

Important Poultry Diseases

Important Poultry Diseases

Research makes the difference



060058 © 2009, Intervet International bv, The Netherlands.

Intervet International bv
P.O. Box 31, 5830 AA Boxmeer
The Netherlands
Phone +31 (0)485 587600
Fax +31 (0)485 577333
E-mail info@intervet.com
www.intervet.com

IMPORTANT POULTRY DISEASES

Contents

<i>Foreword</i>	3
<i>Infectious respiratory Diseases</i>	
Chronic Respiratory Disease	7
Coryza	10
Aspergillosis	12
Newcastle Disease	14
Infectious Bronchitis	16
Infectious Laryngotracheitis	18
Avian Influenza	20
Pneumovirus	22
<i>Neoplastic Diseases</i>	
Lymphoid Leucosis	27
Marek's Disease	29
<i>Avian Adenoviral Diseases</i>	
Inclusion Body Hepatitis	35
Egg Drop Syndrome '76	38
<i>Miscellaneous viral Diseases</i>	
Fowl Pox	43
Avian Encephalomyelitis	45
Infectious Bursal Disease	47
Reovirus infection	49
Malabsorption Syndrome	51
Infectious Anaemia	53
<i>Miscellaneous Bacterial Diseases</i>	
Infectious Synovitis	57
Fowl Cholera	59
Pullorum Disease/Fowl Typhoid	61
Ornithobacterium Rhinotracheale infection	63

Infectious Viral Diseases of ducks

Duck Virus Hepatitis	67
Duck Plague	69

Parasitic Diseases

Coccidiosis	73
Endoparasites	78
Blackhead	80

Deficiency Diseases

Riboflavin	85
Vitamin E	86
Vitamin D3	87

Foreword

We first published the "Important Poultry Diseases " in 1972 and, ever since, it has remained the most sought after of all our publications.

However, in the intervening years new causes of disease such as chicken anemia virus and pneumovirus were identified. Therefore, Intervet Research teams developed new vaccines to combat the changed pattern of diseases. Many of these are inactivated vaccines, in either single or combined form, resulting in new vaccination programmes. We have, therefore, produced this fourth edition of the handbook to cover these developments. It is intended as a pocket reference book for people working with poultry.

It is not highly academic as it is written from a practical angle to assist with every day problems in the field.

Intervet Research continues to co-operate with the poultry industry, worldwide, to stay ahead of disease problems.

The farmer who uses an Intervet vaccine can do so knowing that it is the best product available.

For detailed information on any of our products contact the local Intervet representative or

INFECTIOUS RESPIRATORY DISEASES

- *Chronic Respiratory Disease (CRD)*
- *Coryza*
- *Aspergillosis*
- *Newcastle Disease (ND)*
- *Infectious Bronchitis (IB)*
- *Infectious Laryngotracheitis (ILT)*
- *Avian Influenza*
- *Turkey Rhinotracheitis/Swollen Head Syndrome*

Chronic respiratory Disease

(CRD) (Airsacculitis)

Cause

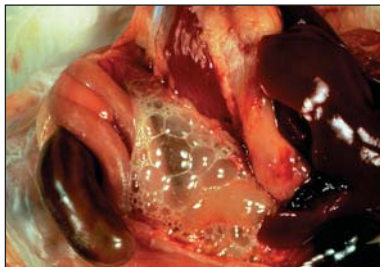
The underlying cause of CRD is *Mycoplasma gallisepticum* (Mg). The condition is frequently triggered by respiratory viruses such as ND and IB and subsequently complicated by bacterial invasion. The main agents involved in the infection are *Mycoplasma gallisepticum* and *E. coli*. Stress caused by moving the birds, by debeaking or other operations or other unfavorable conditions e.g. cold or bad ventilation, make the birds more susceptible.

Transmission

The main problem is that parent birds infected with *Mycoplasma gallisepticum* can transmit the organism through the egg to their offspring. In addition, infection can occur by contact or by airborne dust or droplets. The incubation period varies from 4 days to 3 weeks.

Species affected

Chickens and turkeys.



Airsacculitis

Clinical signs

Young chickens (broiler chicks or layer pullets) will show respiratory distress. The birds frequently show a lack of appetite, decreased weight gain and increased feed conversion ratios.

In adult birds the most common symptoms are sneezing, coughing and general signs of respiratory congestion. In laying birds a drop of egg production between 20-30 % can occur.

CRD does not normally cause an alarming number of deaths. The effect is more of a chronic nature causing reduced weight gain and feed conversion ratios in broilers and lower egg production in breeders and layers. In this way the overall economic loss can be very great in broilers but less dramatic in breeders and layers.

Internal lesions

A reddish inflamed trachea and/or cheesy exudate in airsacs, especially in complicated cases (e.g. with secondary *E. coli* infections) are observed. In mild Mg infections the only lesion might be slight mucus in trachea and a cloudy or light froth in the airsacs.

Turkeys with Mg infection usually have swollen sinuses under the eyes.

Diagnosis

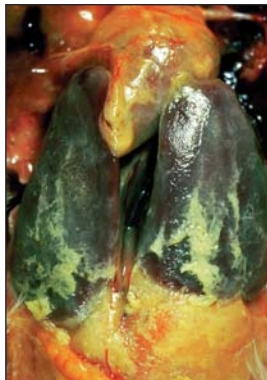
Diagnosis of Mg infection can be made by blood testing of chickens, post-mortem examination and ultimately by isolating the causative Mg organism from tracheas or airsacs of affected birds.

Differential diagnosis

Respiratory virus infection (Newcastle disease or infectious bronchitis) with secondary infection (*E. coli*, etc.) can give similar lesions.

Treatment

Treatment of Mg-infected chickens or turkeys with suitable antibiotics or chemotherapeutics has been found to be of economic value. However, control by medication or vaccination and eradication of Mg infections has been by far the most effective method of combating the disease. Fertile eggs from infected birds can be treated with antibiotics such as tylosin to eliminate the *Mycoplasma gallisepticum* organisms. Methods used are the injection of fertile eggs or egg dipping. Blood serum testing of breeder chickens for Mg antibodies has become a routine to test flocks for a Mg infection.



Pericarditis, peritonitis and perihepatitis is frequently observed in birds with CRD

Infectious Coryza

Cause

The bacterium causing this disease is *Hemophilus paragallinarum*.

Transmission

The disease spreads from bird to bird and flock to flock by contact and airborne infected dust particles and via the drinking water.

Spread by equipment and personnel has also been reported. The incubation period varies from 1 to 3 days.

Species affected

Chickens appear to be the only natural hosts of *H. paragallinarum*.

Clinical signs

The main signs of the disease are inflammation of eyes and nose with foul-smelling discharges, conjunctivitis, sneezing and facial swellings. Feed and water intake is reduced, leading to loss of weight. Egg production in laying birds will drop.

Mortality will vary with the virulence of the infection but is generally low.

Diagnosis

A field infection produces similar symptoms to chronic respiratory disease, a diagnosis is difficult to establish. The most certain diagnosis may be obtained by the isolation of the organism from the sinus or airsac exudate from affected birds. This procedure must be carried out in the laboratory.

Treatment and control

Treatment with antibiotics can be given to subdue clinical infection, but eradication and prevention are the most desirable means of control of coryza. Vaccines have been developed, but are only used in areas where the disease is endemic and cannot be eradicated.



Typical facial edema

Aspergillosis (Fungal Pneumonia)

Cause

The disease is caused by a fungus, *Aspergillus fumigatus*.

Transmission

Transmission is by inhalation of fungus spores from contaminated litter (e.g. wood shavings) or contaminated feed. Hatcheries may also contribute to infection of chicks.

