their body temperatures. The heat can be supplied by a broody hen or, more usually, by brooders using electricity, gas or oil as fuel. As the chicken grows its downy coat is replaced by feathers, the brooding temperature can be gradually reduced until supplementary heat is discontinued at about 4 weeks. The brooder must be capable of providing a steady temperature of 35°C, even in the coldest conditions. Simple electric hobby brooders can be obtained from poultry equipment suppliers and will successfully brood up to 50 chicks. During the winter it may be necessary to provide heat on very cold nights in the fifth week. Generally supplementary heat can be discontinued at the end of the fourth week. Chickens will chirp loudly the temperature is too low. If they move well away from the heat source and start panting they are too hot. Ideally they should be fairly quiet and spaced evenly under and around the heat source.

The area where the chicks are to be brooded should be clean, dry and the floor covered with dry absorbent litter material to a depth of 50 mm. Place a surround of cardboard, metal sheeting or hardboard around the brooding area. The surround should be about 450 mm high to protect the chickens from draughts, and the area enclosed should provide at least 50 square centimetres of floor space for each bird. For the first 2 days the litter in the brooding area should be covered with newspaper. Feed should be sprinkled on the paper and clean

fresh water provided. Ideally the water should be in specially designed drinkers consisting of a plastic jar inverted into a shallow circular trough. The drinkers hold about 2 litres of water and the shallow troughs are designed so that the chickens cannot drown in them.

Brooding temperatures for domestic fowl

Age in days	Temperature at chick height °C
1-3	35
4	34
7	30
14	25
21	22
28	20
>35	18

Ordinary flat dishes can also be used but if the water is too deep the chickens may drown. A large stone or block of wood placed in the centre of the pan will usually prevent drowning by reducing the amount of water in the vessel without restricting access by the birds.

Place feeders and drinkers near the heat source and, for the first 2 days, sprinkle food liberally on the newspaper to encourage the chickens to eat. Also dip each chicken's beak in the water as it is placed in the brooder to encourage it to drink. With large numbers it will not be possible to do this with all of the chickens but it is generally worthwhile dipping the beaks of 10 per cent of the flock.

The newspaper can be removed after 3 days, the feeders and drinkers moved further away from the heat source and the surround gradually expanded until it can be removed completely at 2 weeks.

Space requirements

Age weeks	Floor space birds/sq cm	Feeder space mm/bird	Drinking space mm/bird
1-4	20	20	10
5-8	10	30	20
9-20	5	50	30

Feeding

Chickens that are to be grown for egg production need chicken starter crumbles or mash from day-old to about 6 weeks. The diet should contain 18% crude protein and a coccidiostat if the chickens are to be reared on the floor. From 6 to 18 weeks, grower pellets or crumbles (16% protein) are generally used for small flocks. Birds should have unrestricted access to food and water. At 18 weeks the pullets should be fed a laying diet, which should be available at all times. It can be supplemented with scratch grain and kitchen scraps.

Turkey

Poults should be started on feed and water at once. It is better to buy poults that are at least 2 days of age. Watch to see that poults actually begin to eat and drink - if some don't, coax them by dipping their beaks in the feed or water or by using bright-coloured marbles in the mash, or even by using day-old chickens to teach them.

Poults that do not get feed and water for 2 or 3 days after hatching will never recover from this early setback. They may never learn to eat or drink. This is why some of the biggest poults in the brood die: they are big because they hatched earliest and hence are the ones that have gone longest without eating.

The brooding comments for chickens apply to turkey poults as well.

Pheasants

It has been the trend in Australia to house breeding stock in individual mating groups and to a much lesser extent, under range conditions. If pairs are housed in individual pens, this permits recording of their breeding and progeny performance, the male will not be able to fight other males or each other, and so fertility should be higher and there will be fewer broken eggs.

Under range conditions, foxes, wild dogs and cats can be fenced out, and raptors can be excluded by covering the pens with wire netting or lightweight plastic mesh or strong shadecloth. The hens should be able to find shelter from over-zealous cocks. Good cover will also encourage them to lay eggs at central points and thus prevent them from being scattered over the yard. Numbers of eggs produced in a season varies with the strain and may be from 20 to 130 per hen. Producers should aim to select breeders with an egg production of at least 65 per season.

Egg collection

The egg laying season lasts a few months. Because of this, production costs are high so all the eggs laid should be given a chance to incubate. Collect eggs at regular intervals and as often as possible during the day. Hens will lay their eggs on the ground in a compound, or on the floor in a building. Reduce egg breakages by providing shelter to encourage laying in central locations. Most eggs from hens raised on good deep little will be clean, but eggs from hens raised under range conditions can be quite dirty.

Incubation

The normal time of incubation for pheasant eggs is 24 to 25 days. The actual time will be governed by factors such as age and method of storing eggs, and management of the incubator, with particular regard to temperature and humidity. Forced-draught machines should operate at a temperature of approximately 37.5°C during incubation. The humidity should be approximately 65% for the first 21 days of incubation, but should then be increased to 90%. If high humidity is not maintained at hatching, the chick will have difficulty penetrating the membrane of the shell which will be too hard and rubbery. Keep moisture trays filled with warm water at all times and increase humidity at hatching time by hanging wet hessian in the incubators, or by using automatic misting sprays in the cabinet of the incubator.

Brooding and Rearing

The method of brooding and rearing will depend on the number of pheasants. Chicks should be provided with about 500 cm^2 of total floor space inside buildings and about 2 m^2 each of ground space where outside runs are used. If chicks are given access to outside runs the tops of the runs must be covered with netting because pheasants are nervous and are able to fly from about 3 weeks of age. The netting on top of outside runs should be covered with branches to help simulate natural conditions. At first brooders should operate at 35°C reducing to 25°C by the time chicks are 3 weeks of age. Where outside runs are used, chicks must still be able to come and go inside the brooder house until they are 6 weeks of age. After brooding, pheasants can be reared on range provided they have adequate shelter. Yards should be enclosed with netting to prevent birds becoming lost. To prevent flying, wing feathers should be clipped at least every 4 weeks or pinioned at day-old.

Handling and Catching

Pheasants can fracture their skull and/or neck trying to escape from a disturbing situation. When caught, they must be handled carefully because their bones can be brittle. Always hold pheasants by both legs, with head and body tucked under your arm. Pheasants can be caught with a net similar to a handfishing net with a heavy gauge, wide mesh. This can be placed over the bird to catch it without causing physical damage. Pheasants, when caught, should be placed in carrying cages covered with a hessian bag to prevent self-injury.

Pheasants are very prone to cannibalism and feather picking, much more than other poultry. Once an outbreak occurs it is very difficult to stop. Cannibalism can start in a flock of pheasant chicks 2 weeks of age. If left untouched, the beaks of pheasants are particularly sharp and much damage can be done in a relatively short time often resulting in many birds being picked to death. You can reduce cannibalism by providing:

- dim lighting for birds raised intensively and plenty of vegetation for birds raised on range.
- well-ventilated buildings that are not overcrowded.
- hanging bundles of straw to pick at to prevent boredom.
- a ration containing high levels of salt (0.5%) and fibre (7%).
- perches in houses and shelter sheds.

Beak-trimming

The severity of beak trimming varies, depending on the methods used. As a guide approximately 1/3 to half of the top beak should be removed. Since beaks grow quickly, frequent trimmings will he necessary. Chicks should be debeaked at either day-old or 2 weeks of age then again every four to six weeks.

