1 VIRAL DISEASES

RANIKHET DISEASE (Newcastle Disease)

Ranikhet disease (RD) is the most common, the most widely prevalent, and economically the most important viral disease of poultry in our country. It is a very severe, sudden, and rapidly spreading disease; and may be seen from 6th to 7th day onward up to 72 weeks. It occurs throughout the year, but is most common in the summer.

Cause

A virus called **paramyxovirus**. These **viruses are of different types**. Some are highly powerful and cause most severe form of the disease, others are moderate, while a certain group is only mildly harmful. In addition, there are some viruses that cause infection without showing any symptoms.

Spread

1. **Virus spreads through the air.**
2. Infection occurs mainly through **inhalation** or **ingestion.**
3. Contaminated feed and water spread infection.
4. Movements of people and equipment also spread infection.
5. Away from the bird, that is, in the shed, virus survives for days to weeks.
   However, in the dead bird or faeces, virus survives for several months.

Symptoms

Depending on the disease-producing power of the virus, symptoms vary.

1. With **very harmful viruses**, first indication is sudden death. Then, symptoms such as depression, weakness, lying down, green diarrhoea, swelling of the face, and nervous signs may appear, ending in exhaustion and death (Fig. 1). Other signs include twisting of the neck, paralysis of legs and arched position of the body. Mortality may occur up to 100% in chicks. In layers, early symptom is shell-less or soft-shelled eggs, followed by complete stoppage of laying.
2. **Moderately harmful viruses** usually cause severe respiratory disease and respiratory symptoms. In adult birds there is marked drop in egg production for several months. Mortality is low.
3. **Mildly harmful viruses** may cause no disease, or only a mild respiratory distress.

Postmortem Findings

1. **Pinpoint haemorrhages on the tips of glands in the proventriculus** (Fig. 2, 3).
2. Enlarged and haemorrhagic caecal tonsils.
3. Haemorrhagic lesions in the intestinal wall (in the lymphoid aggregates) (Fig. 4, 5, 6, 7, 8).
4. Spleen shows necrosis (white spots of dead tissue) on its outer surface, and also on the cut surface.
5. Marked congestion of trachea, often with haemorrhages. The airsacs may be inflamed (airsacculitis) and appear cloudy and congested. Airsacs may even contain cheesy (caseous) material.

Diagnosis

1. From the characteristic postmortem findings.
2. Confirmation depends on various laboratory tests, such as HI and ELISA, and also on isolation of the virus and its characterization.

Treatment

There is no treatment.

Control

1. Timely vaccination with live and/or inactivated (killed) vaccine is the only reliable control method.
2. However, under field conditions vaccination alone is not sufficient to control RD. It must therefore be accompanied by good hygiene, good management, and good biosecurity practices.

Fig. 1. **Ranikhet disease** in a **12-week-old grower chicken**. Note the bird is depressed, its eyes are closed and the head is drooping.

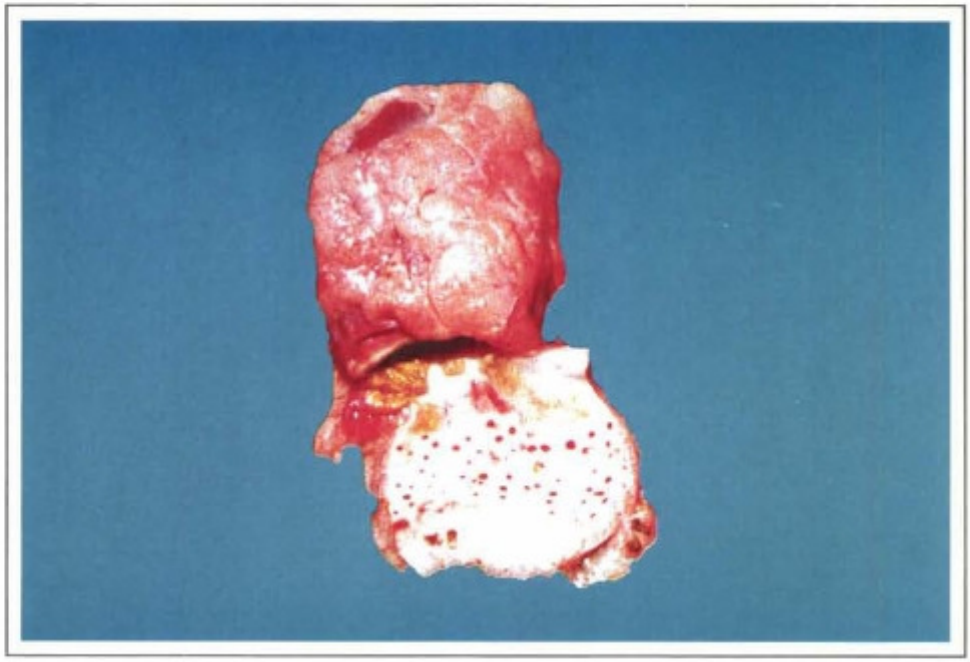

Fig.2. **Ranikhet disease** in a **45-day-old broiler chicken**. Note the presence of haemorrhages on the tips of glands in the proventriculus. **This is diagnostic of Ranikhet disease.**

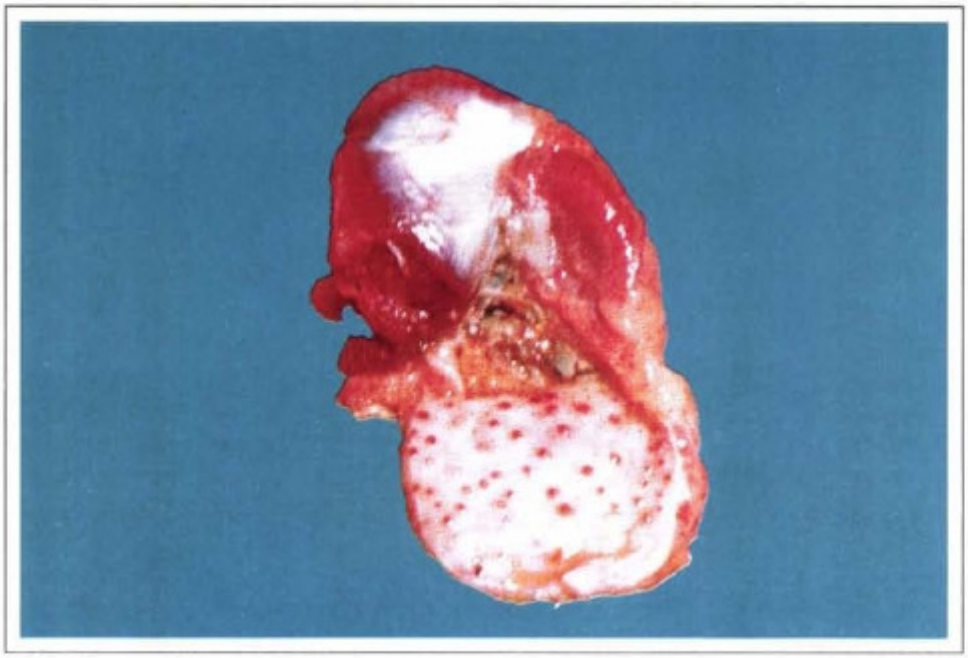

Fig. 3. **Ranikhet disease** in a **63-week-old layer chicken**. Note prominent haemorrhages on the tips of glands in the proventriculus.

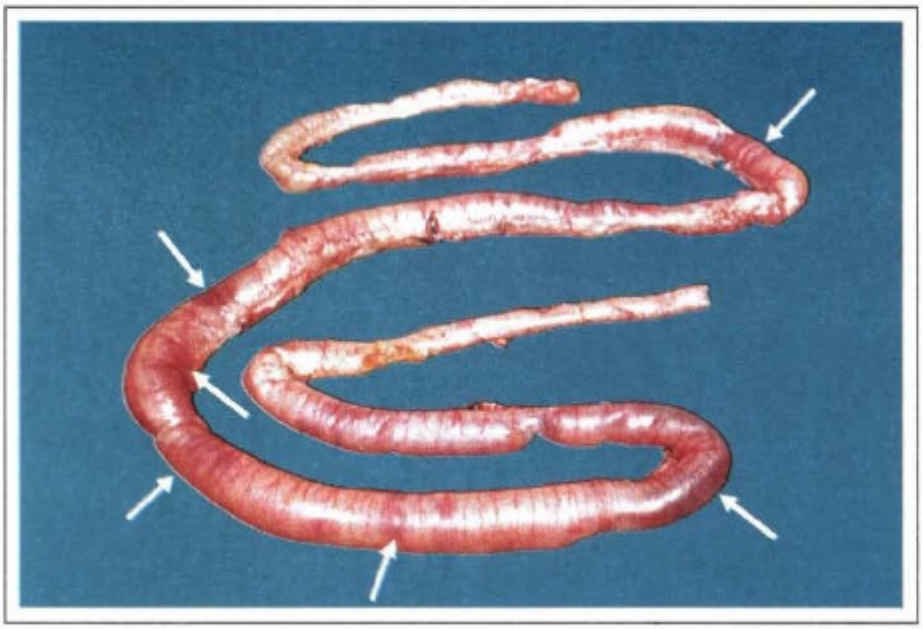

Fig. 4. **Highly powerful (virulent) form of Ranikhet disease (intestinal form)** in a **36-day-old broiler chicken**. Note prominent haemorrhagic lesions (changes) in the small intestine (white arrows). **This is typical of the virulent form of Ranikhet disease.**

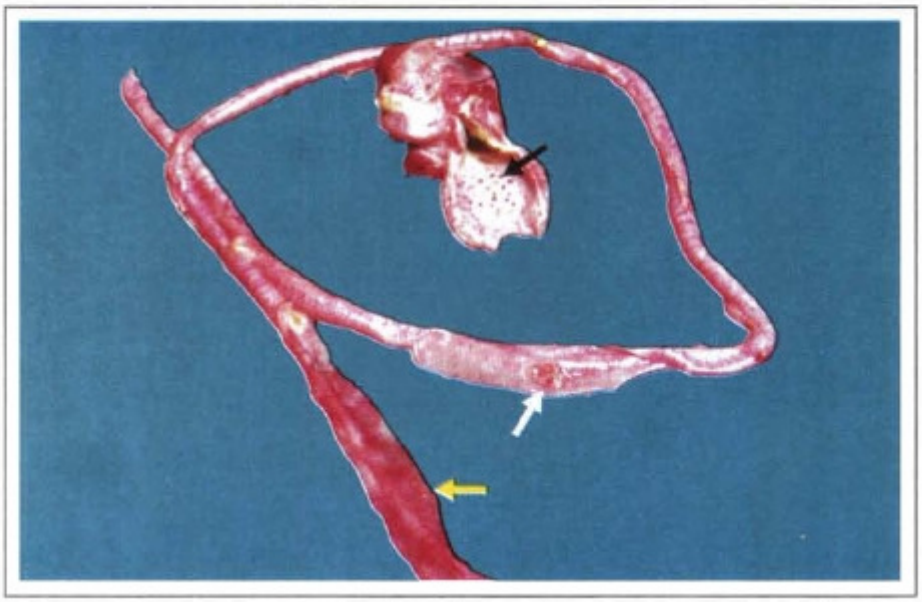

Fig. 5. **Highly powerful (virulent) form of Ranikhet disease (intestinal form) from the same 36-day-old broiler chicken** shown in Fig. 4. Note pinpoint haemorrhages in the proventriculus (black arrow); and one well-defined almost circular haemorrhagic lesion (white arrow) and other diffuse haemorrhages (yellow arrow) in the small intestine. **These lesions are characteristic of highly powerful disease-producing Ranikhet disease viruses (viscerotropic velogenic).**

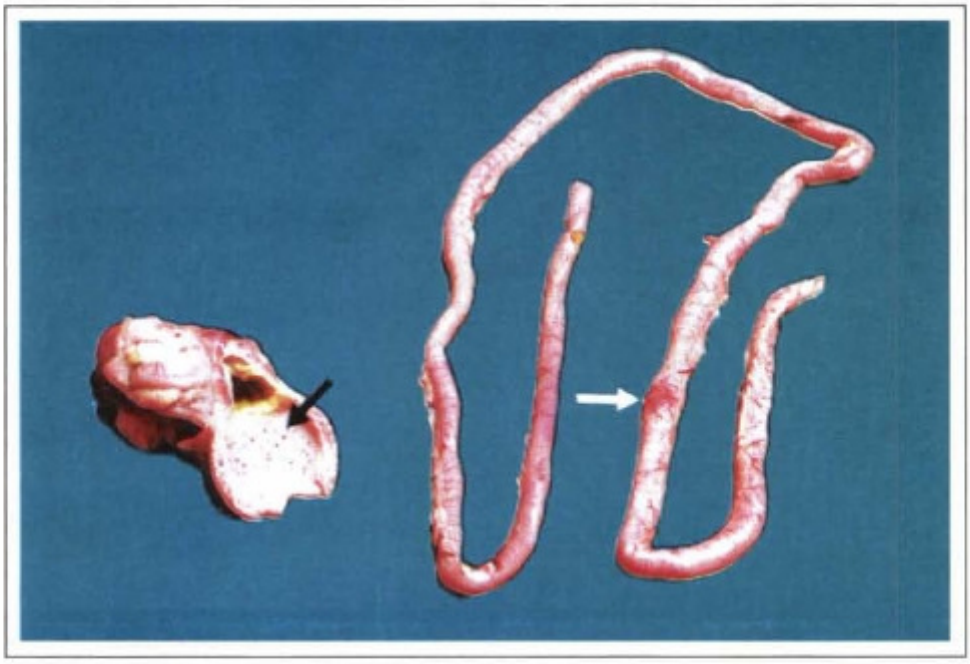

Fig. 6. **Highly powerful (virulent) form of Ranikhet disease (intestinal form)** in a **45-day-old-cockerel**. Note pinpoint haemorrhages in the proventriculus (black arrow) and one distinct haemorrhagic patch in the small intestine (white arrow). This is typical of the virulent form of Ranikhet disease.