Species affected

Young chicks are very susceptible, older chickens are more resistant to infection. Turkey poults, pheasant chicks, quail chicks, ducklings, and goslings may also become infected.

Clinical signs

Infected chicks are depressed and thirsty. Gasping and rapid breathing ("pump handle breathing") can be observed.

Mortality is variable, from 5 to 50 %.

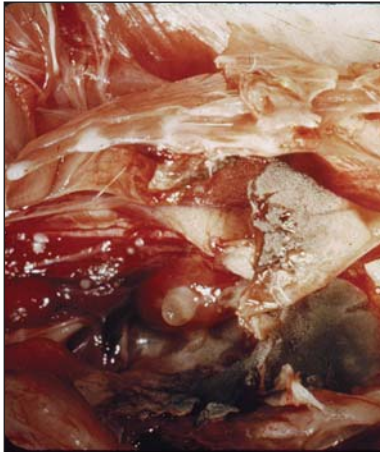
Gross lesions involve the lungs and airsacs primarily. Yellow-white pinpoint lesions can be found. Sometimes all body cavities are filled with small yellow-green granular fungus growth.

Diagnosis

The presence of *Aspergillus fumigatus* can be identified microscopically or sometimes even with the naked eye in the air passages of the lungs, in the airsacs or in lesions of the abdominal cavity.

Treatment and control

There is no treatment for aspergillosis. Affected chicks should be removed and destroyed. Strict hygiene in breeder and hatchery management is necessary. Choice of litter material is important so that no spore-bearing wood shavings are used.



*Gross lesions
of the lungs*

Newcastle Disease (ND)

Cause

Newcastle disease is caused by a paramyxovirus. Only one serotype of ND is known. ND virus has mild strains (lentogenic), medium strength strains (mesogenic), and virulent strains (velogenic).

The strains used for live vaccines are mainly lentogenic.

Transmission

Newcastle disease virus is highly contagious through infected droppings and respiratory discharge between birds. Spread between farms is by infected equipment, trucks, personnel, wild birds or air. The incubation period is variable but usually about 3 to 6 days.

Species affected

Chickens and turkeys.

Clinical signs

Newcastle disease causes high mortality with depression and death in 3 to 5 days as major signs. Affected chickens do not always exhibit respiratory or nervous signs.

Mesogenic strains cause typical signs of respiratory distress.

Labored breathing with wheezing and gurgling, accompanied by nervous signs, such as paralysis or twisted necks (torticolis) are the main signs. Egg production will decrease 30 to 50 % or more, returning to normal levels in about 2 weeks. Eggs may have thin shells and eggs without shells may also be found. In well-vaccinated chicken flocks clinical signs may be difficult to find.

Internal lesions

Inflamed tracheas, pneumonia, and/or froth in the airsacs are the main lesions. Haemorrhagic lesions are observed in the proventriculus and the intestines.

Diagnosis

Is made by virus isolation from tracheal or cloacal swabs together with blood testing to demonstrate high antibody levels. Infectious bronchitis or infectious laryngotracheitis can give similar clinical signs, but lesions, blood tests, and virus isolation tests are decisive.

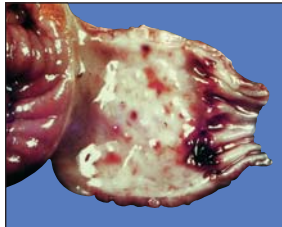
Treatment and control

There is no treatment for Newcastle disease. Vaccination against ND with live and/or inactivated (killed) adjuvant vaccines is the only reliable control method.

Neurotropic form of ND



Haemorrhagic proventriculus



Infectious Bronchitis (IB)

Cause

Corona-virus is the causal agent. Several different serotypes of IB virus are known to exist.

Transmission

The virus is transmitted from bird to bird through the airborne route. The virus can also be transmitted via the air between chicken houses and even from farm to farm.

Species affected

Only chickens are susceptible to IB virus.

Clinical signs

In young chicks IB virus infection causes a cheesy exudate in the bifurcation of the bronchi, thereby causing asphyxia, preceded by severe respiratory distress ("pump handle" breathing). In older birds IB does not cause mortality. Egg production will decrease dramatically, deformed eggs with wrinkled shells will often be laid.

*Respiratory
symptoms of IB
in chickens*



Internal lesions

Mucus and redness in tracheas, froth in airsacs in older chickens. In young chicks a yellow cheesy plug at the tracheal bifurcation is indicative of IB infection.

Diagnosis

There are three main factors to be considered in order to arrive at a diagnosis.

- a. The clinical picture including post-mortem findings in the flock.
- b. Isolation of the virus in the laboratory.
- c. A rising antibody titre when the serum is tested against a known strain of bronchitis virus.

Treatment and control

There is no treatment for infectious bronchitis.

Secondary bacterial infections may be prevented by, or treated with antibiotics.

Prevention by vaccination is the best method to control IB.



Misshapen, shellless and normal eggs



Infectious Laryngotracheitis

(ILT)

Cause

ILT is caused by a virus belonging to the herpes group. Only one serotype is known.

Transmission

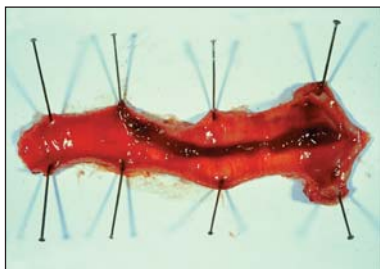
Field infection occurs from bird to bird by the respiratory route. Most outbreaks of ILT on farms are traced back to transmission by contaminated people or equipment (visitors, shoes, clothing, egg boxes, used feeders, waterers, cages, crates etc.). The incubation period varies from 4 to 12 days.

Species affected

Chickens and pheasants are natural hosts for ILT.

Clinical signs

Respiratory distress is usually quite pronounced due to build up of blood, sloughed tracheal lining and even caseous exudate in larynx and trachea. When a caseous plug occludes the larynx or trachea, the affected chickens will have extreme difficulty breathing ("pump handle" breathing) and will frequently die from suffocation. Mortality is approximately 1 % per day in a typical ILT outbreak. Milder forms of ILT outbreaks occur where less virulent strains of ILT virus are involved. Conjunctivitis and respiratory sounds (wheezing) can be observed, with little or no mortality in such cases.



*Haemorrhagic
lesions in trachea*

The disease spreads through a chicken house more slowly than either IB or ND. Egg production in laying flocks will usually decrease 10 to 50 %, but will return to normal after 3 to 4 weeks.

Diagnosis

In a chicken flock, spreading of respiratory distress, with possible coughing up of blood and mortality is indicative of ILT. Bloody mucus and cheesy exudate can be found in larynx and trachea. In the laboratory a definite diagnosis can be made by histological examination of tracheal tissues or virus isolation from tracheal mucus in embryonated chicken eggs.

Treatment and control

Prevention of ILT by vaccination with mild eye-drop vaccine is by far the best control method. Sometimes such vaccines are applied by drinking water or spray methods with variable success. Even when an outbreak of ILT has been detected in a chicken flock, immediate vaccination is advisable to stop the spread of infection.

*Some birds show
symptoms of gasping
with the head extended
and the beak open*



Avian Influenza

Cause

Avian influenza is caused by a myxovirus. There are several serotypes.

Transmission

Airborne virus particles from the respiratory tract, droppings, and people-carrying virus on their clothing and equipment are the main routes of transmission.

Species affected

Turkeys and ducks are mainly affected but chickens, geese, and wild birds can also be infected.

Clinical signs

Clinical signs may vary, depending on the type of influenza virus. Respiratory disease with mortality in turkeys has been observed, but a drop in egg production without clinical signs has also been seen in chickens. Swelling of the head and neck, swollen sinuses with nasal discharge can be seen with respiratory involvement.