Flight Control

Pheasants are very good fliers, so they should be kept in completely covered yards or buildings. If they escape after a disturbance, they can fly away too far and become lost.

Clipping. Wing clipping temporarily prevents flying. The primary wing feathers of one wing are clipped short. Birds can be clipped after they reach 6 weeks of age,

but it will be necessary to reclip feathers about every four weeks. Obviously, clipping has definite disadvantages - the flock is regularly disturbed and the job is time consuming.

Brailing. Brailing is another method or flight control. A brail is a thin, pliable leather strap cut with a small T-shaped strap at one end. The smaller strap is fastened around the pheasant's "forearm" with a paper clip. The longer section of the strap is then passed beneath the wing and pushed up between the outermost flight feathers, before being bent back and secured with the paper fastener. The ends of the paper fastener should be curved back with a pair of pliers so that they can be bent under the fastener head. Some limited movement should be allowed to the brailed wing. To give both wings some exercise, with breeders it is advisable to change the brail to the other wing about every four months.

Taping. Taping wings can be used as an alternative to brailing. Two lengths of 6 mm linen tape should be laid side by side and knotted to divide the double tape into one-third and two-thirds of its length. Apart from the long end of the tape being double instead of single, the same technique should be used for securing the wing, as in brailing. Use a suitable knot to locate the tape on the pheasant's elbow.

Pinioning. To pinion a pheasant is to remove the first joint of one of its wings. The operation can be done at day-old with a beak trimming machine with virtually no injurious effects to the bird.

Diseases

The commonest diseases that affect backyard poultry in Australia are Marek's disease (especially after introducing some newly purchased chickens), leucosis, coccidiosis, egg yolk/*E. coli* peritonitis, *Cnemidocoptes* infection (especially on the legs and peri-cloacal area), internal parasitism, fowl tick fever and coryza.

Marek's Disease

Definition: Marek's disease is a contagious lymphoproliferative and neuropathic disease of the domestic chicken, caused by a cell-associated herpesvirus.

Synonyms: MD; acute leucosis; neural leucosis; range paralysis; ocular leucosis; "grey-eye"; neural lymphomatosis.

Aetiology: Herpesvirus.

Transmission: By aerosol in feather dander, dust, faeces and saliva. The virus is not egg-transmitted.

Signs: Lameness, droopy wing, incoordination, weakness. Enlarged feather follicles, grey eye, constricted pupil. The disease is mainly recognised in three forms:

a. Classical form, characterised by variable degrees of paralysis of the legs and wings, a low incidence of lymphomas and a low mortality rate extending over a period of several months;

b. Acute form, characterised by a high incidence of lymphomas and a rapid rise in mortality without marked premonitory signs; and

c. Transient paralysis, an encephalitic form of MD.

Lesions: Tumours in viscera and nerves.

Pathogenesis: MDV infection is usually acquired by inhalation of infective feather debris, dander or dust. Virus is transported away from the lungs in phagocytic cells.

Diagnosis: Affected bird 2-16 weeks of age. Tumours in nerves, brain, spinal cord, liver, spleen, kidney, gonads, heart, lungs, GIT, pancreas, skin, muscle and eye.

Treatment: None.

Prophylaxis: Incidence can be significantly lowered by:

1. rearing in isolation;
2. strict sanitation;
3. good management;
4. elimination of stress, control of other diseases; and
5. incineration of dead birds.

Prevention: Hygiene plays an important role in the control of infectious diseases in intensively-reared poultry, and the principles of hygiene are particularly important in the control of MD. It is vital that young vaccinated chicks are not exposed to large amounts of MDV, as this can overwhelm the developing immunity. Exposure to virulent strains of MDV must be minimised and even avoided during the first week of the chick's life.

It is important to have single-age brooding rooms and rearing sheds. On a multi-age site, young birds in the rearing stages should be as far away as possible from older birds and tended by separate attendants.

Vaccination

A live-virus HVT vaccine for backyard flocks will soon be available. The vaccine is given as a single subcutaneous injection to day-old birds.

AVIAN LYMPHOID LEUCOSIS

Definition: Avian lymphoid leucosis is a naturally occurring neoplastic disease of the sexually mature chicken, and is caused by replication-competent members of the exogenous leucosis retrovirus subgroups (A-D, F & G). Only subgroups A and B are common.

Synonyms: ALL; visceral leucosis; VL; "big liver"; haemangioma; water-belly; visceral lymphomatosis; osteopetrosis; marble-bone disease.

Aetiology: RNA retroviruses.

Transmission: Vertical, lateral, genetic.

Signs: Formation of lymphoid tumours, particularly in the liver and spleen. Affected birds may die without preliminary signs. A wide range of non-specific signs depending on strain of virus. Death following a progressive emaciation, weakness and abdominal distension is most common. Less commonly, thickening of long bones (osteopetrosis), connective tissue tumours, myeloid tumours. Reduced egg production.

Lesions: Diffuse or focal (or both) tumours consisting of large lymphoid cells are seen in many visceral organs particularly the liver and spleen.

Pathogenesis: The earliest changes may be observed in the cloacal bursa, where nests of larger-than-normal lymphoblasts occur in an otherwise normal follicle. Transformed B-cells metastasise to the liver, spleen, gonads and other organs, leading to the death of the host at the onset of sexual maturity. These lymphoblasts have a large vesicular nucleus with one or more very prominent nucleoli and scattered strands of rough endoplasmic reticulum. ALL viruses are nondefective and competent for replication. They do not possess an oncogene and fail to transform any target cell thus far tested in cell cultures. The genome of ALL viruses possesses only those genes necessary for encoding the replication and structural functions of the virus. In lymphoid tumours, the ALL provirus is integrated adjacent to a specific cellular gene, similar to the oncogenic C-myc. Thus does the virus transform the cell, a rare event. Because transformation is rare, all ALL tumours are clonal.

Diagnosis: Based on flock history, detection of virus, and serology. Affected birds are 16 weeks and older. Differentiate from MD, PD, TB, blackhead, coligranuloma, REV.

Treatment: None.

Control: Eradicate ALV from breeders and terminate hens that carry the virus.

Prophylaxis:
1. brood in isolation;
2. strict sanitation;
3. good management;
4. elimination of stress, control of other diseases;
5. incineration of dead birds;
6. control of biting insects; and
7. buy resistant strains of birds.

DIFFERENTIAL DIAGNOSIS OF AVIAN LYMPHOPROLIFERATIVE DISEASES.

The differential diagnosis of MD, ALL and RE is shown in Figure 2.

MD can occur at any time after four weeks of age, but is most common between 12 and 24 weeks. It may be seen in older birds, particularly in flocks vaccinated against MD. ALL is a disease of sexually mature chickens and is not seen in birds under 16 weeks of age. Myeloid and erythroid leucoses are also diseases of the adult chicken, although myelocytomatosis and erythroblastosis may be seen in younger birds. Paresis or paralysis are commonly observed in MD in a flock. Signs of ALL are non-specific and include wasting. MD usually affects more than 5% of a flock, whereas ALL occurs at a much lower incidence. In adult flocks vaccinated against MD, MD may occur at a very low incidence and may be difficult to differentiate from ALL. Myeloid and erythroid leukoses occur sporadically.