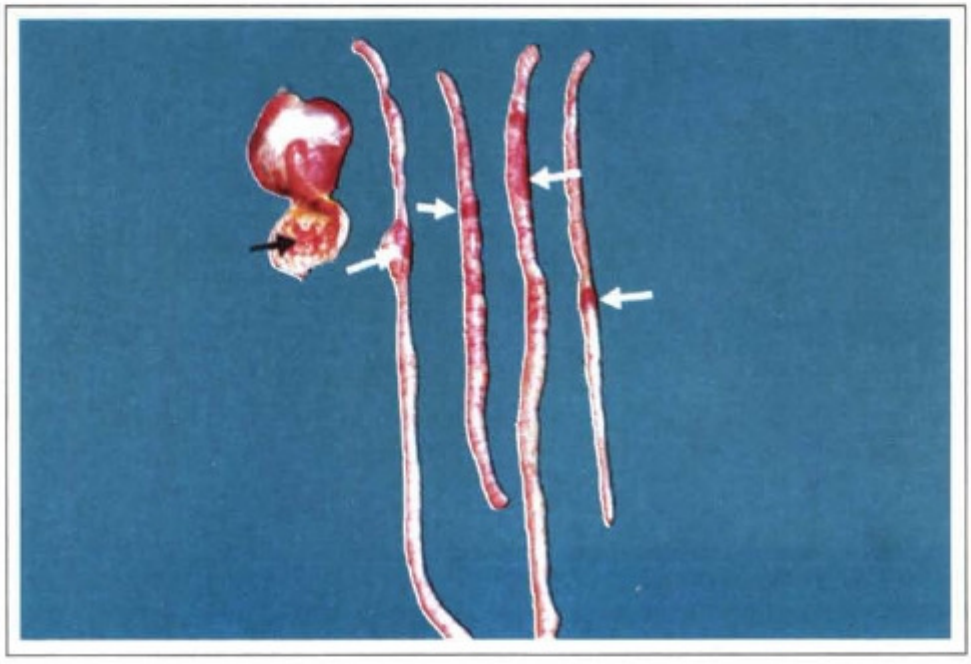

Fig. 7. **Highly powerful (virulent) form of Ranikhet disease (intestinal form)** in **another 45-day-old cockerel**. Note haemorrhages in the proventriculus (black arrow) and well-defined haemorrhagic lesions in the small intestine (white arrows), typical of the virulent form of Ranikhet disease.

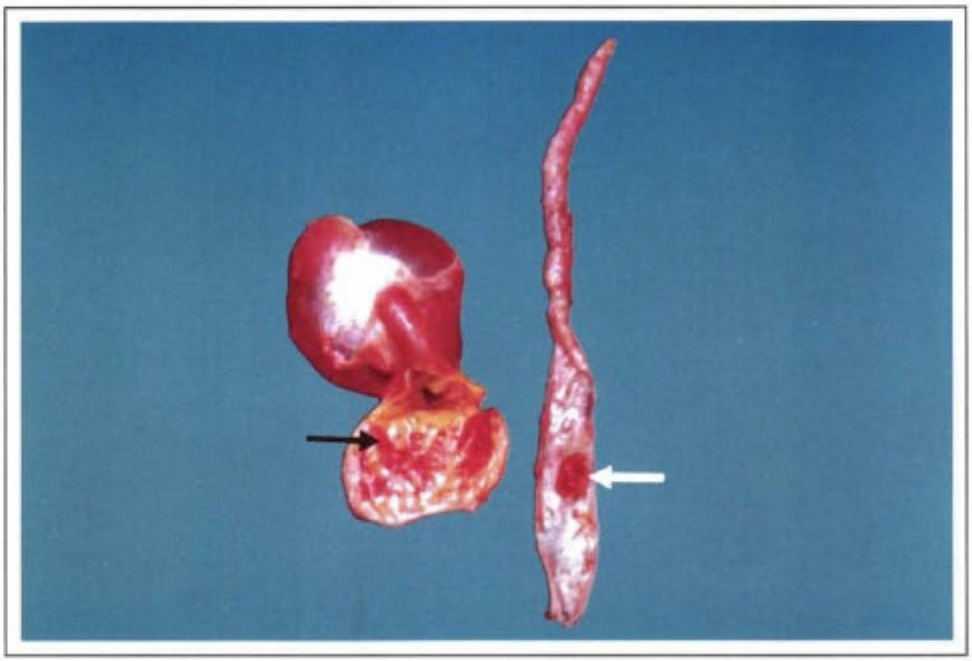

Fig. 8. A close-up picture of the **highly powerful (virulent) form of Ranikhet disease (intestinal form)** from the same **45-day-old cockerel** shown in Fig. 6. Note prominent haemorrhages in the proventriculus which tend to diffuse (black arrow) and one well-defined circular haemorrhage in the small intestine (white arrow), **typical of the virulent form of Ranikhet disease.**

GUMBORO DISEASE (Infectious Bursal Disease)

Gumboro disease is a sudden and severe, highly contagious viral infection of **young chickens**. Next to Ranikhet, it is the second most important disease of poultry, and every year inflicts heavy mortality. The disease is of great economic importance because, besides causing heavy mortality, it produces severe and prolonged suppression of the natural immune responses (immunosuppression). Immunosuppression, in turn, can lead to vaccination failures, ***Escherichia coli*** infection, and gangrenous dermatitis. The deaths resulting from these other diseases in many cases are greater than those from Gumboro disease itself. Gumboro disease occurs throughout the year, and usually **affects young chickens between 18 to 40 days of age**. Rarely, however, in layers, it may be seen even up to 14 weeks.

Cause

A virus - called **birnavirus**. The virus has **two main serotypes: 1 and 2. Only serotype 1 produces disease.** It has several strains. Some strains are so mild that they do not produce disease, while others are so harmful that they cause up to 50% mortality. **The viruses have an attraction for cells of bursa and cause depletion of this organ**. Virus in the sick birds is excreted in faeces for 10-14 days. **It is very stable** and remains highly infectious in the poultry environment for many months.

Spread

The most common route of infection is by mouth. The virus being resistant to heat and disinfectants continues to linger on at the farm, and poultry houses remain infective for **up to 122 days after an outbreak**. Water, feed, and droppings in the infected sheds are infectious even after 52 days.

Symptoms

Symptoms vary, depending on the disease-producing power of the virus; and age and maternal antibody level of the chick.

1. **Severe form** is seen in chicks between 3 and 6 weeks of age (**between 21 and 42 days**). One of the earliest symptoms is the tendency for some birds to pick at their own vents. Other symptoms include depression, white watery diarrhea, soiled vents, loss of appetite, ruffled feathers, unwillingness to move, trembling, closed eyes (Fig. 9), lying down in exhaustion, and finally death. The number of birds affected may vary from 10 to 100%, and mortality from 0 to 20%, sometimes reaching 50%. Strains of the most severe type of Gumboro virus cause 90 to 100% mortality.
2. **Mild form** may not show any symptoms except poor growth.
3. The **course of the disease** in individual chicks **is short (5-7 days)**, leading rapidly to death or recovery.

Postmortem Findings

1. **Swollen (oedematous) bursa in the early stages** that may be **double in size and weight** by the 4th day (Fig. 10).
2. The **bursa** first shows cheesy mass within its lumen, and later on **small and large haemorrhages on its inner surface**. Sometimes **widespread haemorrhages** are present **throughout the bursa** (Fig.11, 12, 13, 14). **This is typical of Gumboro disease.**
3. Kidneys are swollen and slightly pale in appearance (Fig. 14).
4. **Haemorrhages in the thigh and breast muscles** (Fig.15, 16, 17). This again is typical of Gumboro disease.

Diagnosis

1. From the characteristic postmortem findings. Changes in the bursa are quite characteristic and confirm the diagnosis.
2. Confirmation of the diagnosis can be made by ELISA and isolation of the virus.

Treatment

No treatment is available for Gumboro disease.

Control

1. Vaccination of parent breeders and/or young chicks is the best method of control.
2. Hygiene and sanitary precautions that are applied to prevent the spread of most poultry infections must also be strictly followed in the case of Gumboro disease.

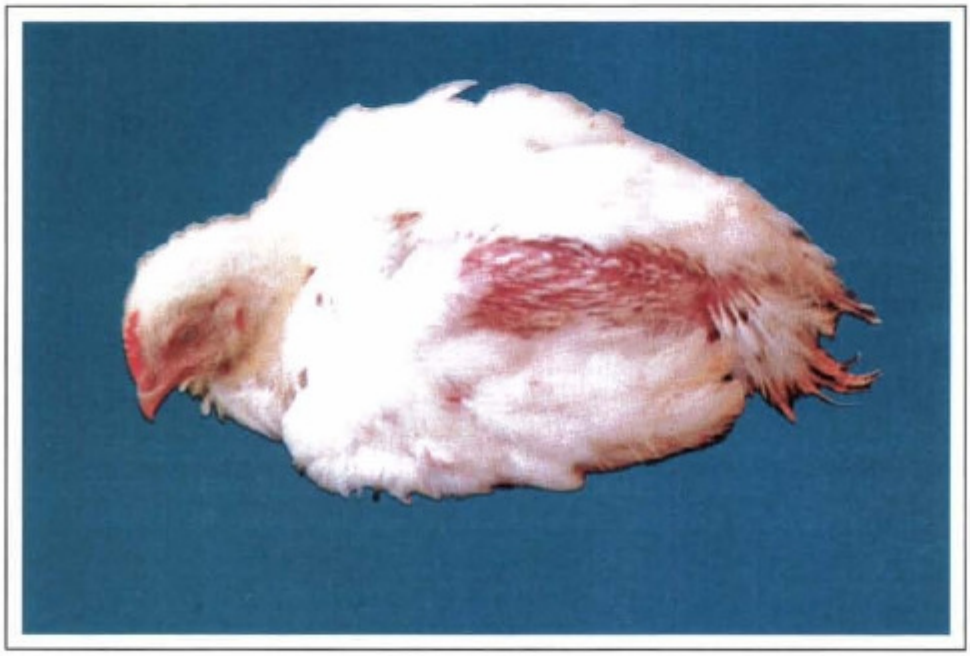

Fig. 9. **Gumboro disease in a 25-day-old broiler chicken**. Note typical posture of the bird. The eyes are closed and head is drooping.

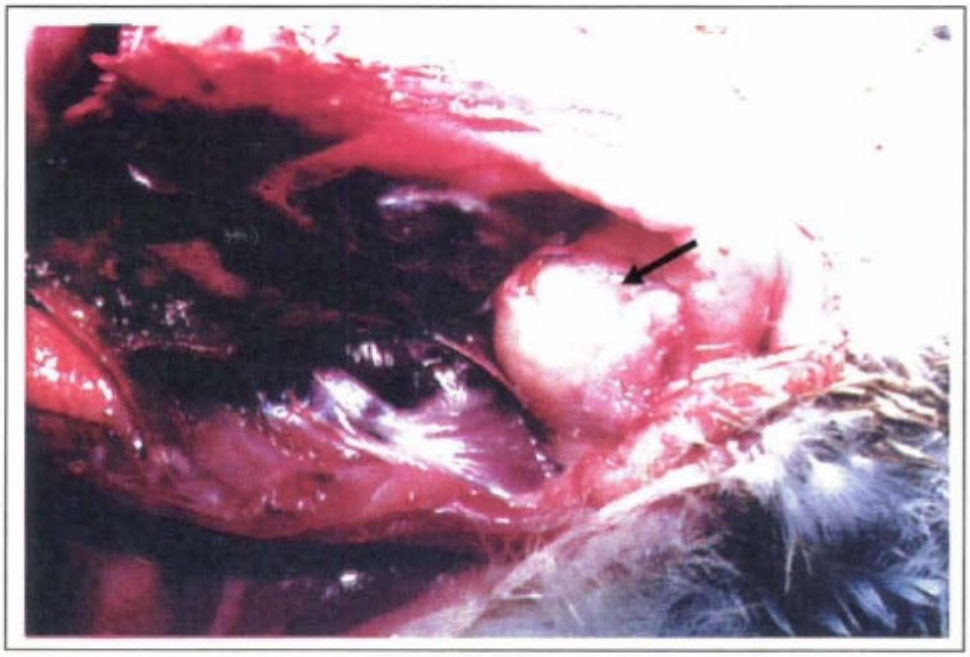

Fig. 10. **Gumboro disease in a 25-day-old broiler chicken**. Note greatly enlarged bursa of Fabricius (arrow). **This is typical of the early stage.**

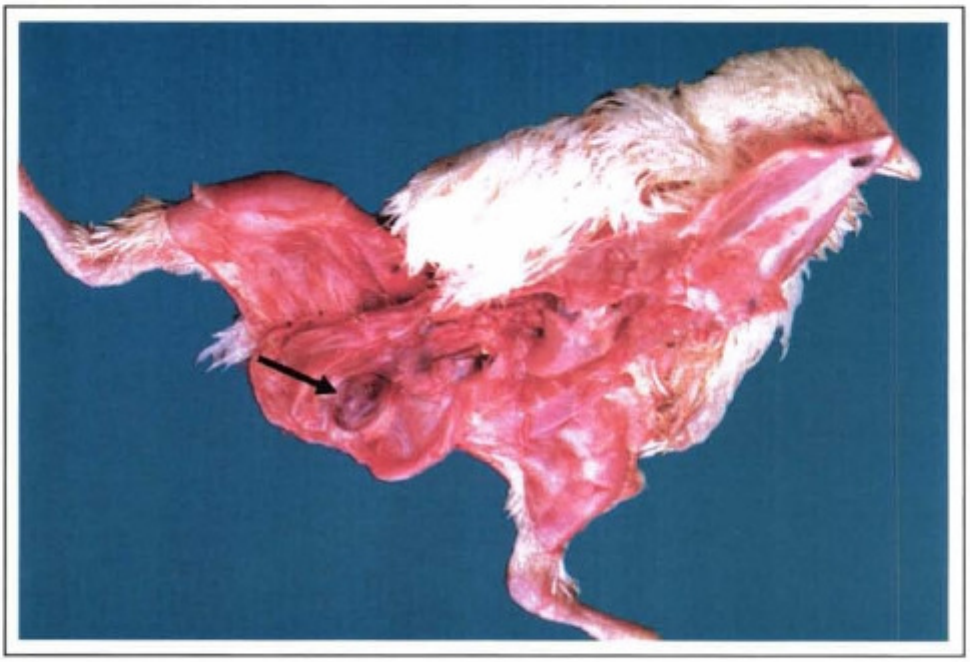

Fig. 11. **Gumboro disease in a 26-day-old broiler chicken**. Note bursa of Fabricius is markedly congested and haemorrhagic, that is, red (arrow), but is slightly reduced in size. **This is typical of the disease in the advanced stage.**