Mortality is usually low. Fowl plague, also an avian influenza, is an exception to the rule in that it causes high mortality in turkeys and chickens.



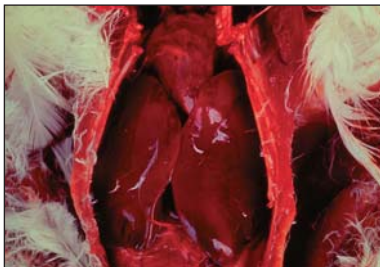
Influenza in turkeys

Diagnosis

A laboratory diagnosis is necessary by serological (agar gel precipitation AGP) or virological methods (virus isolation). Avian influenza can be confused with Newcastle disease, fowl pox, Mycoplasma infection, Staphylococcus, or other respiratory or systemic infections.

Treatment and control

There is no treatment for avian influenza. Antibiotics will help prevent secondary bacterial infections.



*AI infected heart
and proventriculus*

Pneumovirus Infections

(Turkey Rhinotracheitis/Swollen Head Syndrome)

Cause

The disease is caused by a pneumovirus.

Transmission

The virus may be transmitted horizontally by contaminated water, personnel and equipment as well as from bird to bird.

Species affected

Turkeys and chickens.

Clinical signs and lesions

In young turkeys sneezing. Rales and nasal discharge, conjunctivitis, swelling of the infraorbital and submandibular sinuses can be seen.

In laying flocks a drop in production may occur along with respiratory distress. Morbidity is high whereas mortality may vary being usually higher in young poults. In chickens the pneumovirus may be involved in the so called "swollen head syndrome" (SHS). In such cases affected chickens may show swelling of the periorbital and infraorbital sinuses, torticollis, cerebral disorientation and depression. Marked egg production losses can be associated with SHS.

At necropsy the lesions seen may vary due to other microorganisms that may complicate the original picture. In cases of SHS apart from oedema in the head also purulent or caseous subcutaneous exudate can be found. Rhinitis, tracheitis and sinusitis are frequently noted in both chicken and turkeys, hence also the name turkey rhinotracheitis (TRT).

Poliserositis affecting the air sacs and pericardium may be due to secondary infections (*E. coli*). The kidneys may be swollen and congested as well as the lungs which may show a fibrinous exudate in the pleural cavity.

Diagnosis

The diagnosis based only on clinical signs is difficult to establish since other agents may be involved. The most certain diagnosis may be obtained by the isolation of the organism from nasal secretions or tissue scraped from the sinus of affected birds. Antibodies can be detected by several serological methods such as the VN test, IFT and ELISA.

Treatment and control

Treatment with antibiotics can be given to control secondary bacterial infections. The use of vaccines should be the best approach to control the disease.

*Young turkey
with conjunctivitis
and sinusitis*



*Swollen head
syndrome*



NEOPLASTIC DISEASES

- *Lymphoid Leucosis*
- *Marek's Disease*

Lymphoid Leucosis

(LL, Big Liver Disease, Visceral Leucosis)

Cause

Lymphoid leucosis (LL) is caused by a retro (leuco) virus.

Transmission

The lymphoid leucosis virus appears to be transmitted through eggs. However, horizontal transmission at a young age may occur. Infected breeders can be detected by testing hatching eggs and cloacal swabs for the presence of the virus. In this manner eradication of lymphoid leucosis will be possible.

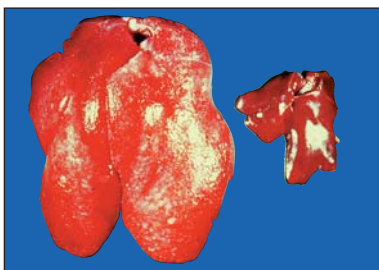
Clinical signs

Visceral tumors are the main feature of lymphoid leucosis. They can be found in liver, spleen, kidneys, and bursa of birds that are in general older than 25 weeks. In affected layer flocks a lower egg production can be observed.

Osteopetrosis is lymphoid leucosis of the bones of legs and wings which become enlarged, but is quite rare. Affected birds have bowed, thickened legs. Lymphoid leucosis can also occur as blood leucosis. Such erythroid and/or myeloid leucemias are also quite rare.

*Lymphoid Leucosis
(big liver)*

*Right: normal
Left: affected*



Affected birds are listless, pale, and are wasting away. Because of the tumours, LL may be confused with Marek's disease, but in LL the nervous system is never involved (no paralysis). LL generally causes birds to weaken, lose weight and eventually die. Histopathological examination is essential for a proper diagnosis.

“J” Virus

Recently, within the known subgroup of exogenous LL viruses (A, B, C and D), a new subgroup denominated “J” has emerged. The new “J” virus shows tropism for cells of the myelomonocytic series, causing tumors, which are identified by histopathology as being myelocytomas. The virus has tropism for meat -type birds. The tumors caused by this virus are normally seen from sexual maturity onward and are frequently located on the surface of bones such as the junction of the ribs, sternum, pelvis, mandible and skull and may be also found in visceral organs.

Treatment and control

No treatment is known. The best control method is the laboratory detection of infected breeders. Breeding leucosis-free offspring from leucosis-free breeders can eventually lead to eradication of the disease.

Marek's Disease

(MD, Neurolymphomatosis)

Cause

Marek's disease is caused by a herpes virus.

Transmission

Main transmission is by infected premises, where day-old chicks will become infected by the oral and respiratory routes. Dander from feather follicles of MD-infected chickens can remain infectious for more than a year. Young chicks are particularly susceptible to horizontal transmission. Susceptibility decreases rapidly after the first few days of age.

Species affected

The domestic fowl.

Clinical signs

Infected birds show weight loss, or may exhibit some form of paralysis. Mortality varies from 5 to 50 % in unvaccinated birds. The classical form (paralysis) with leg nerve involvement causes a bird to lie on its side with one leg stretched forward and the other backward.

When the gizzard nerve is involved, the birds will have a very small gizzard and intestines and will waste away.

Mortality usually occurs between 10 and 20 weeks of age.



MD leg paralysis

Diagnosis

The presence of tumours in liver, spleen, kidneys, lungs, ovary, muscles, or other tissues is indicative of MD, but they can also be indicative of lymphoid leucosis. However, nerve involvement, either grossly (swelling of leg, wing or other nerves) or microscopically, is typical of MD.

Eye involvement can be visible as an irregular constriction of the iris (ocular lymphomatosis).

Skin involvement (skin leucosis) often consists of tumours of feather follicles or in between follicles. Skin leucosis is a reason for broiler condemnation in certain parts of the world. A proper diagnosis to differentiate MD from LL requires histological examination.

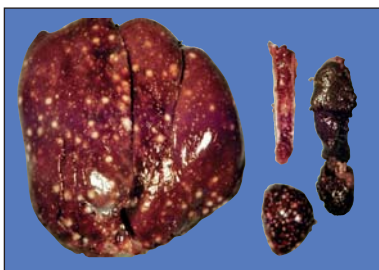
The paralysis is caused by lesions and enlargements of the affected nerves. The picture shows an enlargement of a sciatic nerve



Treatment and control

Vaccination of day-old chicks is an effective mean of control. It has been demonstrated that MD vaccine only prevents the appearance of Marek's disease tumours and paralysis. It does not prevent the birds from becoming infected with MD-virus. It is therefore of major importance to maintain high hygienic and sanitary measures by good management to avoid early exposure of young chickens.