Neural enlargement is often seen in MD, and, occasionally, in REV, but not in ALL. Gonad involvement is often a feature of MD, while the liver and spleen are more commonly affected in ALL. When diffusely affected, the liver in ALL is usually very large, not the case with MD, except in adults when in both diseases the liver may be enlarged. In many birds MD affects the skin, skeletal muscle and proventriculus, sites rarely affected by ALL.

Tumour involvement of the cloacal bursa is rarer in MD than in ALL. When it does occur, it is diffuse (interfollicular) in nature, as compared with the nodular (intrafollicular) tumours of ALL.

Infectious Bronchitis

Definition: An acute, highly contagious viral respiratory disease of the domestic fowl characterised by respiratory signs (gasping, coughing, tracheal rales and sneezing); negligible mortality in layers, unless complicated by other infectious agents; a marked egg production drop in layers; and infection of the urogenital system. In Australia the disease is often seen as a nephritis, affecting young birds 3-6 weeks of age.

Synonyms: IB, bronchitis, cold.

Aetiology: Coronavirus.

Transmission: Considered the most contagious disease known. Once introduced to a population, the virus will infect ALL susceptible chickens, regardless of the level; of sanitation or precaution. Inhalation of virus-contaminated aerosols expelled by infected chickens. Recovered birds may carry and excrete the virus for up to 60 days. All ages are susceptible, but the most severe signs are seen in birds 1-8 weeks old. There is no vertical transmission. Elsewhere in the world the virus has been shown to be capable of permanently damaging the oviduct of young chicks, but only those with no maternal antibody. Infected embryos die before hatching.

Signs:

Chicks: Rapid onset and exceptionally rapid spread, morbidity 1005, mortality nil in uncomplicated cases. Feed and water consumption declines. Gasping, coughing, tracheal rales, oculo-nasal discharge. Nephrogenic strains may cause high mortality. Signs due to renal involvement are typically those of increased fluid intake and excretion, soiled vent feathers, depression and 10-15% mortality commencing several days after first respiratory signs. Chicks that have had IB or a severe reaction to vaccination may develop airsacculitis and CRD in concert with MG. **There are no nervous signs.** As chickens mature they become less susceptible ti the nephrogenic effects of the virus.

Adults: Coughing, sneezing, rales. Oculo-nasal discharge rare. Drop in egg production of up to 100%, with soft-shelled, mis-shaped and rough-shelled eggs. Production is regained after 4-6 weeks, but at a lower level. The albumen of eggs is watery. Low egg quality and mis-shapen eggs may persist for many weeks after the disease subsides. **There are no nervous signs**.

Lesions: Conjunctivitis and sinusitis, tracheitis and bronchitis with serous, catarrhal or caseous exudate. With renal involvement, there is dehydration and the kidneys are pale and swollen with lobulation apparent. The ureters may be distended with uric acid crystals.

Diagnosis: Rapid onset, rapid spread, signs, lesions, egg production drop, no nervous signs, positive virus isolation, positive serology. Differentiate from mild ILT, coryza, ND, AI, fowl cholera, EDS'76, aspergillosis.

Prognosis: Egg production will be slow to recover and deformed eggs may be produced for months after infection.

Treatment: Antibiotics for 3-5 days after signs appear are optional, and usually given to prevent airsacculitis (waste of money). Raise the temperature 3°C for brooding chicks and ensure that overcrowding does not occur. Ensure a warm draft-free and ammonia free environment. Electrolyte replacer (2g sodium chloride and 4g potassium citrate per litre of water) for up to 10 days will minimise losses due to renal involvement.

Prophylaxis: Vaccination.

Infectious Laryngotyracheitis

Definition: A highly contagious infectious viral disease of chickens, characterised by severe respiratory distress and bloody tracheal exudates.

Synonyms: ILT, LT, trach, laryngo, laryngotracheitis.

Epidemiology: Transmission is by aerosols generated by infected birds. Recovered birds become lifelong carriers and shed the virus intermittently. Egg transmission does not occur. Spread is less rapid than with other respiratory viruses. All ages are susceptible, but the disease is more severe in birds less than 6 weeks old. Rapid onset, lasts 10-14 days, mortality 10-70%. Amazon parrots may also be affected by this virus. Peasants and waterfowl may be infected experimentally.

Aetiology: Herpesvirus.

Signs: The severe form of the disease is characterised by sudden onset and rapid spread through the flock. Affected birds show a marked dyspnoea with gasping, moist rales and coughing. When the birds cough they spit out blood-stained mucus, staining the feathers of the face and breast. Death may occur 2 days after onset of clinical signs. A phenomenon called "cycling" is characteristic of this disease. In the first wave up to 70-80% of birds are affected, followed in 10-12 days by a second wave when the remainder of the flock is infected. There is a drop in egg production in laying birds. In mild outbreaks the spread is slower and fewer birds in the flock are affected. In the chronic phase caseous deposits may be seen in the trachea, larynx and nasal cavity.

Lesions: Confined to mucous membranes of the larynx and trachea with pseudomembrane formation. Sloughing of surface epithelium causes excessive bleeding with free blood in the lumen. Later, yellow cheesy diphtheritic membranes form and these can slough, leaving no free blood. However, they may obstruct the trachea. The pseudomembranes in subacute to chronic stages can be removed without bleeding,

as opposed to avian pox infection, which, if removed, leave a raw, bleeding surface. There may be a conjunctivitis, with serous oculonasal discharge. Intranuclear inclusion bodies are present sloughed tracheal epithelial cells in the early stages of disease.

Diagnosis: There are no nervous signs. Severe haemorrhagic disease. Serology. Absence of other diseases which can cause similar signs: ND, AI, avian pox, fowl cholera, coryza, aspergillosis.

Treatment: Nil.

Control: When infected flock is depopulated, clean and disinfect the premises, rest premises for 6-8 weeks, repopulate with healthy ILT-negative birds.

Prophylaxis: Vaccination.

Avian Pasteurellosis

Definition: Avian pasteurellosis (avian cholera) is an acute to chronic disease affecting poultry and wild birds characterised by high morbidity and mortality in the acute form, and respiratory disease, malaise in more chronic forms. Sporadic occurrence. **The most feared disease of turkeys.**

Synonyms: Fowl Cholera; avian cholera; pasteurellosis; FC; goose septicaemia.

Aetiology: *Pasteurella multocida.*

Transmission: Bird-to-bird contact, ingestion of contaminated food or water, and possibly by aerosol via naso-pharyngeal mucosa. Wild birds and rodents can assist bird-to-bird transmission. Thus infection can come from flock additions, free-flying birds, infected premises, predators or rodents. Once introduced to a poultry flock, the disease spreads horizontally in the following ways:

1. picking at the oculo-nasal discharge of infected birds;
2. contamination of food and water by infective discharges;
3. cannibalism of dead or weakened birds.

Spread from flock to flock occurs in the following ways:

1. spread by personnel, contaminated equipment, wild birds;
2. artificial insemination in turkeys;
3. predators

P. multocida persists for several months in bone marrow.

Susceptibility: Birds of all species. Young adult poultry, especially turkeys, chickens and ducks, are most commonly affected. Recovered birds remain carriers. Breeder birds

coming into lay experience greatest incidence of disease. Scavenger species such as crows and gulls are commonly found to have died from this disease, but deaths of raptors are rare.

Signs: In chickens there may be an acute explosive outbreak, with hens dying on the nest. In the peracute form, death is only sign. In acute or sub acute forms, birds will be depressed with an oculo-nasal discharge. As the disease progresses, the birds lose weight, become lame (arthritis) and develop a "rattle" from exudate in the trachea and bronchi. In the chronic form, the disease may be localised in the wattles and comb, and there may be involvement of the middle ear.