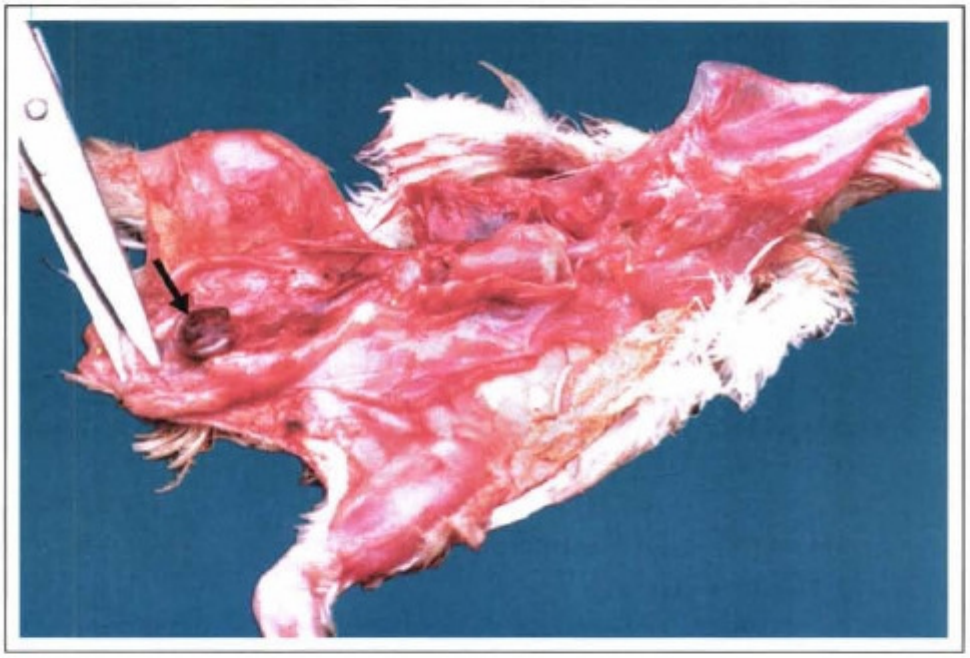

Fig. 12. **Gumboro disease** in another **26-day-old broiler chicken**. The opened bursa of Fabricius is congested and haemorrhagic, but slightly reduced in size (arrow). This is typical of the disease in advanced stage.

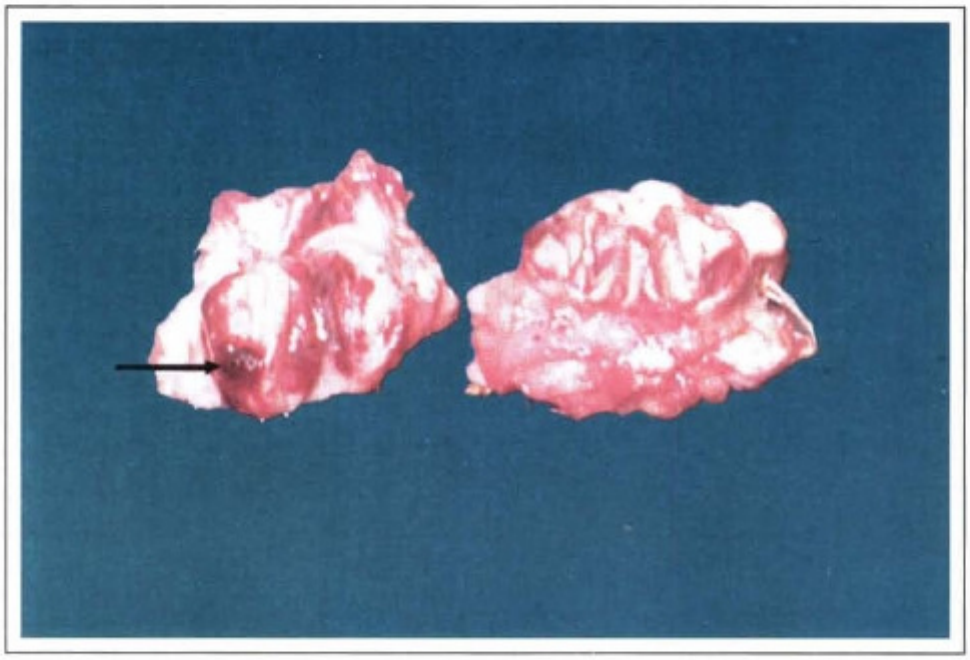

Fig. 13. **Two bursae** from **two different 25-day-old broiler chickens** suffering from **Gumboro disease**. Right bursa is enlarged and oedematous. That is, it contains fluid and is therefore swollen. Left bursa is not only enlarged, inflamed, and oedematous, but it also reveals haemorrhages (arrow).

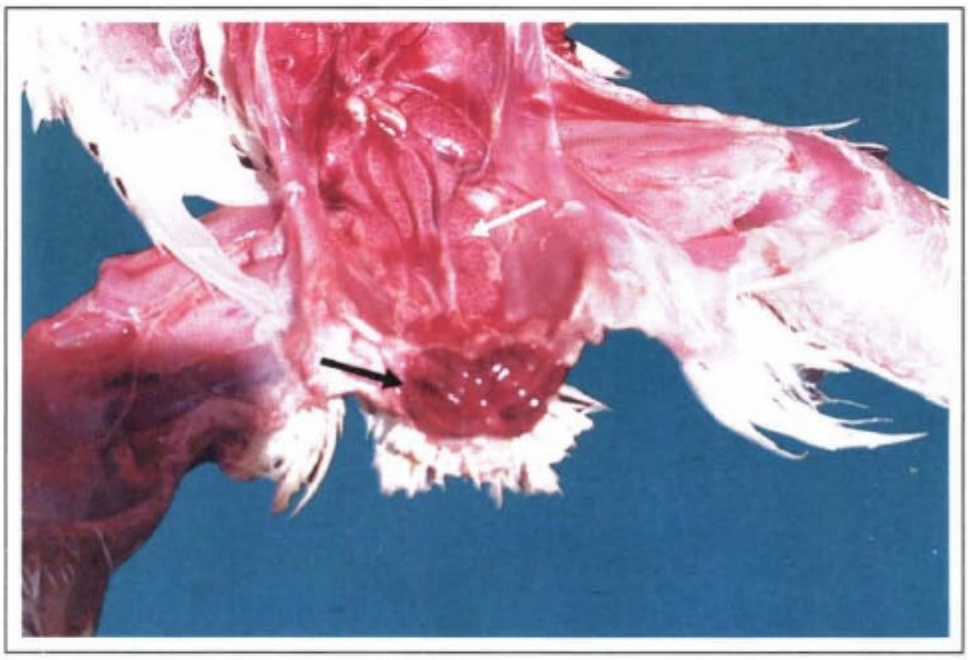

Fig. 14. **Gumboro disease** in a **26-day-old broiler chicken** showing markedly haemorrhagic bursa of Fabricius (black arrow). Bursa is cut open to show haemorrhages. Note also that kidneys are swollen, slightly pale and gouty because of urate deposits (white arrow).

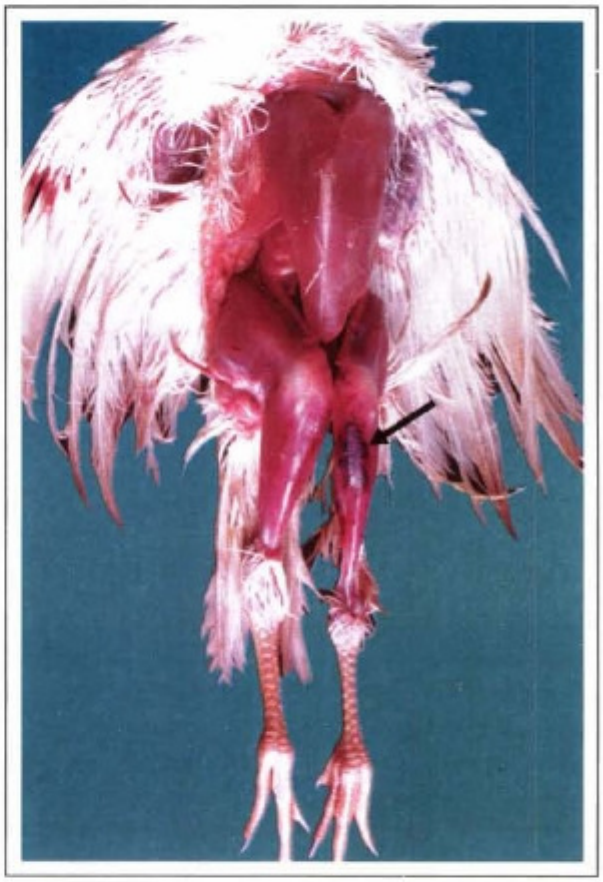

Fig. 15. **Gumboro disease** in a **7-week-old grower chicken**. Note haemorrhages on both the legs, but are more marked on the right (arrow). **This is characteristic of Gumboro disease.**

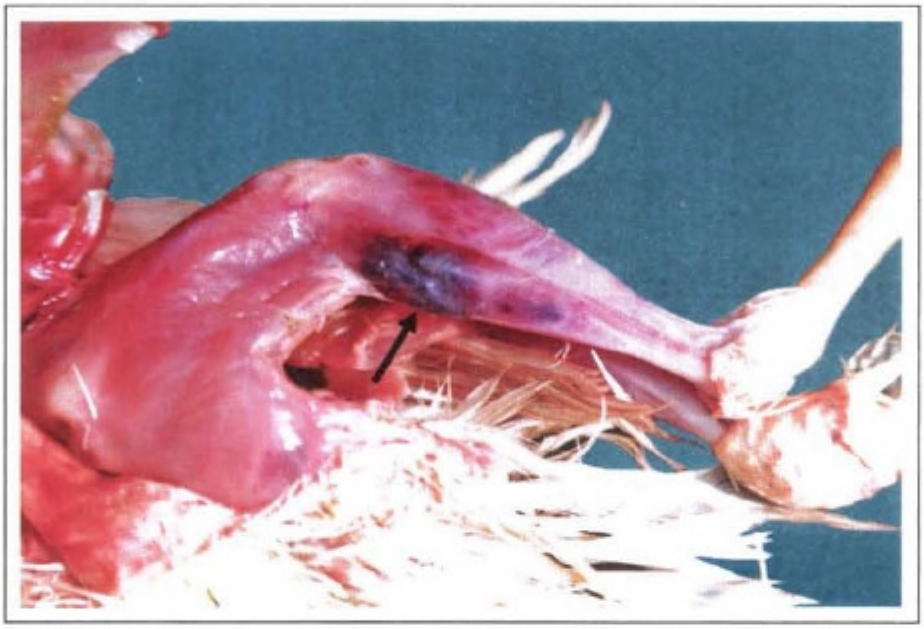

Fig. 16. **Gumboro disease** in the same **7-week-old grower chicken** shown in Fig. 15. A close-up picture of the right leg shows a big haemorrhage (arrow).

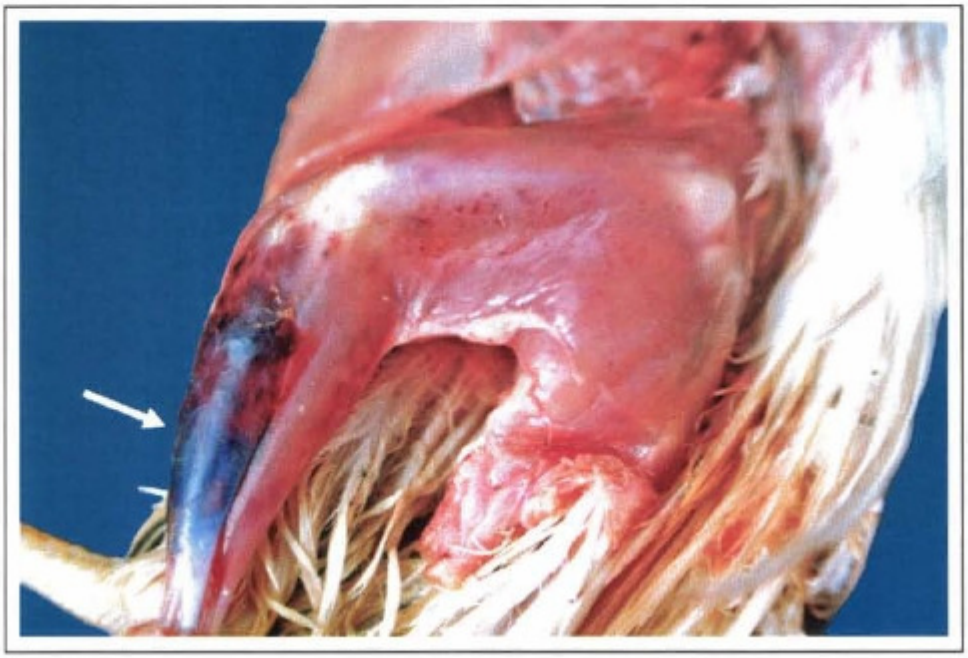

Fig. 17. **Gumboro disease** in the same **7-week-old grower chicken** shown in Fig. 15. A close-up picture of the left leg exhibits a very big haemorrhage, involving almost the entire leg (arrow), and other numerous minute haemorrhages (upper side).