*Tumors may be
observed in the ovaries.
In the acute form
visceral tumours are
most common*



AVIAN ADENOVIRAL DISEASES

- ***Inclusion Body Hepatitis***
(Hydropericardium-Hepatitis Syndrome)
- ***Egg Drop Syndrome 1976***
(EDS '76)

Inclusion Body Hepatitis

(Hydropericardium-Hepatitis Syndrome)

Cause

The disease is caused by an avian adenovirus (for example the Tipton strain) and is usually simultaneously accompanied by other immunosuppressive diseases such as infectious bursal disease or infectious anaemia. There are 12 known serotypes of avian adenoviruses that may be involved in the development of this disease.

Transmission

Egg transmission is an important factor.

Horizontal transmission from bird to bird by contact with droppings.

Once the bird becomes immune, the virus can no longer be isolated from the droppings.

Species affected

Chickens, turkeys and pheasants and possibly other birds can be affected by avian adenovirus.

Clinical signs

Chickens with inclusion body hepatitis are affected at usually 5 to 7 weeks of age. The birds are listless, with ruffled feathers. Mortality is usually quite severe, up to 25 % in the first 10 days of the disease.

Internal lesions

Affected chickens have mottled livers, many with pinpoint necrotic and haemorrhagic spots. Pale bone marrow and, in some cases in presence of infectious anemia, gangrenous dermatitis can be seen. Kidneys are pale and swollen. The spleen is usually quite small (atrophy).

If Gumboro disease (infectious bursal disease) has been present in the birds, even if subclinical, the bursa of Fabricius will be very small (atrophic). Such chickens are immune-suppressed and usually have more severe cases of inclusion body hepatitis and/or infectious anaemia.

Mature birds do not have clinical signs of adenovirus infection, they only start showing antibodies in their blood.

Hydropericardium-Hepatitis Syndrome

HHS was reported for the first time in 1987 in Pakistan and was referred to as “Angara disease”. The disease has meanwhile been reported from several other countries, including India and countries in the Middle East and Latin America.

Hydropericardium-hepatitis syndrome is caused by a virus belonging to the family of the fowl adenoviruses (FAV).

Despite of the diversity in the geographical distribution of the disease, in all areas the infection is caused by a virus belonging to FAV serotype 4. There are three features which underline that this condition is a new disease, different from the known IBH. IBH and hydropericardium accompany this syndrome. Once outbreaks of HHS occur, it remains a problem for the poultry industry. And while IBH is shown to be caused by strains belonging to various FAV serotypes, HHS is, contrary to this, caused by FAV serotype 4. The infected flocks show high mortality rates and beside the lesions typical for IBH, a marked hydropericarditis is found in the affected birds.

Diagnosis

Typical mottled livers with pinpoint lesions, pale bone marrow and kidneys, small spleen and bursa are good indications of the disease. In the case of HHS the typical lesion (hydropericardium) is also found. Histological examination (intranuclear inclusion bodies) of liver and/or virus isolation are helpful means of diagnosis.

Treatment and control

No treatment exists. Antibiotics can be used to prevent secondary bacterial infection and possible gangrenous dermatitis.

The best method of control is to ensure adequate immunity against other immune suppressive diseases (e.g. infectious bursal disease). Chickens may be vaccinated s.c. during the first two weeks of life with an inactivated oil-emulsion vaccine.

*Affected chickens
have mottled livers,
pale bone marrow
and sometimes gang-
renous dermatitis
can be seen*



Egg Drop Syndrome 1976

(EDS '76)

Cause

The disease is caused by an avian adenovirus (strain BC14, virus 127), the EDS virus does not belong to any of the 12 fowl adenoviruses.

Transmission

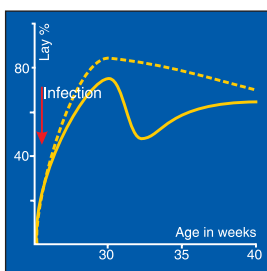
The virus is transmitted through the egg to a few birds in a flock, these birds carry the virus until the flock comes into lay at which time they begin to excrete virus and infect birds kept in the same house.

Horizontal spread through infected litter can and does occur once a flock shows the disease but it seems that the virus is not very infectious or the level of virus excretion is low.

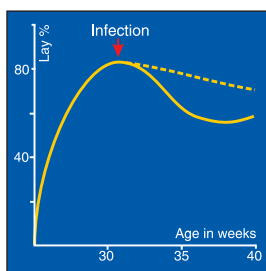
Species affected

Only chickens are susceptible to clinical disease due to EDS virus; however, the virus is widespread in ducks but does not cause any problems.

Non-peaking effect of early EDS '76 infection



Egg drop by infection during lay



Clinical signs

EDS '76 affects only layers and breeders at the start of or during their egg production.

Affected flocks show a failure to reach peak egg production or a drop in egg production accompanied by an inferior eggshell quality and in the case of brown eggs, a loss of shell color.

Affected birds may also appear to be anaemic, may show a transient diarrhoea and sometimes the food intake may be reduced. No increased mortality or other symptoms are observed.

Internal lesions

No specific internal lesions have been observed.

Diagnosis

Clinical signs provide the diagnosis for EDS '76. Virus isolation and antibody tests can confirm this.



*Misformed and
soft shelled eggs*

Differential diagnosis

Infectious bronchitis and to a lesser extent Newcastle disease and infectious laryngotracheitis will have to be considered. Proper diagnostic tests such as the antibody tests will eliminate doubt.

Treatment and control

There is no treatment against egg drop syndrome 1976. Vaccination with an inactivated vaccine before point of lay is the only available, effective method for the control of EDS '76.

MISCELLANEOUS VIRAL DISEASES

- *Fowl Pox*
- *Avian Encephalomyelitis*
- *Infectious Bursal Disease*
- *Reovirus Infectious*
- *Malabsorption Syndrome*
- *Infectious Anaemia*

Fowl Pox

(Avian Pox, Avian Diphtheria)

Cause

Fowl pox is caused by a pox virus.

Transmission

Introduction of infected or “carrier” birds in a susceptible flock will cause an outbreak by direct contact and water or feed transmission.

Mosquitoes and other flying insects can also transmit the virus from bird to bird and also transmit the disease to near-by flocks. The incubation period varies from 4 to 20 days.

Species, affected

Chickens, turkeys, pheasants and pigeons can be affected by different fowl pox virus strains.

Clinical signs

The lesions of fowl pox can be external (mainly on the head) or internal (“wet pox”) in the mouth, oesophagus and/or trachea, they can also be found on other parts of the body (skin of legs, cloaca etc.). The lesions on the head, combs, and wattles are usually wart-like in appearance, yellow to dark brown in color. The internal lesions in the mouth, oesophagus and/or trachea are yellow-white and cheesy in appearance. Affected birds will be depressed, lack appetite and when “wet pox” is present they breath laboriously.

Mortality

Mortality is variable, from a low 1 to 2%, when slight head lesions are present, to over 40% when the diphtheritic form (“wet pox”) is more prevalent.

Reduced egg production can be observed in laying birds, this will return to normal in a few weeks.

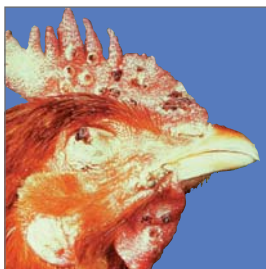
Diagnosis

Wart like lesions of the head particularly of the comb and around the eyes or yellow cheesy lesions of the mucous membranes of the nasal and buccal cavities are suggestive of fowl pox. A definitive diagnosis can be made in a diagnostical laboratory by histological examination (inclusion bodies) or virus isolation in embryonated chicken eggs.

Treatment and control

It is difficult to treat affected birds. Treatment of local lesions with disinfectant and/or removal of the diphtheritic membranes from the throat to improve respiration has been practised.