Affected turkeys are lethargic, off their feed and die. The heads of affected birds are cyanotic, and **the death rate increases logarithmically**.

Lesions: Vary from bird to bird. There may be no lesions in cases of peracute death, but usually there is splenomegaly and hepatomegaly, with widespread petechiation, especially in the heart, mucous membranes and fat in the abdominal area. Focal necrosis of the liver is common. In turkeys, the most striking feature of the disease is a very solid, red-purple pneumonia with granulomas, and this is considered pathognomonic. Other lesions in turkeys are cyanosis of the head, apteria and tissues. In the chronic form, there may also be a fibrinous pericarditis and peritonitis and caseous plugs in the airsacs, sinuses, lungs, wattles and combs.

Wild birds: Waterfowl that have died acutely have substantial amounts of fat. Petechiae are found on the surface of the heart and gizzard and there may be focal area of necrosis on the liver. The longer the survival time, the more numerous and dramatic are the lesions. Because the birds have died so quickly, the upper GIT usually contains recently digested food. In ducks, these findings **are similar to those seen in duck plague (DVE), and illustrate the need for a laboratory diagnosis.** The lower GIT contains a syrupy, yellowish fluid that is heavily laden with *P. multocida*.

Diagnosis: History, sudden death. Culture of *P. multocida* from affected lungs, and from liver (takes 3-5 days). Avian influenza and Newcastle disease should be excluded. Big Liver and Spleen disease in broiler parents. Turkey rhinotracheitis produces a much lower mortality and more copious discharge. Erysipelas causes sudden death in turkeys, but the pneumonia is mild. Chlamydophilosis in turkeys does not result in an obvious pneumonia, but can produce similar impressive mortalities. Chronic fowl cholera may be confused with colisepticaemia.

Prognosis: Poor in acutely affected birds.

Treatment:

1. Minimise the amount of bacterial challenge in the flock through rodent control, sanitation and quarantine.
2. Maximise immunity. Vaccination will not totally prevent a cholera outbreak, but will minimise its effects on the flock. Use live attenuated or inactivated *P. multocida* vaccine. In Australia, inactivated vaccines are used, usually at 18 and 26 weeks of age (see vaccination).
3. Be ready to institute effective therapy and treat aggressively if an outbreak starts.

Tetracycline at 400-600 ppm in feed. Injection of sick birds with Terramycin LA (20 mg/kg) or peniclilin/streptomycin.

Control: Rapid, accurate diagnosis.
Sanitation
Rotate range areas
Effective rodent control - see Rodent Control
Eliminate poorly drained areas
Sanitary feeding and watering equipment
Dispose of carcasses promptly and properly
Screen out wild birds
Bacterin vaccination
Quarantine
All-in, all-out rearing of pullets.

Coryza

Definition: An acute to subacute disease of chickens, pheasants and guinea fowl characterised by conjunctivitis, oculonasal discharge, swollen infraorbital sinuses, facial oedema, sneezing and airsacculitis.

Synonyms: Infectious coryza, haemophilosis, roup

Aetiology: *Haemophilus paragallinarum.*

Epidemiology: Chronically ill or healthy carrier birds are the main reservoirs of infection. All ages are susceptible. In chickens over 14 weeks of age the disease is more severe, especially in roosters. Infection is by droplet inhalation, contact, drinking water, ingestion. Can act secondarily or in concert with other respiratory pathogens.

Signs: **Uncomplicated coryza:** There is a drop in egg production, with depression, especially in roosters, and a serous oculonasal discharge which later becomes mucoid and even purulent. The infraorbital sinuses becomes distended and there is oedema of the wattles. There is an airsacculitis in 60% of cases.

Complicated coryza: Usually complicated with MG and/or MS. The signs are the same as those seen in uncomplicated coryza, but are more persistent. There is a continuous nasal discharge with caseous plugs in the nasal passages. Rales are more common, and there is a more severe airsacculitis.

Lesions: Acute catarrhal inflammation of the mucous membranes of the nasal passages and sinuses. Subcutaneous oedema of the face and wattles. Conjunctivitis. Lesions may be limited to infraorbital sinuses. There may be a tracheitis, pneumonia or airsacculitis.

Diagnosis: History, signs and lesions, elimination of other diseases which cause similar signs and lesions. Differentiate from mycoplasmosis, avian pox, localised chronic form of avian pasteurellosis, avitaminosis A.

Prognosis: Recovery in 10 to 14 days.

Treatment: Treatment of this disease is complicated by:
* slow spread in a flock;
* recovered and/or immune birds may be carriers; and
* mixed bacterial infections and secondary bacterial infections.

Birds respond to treatment but relapse may occur when treatment is discontinued. Individual bird treatment better.

Flumequine at 12mg/kg BW for 10 days
Chlortetracycline + Erythromycin - 1g/litre DW
Nalidixic acid
Sulphonamides with trimethoprim
Streptomycin
Spectinomycin
Tylosin.

Control: Depopulate, if necessary, to eliminate all carrier birds. Clean and disinfect, leave vacant for 2-4 weeks, repopulate with coryza-free chicks. Quarantine. All-in all-out policy.
Controlled exposure of pullets prior to onset of lay.
Vaccination.

Colibacillosis

Definition: An infectious disease of birds in which *Escherichia coli* is the primary or secondary agent.

Synonyms: Colibacillosis, *E. coli* infection.

Aetiology: *Escherichia coli.*

Epidemiology: The organism is normally present in the GIT of birds and mammals and is consequently passed in the faeces. Birds are continuously exposed via contaminated faeces, water and dust. It is possible that these "normal" commensals may, in time of stress or whenever the host's resistance to disease is lowered, cause disease. The organism can be isolated from the eggs of normal hens, and progeny may be infected either *in ovo*, or from poor nest box hygiene.

E. coli causes secondary infections following primary viral or mycoplasmal infections or adverse environmental conditions. In normal psittacine birds coliforms are absent, so that the isolation of *E. coli* from the intestine or faeces of a psittacine bird with signs of enteritis should be regarded as significant.

Signs and lesions: **Airsacculitis**: Respiratory signs vary in severity. It may be primary when it is associated with dusty litter, excessive ammonia or other adverse environmental condition. It is more usually a secondary pathogen consequent to, or in concert with, *Mycoplasma* spp.
Omphalitis: Navel is moist and yolk sac is infected, watery and has an unpleasant odour. A peritonitis will be present, and the gall bladder full, indicating that the chick has not eaten.
Salpingitis: Adult layers can get a distended oviduct filled with cheesy exudate which has a foul odour. A peritonitis may also be evident.
Coligranuloma: Granulomas in liver, spleen or other organs, and rarely in the intestinal wall. Can be confused with tuberculosis.
Arthritis: A rare cause of arthritis in adult chickens and turkeys.

Diagnosis: Isolation and characterisation as a serovar recognised as a pathogen. Eliminate the possibility of other pathogens acting primarily or in concert with *E. coli*. When *E. coli* acts secondarily to another pathogen, the signs/lesions should be described as secondary colibacillosis.

Prognosis: Usually has a low incidence, therefore good prognosis for flock. If secondary, depends on primary pathogen.