MAREK'S DISEASE

Marek's disease (MD) is a tumour-causing viral disease of chickens. It is characterized by marked enlargement of the nerves, or marked enlargement of the liver, spleen, and kidneys due to diffuse growth of certain cells. It is an economically important disease. Although Marek's disease has been effectively controlled by vaccine, sporadic and sometimes serious losses still continue to occur from it. The disease therefore needs adequate attention. Marek's disease appears almost exclusively **confined to female birds**. The author has observed that the disease usually begins in growers when they approach **sexual maturity**, that is, **between 17-20 weeks of age**. The disease may then continue to inflict mortality **even up to 40 weeks (a grower-to-layer disease).**

Cause

A virus - called **herpesvirus**. The **virus is of three different types**: (1) mildly harmful, (2) harmful, and (3) very harmful. **Birds may become infected early in life, and remain infected until death.**

Spread

1. The virus spreads rapidly from infected to uninfected birds. **Cells from the feather follicles are the most important source of infection**. The virus is present in a free form in cells shed from the feather follicles.
2. Marek's disease is highly contagious. Virus spreads through the air. **Inhalation through the respiratory tract is the most important route of infection**.

3. **Once contracted, the infection persists throughout the life of the chicken, and infected birds continue to contaminate the environment by shedding the virus.** Continued shedding of the virus by infected birds and hardiness of the virus are responsible for prevalence of the infection. **The virus survives for months outside the birds.**

Symptoms

Marek's disease affects chickens from about 6 weeks of age. It occurs usually **between 12 and 24 weeks of age**, but older birds may also be affected. Clinical disease occurs mainly in **two forms**: (1) **Classical Marek's disease**, and (2) **Acute Marek's disease**.

Classical Marek's Disease

In our country this form is now rarely seen. It used to be common once upon a time. The symptoms depend on which nerve is affected. Involvement of brachial and sciatic nerves is common, and leads to paralysis of the wings and legs. A particularly characteristic posture is that in which the bird lies on its side with one leg stretched forward and the other backward as a result of leg nerve involvement. Mortality varies, but is rarely more than 10-15%.

Acute Marek's Disease

This is the most common form of Marek's disease encountered in our country. Mortality in this form is usually much higher than in the classical form. Mortality of 10-30% of the flock is common, and outbreaks involving up to 80% of the flock are recorded. Many birds die suddenly without preceding symptoms. Others appear depressed before death, and some show paralytic symptoms similar to those seen in the classical form. A particularly characteristic posture is that in which the bird lies on its side with one leg stretched forward and the other backward (Fig. 18, 19). Non-specific signs such as weight loss, paleness, shrunken combs, loss of appetite, and diarrhoea may be observed, especially in birds in which the course is prolonged.

Postmortem Findings

1. **In classical Marek's disease**, the characteristic finding is marked enlargement of one or more nerves. Nerves commonly affected are sciatic and brachial. Affected nerves are up to 2-3 times the normal thickness (Fig. 20).
2. **Acute Marek's disease** is characterized by marked enlargement of the liver, spleen, kidneys, lungs, gonads (ovary, testes), proventriculus, and heart. In younger birds, liver enlargement is moderate, but in adult birds the liver is greatly enlarged, which is similar to that in lymphoid leukosis. **A characteristic postmortem finding of Marek's disease is marked enlargement of the liver (21, 22, 23, 24, 25, 26, 27) and spleen (28, 29, 30), several times their normal size, showing white spots of cancerous tissue on their surface**. The other typical finding in our country is significant enlargement of the proventriculus. When opened, its wall is greatly thickened and the internal lining shows irregular, somewhat diffuse, blotchy haemorrhages, quite different from those seen in Ranikhet disease (Fig. 31, 32, 33, 34, 35).

Diagnosis

Diagnosis is based on the characteristic postmortem findings. That is, from the markedly enlarged liver and spleen, and the presence of tumours in various other internal organs.

Treatment

There is no treatment for Marek's disease.

Control

Control is based on management methods, which include: (1) isolation of growing chickens from sources of infection, (2) vaccination, (3) use of genetically resistant stock.

Fig. 18. **Marek's disease**. Characteristic posture of a **29-week-old layer chicken** showing extension of one leg and retraction of the other. This is typical of Marek's disease.

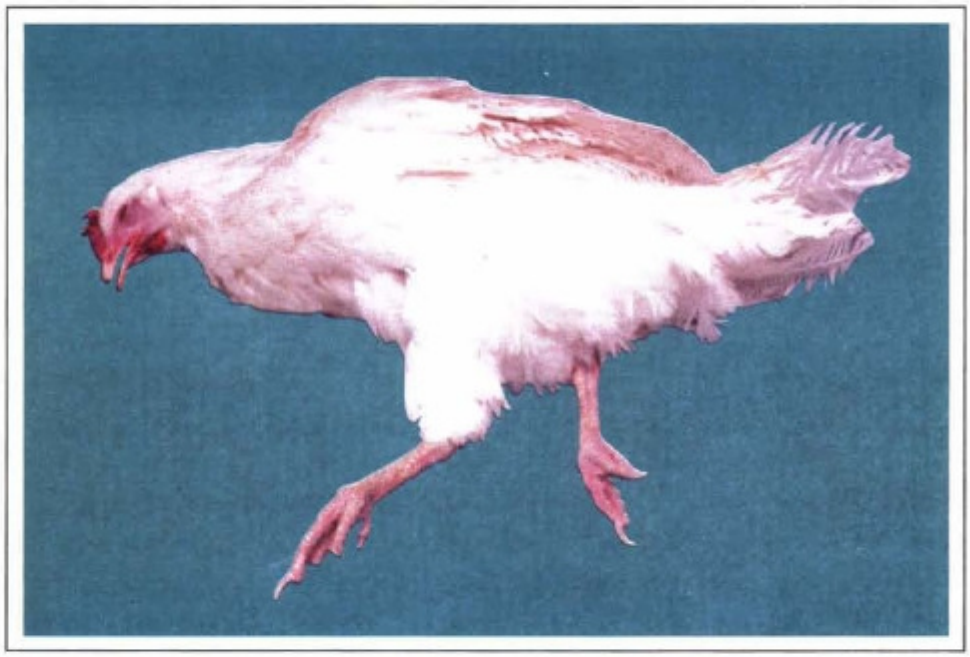

Fig. 19. **Marek's disease**. Another **29-week-old layer chicken** showing a somewhat similar posture typical of Marek's disease.

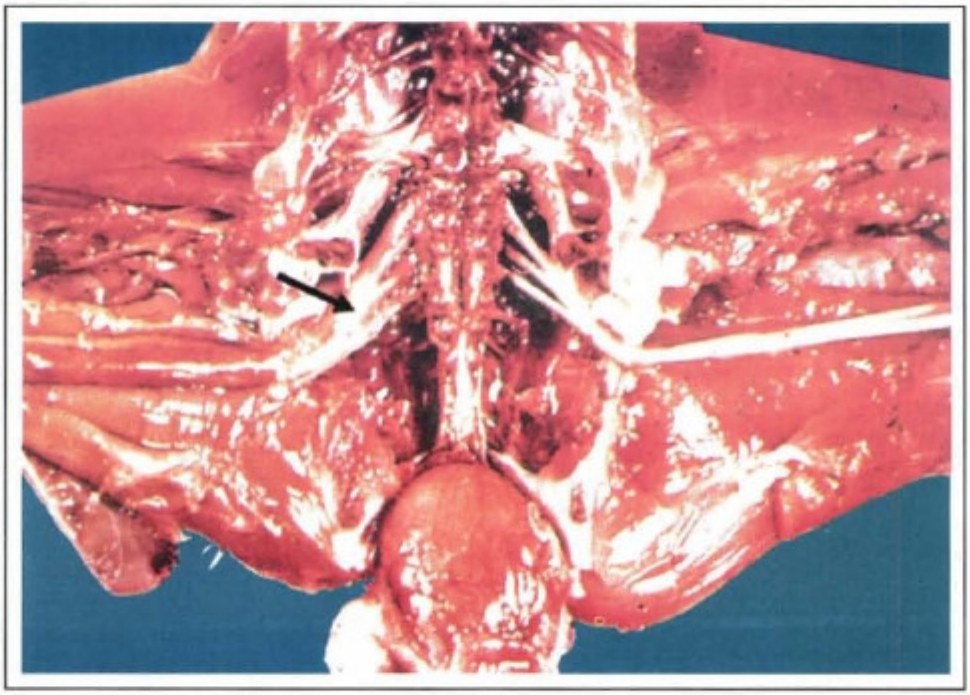

Fig. 20. **Marek's disease (classical form).** Note enlargement of nerves in the left sciatic plexus (network) in a **hen** (arrow). **This is characteristic of the classical form.** The right sciatic plexus is normal.

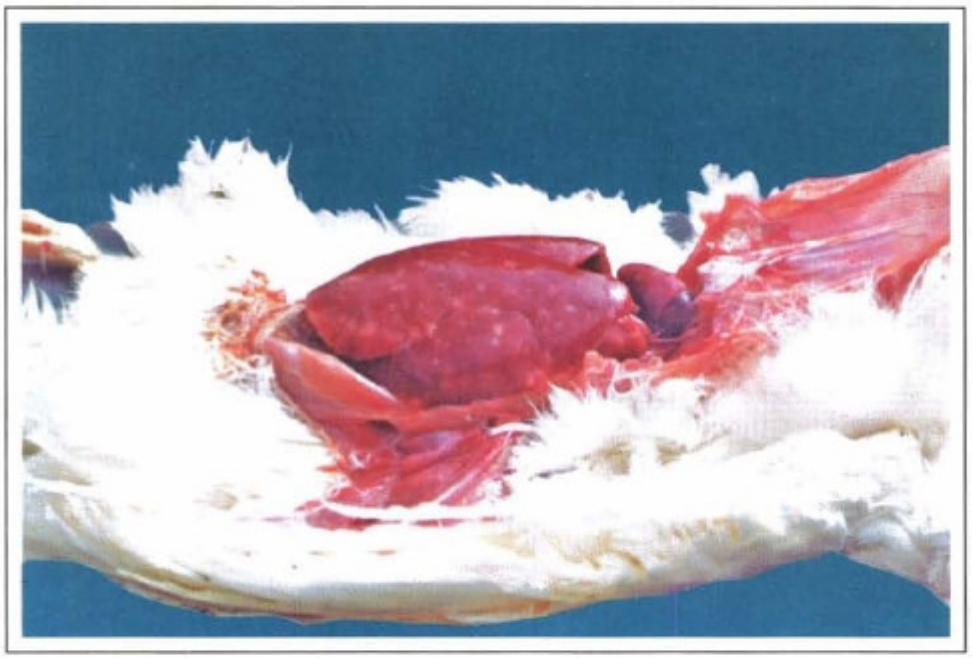

Fig. 21. **Marek's disease (acute form)** in a **25-week-old layer chicken**. Note liver is greatly enlarged and shows numerous tumour lesions (yellowish spots).

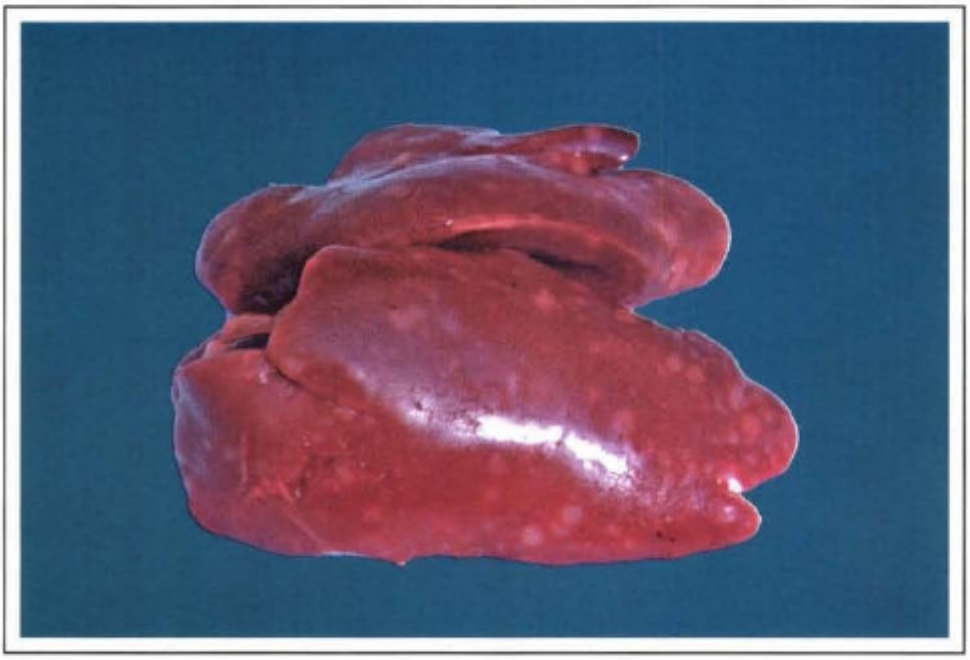

Fig. 22. **Marek's disease (acute form)** from the **same 25-week-old layer chicken** shown in Fig. 21. A close-up picture of the liver shows tumour lesions more distinctly (yellowish spots).