Preventive vaccination using a live vaccine is by far the most succesful control method. Even when an outbreak of fowl pox has been diagnosed, it is advisable to vaccinate the flock immediately to stop further spread of the infection.



*Fowl pox lesions on the head
and appendages*

Avian Encephalomyelitis (AE) or Epedemic Tremor

Cause

Avian encephalomyelitis (AE) is caused by an enterovirus belonging to the picornavirus group.

Transmission

Egg transmission is the major route of transmission of AE virus. Infected breeders will transmit the AE virus for several weeks and cause a decrease in egg hatchability. Infected chicks that hatch will show clinical signs of the disease and spread the infection in the incubator to other newly hatched susceptible chicks. Young chicks can also be infected on the farm. The incubation period varies from 5 to 14 days depending on the route of infection.

Species affected

Primarily chickens are susceptible to AE, but turkeys and pheasants have been reported as natural hosts.

Clinical signs

The disease is mainly seen in young chicks, between 1 and 3 weeks of age. Affected chicks sit on their hocks, do not move well, and many fall on their sides. A fine, rapid trembling of the head and neck can be seen, but especially felt when affected chicks are held in the hand. In laying and breeding flocks, AE virus infection causes a marked drop in egg production which returns to normal in about 2 weeks. Mortality in naturally infected chicks varies and can be as high as 75%.

Diagnosis

Clinical tremors in chicks, together with a drop in production and hatchability in the parent breeders, is indicative of AE infection. Chicks will not have gross lesions, but histological examination of brain, proventriculus and pancreas reveals typical lesions of AE. This will also differentiate the diagnosis of AE from encephalomalacia (Vitamin A deficiency, crazy chick disease). Laboratory testing of blood serum from breeder flocks, or their hatching eggs, can determine if an infection occurred.

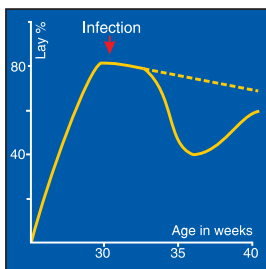
Treatment and control

Preventive vaccination of breeder pullets with live AE vaccine before egg production is the only effective means of AE control. If a breeder flock has not been, or has been inadequately, vaccinated against AE and an outbreak occurs, it is advisable to stop hatching eggs from the flock for several weeks until the breeders have acquired immunity and no longer transmit AE virus through their eggs.

AE infected young chickens



Effect of AE on egg production



Infectious Bursal Disease

(IBD, Gumboro Disease)

Cause

The disease is caused by a birna virus of serotype 1. Virus strains can be divided in classical and variant strains. The virus is very stable and is difficult to eradicate from an infected farm.

Transmission

IBD virus is very infectious and spreads easily from bird to bird by way of droppings. Infected clothing and equipment are means of transmission between farms.

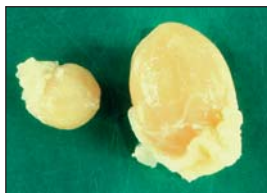
Species affected

Chickens and turkeys appear to be natural hosts.

Clinical signs

Clinical IBD occurs usually between 4 and 8 weeks of age. Affected birds are listless and depressed, pale and huddling. Mortality varies. Usually new cases of IBD have a mortality rate of about 5 to 10% but can be as high as 60% depending on the pathogenicity of the strain involved. In subsequent infection on the same farm, mortality is lower and eventually, with successive attacks, there is no mortality noted.

The subclinical form caused by the immunosuppressive effect of the IBD virus is now of more economic importance in that the immune system of the bird is damaged. Gumboro disease related diseases such as inclusion body hepatitis are more frequent in these birds. In broilers this form of the disease results in bad performance with lower weight gains and higher feed conversion ratios.



Normal (right) and affected bursa three days post infection

Diagnosis

In acute cases the bursa of Fabricius is enlarged and gelatinous, sometimes even bloody. Muscle haemorrhages and pale kidneys can be seen. Infection by variant strains is usually accompanied by a fast bursal atrophy (in 24-48 hours) without the typical signs of Gumboro disease. Also in chronic cases the bursa is smaller than normal (atrophy). The bursa destruction is apparent on histologic examination. The lack of white blood cells (lymphocytes) results in a reduction in the development of immunity and decreased resistance of the birds to other infections. Typical signs and lesions are diagnostic of IBD. Histopathological examination, serology and/or virus isolation are helpful tools. IBD can be confused with sulfonamide poisoning, aflatoxicosis, and pale bird syndrome (Vitamin E deficiency).

Treatment and control

No treatment is available for IBD. Vaccination of parent breeders and/or young chicks is the best means of control. The induction of a high maternal immunity in the progeny of vaccinated breeders, together with the vaccination of the offspring is the most effective approach to successful IBD control.



*IBD infected chicken
(right)*

Reovirus Infections

Cause

Reovirus infections, also known as viral arthritis/tenosynovitis, is caused by an avian reovirus.

Transmission

The virus may be transmitted by droppings from bird to bird. Egg transmission is also a factor when breeder flocks become infected during egg production.

Reovirus is a common inhabitant of the intestines of birds and not all strains are pathogenic.

Species affected

Chickens, turkeys and possibly pheasants are natural hosts.

Clinical signs

The first signs of reovirus infection are usually observed in broiler breeder chickens between 6 and 10 weeks of age. The birds are reluctant to walk and when forced up have a painful, trembling gait. A distinct swelling of the tendons of the shanks and also above the hock joint can be observed. Affected birds have malpositioned feathers, especially on the wings.

Internal lesions

The hock joint may be somewhat swollen, but usually not as severely as with *Mycoplasma synoviae* or Staphylococcus infections. Upon opening the legs the tendons usually appear discolored, brown or blood-tinged, with straw coloured fluid between them.

Ruptured tendons may occur and, in older broiler breeders (29-30 weeks old), one may feel a hard scarry knot in the tendon above the hock joint. When the infection is complicated by *Ms* or Staphylococcus the fluid may appear yellow and creamy. In commercial egg-laying breeds of chickens, the disease is not as common as in broiler breeders and broilers.

Diagnosis

Leg problems in broilers or broiler breeders associated with swelling of shank tendons or tendons above the hock joint sometimes accompanied by ruptured tendons, are indicative of reovirus infections.

A positive blood test with the agar gel precipitation (AGP) test is of some value as an indication of exposure to reovirus, but does not constitute proof of diagnosis. Histopathological examination of affected tissues and isolation of virus from such tissues are needed for a definite diagnosis.

Treatment and control

Reovirus infection cannot be treated successfully, but antibodies are of help in preventing secondary bacterial infections, particularly Staphylococcal infections.

Vaccination of broiler breeders with certain live and inactivated vaccines to ensure maternal immunity in their offspring appears to be of benefit to the birds themselves.

VA infected birds have malpositioned feathers



Swollen tendons



Malabsorption Syndrome

This complex disease has been reported under various names such as helicopter disease, femoral head necrosis, brittle bone disease, infectious proventriculitis, pale bird syndrome, running disease and stunting disease.

Cause

The malabsorption syndrome appears to be a disease complex involving avian Reoviruses and other viral and bacterial agents which may affect the digestive system resulting in nutritional and deficiency signs and lesions.

Transmission

Only circumstantial evidence is present at the moment to indicate that the causal organism(s) may be vertically transmitted. Horizontal transmission also seems to play a role on infected sites.

Species affected

Chickens and possibly turkeys.

Clinical signs

The disease is mainly observed in broiler flocks. Many affected broiler flocks have a history of diarrhoea, beginning as early as a few days of age and lasting until 10-14 days of age. Light or dark brown, foamy droppings can be found with undigested food particles. Several affected broilers in a flock may exhibit malpositioned feathers, especially on the wings. Early rickets with extreme paleness of legs and heads can be observed.