Treatment: Chickens with omphalitis and adults with salpingitis are culled. Treatment to protect remainder of flock:

Tetracycline in feed at 300-400 ppm is drug most likely to give cost-beneficial results. OTC or CTC 0.25-0.5 g/litre drinking water for 5-7 days may be given.
Trimethoprim/sulphadiazine 0.25 mL/L drinking water for the first 5-7 days of life.
Amoxycillin trihydrate 4-11mg/kg or 75mg/litre drinking water for 5-7 days
Furaltadone 0.63g/litre drinking water for 5-7 days

Control: Principles of good management, egg collection, hatchery hygiene.

Avian Mycoplasmosis

Organisms of the genus *Mycoplasma* are important causes of respiratory disease in birds. Three are known to be of significance in poultry: *Mycoplasma gallisepticum*, associated with chronic respiratory disease/air sac syndrome of chickens and infectious sinusitis of turkeys; *M. synoviae*, the cause of infectious synovitis in chickens and turkeys; and *M. meleagridis*, associated with air sac disease in turkeys. These diseases are rarely seen in backyard flocks in Australia.

Aspergillosis

`Definition: Aspergillosis is an acute fatal respiratory disease of young birds characterised by the presence of miliary nodules in the lungs, and fungal growth on the airsacs. Aspergillosis is a common cause of respiratory disease in ostriches, penguins, raptors, turkeys and many other domestic, captive and wild bird species (especially raptors).

Synonyms: Brooder pneumonia

Aetiology: *Aspergillus fumigatus*, ubiquitous fungus.

Epidemiology: Chicks or poults may become infected as embryos through dirty or sweating egg shells. The fungus grows while the egg is incubated. The egg may actually explode in the hatcher and expose other eggs to high numbers of infective spores. The fungus may grow in air conditioning systems in hatcheries, infecting chicks and poults after hatching.

Transmission: By aerosol - inhalation of spores, inhalation of an overwhelming number of spores.

Signs: Primarily affects the lower respiratory tract, but can affect the upper respiratory tract.

Young chicks are usually affected, hence the name "brooder pneumonia". Older birds may be affected if immunodeficient. Chicks as young as 5 days of age may show signs. Birds may be dyspnoeic and gape. **They do not cough**. Other signs include encephalitis and conjunctivitis.

Chronic aspergillosis in psittacine birds probably results from immune suppression secondary to illness or prolonged antibiotic therapy. Birds lose weight and have an exercise intolerance. **Dyspnoea occurs late in the disease process**.

Pathology: Lesions in the viscera appear as yellowish nodules about the size of rice grains. Air sac lesions may be greenish blue - similar to that on mouldy bread. In some birds the fungus may have spread from the lung to the aorta (seeking oxygen), penetrated the aorta and precipitated haemorrhage with death.

Diagnosis: Clinical signs and lesions. Stained smears from lesions show typical dichotomously

branched septate hyphae. Histological examination of lesions. The respiratory form of

aspergillosis is not easy to misdiagnose. The nervous lesions may be similar to those of encephalomalacia and verminous encephalitis. Culture of *A. fumigatus* is not diagnostic in the absence of lesions.

Prognosis: Poor

Treatment: On a flock basis, nil.
Individually, treatment may be attempted as follows:

1. **Amphotericin B injectable** (*Fungizone intravenous* - Bristol-Myers Squibb - 50mg powder for reconstitution.) Reconstitute in 10 mL sterile water. Take 1.5 mL of fungizone (5 mg/ml) and make up to 5 ml with sterile water. Nebulise at 1.5 mg/kg daily for 20 minutes for 4 days. Repeat in 4-6 weeks. Can be used in combination with ketoconazole, itraconazole or flurocytosine. Cam also be given IV at 1.5 mg/kg TID for 3-5 days. Potentially nephrotoxic and may cause bone marrow depression. Monitor enzymes.

2. **5-fluorocytosine** (*Ancotil* - Roche) 250mg tabs. 50-100 mg/kg PO BID (start at 50 and work up to 100). Also available as an infusion solution 2.5 g/250 ml. Potentially nephrotoxic. Monitor enzymes.

3. **Ketoconazole** (*Nizoral* - Janssen-Cilag) 200 mg Tabs.
Dissolve ¼ Tab in 0.2 mL HCl and 0.8 mL water
10-30 mg/kg BID for 21 days

4. **Itraconazole** (Bristol-Myers Squibb) is supposed to be more specific against *Aspergillus.* Higher plasma concentrations if dissolved in 0.1 N HCl

5. **Enilconazole**

   a. Clinafarm Spray (Janssen Pharmaceutical - Marketed by SmithKline Animal Health Products). 150mg enilconazole/mL - 1 litre bottle. Dilute 1 part of Clinafarm Spray with 99 parts of water. Clinafarm Spray may be mixed with formalin. Nebulise Clinafarm Spray solution at a rate of 10 litres per 3000 m^3, ensuring that all walls and floors are moistened. The solution may be sprayed onto a surface of 750 m^2. Disinfection of buildings and equipment, especially air-conditioning systems; Antifungal.

   b. Clinafarm Smoke (Janssen Pharmaceutical - Marketed by SmithKline Animal Health Products). 5g enilconazole per smoke generator; Packet containing 6 generators. Remove the cover and light the wick. One generator is sufficient for a room of 50 m^3. Clinafarm Smoke is used in areas which cannot be moistened. Disinfection of buildings and equipment, especially air-conditioning systems; Clinafarm Smoke cannot disinfect walls - use Clinafarm Spray for walls. Antifungal.

Control: Sanitation. Avoid locating hatcheries near installations which exhaust organic dusts into the air. Filter incoming air. Avoid brooding on old litter, straw or maize stalks. Clean and disinfect hatcheries with Clinafarm Spray and Smoke. With caged birds avoid mouldy

feed, do not store seed in refrigerator, and do not allow access to grass clippings.

Prophylaxis: Collect only clean eggs for hatching, and do not clean dirty or soiled eggs. Eggs should be collected frequently and the shell surface should be disinfected. Contamination of the hatchery and its air-conditioning system commonly occurs. An adequate cleaning and disinfection system should be in place.

* beware of "good deals" on shavings and litter;
* ensure that the litter has not been stored under conditions that promote fungal growth;
* do not expose chicks or poults to mouldy conditions in the litter or feed;
* do not top-dress the litter while birds are in the house;
* avoid the creation of excess dust in the house.
* Sanitation.
* Filter the air supply to hatcheries.
* Do not place penguins or ostrich chicks on or near straw or hay.
* Avoid stress.
* Avoid long term glucocorticoid treatment.
* Discard mouldy or dusty feed.

Metabolic Bone Disease

Rickets is occasionally seen, but is relatively rare both in commercial and backyard flocks.

Urolithiasis

Urolithiasis primarily effected laying flocks and is characterized by visceral gout and "stones" in the kidneys. The stones plug the ureter of at least one kidney, leading to visceral gout in the effected kidney and hypertrophy of the remaining kidney. Eventually, both kidneys are involved and the bird dies.

Precipitation of stones may be due to either infection (by nephropathogenic strains of infectious bronchitis virus in the domestic fowl) or an imbalance of dietary minerals, particularly calcium and phosphorus. The role of calcium and phosphorus in predisposing pullets and laying hens to urolithiasis is primary. Urolithiasis is reliably produced in pullets and laying hens by feeding 4- to 8-week-old pullets diets containing more than 3% calcium and less than 0.45% available phosphorus. This is commonly the case when laying rations are fed to pullets before they are old enough to handle these levels of nutrients.