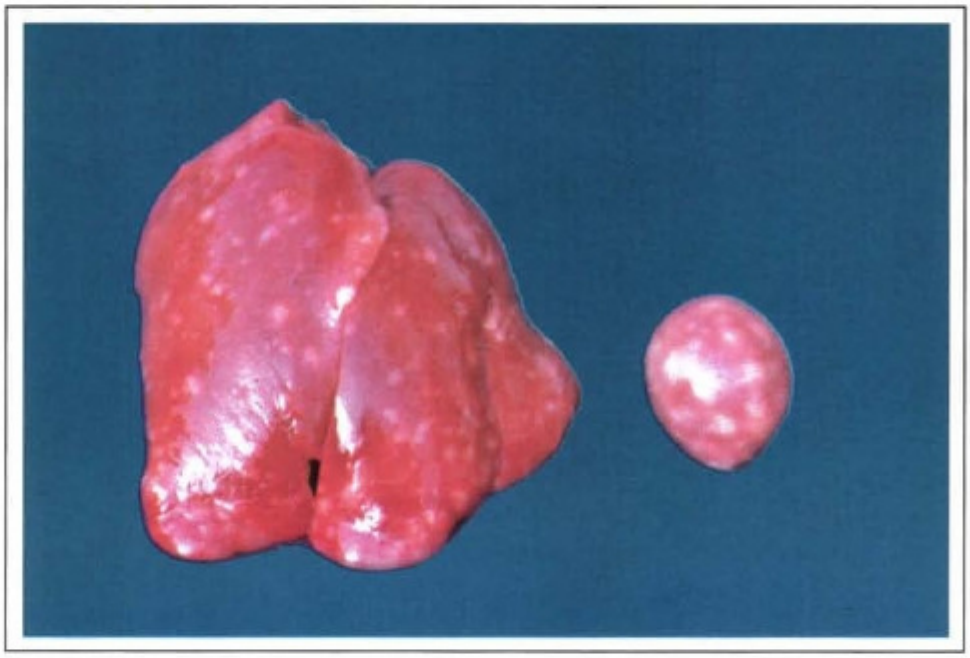

Fig. 23. **Marek's disease (acute form)** from another **25-week-old layer chicken**. Both liver (left) and spleen (right) show numerous tumour lesions (yellowish spots).

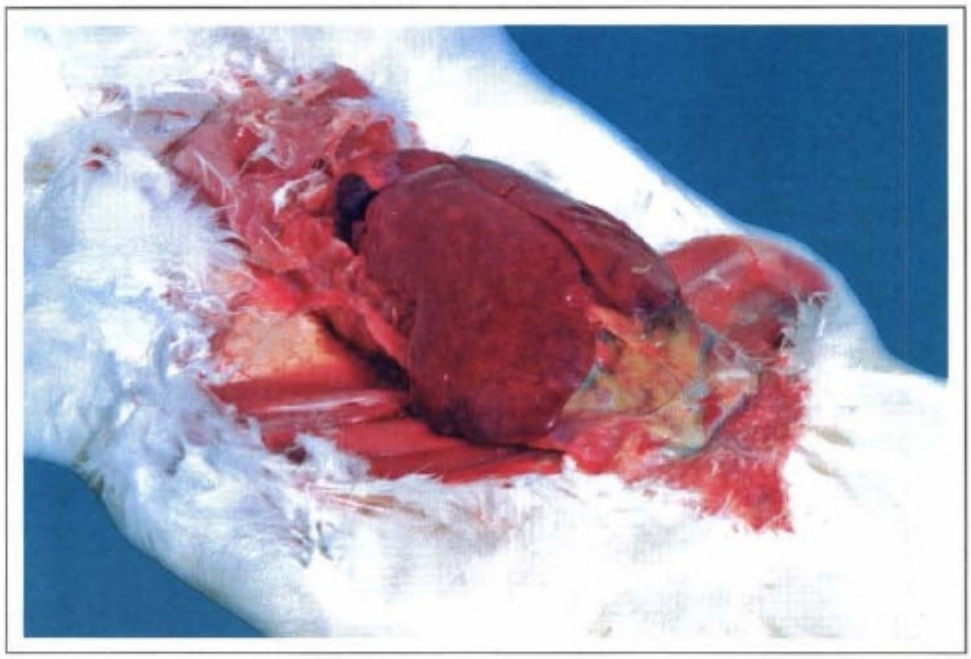

Fig. 24. **Marek's disease (acute form)** in a **20-week-old layer chicken**. Note liver is markedly enlarged and shows the presence of tumour lesions throughout (faintly yellowish spots).

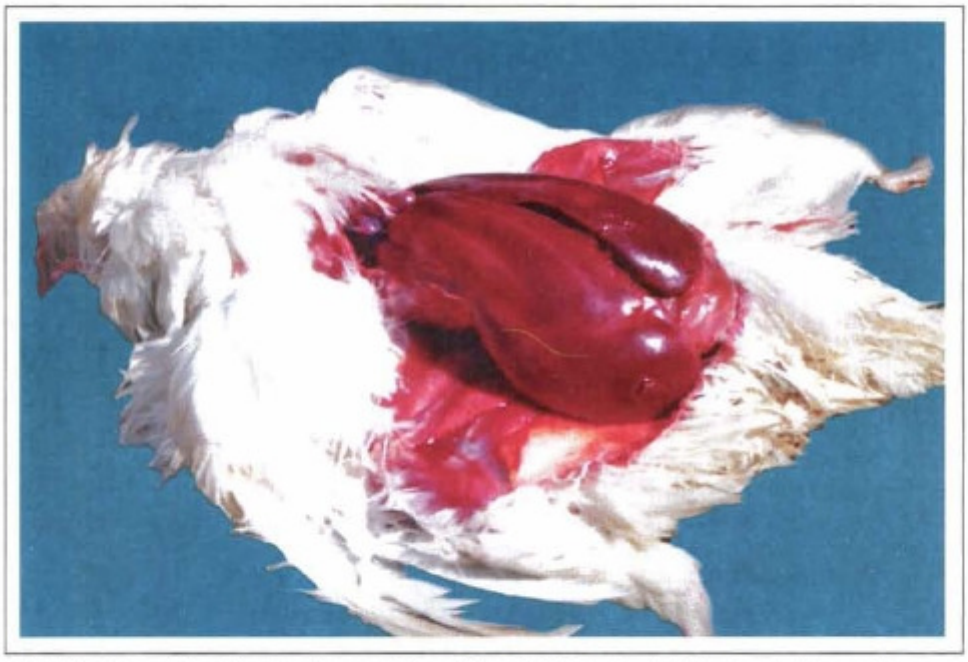

Fig. 25. **Marek's disease (acute form)** in a **29-week-old layer chicken**. Note a massive increase in the size of liver.

Fig. 26. **Marek's disease (acute form)** in a different **29-week-old layer chicken**. Note the affected liver (right) is enlarged to several times its normal size. Compare it with a normal liver on the left, from a different bird of the same age. Note also the presence of a distinct tumour on the left border of the affected liver (arrow).

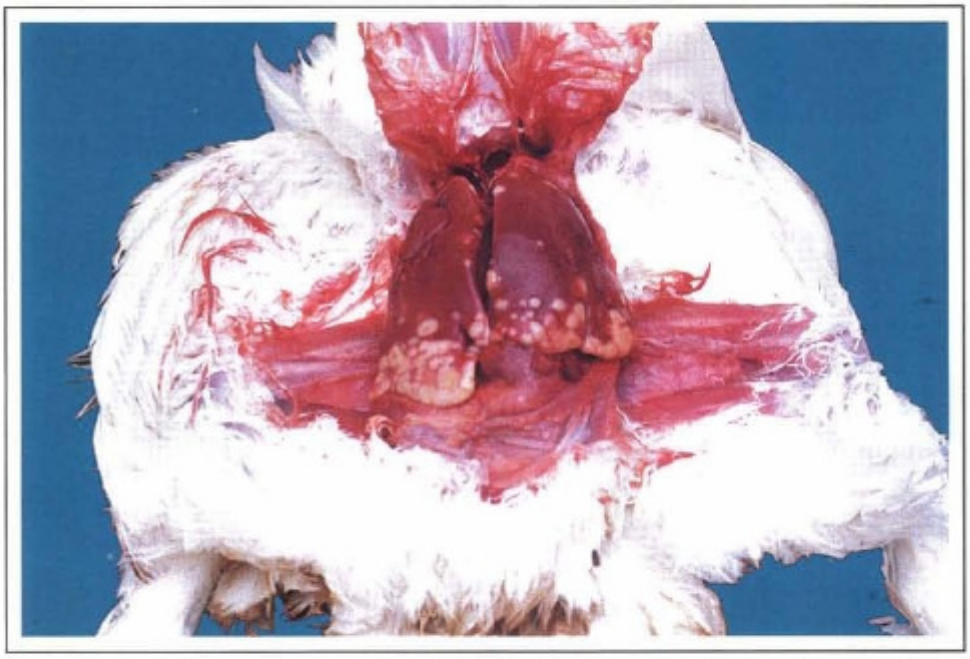

Fig. 27. **Marek's disease (acute form)** in a **27-week-old layer chicken**. Note liver shows numerous tumour lesions at its lower end (yellowish areas).

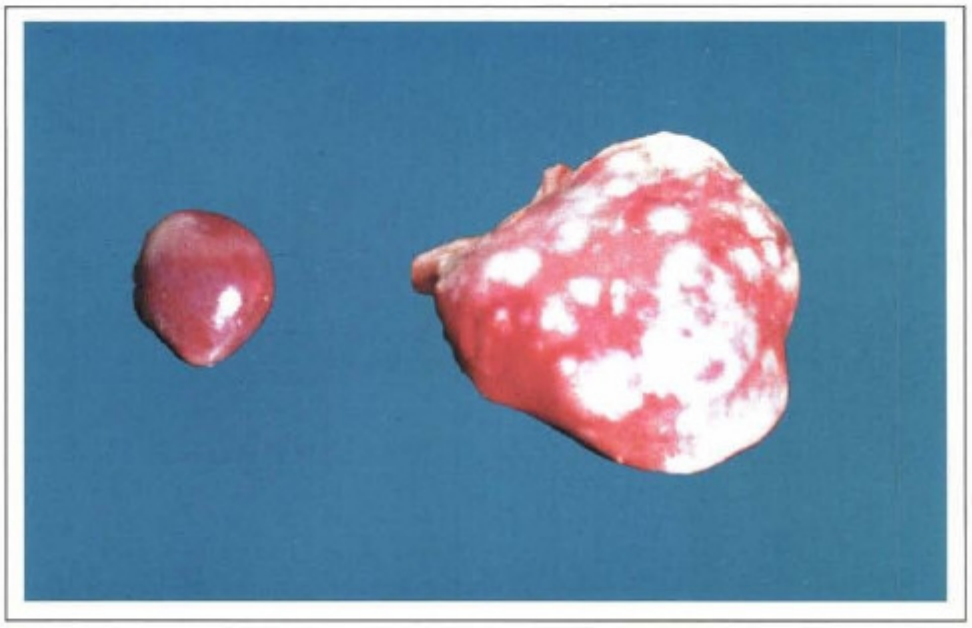

Fig. 28. **Marek's disease (acute form)** from the same **29-week-old layer chicken** shown in Fig. 26. The close-up picture shows affected spleen on the right and normal spleen on the left. Note the affected spleen is enlarged to several times its normal size and shows numerous distinct tumour lesions (diffuse whitish areas).

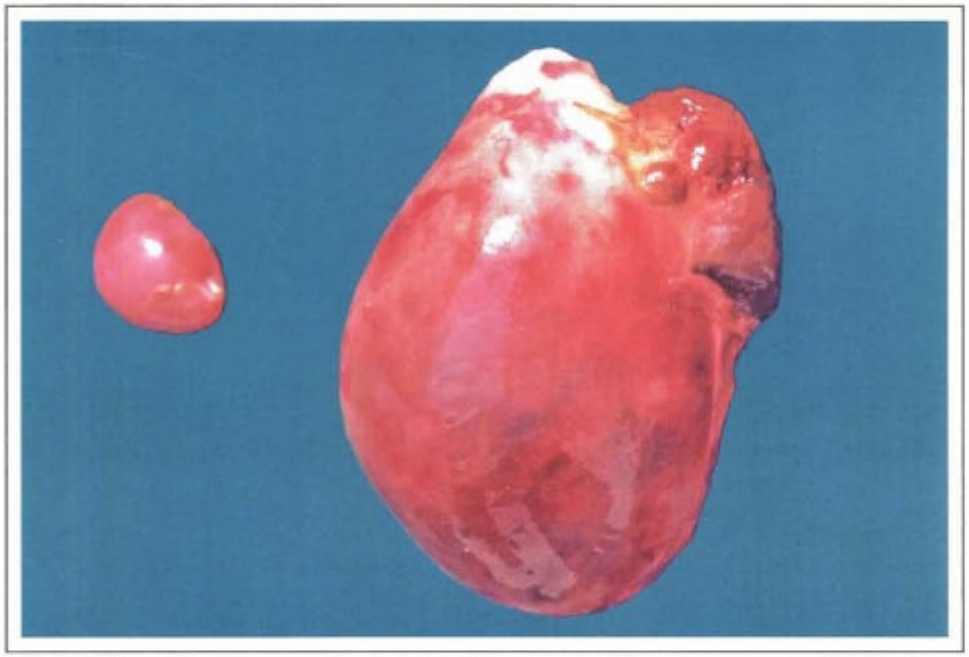

Fig. 29. **Marek's disease (acute form)** in a **20-week-old layer chicken**. Note tremendous enlargement of the affected spleen (right). On the left is a normal spleen from a layer bird of the same age. Weight of the **normal spleen was 7 gram, whereas that of the affected spleen was 125 gram!**