Encephalomalacia is also regularly found. At a later age (5-6 weeks) osteoporosis becomes clinically evident, frequently unilateral causing the birds to limp. Later an important effect is the delayed growth of the affected birds. Mortality is variable and in general as low as 4 %.

Diagnosis

The clinical disease is characterized by one or more of the following lesions: enteritis with watery brown and foaming contents and the presence of undigested food in the intestine. Mucosal and submucosal proventricular lesions.

Pancreatic inflammatory infiltration with degenerative changes have been found. Osteoporosis and osteomyelitis, femoral head necrosis whereby the bone of the epiphysis of the femur is unusually soft.

Since the causal agent may differ it is difficult to base a diagnose on virus isolation or serology.

Treatment and control

Treatment is impossible, vaccination against reovirus in the breeders helps to reduce problems in the progeny.

Strict hygienic and sanitary measures will reduce the incidence of the disease.

Infectious Anaemia

Cause

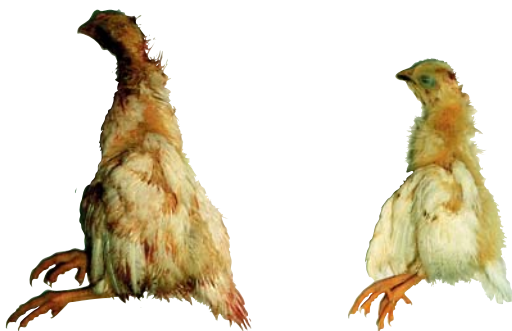
Infectious anaemia is caused by a very resistant small virus known as CAV (Chicken Anaemia Virus).

Transmission

The major mode of transmission of infectious anemia is vertical transmission from infected breeder hens. Horizontal transmission from bird to bird or by infected equipment, clothing, etc. is also possible.

Clinical signs and lesions

CAV causes a syndrome in young chicks up to approximately 3 weeks of age. Adult birds may get infected but will not develop clinical signs. The disease is characterized by increased mortality and anaemia associated with atrophy of the haematopoietic tissues in the bone marrow. Subcutaneous and intramuscular haemorrhages can be found accompanied with atrophy of the lymphoid system. Affected birds may show focal skin lesions (also known as blue wing disease). Mortality rates vary from 20 % to 70 %. Affected flocks will show poor growth reflected in economic losses.



A marked difference in size and growth retardation is observed between healthy (left) and infected (right) birds.

Diagnosis

The diagnosis can be based on the clinical signs and pathological findings in affected birds. Blood serum testing for specific CAV antibodies can be carried out (IFT, VN, ELISA). Virus isolation is also possible but it is time-consuming and expensive.

Treatment and control

No treatment is available for infectious anaemia. Maternally derived antibodies can offer protection. The induction of high maternal immunity in the progeny by vaccinating breeders is the best approach to successful CAV control.

MISCELLANEOUS BACTERIAL DISEASES

- *Infectious Synovitis*
- *Fowl Cholera*
- *Salmonella Pullorum Disease/
Fowl Typhoid*

Infectious Synovitis

Cause

This disease is caused by *Mycoplasma synoviae* (Ms).

Transmission

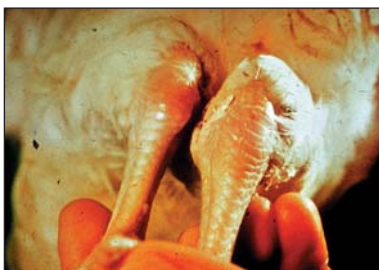
The major mode of transmission of Ms is vertical (egg) transmission from Ms-infected breeder hens. Horizontal transmission from bird to bird and by infected equipment, clothing, shoes, egg boxes and other fomites.

Clinical signs and gross lesions

Vary from asymptomatic infection to mild respiratory signs, airsacculitis and synovitis, an inflammatory swelling of the joints of legs and wings and inflammation of the sternal bursa ("breast blisters").

Creamy exudate in joints extending into tendon tissues is indicative.

Airsacculitis with frothy to cheesy exudates in the airsacs can be found, especially if secondary infection with *E. coli* is present.



Swollen hock-joint

Diagnosis

Blood serum testing for specific Ms antibodies with Ms antigen and the findings of specific lesions are indicative of Ms infections. Isolation of the causative Ms organisms is decisive for diagnosis.

Differential diagnosis

Staphylococcus arthritis can also cause swollen joints with a creamy exudate sometimes extending into the tendon sheaths. Reovirus infection can also cause swelling of joints and tendon sheaths, but the exudate is more watery or blood-tinged, unless secondary Staphylococcus infections occur.

Treatment and control

Ms infections can be treated with antibiotics with variable degrees of success (tetracycline, erythromycin, tylosin, tiamulin). However, control of Ms has been largely successful by blood testing of breeder chickens and elimination of positive Ms reactors.

Fowl Cholera (Pasteurellosis)

Cause

Fowl cholera is caused by a bacterium: *Pasteurella multocida* (several serotypes).

Transmission

Transmission of fowl cholera is mainly from bird to bird by water or feed contamination. Rodents (rats and mice) also appear to play a role in contamination of water and feed with *Pasteurella multocida*.

Species affected

Chickens, turkeys, game birds and other bird species are susceptible.

Clinical signs

Affected birds are depressed and have decreased appetite. Egg production will drop 5-15 % and mortality will be high in acute fowl cholera. Birds that die from acute fowl cholera frequently have bluish combs and wattles. Chronic fowl cholera will not cause high mortality, although there will be an increase in deaths. Swollen wattles is a feature of chronic fowl cholera.



Fowl cholera

Internal lesions

Gross lesions in acute cases are mainly internal haemorrhage and congestion of liver, spleen and kidneys. In chronic fowl cholera cheesy exudates can be found between the intestines, and on liver and heart.

Treatment and control

Treatment with appropriate antibiotics or chemotherapeutics can be successful in halting mortality and restoring egg production. However chronic carrier birds have been found in flocks of chickens after treatment. If clinical fowl cholera with mortality reappears in such flocks, one must treat again. Rodent control is also very important to prevent reintroduction of the infection. Vaccines, both inactivated bacterins as well as live vaccines are available.

Pullorum Disease and Fowl Typhoid

Cause

Pullorum disease is caused by a bacterium, *Salmonella pullorum*. Fowl typhoid is caused by *Salmonella gallinarum*, which is related to, but not identical to, *S. pullorum*.

Transmission

Pullorum can be transmitted by infected (carrier) breeder hens through their eggs. Chicks that hatch from such infected eggs will have typical pullorum disease (white diarrhoea) and high mortality. Infected chicks can also infect other chicks via droppings.

Fowl typhoid is more a disease of adult chickens, with high mortality and morbidity. Horizontal transmission is important with fowl typhoid through infected droppings, dead bird carcasses, and infected clothing, shoes, utensils and other fomites.

Species affected

Chickens, pheasants, ducks, geese and guinea fowl can contract both pullorum and fowl typhoid.

Clinical signs

Pullorum in chicks causes typical white bacillary diarrhoea, with pasted cloacas and high mortality. Infected adult breeders do not have clinical signs of the disease but have internal lesions in the ovary (miss-shaped, dark coloured follicles). Fowl typhoid in adult chickens causes listlessness and sulfur-coloured diarrhoea. The birds have generalized infection with swollen livers, spleens, and kidneys and haemorrhages in such tissues. Mortality is usually high: 25 to 60 %.

Treatment and control

Treatment of pullorum disease will not bring about a cure and is undesirable from a standpoint of eradication. It is far more practical to control the disease by elimination of infected carrier breeder hens.