There are three methods to avoid the nutritional factors that predispose laying chickens to urolithiasis:

1. withhold diets high in calcium and low in phosphorus until pullets are old enough to handle these levels of nutrients;
2. increase phosphorus levels to about 0.6 % available phosphorus to reduce the calcium/phosphorus imbalance. Because phosphorus is expensive, this method is often neglected; or
3. acidify the urine of the chicken to create conditions that do not favor the formation of stones. This has been accomplished with ammonium chloride, although the acidosis produced produces polydipsia, polyuria, and wet litter. Better effects were obtained with the addition of 0.6% methionine hydroxy analog to an otherwise complete diet. This acidified urine leads to improved excretion of calcium and reduced urolithiasis.

Hypovitaminosis A ("Nutritional Roup")

Birds don't obtain Vitamin A from plants, but obtain its precursors, the Carotenoids. These are generally yellow/orange pigments of which beta carotene is the most important.

Vitamin A is required for -

- Vision
- Normal growth and repair of epithelial tissue
- Disease resistance
- Reproduction
- Growth, especially of bones

Clinical signs

Signs of vitamin A deficiency depend on which organ system is affected (the reproductive, digestive, or respiratory tracts) and which microorganism or combination of microorganisms is invading the patient.

The respiratory system is the most often affected. At first small white plaques appear on the roof of the mouth and/or at the base of the tongue. The plaques may become infected, forming large abscesses. The abscesses can distort the glottis, causing laboured breathing and eventually mechanical suffocation. The choana may be blocked and when this happens the bird shows a profuse nasal discharge and swelling of the facial sinus below the eyes.

Birds also show sneezing, wheezing, nasal discharge, crusted or plugged nostrils, unthriftiness lethargy, depression, diarrhoea, tail-bobbing (due to respiratory difficulty), poor feather color, swollen facial sinuses, ocular discharge, lack of appetite, gagging, foul-smelling breath and "slimy mouth".

Because the epithelial tissues are compromised, secondary bacterial infections commonly occur. Thus the life-threatening bacterial infections must be treated at the same time as the vitamin A deficiency.

Poultry

Usually appears in chicks or poults under 7 weeks of age. If the starter ration is markedly deficient in vitamin A, chicks become stunted and the growth rate declines very rapidly after about 3 weeks. Affected chicks are unthrifty, droopy, ataxic and have ruffled feathers. As the deficiency advances, they develop nasal discharge, the facial sinuses enlarge, and pustules form in the mouth, oesophagus and crop, with a sticky exudate from the nostrils and eyes. The lower eyelids jut outward and a cheesy exudate may accumulate behind them. Mortality may reach 100 percent if the deficiency is not corrected. Vitamin A deficiency in poults is usually more severe than in chicks. In adult poultry inadequate vitamin A causes production decline and reduced hatchability.

In laying hens there is a decreasing egg production and unthriftiness. Inflammation of the eyes or sinuses and the eyes and sinuses may be swollen. Mucoid or cheesy exudate accumulates in the conjunctival sac. Nasal or ocular discharge may be present.

In poultry, the term “nutritional roup” refers to clinical signs characterized by whitish pustules, patchy thickenings (keratinization), and stringy exudate on epithelial surfaces. These lesions are most obvious on the linings of the nasal passages, pharynx, and esophagus. The patchy thickenings are squamous epithelial cell metaplasia, or keratinization. Other findings include urate deposits in the kidneys, ureters, cloaca, and bursa of Fabricius. The condition is reversible by correcting the vitamin A deficiency, especially when detected before severe kidney damage occurs.

Lesions

In young birds: conjunctivitis with a sticky exudate. Excessive urates in the ureters, in collecting tubules of the kidneys and bursa of Fabricius.

Laying hens: 1-3 mm pustule-like lesions in the mucosa of the mouth, pharynx, esophagus and crop. Mucoid exudate in nasal passages. Conjunctival sacs or sinuses contain mucoid or caseous exudate and may be distended. There may be a delicate pseudomembrane lining the trachea.

Diagnosis

Based on clinical signs, dietary history, lesions and response to vitamin A. The disease should be differentiated from trichomoniasis and candidiasis.

Treatment

If severe, IM vitamin A. Blocked sinuses may need to be cleared by removal of keratinized material. Ensure diet contains vitamin A 4000 IU/kg feed. Disease stresses such as coccidiosis, fungal infections, worms, and other intestinal disorders increase the need for vitamin A above the minimum requirement. It is possible to overdose with vitamin A - the signs are similar to those of deficiency.

Avoid storing feed for long periods.

Vitamin A supplementation is recommended as adjunct therapy for infections of the gastrointestinal tract.

Encephalomalacia ("crazy chick disease")

This is caused by a deficiency of vitamin E. This can result from:

1. long term unsupplemented all-seed diets;
2. maldigestion or malabsorption syndromes;
3. diets high in polyunsaturated fats; or
4. ingestion of rancid fat or oil.

A deficiency of vitamin E can produce:

- White muscle disease - a muscular dystrophy - see **exudative diathesis**.
- Low hatchability due to a weakness in the pipping muscle of the chick
- Splayed legs
- Brain disfunction - incoordination
- Infertility
- Oedema - swelling around the neck, wings or breast - see **exudative diathesis**

Signs

1. paralysis or paresis;
2. tremors, staggering, stumbling, incoordination and torticollis;

Lesions

Swollen cerebellum with congested, haemorrhagic or necrotic areas. In turkeys poliomalacia of the lumbar spinal cord may occur.

Treatment

1. There is no cure for an encephalomalacia-affected chick;
2. Flock treatment with vitamin E will prevent new cases.
3. Supportive therapy with additional vitamin E;
4. Addition of antioxidants in the feed to prevent oxidation (loss) of vitamin E.

Exudative Diathesis, Muscular Dystrophy

Vitamin E deficiency contributes to metabolic disorders other than encephalomalacia, namely exudative diathesis, muscular dystrophy, and lowered reproductive performance of breeding hens. Deficiency of vitamin E has a metabolic role in these conditions but is not singly responsible as in encephalomalacia. Inorganic selenium can prevent muscular dystrophy.

Signs

Oedema along the ventrum of the thorax, abdomen and mandible. The edematous skin is red-black or blue-black. Oedema causes difficulty in walking. The birds may have locomotor problems.

Lesions

Blood-stained oedema in the skin and subcutis. In domestic fowl chicks there are white to yellow muscle fibres in skeletal muscles of the breast or legs. In poults, the musculature of the gizzard may contain gray areas of muscle degeneration.

Gout

Gout is seen in man and the apes but not in other mammals. Two forms of gout are seen in birds: articular and visceral. Articular gout is the extracellular deposition of urate crystals in and around the joint spaces, producing a specific arthritis.

Any joint may be affected. Visceral gout is the extracellular deposition of urates on the serosal membranes of the liver and kidney. Articular and visceral gout do not occur in the same individual - it is one or the other. Birds and some reptiles are uricotelic and lack the enzyme uricase, thereby leaving uric acid as the main end product of nitrogen metabolism, and making birds susceptible to developing gout..

Articular gout

Spontaneous articular gout is relatively uncommon in poultry and appears to have a heredity basis. Gross lesions are usually soft swellings around the feet, especially around the metatarsophalangeal and interphalangealjoints, though other sites, including the skin can be involved. Incision of the gouty joints reveals a semisolid paste-like material that contains much urates.