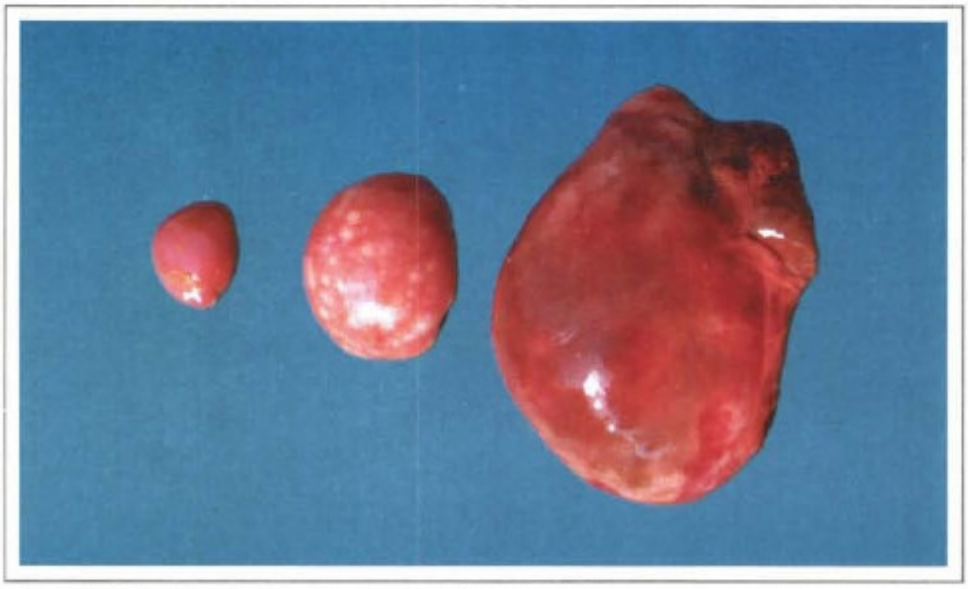

Fig. 30. **Marek's disease (acute form)**The big affected spleen on the right is from the same **20-week-old layer chicken** shown in Fig. 29. Spleen in the middle is from **another 20-week-old layer chicken** affected with Marek's disease. Although it is only moderately enlarged, it shows the presence of tumour lesions throughout (yellowish spots). Weight of the normal spleen (left) was 7 gram, that of the middle 25 gram, and of the affected on the right **125 gram.**

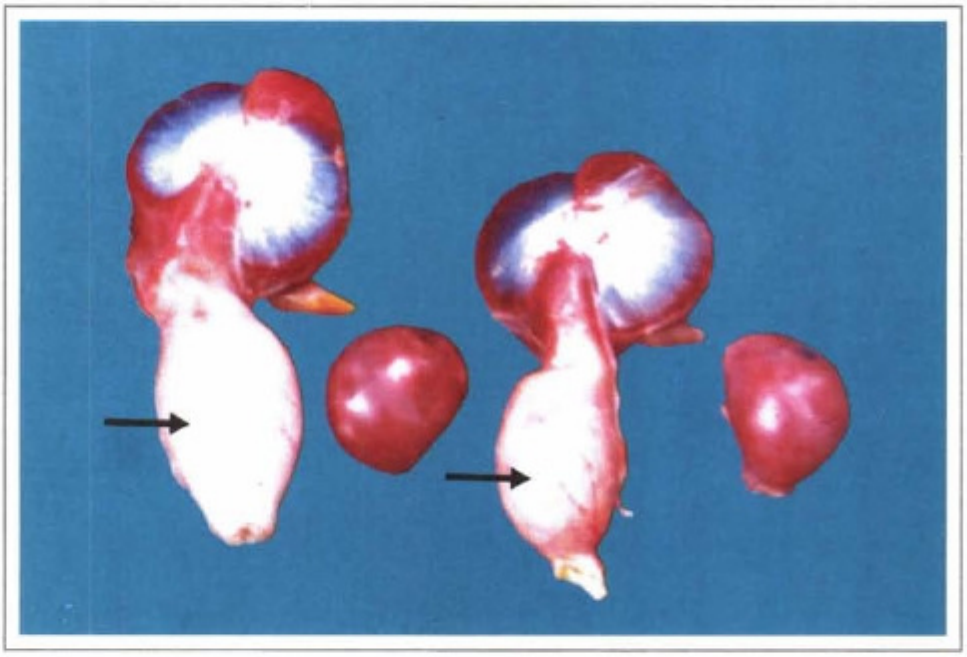

Fig. 31. **Marek's disease (acute form)** in a **22-week-old layer chicken**. Note two markedly enlarged, thickened, and firm proventriculi (arrows). **Such an enlargement of proventriculus is characteristic of Marek's disease.** The spleens are also enlarged and show distinct tumour lesions (yellowish areas).

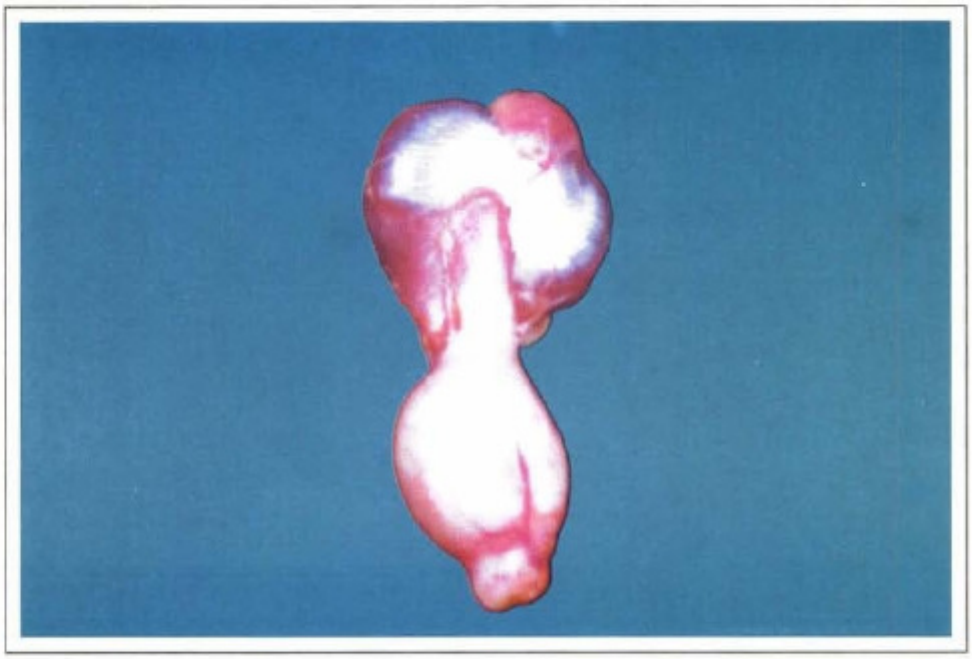

Fig. 32. **Marek's disease (acute form)** in a **29-week-old layer chicken**. Note enlarged proventriculus. When opened, it showed irregular, somewhat diffuse, blotchy haemorrhages (see Fig. 33).

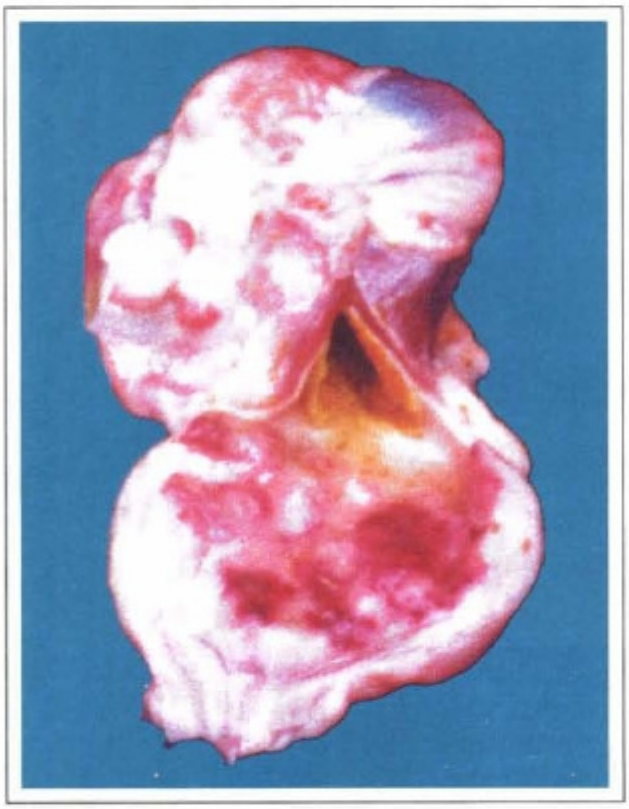

Fig. 33. **Marek's disease (acute form)** from the same **29-week-old layer chicken** shown in Fig. 32. The proventriculus is now cut open and reveals irregular, somewhat diffuse, blotchy haemorrhages. Its wall is thickened.

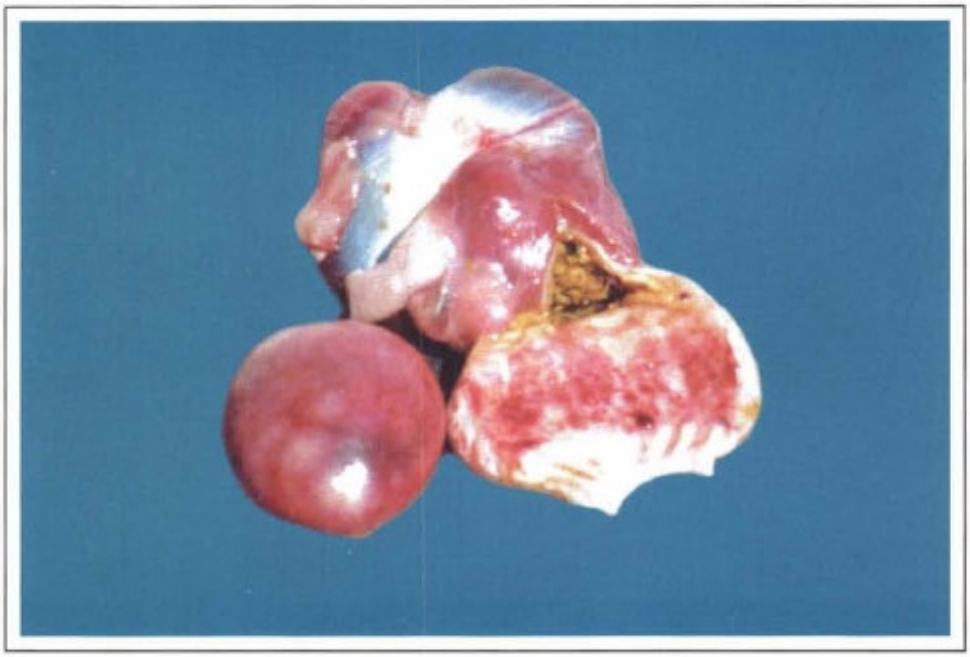

Fig. 34. **Marek's disease (acute form)** in a **19-week-old grower chicken**. Note proventriculus (on the right) is thickened and shows diffuse haemorrhages on its inner surface. Spleen, on the left, is greatly enlarged and shows tumour lesions on its surface (faintly yellowish areas).

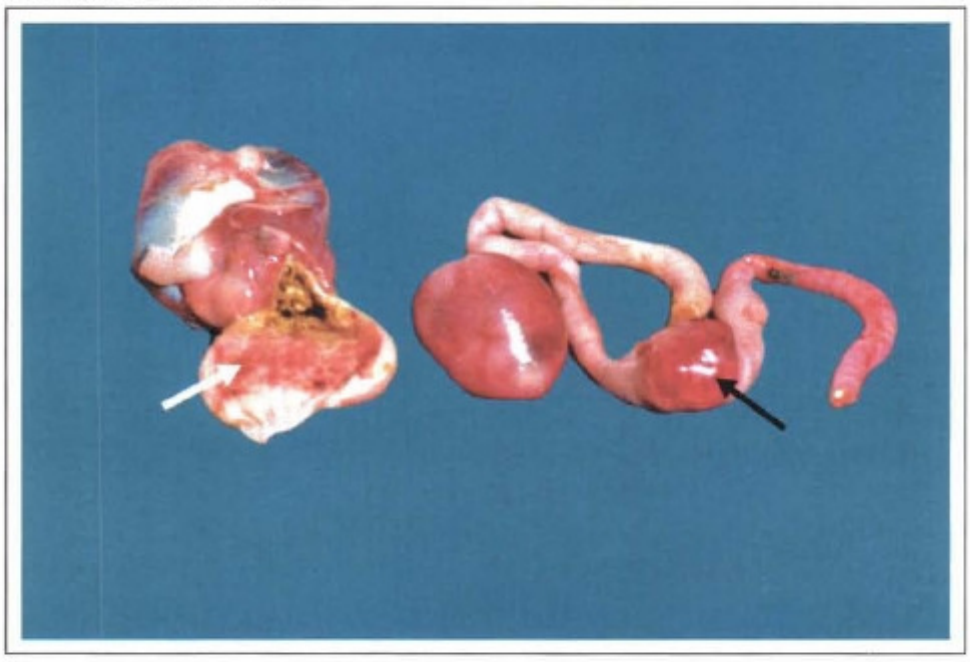

Fig. 35. **Marek's disease (acute form)** from the same **19-week-old grower chicken** shown in Fig. 34. Note that, besides diffuse haemorrhages in the proventriculus (white arrow) and enlarged spleen, small intestine shows the presence of a tumour (black arrow).

FOWL POX

Fowl pox is a viral disease characterized by skin lesions (changes). Although there is another form of the disease, in which changes appear in the mouth and upper respiratory tract, this form of the disease is also usually associated with skin changes in some birds. The disease was once widely prevalent, but with the arrival of vaccination its incidence is greatly reduced. However, still in some areas it continues to be of considerable economic importance.

Cause

A virus - called **avipoxvirus**. Fowl pox virus infects birds of both sexes, of all ages and breeds.