Blood testing of breeder chickens by the serum plate or tube agglutination test with suitable *S. pullorum* antigen will detect infected carrier birds which can then be culled. Such control measures will stop the incidence of egg-transmitted pullorum disease. If hatching eggs from tested pullorum-free breeders are kept free from contamination through infected eggs from infected breeders or through contaminated equipment, chickens can remain after treatment. The best control method is eradication of infected birds. Breeder flocks should be blood tested with antigen for typhoid. The typhoid carriers can then be eliminated.

Vaccination for fowl typhoid with a special *S. gallinarum* (9R strain) has been practiced in several countries, but it should be discouraged in breeders when an eradication programme is in operation.

S. pullorum infected internal organs



Ornithobacterium rhinotracheale (OR) Infection

Cause

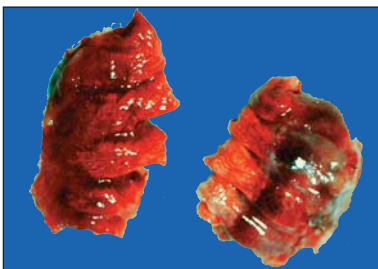
Ornithobacterium rhinotracheale is a gram-negative rod responsible for causing infections in chickens and turkeys of all ages.

Transmission

Horizontal transmission from bird to bird and by infected fomites. Infected breeder hens may transmit the disease agent vertically to their offspring.

Clinical signs and gross lesions:

OR may cause a respiratory disease in turkeys, usually between 2-6 and 12-20 weeks of age. The birds may show respiratory disease with watery eyes and swelling of the sinus infraorbitalis. Broilers may also show respiratory signs after infection at around 4 weeks of age. A severe purulent pneumonia, accompanied by airsacculitis and pericarditis may be found in broilers as well as in turkeys. Affected birds also show growth retardation. Concomitant viral infections may intensify the severity of the lesions.



*Pneumonic lungs
caused by OR infection*

Diagnosis

Blood serum testing using a commercially available ELISA for the detection of specific antibodies against OR and the finding of specific lesions are indicative of OR infection. Isolation of the organism and its biochemical determination may be attempted but care should be taken that these are carried out using appropriate methods in order to avoid unreliable results. Differential diagnose with bacteria causing similar disease patterns is recommended (*E. coli*, *P. multocida*, MG).

Treatment and control

OR infections may be treated with broad -spectrum antibiotics with variable degrees of success. An inactivated vaccine for broiler breeders and turkey poults is available.

INFECTIOUS VIRAL DISEASES OF DUCKS

- *Duck Hepatitis*
- *Duck Plague*

Duck Virus Hepatitis

Cause

Duck virus hepatitis is caused by a picornavirus. It is possible that there are more serotypes.

Transmission

The disease can spread rapidly to all susceptible ducklings in the flock via faeces. The incubation period varies from around 24 hours to a few days.

Species affected

Ducklings under 6 weeks of age are susceptible.

Clinical signs

The disease has a very short course, with all affected birds dying within a few days.

Signs, if seen at all, may include somnolence and convulsions, followed by quick death. Mortality up to 95 %.

Internal lesions

Principal lesions found in the liver, showing fatty degeneration, yellowish and with many small or bigger haemorrhages.

*Signs may include
somnolence and
convulsions, followed
by quick death*



Diagnosis

Sudden death in small ducklings is highly suggestive. Virus isolation can confirm this diagnosis.

Treatment and control

Serum therapy is possible.

Strict isolation during the first 4-5 weeks can prevent infection. Ducklings can be protected by maternal antibodies. To provide for this, parent stock has to be vaccinated, preferably twice.

This will protect the progeny for two weeks, and mitigate infection afterwards.

Ducks without maternal antibodies should be vaccinated at day old.

Principal lesions found in the liver, showing fatty degeneration, yellowish and with many small or bigger haemorrhages.



Duck Plague

(Duck Virus Enteritis)

Cause

Duck plague is caused by a herpes virus. Only one serotype is known. There is a difference in virulence between strains.

Transmission

Duck plague virus is excreted by affected birds through faeces and other body discharges. Via soiled drinking water, contaminated pond water or open water other birds are infected.

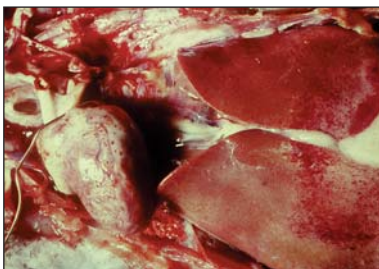
Species affected

Ducks, geese and swans, of all ages.

Clinical signs

High mortality up to 100 %, sometimes sudden death. Droppy appearance, slow movements with hanging wings. Birds show bloody nasal discharges and conjunctivitis, diarrhoea and they may make a hoarse noise. The birds are very thirsty. Birds often die in a rather characteristic position, with the neck twisted downwards, sideways or backwards. In laying flocks egg production may drop 50 % or more.

*During the disease
vascular damage,
tissue haemorrhages,
digestive mucosal
eruptions, lesions of
lymphoid organs as
well as degenerative
changes in
parenchymatous
organs can occur.*



Internal lesions

Haemorrhagic enteritis, haemorrhagic or pseudo-membranic pharyngitis, oesophagitis and cloacitis, haemorrhagic ovaritis.

Diagnosis

The gross lesions are rather characteristic for duck plague. Virus isolation and neutralization confirm the diagnosis to distinguish the disease from avian cholera (*Pasteurella multocida*) or duck hepatitis.

Treatment and control

There is no treatment known. Prevention should include clean drinking water and keeping wild, free-flying waterfowl away. Vaccination with adapted strains can provide a reliable protection. Even in an infected flock emergency vaccination can limit the damage, due to an interference phenomenon between the vaccine virus and the field virus.

PARASITIC DISEASES

- ***Coccidiosis***
- ***Endoparasites***
- ***Blackhead***

Coccidiosis

Cause

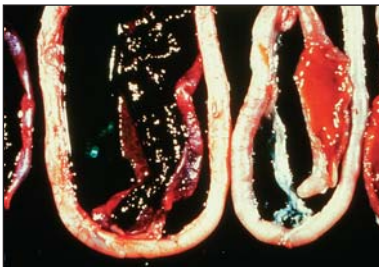
Coccidiosis is caused by protozoa, unicellular parasites. In chickens there are 9 different species of coccidia of which the main 5 are *Eimeria acervulina*, *Eimeria necatrix*, *Eimeria tenella*, *Eimeria maxima* and *Eimeria brunetti*.

Transmission

Infected droppings, containing oocysts of coccidia are the main means of transmission, between birds. The incubation period is 4 to 6 days.

Species affected

Chickens have their own specific coccidiosis types which do not cross-infect other bird species.



Eimeria tenella

Clinical signs/Diagnosis

Coccidiosis can be divided into 2 groups:

The caecum is involved (Caecal coccidiosis).

Mainly caused by *E. tenella* in chickens up to 12 weeks.

Mortality may run as high as 50 %. Infected birds are listless, have bloody droppings, a pale comb and show a lack of appetite. Laboratory examination will show haemorrhages in the caecal wall. After severe bleeding a core will be formed in the lumen.

The small intestine is involved (small intestinal coccidiosis).

Caused by *E. acervulina*, *E. brunetti*, *E. maxima*, *E. necatrix*.

E. acervulina

May affect birds of any age.

E. acervulina is not normally very pathogenic, but in some cases considerable mortality may be seen.

Birds infected show loss of weight, combs may be shriveled and a drop or even cessation of egg production in layers may be seen.