In domestic fowl lines highly susceptible to developing gout were fed 80% protein for 4 weeks, the chickens developed tophi, and at no time was any visceral gout observed. When the birds were returned to a 20% diet, the tophi regressed. Chicken lines which normally have high levels of plasma uric acid have plasma uric acid levels 3 times higher than chicken lines with normal plasma uric acid levels, irrespective of the concentration of protein in the diet. In addition, renal excretion of uric acid in genetic lines with normally high plasma uric acid levels is only 40% that of normal lines.

Besides plasma uric acid (PUA) levels and protein concentration of the diet, other factors may contribute to gout. Increasing dietary sodium or potassium levels increases plasma uric acid while increasing dietary chloride decreases plasma uric acid. The feeding of animal protein also increases PUA levels, whereas vegetable protein does not.

Dietary treatment of articular gout in susceptible birds should include reducing protein levels to less than 20% and preferably to less than 12%, reducing sodium and potassium levels each to 0.3% to 0.15% in adults, and maintaining chloride to not less than 0.45%.

Affected caged birds and raptors will fly reluctantly and act lame when at rest, shifting their weight from one leg to the other. On palpation, one may feel doughy swelling around the joints and tendon sheaths. The feet may be swollen and look like bumblefoot.

Visceral gout

Visceral gout is differentiated from articular gout by the presence of chalk-like urate deposits in the viscera and a lack of tophi in the joints. While urate deposits may be present in the joints, the characteristic tophi of articular gout are not present.

Uric acid is insoluble in water, so that it must be maintained in solution in the blood by some agent other than its solubility, perhaps by carrier proteins or some other mechanism. Since gouty deposits are not usually seen in blood vessels but instead on serous surfaces, the solubility of uric acid in blood must remain intact even when gout occurs.

Visceral gout may be divided into two forms:

5. nephrotoxic, where impairment of kidney function takes place due to chemical insult; and .

6. obstructive. Where obstruction of the ureters occurs, blocking urine flow and leading to a failure of uric acid excretion. While some nutritional regimes can lead to obstruction of the ureters, the effect of nutrition on visceral gout is usually not primary. Treatment can include the dietary regime recommended for articular gout, but resolution of the primary insult is essential.

As the disease progresses, there may be an increased water intake. Affected raptors may stand in their water bowl and drink continuously, before becoming lethargic and dying.

Treatment

1. Food and water containers should be placed within easy reach;

2. Lower the protein content of the food.
3. Provide vegetables and fruit;
4. Supplementation with vitamin A;
5. Oral administration of 1% sodium bicarbonate solution
6. Colchicine (*Colgout* - Aventis Pharma Pty Ltd - 0.5 mg tabs)) is also effective in acute gout and a clear response to this drug may be valuable in supporting the diagnosis.

   The dose is 0.04 mg/kg SID increasing gradually to BID. Be aware that toxicity (hypertension, hypothermia, muscle weakness) can occur as the dose is increased. Colchicine inhibits the tubular excretion of penicillins and potentiates CNS depressants.

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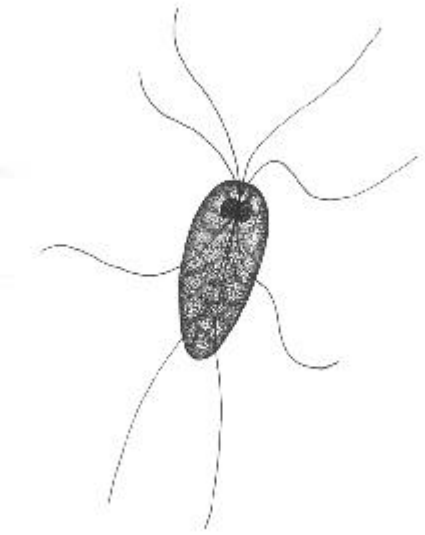
Hexamitiasis

Definition: A protozoan disease of turkey poults, ducks pigeons, gamebirds and king parrots characterised by acute to chronic catarrhal enteritis and a foamy/watery diarrhoea.

Aetiology: *Hexamita meleagridis* in galliform birds. *H. columbae* in pigeons.

Epidemiology: Hexamita inhabits the small and large intestines, particularly the caeca, caecal tonsils and bursa of Fabricius. It reproduces by binary fission and a cystic stage is shed to the environment. It is a common parasite of poultry,. Transmission is via the faecal oral route. Recovered birds are often carriers and shed the parasite in their faeces which contaminate the environment. Susceptible birds then ingest the parasite. When consecutive broods of poults are reared on the same premises, and management is poor, the disease seems to get worse with each successive batch.



Signs: Affected poults are very nervous and have ruffled feathers, a stilted gait and watery diarrhoea. They continue to eat but lose weight. They huddle together and eventually die. King parrots have a history of a chronic, intractable diarrhoea, with loss of weight. In pigeons, a loss of flight performance is recorded..

Lesions: Dehydration, emaciation. The intestine is flabby and ballooned and the contents are watery. King parrots have a greatly thickened intestine, akin to a plastic hose.

Diagnosis: Identify the organism. Examine a **freshly prepared** intestinal smear **IMMEDIATELY**. If you make the smear and then answer the telephone, you will not see any motile organisms when you return. You are unlikely to see any motile organisms in a bird that has been dead for more than an hour. Differentiate from coronaviral enteritis (turkeys), capillariasis (king parrots, pigeons). *Hexamita* swims in a rapid, smooth, linear fashion and can be difficult to maintain in the field of view.

Treatment: Spartrix Boehringer Ingelheim Pty Ltd. Carnidazole 10mg. ½ tablet squab, 1 tablet adult. Dose 30mg/kg. Single dose.
Carnidazole 20 mg/kg body weight once in combination with ronidazole in water for 7 days.
Ronivet Vetafarm. Ronidazole 4%. 6-10 mg/kg orally SID for 6-10 days.
Emtryl Mill Pack Rhone-Poulenc Animal Nutrition Pty Ltd. Dimetradizole 100%. 3g/5L drinking water 7 days. Lorikeets and finches - 1.5g/5L.
Flagyl S Suspension May & Baker Pharmaceuticals. Benzoyl metronidazole. Metronidazole 40mg/mL, 100mL. Dose 50-60 mg/kg orally SID for 5 ys.

Control:

1. Short periods of depopulation with thorough cleaning and disinfection of premises.
2. Single age flocks.
3. Prevent contact of cage birds with wild birds.
4. Antihistomonal drugs will prevent hexamitiasis.

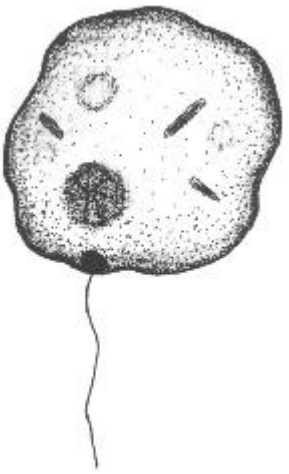
Histomoniasis

Definition: A protozoan disease of gallinaceous birds characterised by inflammation of the caeca and liver.

Synonyms: Blackhead, infectious enterohepatitis, enterohepatitis.

Aetiology: *Histomonas meleagridis*, assisted by anaerobic bacteria.