Spread

1. **Spread** of the virus from one bird to another **by direct contact is the main method of spread**. Most of the spread is the result of birds picking, fighting, or scratching one another. Some break in the skin is required for the virus to enter the cells, grow, and cause disease.
2. Infection also occurs by **mechanical spread of virus** to the injured skin. Individuals handling birds at the time of vaccination may carry the virus on their hands and clothes, and may unknowingly deposit the virus in the eyes of susceptible birds.
3. **Mosquitoes** are known to transmit the disease and produce eye infection. Mosquitoes can infect a number of birds after a single feeding on a bird infected with fowl pox virus.
4. In a contaminated environment, presence of virus in the air from **feathers and dried scabs** containing fowl pox virus, may cause skin and respiratory tract infection. The virus can survive in dried scabs for months or even years.
5. **Bad sanitary and hygienic conditions help in the spread of the disease.**

Symptoms

Fowl pox can occur at any age. There are **two forms of the disease**: (1) **skin** or **cutaneous form** (**dry pox**), and **diphtheritic form** (**wet form**).

In the **skin form**, lesions (changes) appear on the unfeathered skin of the head, neck, comb, wattles, eyelids, legs, and feet. The lesions on the head, combs, and wattles are usually **wart-like (nodular) in appearance**, and yellow to dark brown in colour (Fig. 36, 37).

In the **diphtheritic form** (**wet form**), small white nodules are observed in upper respiratory and digestive tracts. These nodules merge together to form **raised-yellow white cheesy patches**. Most lesions are found in the mouth, but may also be present in the **larynx**, trachea, and oesophagus. These lesions cause **difficulty in breathing.** Recently it has been found that most cases of diphtheritic fowl pox are characterized by the **formation of massive yellow cheesy necrotic masses in the larynx and adjacent trachea** (Fig. 38, 39, 40). The bird in such cases dies suddenly from asphyxiation (lack of oxygen). Lesions in the nares (nose) give rise to nasal discharge, while those on the conjunctiva to eye discharge.

Fowl pox usually causes weakness and poor weight gain. In layers, egg production is temporarily stopped. Mortality is low (1 to 2%) when skin lesions are present, but may be as high as 50% with the diphtheritic form, but is usually low.

Diagnosis

Wart-like lesions of the head particularly of the comb and around the eyes, or **yellow cheesy lesions in the larynx and adjacent trachea on postmortem, are diagnostic of fowl pox**.

Treatment

There is no satisfactory treatment.

Control

1. **Fowl pox can be prevented by vaccination**. Therefore, preventive vaccination using a live vaccine is the most successful 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 infection.
2. **Precautions** should be taken **to minimize the spread of the vaccine virus**, both on the birds and in the environment. Being a live virus, it is capable of spreading the disease and therefore must be handled carefully.
3. **Carcasses of dead or affected birds should be buried or burnt**. After removal of the birds the house should be thoroughly disinfected, although the virus remains in the infected scabs and is difficult to clear from certain premises.

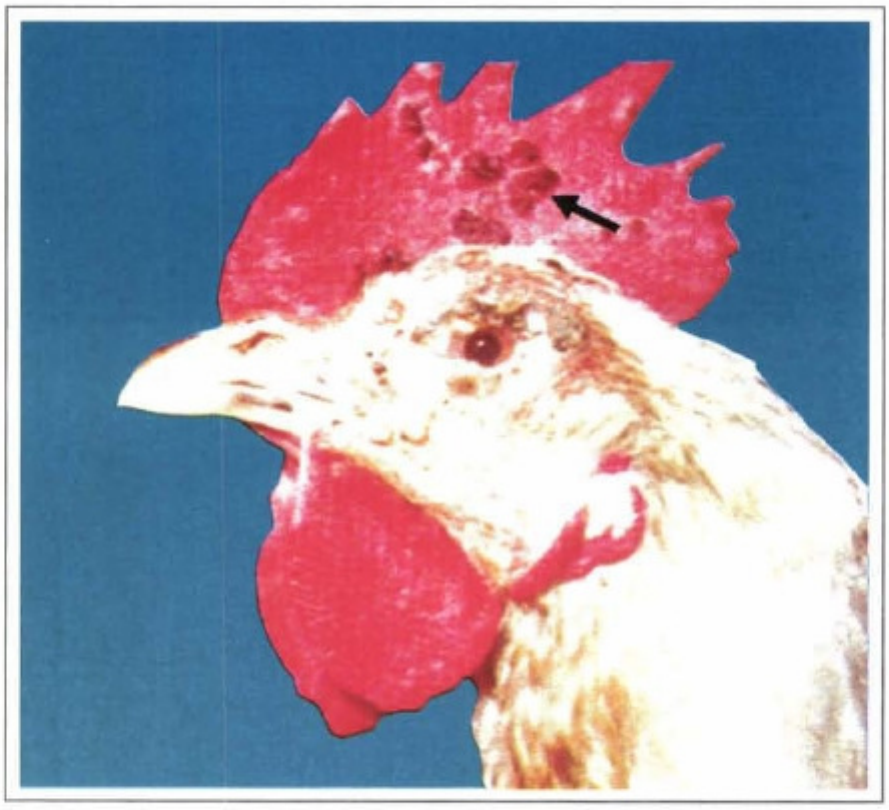

Fig. 36. **Fowl pox (skin form).** Note lesions on the comb (arrow) and eye of a **chicken**.

Fig. 37. **Fowl pox (skin form)**. Note lesions on the comb of a **chicken** (arrow).

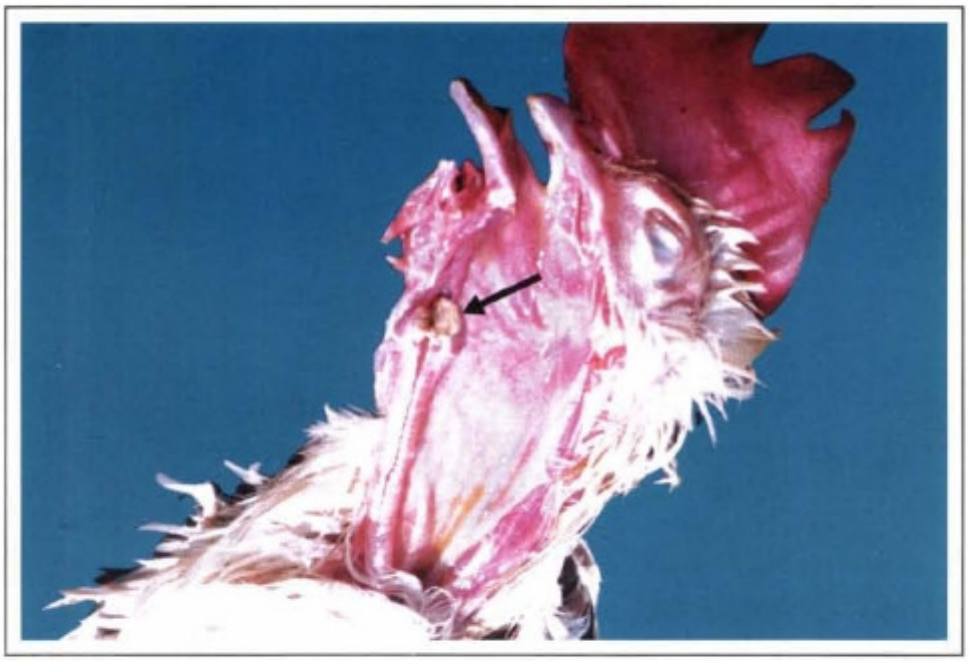

Fig. 38. **Fowl pox (diphtheritic form)** in a **40-week-old layer chicken**. Note the presence of cheesy plaques (masses) in the larynx and adjacent trachea (arrow).

Fig. 39. **Fowl pox (diphtheritic form)** in another **40-week-old layer chicken**. Note the presence of cheesy plaques (masses) in the larynx and adjacent trachea (arrow).

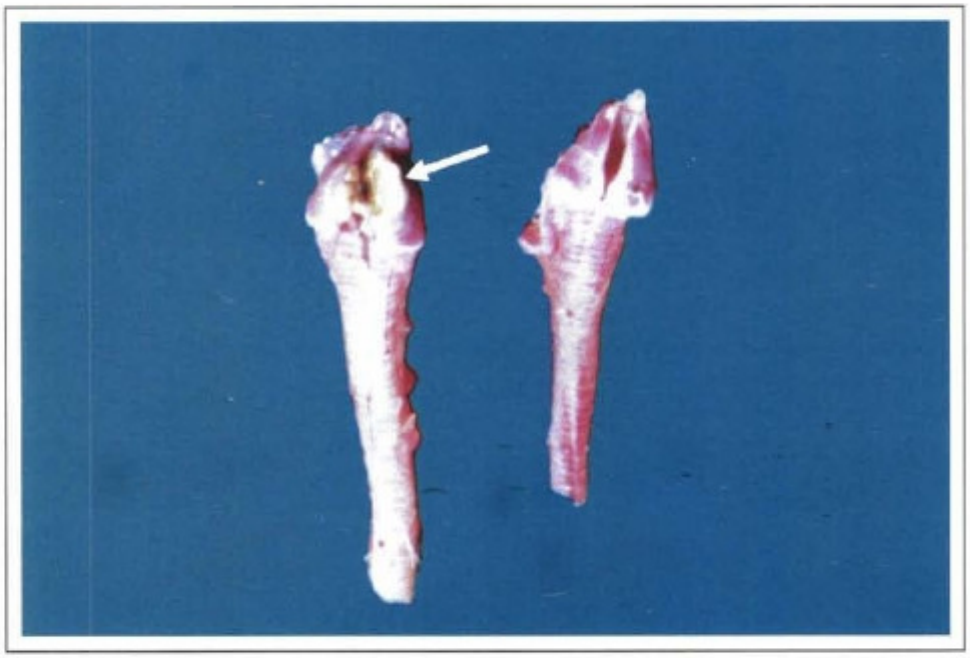

Fig. 40. **Fowl pox (diphtheritic form)** from a different **40-week-old layer chicken**. Note the presence of cheesy plaques in the affected larynx and adjacent trachea (arrow). Larynx and trachea on the right are normal.

INFECTIOUS BRONCHITIS

Infectious bronchitis is a sudden, rapid, highly contagious disease of chickens characterized by abnormal respiratory sounds, coughing, and sneezing. **It is an important disease of young chicks, causing high mortality**. The virus may also affect kidneys. In laying birds, the disease causes great economic loss through reduced egg production and poor eggshell quality. Both in broilers and layers, infection causes poor weight gain and feed efficiency. Birds of all ages are susceptible but the disease is most common in young chicks, causing some mortality.

Cause

A virus - called **coronavirus**. The virus survives outside the bird under farm conditions for weeks to months. However, it is rapidly killed by common disinfectants.

Spread

1. **By the air**. As the virus is easily spread by air, **inhaling infected air** is the most important means of spread. However, **spread through infected faeces** may also be important.
2. Spread by **people and virus-contaminated materials** can occur.
3. **Carriers** (birds which carry infection without showing symptoms) also spread the disease. Birds may shed the virus for up to 4 weeks after recovery.

Symptoms

1. The **respiratory form** is the most common in birds of all ages. The symptoms include abnormal respiratory sounds, severe respiratory distress (Fig. 41), gasping and sneezing, watery nasal discharge, and sometimes eye discharge and facial swelling.
2. In the **reproductive form**, there is reduced egg production. Drop in production may sometimes be more than 50%. Egg production may become normal after 3-4 weeks, but there is a fall in egg quality. **Eggs** may be smaller, **deformed** (Fig. 42), **shell-less** (Fig. 43), or have calcium containing deposits on the surface. Inside, the albumen loses its viscosity. That is, the albumen is thin and watery, without clear-cut demarcation between the thick and thin albumen of the normal fresh egg.
3. In the **kidney form**, there is marked depression usually with respiratory symptoms, and mortality as high as 30% in the severe form.

Postmortem Findings

In the **mild respiratory form**, there is excess mucus in the respiratory tract. In the **more severe form**, a caseous plug may be found in the lower trachea or bronchi of chicks that die. In the **kidney form**, the kidneys are swollen and pale. The ureters are distended with urates. In some birds there is visceral gout, in which case white granular material (urates) may coat the internal organs (see 'visceral gout').

Diagnosis

1. Symptoms and postmortem findings may be suggestive, but are not diagnostic.
2. Infectious bronchitis must be differentiated from Ranikhet disease and infectious coryza. Ranikhet disease is more severe, and in layer flocks drop in egg production is more than with infectious bronchitis. Infectious coryza can be differentiated on the basis of facial swelling which occurs only rarely in infectious bronchitis.

Treatment

There is no specific treatment for infectious bronchitis. In mixed infections, use antibiotics against ***E. coli*** and mycoplasma. In the kidney form of disease, administer electrolytes in the drinking water.

Control

1. Management can be helpful if proper attention is given to maintaining proper ventilation of chicks.
2. As treatment is of no value and because this virus is so commonly present everywhere and spreads rapidly, control depends on increasing resistance of flocks by vaccination. Both live and killed vaccines are available and both have been shown to be of value.

Fig. 41. **Infectious bronchitis** in an **8-day-old broiler chicken**. Note the symptom of severe respiratory distress.

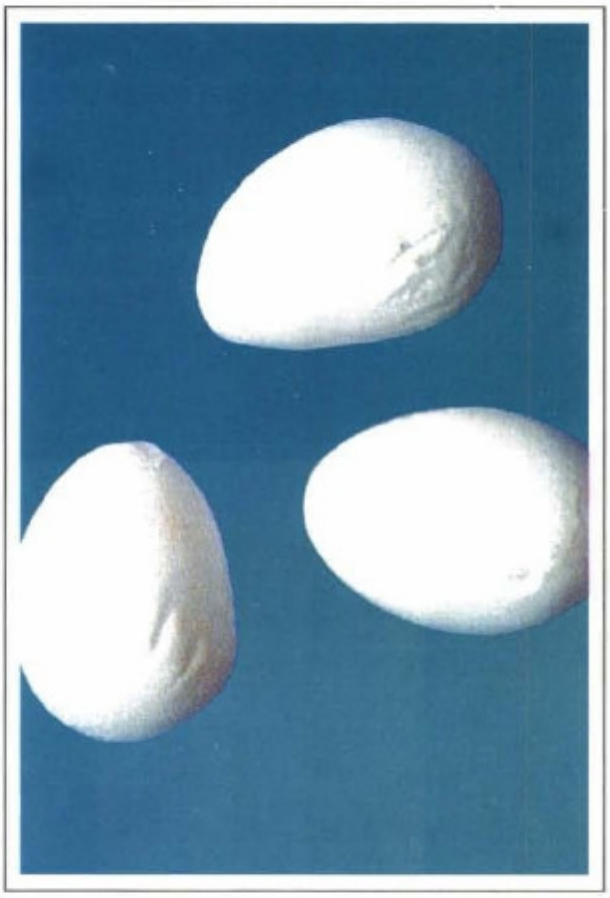

Fig. 42. **Infectious bronchitis**. Note deformed eggs.