At necropsy, haemorrhagic lesions of *E. acervulina* are seen throughout the upper portion of the affected intestine and also grey or whitish patches may be present.

E. brunetti

May affect birds of any age.

E. brunetti is definitely pathogenic, in severe infections mortality can be high. Birds infected show emaciation and diarrhoea.

At necropsy a white cheese-like material is found in the lumen of the lower intestine and rectum.

The caeca and cloaca are inflamed. The gut wall is thickened.

E. maxima

May affect birds of any age.

E. maxima is less pathogenic than *E. acervulina*, *necatrix* and *brunetti*, mortality is generally low.

Diarrhoea, loss of weight and a drop in egg production of layers, will be seen; bloody droppings are common.

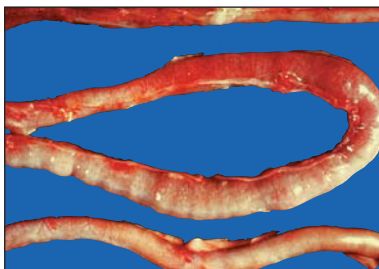
At necropsy the lower portion of the small intestine is dilated and the wall is thickened; the gut is filled with thick mucus, grayish, brownish or pinkish in color.

E. necatrix

Mainly in chickens up to 4 months of age.

E. necatrix is very pathogenic. Infection with *E. necatrix* may result in a two stage clinical outbreak of coccidiosis. In the acute stage mortality may be high in the first week after infection.

In the chronic stage blood may be seen in the droppings, the birds are listless and lose weight. In layers a drop in egg production will be observed. At necropsy the middle portion of the intestine is affected, haemorrhage will be seen. The unopened intestine looks spotty, white areas (schizonts) intermingled with bright or dull red spots (haemorrhages) will be observed.



Eimeria nectarix

Treatment and control

This heading is most appropriate in the case of coccidiosis as there is no disease group in poultry where both control and treatment are employed more.

The well established principles of good management and husbandry are of basic importance.

It is common practice to include low levels of chemotherapeutics in the feed of birds. These chemicals are referred to as coccidiostats and as such keep in check the development of the parasites so that a pathological situation does not develop. It should, however, be taken into account that coccidia can develop a resistance to all chemicals so far used for this purpose and for this reason it is necessary to change from one chemical to another periodically. Treatment of infected flocks may be carried out by the administration of coccidiostats at a higher therapeutic level to the affected birds. There are certain products available which are specifically designed for treatment and which are not satisfactory for prevention. These chemicals are sometimes referred to as coccidiocidal agents.

Whenever administering these products, particular attention should be paid to the dosage recommendation of the manufacturer.

Endoparasites

Worms living in the intestines of chickens fall mainly into four categories.

Roundworms (Ascarids), usually 5 to 7 cm (2-3 inches) long.

Hairworms (Capillaria), only measure 1-1.5 cm long.

Caecal worms (Heterakis), usually 1.5 cm long.

Tape worms, usually 7 to 10 cm long, consisting of many small segments.

Roundworms



Hairworms



Vaccination to prevent coccidiosis is also possible

Tapeworms



Clinical signs

Mature roundworms are not a major cause of the disease, but the larvae can damage the intestinal lining, causing enteritis, anaemia, decreased egg production and at times eggs with pale yolks.

Capillaria cause more damage to the intestinal lining and can cause enteritis and anaemia with decreased egg production and the appearance of pale egg yolks ("platinum yolks").

Caecal worms are found in the caeca and do not cause serious damage, except that their eggs can transmit blackhead - mainly in turkeys (see blackhead, page 80).

Tape worms are infrequently found and do not cause serious damage, except that they use the nutrients of the host chicken.

Diagnosis

Examination of the intestinal contents will reveal roundworms, caecal worms, and tape worms without difficulty. Capillaria can usually be found when intestinal contents are washed through a fine mesh sieve.

Treatment and control

Roundworms and caecal worm infections can be treated with piperazine. Piperazine is not effective against tape worms and capillaria for which other anthelmintics are required.

Blackhead

(Histomoniasis, Enterohepatitis)

Cause

A protozoan parasite, *Histomonas meleagridis*.

Transmission

Direct transmission by infected water, feed, or droppings has been proved.

Indirect transmission by infected eggs of the caecal worm.

Heterakis gallinarum, is also a major factor. Raising turkeys and chickens on wire and indoors decreases the incidence of blackhead.

Species affected

Chickens, turkeys, and peafowl are natural hosts to blackhead infection.

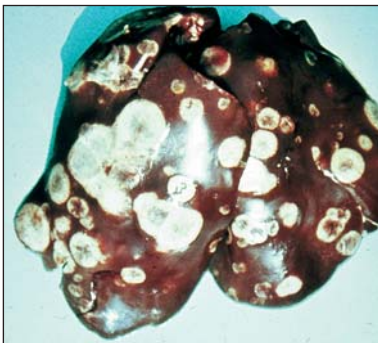
Clinical signs

Affected birds are depressed, stand or sit with ruffled feathers, and have yellowish diarrhoea. Darkening of head parts, especially in turkeys, gave the name to the disease (black-head). Gross lesions include circular necrotic areas in livers with a crater-like center and cheesy cores in the caeca.

Blackhead can cause high mortality, particularly in young turkey poults, but the disease can also affect older birds. In chickens the mortality from blackhead infection is usually lower, young chickens being the most susceptible.

Treatment and control

Treatment with protozoan chemotherapeutics is usually effective. Such drugs can also be given at preventive levels in turkeys starter and grower feed. Growing turkeys on wire and indoors can reduce the incidence of blackhead to a large extent, but even so, strict hygiene and elimination of caecal worms are important control measures.



*Necrotic liver infected
by Histomonas
meleagridis*

SOME IMPORTANT VITAMIN DEFICIENCY DISEASES

- *Riboflavin*
- *Vitamin E*
- *Vitamin D3*

Riboflavin (Vitamin B2)

Deficiency (Curly Toe Disease)

Clinical signs

Young chicks, as early as 1-week-old, exhibit curling of the toes, inability to walk and sometimes diarrhoea.

Treatment and control

Administering vitamin B preparations brings a rapid cure. Only in advanced cases will birds be dehydrated and emaciated, requiring further treatment.

It is important to ensure adequate vitamin B levels not only in starter and grower diets, but also in the diet of parent breeders.



*Curly toe caused by
riboflavin deficiency*

Vitamin E Deficiency

(Crazy Chick Disease, Encephalomalacia)

Clinical signs and gross lesions

Vitamin E deficiency in chickens affects the brain, causing degeneration, oedema and haemorrhage, especially in the small brain (cerebellum).

Affected young chicks appear unable to walk, they fall on their sides or stand with their heads between their legs.

The cerebellum shows gross swelling, with yellow or brown discoloration and pinpoint haemorrhages may be observed.

Encephalomalacia can also be found in mature chickens.

Treatment and control

Adequate levels of vitamin E and selenium in the diet of chickens and their parent breeders is of prime importance.

Treatment of affected birds with vitamin E preparations (alpha-tocopherol) is effective if the condition is not too far advanced.

*Brain lesions
caused by vitamin E
deficiency*



Vitamin D3 Deficiency

(Rickets, “Rubber Legs”)

Clinical signs and gross lesions

Young chickens, 2 to 5 weeks of age, with vitamin D3 deficiency are unable to stand and have very soft, pliable, legs and beaks. The rib joints are swollen like beads and curved inward, the breastbone often twisted.

In layer chickens, vitamin D3 deficiency causes soft-shelled eggs and a drop in production.

Treatment and control

Vitamin D3 can be given as treatment, usually in combination with calcium and phosphorus.



Rubber legs