Epidemiology: The disease may seriously affect turkeys, grouse and peafowl of all ages, but mainly less than 12 weeks. The infection usually remains latent in chickens, guinea-fowl, quail and pheasant. The organism has an extracellular, asexual life cycle - it multiplies by binary fission. Histomanads have a non-amoeboid form and are then nearly spherical with a diameter of 3-16µm. Most of the time they are amoeboid with a flagellum 6-11µm long. The caecal worm, *Heterakis gallinarum*, acts as a paratenic host enabling the flagellate to pass the acid environment of the proventriculus.



Signs: Increased thirst, decreased appetite, brownish-black to watery yellow droppings, drowsiness and weakness. The skin of the head may be black ("blackhead") but are more often not. In chickens there may be some blood in the faeces.

Lesions: Caecal wall becomes swollen, hyperaemic and oedematous. Blood and fibrinous exudate enters the caecal lumen. If the caecal wall ulcerates a generalised peritonitis will result. Caecal cores eventually form, if the bird survives. From the tenth day after infection yellowish, circumscribed, depressed, characteristic ring lesions develop in the liver. Infected chickens usually lack the liver lesions.

Diagnosis: History, signs, lesions, presence of causative organism. Look at the **edges** of a caecal lesion - straight examination in saline on a glass slide. Differentiate from TB, caecal coccidiosis, salmonellosis,

Prognosis: With treatment in the early stages, excellent.

Treatment: **Treatment**: Turkeys - dimetradizole (*Emtryl*) 6-12g/10L drinking water for up to 12 days. The response is slow to minimal. Can go to 2g/L for 5-7 days. Emtryl in feed 500ppm.
Others - 3g/5L drinking water 7 days.

Control:

1. Do not run turkeys (or other susceptible birds) and chickens on same range.
2. Ensure that turkey range has been free of chickens for at least 4-5 years.
3. Worm turkey flocks regularly to suppress *Heterakis gallinarum*. Levamisole 300ppm 1 day, mebendazole 60ppm 6 days.
4. Rotate turkey range if possible.
5. Prevent access to earthworms.
6. Add histomonastat to ration:

Emtryl Mill Pack Dimetridazole (100%). Rhone-Poulenc Animal Nutrition Pty Ltd. In feed turkeys 125-250ppm. Chickens 75ppm.

Coccidiosis

Definition: Coccidiosis is an enteropathy due to infection by one or more species of host-specific coccidia. It is usually a disease of young birds kept under crowded warm, moist conditions which allow the rapid build up of sporulated oocysts in the environment.

Aetiology: The species of coccidia affecting chickens and turkeys are:

CHICKENS	TURKEYS
*Eimeria tenella**	*Eimeria adenoides**
*Eimeria necatrix**	*Eimeria meleagrimitis**
*Eimeria acervulina**	*Eimeria gallopavonis**
*Eimeria brunetti**	*Eimeria meleagridis*
*Eimeria maxima**	*Eimeria dispersa*
Eimeria mivati	*Eimeria innocua*
Eimeria hagani	*Eimeria subrotunda*
Eimeria praecox	

* Considered the major cause of clinical outbreaks

The genera *Isospora, Tyzzeria and Wenyonella* are associated with coccidiosis in wild birds. In these the infections are usually asymptomatic, either because the species of coccidia are not pathogenic, or because the bird has developed immunity in the face of low level challenge. Coccidiosis is rarely seen in psittacine or passeriform birds but can affect columbiform birds.

The various coccidia are differentiated as follows:

GENUS	SPOROCYSTS	SPOROZOITES
Eimeria	4	2
Isospora	2	4
Wenyonella	4	4
Tyzzeria	-	8
Cryptosporidium	-	4

Geese - *E. truncata* - nephritis
Duck - *Tyzzeria perniciosa* - anterior SI (may involve all SI).

Epidemiology: Coccidia are present in the litter of all used chicken houses, and may be transported to houses by humans, equipment, animals and insects. Susceptible chickens eat sporulated oocysts and become infected. If the infection is moderate, the bird may not show signs and will become immune, excreting oocysts into the environment whenever reinfection occurs. Outbreaks of clinical coccidiosis occur whenever susceptible birds ingest large numbers of sporulated oocysts. Coccidia require 6-8 days to complete their life cycle in the chicken, and signs do not appear until days 4-6 after infection. There is no breed incidence, and outbreaks occur all year round if birds are reared in confinement. All ages are susceptible if immunity has not developed. Non-immune birds are generally young birds. The tables show typical life cycles for *E. tenella.*

Signs: Clinically, coccidiosis is well recognised with diarrhoea (with or without blood or mucous) as the primary clinical sign. Weight loss, dehydration, depression, ataxia, incoordination and sudden death are common sequelae. A soiled vent and swollen abdomen (due to distended intestines) may also be seen. The diarrhoea may be mild to severe and will range from bloody and greenish to watery and brown. The first signs are depression, huddling and ruffled feathers. Wet, blood-stained or mucous-containing faeces are commonly seen. Chickens sometimes let out a cry during acute attacks of diarrhoea. Affected birds are dehydrated (diarrhoea) and anaemic (blood loss). Birds which survive will take 1-2 weeks to recover, but never make up the production loss.

Lesions: Enteritis, typhlitis. Nephritis in geese (*E. truncata*). Ballooned small intestine that has erythematous mucosa and haemmorhages or it may appear normal. Impression smears of the intestine from several sites will reveal oocysts, as long as the infection has reached the stage of oocyst formation..

Diagnosis: Signs and lesions, demonstration of oocysts. Blackhead, pullorum disease and paratyphoid could be confused with chronic E. tenella infection in chickens. Necrotic enteritis and acute capillariasis could be confused with upper and mid-intestinal coccidiosis in chickens. In the chicken, diagnosis is based on the portion of intestine affected; the lesions and oocyst size, shape, colour.

Anterior a: *E. acervulina*. Causes enteritis in the proximal **a** of the small intestine. (*mivati, praecox* and *hagani* also affect this part of the intestine, but are relatively apathogenic). In severe cases lesions may extend further down the tract. The enteritis can be mild or severe, leading to marked thickening of the mucosa. Transverse white to grey striations (plaques) may be seen in the mucosa but may go unrecognised if they have coalesced.

Middle a: *E. necatrix* causes a severe enteritis in the middle **a** of the small intestine, and throughout the intestine if the infection is severe. The intestine is often dilated ("ballooned"), purplish-red and covered in petechial haemorrhages and white to yellow foci (schizonts). Oocysts develop in the caeca, but mortality usually precedes formation of oocysts. High mortality.

E. maxima causes a mild to severe enteritis in the middle **a** of the small intestine, sometimes with thickening and ballooning of the intestinal wall. Very large oocysts present.

Posterior a: *E. brunetti* affects the lower small intestine, rectum and proximal caeca. Caseous cores may be present in the caeca and rectum. *E. tenella* causes a marked typhlitis, with blood in the caeca and faeces in early cases and cheesy caecal cores containing numerous oocysts in later stages (days 8-10).

In the chicken, coccidiosis needs to be differentiated from:

maxima, necatrix	- ulcerative enteritis,
acervulina	- enteritis
chronic tenella	- histomoniasis, salmonellosis
brunetti	- necrotic enteritis

In the turkey, the following areas of the small intestine are infected by the respective coccidia:

E. meleagrimitis	-	anterior SI
E. adenoides	-	lower SI and caecae
E. meleagridis	-	caecae (meronts mid SI)
E. gallopavonis	-	rectum, posterior SI

Prognosis: Good on flock basis, with treatment.

Treatment: Use a coccidiocide such as Amprolium plus or Baycox.