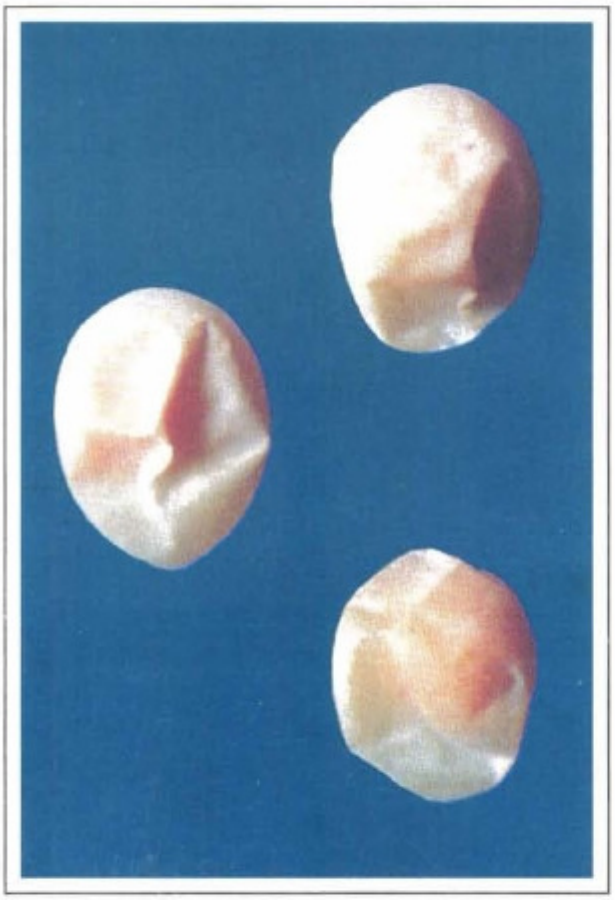

Fig. 43. **Infectious bronchitis**. Note shell-less eggs.

LYMPHOID LEUKOSIS

Lymphoid leukosis is a tumour-producing viral disease of chickens. Lymphoid leukosis is characterized by marked enlargement of the liver. The disease occurs any time after 16 weeks of age. The incidence is highest at about sexual maturity. Lymphoid leukosis is usually a problem of laying hens.

Lymphoid leukosis and Marek's disease closely resemble each other in their involvement of liver and other organs. **Whereas, today, Marek's disease is a burning problem of the poultry industry, lymphoid leukosis has been largely controlled through breeding and is now uncommon.**

Cause

Alpharetroviruses, cause of lymphoid leukosis, are widely prevalent in commercial chickens. Infection occurs in all chicken flocks. By sexual maturity, most birds are exposed. However, the incidence of clinical disease is usually low.

Spread

Lymphoid leukosis virus is transmitted both **vertically** from hen to progeny through the egg, and also **horizontally** from bird to bird by direct or indirect contact. Usually only a small number of chicks are infected vertically, and the majority become infected by contact with vertically infected chicks during rearing.

Symptoms

1. The symptoms are non-specific.
2. The birds are usually pale and emaciated, and show loss of appetite.
3. The comb may be pale, shrivelled, and of bluish discoloration (cyanotic).
4. Diarrhoea may occur and wattles may be pale.
5. The abdomen is usually very large because of the massive liver.

Postmortem Findings

1. **The liver is greatly enlarged** (Fig. 44).
2. The spleen, **bursa of Fabricius**, kidneys, and ovary are also usually enlarged.

Diagnosis

Postmortem findings may suggest lymphoid leukosis. However, as lymphoid leukosis closely resembles Marek's disease, both diseases can be differentiated with difficulty. Diagnosis is possible in most cases on careful microscopic examination. **However, when distinct lymphoid tumours are present in the bursa, a diagnosis of lymphoid leukosis can be made. Such tumours in bursa are absent in Marek's disease.**

Treatment

No treatment or vaccines are available.

Control

1. Control must be based on high standards of hygiene and flock management to reduce infection from the environment.
2. As the infection is egg transmitted, it cannot be prevented by rearing chicks in isolation.

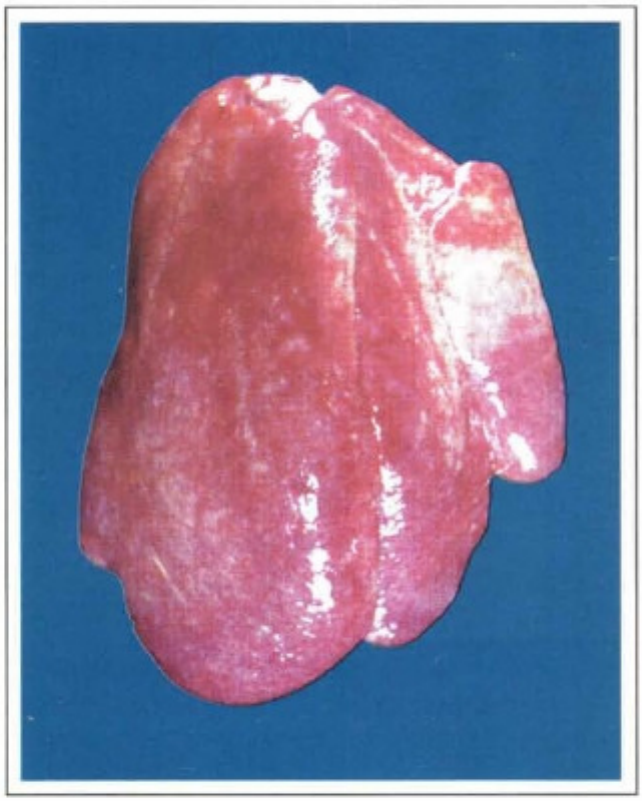

Fig. 44. **Lymphoid leukosis** in a **hen**. Note marked enlargement of the liver.

AVIAN ENCEPHALOMYELITIS

Avian encephalomyelitis is a viral disease of chicks. It is characterized by muscular incoordination and rapid tremors, especially of the head and neck. Because of the trembling and shaking, it is also called **'epidemic tremor'**. Its economic importance is mainly due to the mortality it causes in chickens.

Cause

A virus called **enterovirus**.

Spread

1. Transmission through hatching egg (**vertical transmission**) is a very important means of spread.
2. **Contaminated water** and **feed** are sources of spread from bird to bird and house to house.
3. Disease can be spread by people and equipment contaminated with virus-containing faeces. **Ingestion is the usual route of entry**. Exposure through the respiratory tract is unimportant.

Symptoms

1. The disease usually occurs **when chicks are 1-2 weeks of age**. Symptoms include depression, muscular incoordination, and tremors. The nervous symptoms may be seen soon after hatching, but are usually

seen at one week of age. The muscular incoordination varies from slight incoordination to sitting on the hocks, or lying down on one side (Fig. 45). The birds are unable to move. In such a condition, death occurs from not reaching to the feed (starvation), or getting crushed by other members of the flock. Birds over 4 weeks of age rarely show avian encephalomyelitis.

2. In layers, there is some drop of egg production, with reduced hatchability.
3. Very mildly affected birds recover completely.

Postmortem Findings

There are no gross lesions (changes) in the young or older birds. Therefore, the disease cannot be diagnosed from postmortem.

Diagnosis

1. The symptoms in young birds, absence of gross changes at postmortem together with the absence of other infectious and nutritional deficiencies affecting the nervous system, are strongly suggestive of avian encephalomyelitis.
2. However, a definitive diagnosis requires help of laboratory tests.
3. In differential diagnosis, it is necessary to rule out vitamin E deficiency (nutritional encephalomalacia; see 'vitamin E deficiency').

Treatment

Treatment is of no value.

Control

1. Under commercial conditions, it is not possible to eliminate infection by high standards of hygiene alone. Thus, control depends on the vaccination of birds, and using hatchery eggs from breeder flocks that are free from avian encephalomyelitis.
2. Live and killed vaccines can be given which produce a lasting and adequate degree of protection.

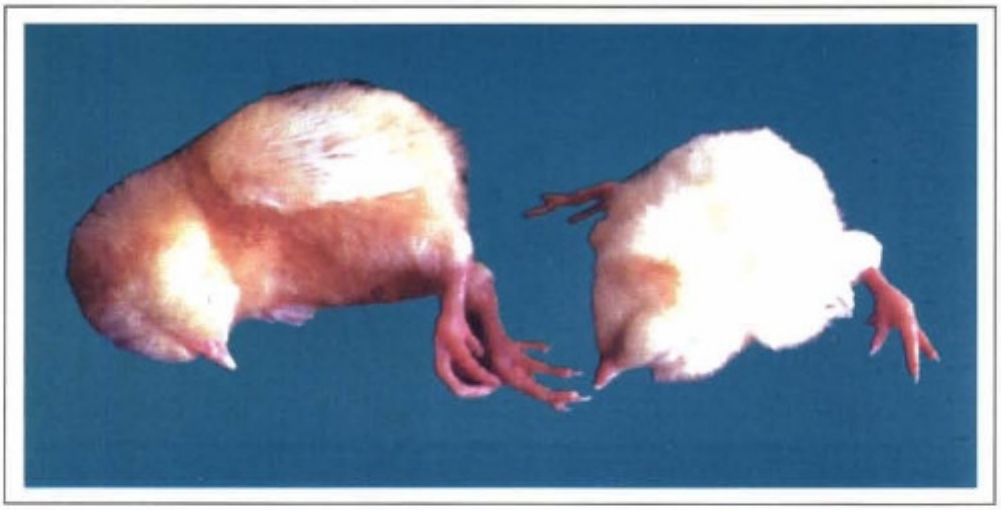

Fig. 45. Muscular incoordination and recumbent (lying down) position of **chicks**. This may be due to **avian encephalomyelitis**, or deficiency of vitamin A, or E.

2 BACTERIAL DISEASES

E. coli Infection (Colibacillosis)

Escherichia coli* infection** or **colibacillosis** is the most common, the most widely prevalent, and economically the most important bacterial disease of poultry in our country. It may be seen right from the day-old chick as **yolk sac infection** up to the adult bird as **egg peritonitis. It occurs throughout the year**. Collectively, infections caused by ***E. coli are responsible for huge economic losses to poultry farmers.

Cause

A bacterium called ***Escherichia coli***. ***E. coli*** normally lives in the digestive tract of birds, but most types (strains) do not produce disease. Certain strains, however, can cause disease in poultry. These diseases include **blood infection (colisepticaemia)** and **yolk sac infection in young chickens**, and **egg peritonitis** (inflammation of the peritoneum, a membrane in the abdominal cavity) **in adult chickens**. All the conditions are collectively called **'Colibacillosis'.**

E. coli causes disease only when there is a serious managemental fault, or some other underlying disease-producing organism like **Mycoplasma** is present. Thus, ***E. coli*** infection occurs when bird's defences have been damaged or defeated.

1. Yolk Sac Infection

Yolk sac infection is the commonest cause of mortality in chicks during the first week after hatching **(early chick mortality). Faecal contamination of the eggs** is the most important source of infection. Bacteria can grow in the hatching egg after entering the shell. Other sources may be infection of the ovary and inflammation of the oviduct (salpingitis). The incidence of infection increases soon after hatching and decreases after about 6 days. ***E. coli*** enters through the unhealed navel. Yolk of chicks is the site of infection. Yolk sac infection can cause up to 100% mortality in chicks during the first week of life, but deaths are usually between 5 to 10%.

Symptoms

Affected chicks are depressed, have swollen abdomens, and show a tendency to crowd together.

Postmortem Findings

The yolk sac blood vessels are dilated and filled with blood (Fig. 46, 49). The lungs are congested and the liver and kidneys are dark and swollen. The most important finding is an inflamed unabsorbed yolk, the yolk being abnormal in colour and consistency (Fig. 47, 48, 50). It is usually very foul-smelling. Peritonitis and haemorrhages on the surface of intestine are most common.

2. Blood Infection (Colisepticaemia)

Blood infection is most common in young chickens, and is a very serious form of ***E. coli*** infection.

Sources of infection

1. Through **inhalation of *E. coli* contaminated dust** via the respiratory tract. This is the most important source of infection.
2. Through **ingestion** of **contaminated drinking water and feed.**

3. ***E. coli*** can be found in litter and faecal matter. Dust in poultry houses may contain between one lakh to ten lakhs ***E. coli*** per gram. The bacteria persist for long periods, particularly when dust is dry.
4. ***E. coli*** can also be introduced into the poultry flocks through **contaminated well water.**

Predisposing Factors

These are those factors which make the bird easily prone to infection, and include:

1. Contaminated water
2. Dry, dusty conditions. ***E. coli*** persist for long periods outside the bird's body in dry, dusty conditions.
3. Overcrowding
4. Poor ventilation
5. Poor litter condition
6. Exposure to ammonia
7. Temperature extremes (too hot or too cold)
8. Stress
9. Feed/water restriction
10. **Immunosuppression**: Certain factors cause immunosuppression. That is, they suppress the natural immune responses. The bird is therefore unable to fight ***E. coli*** and succumbs to infection. For example, viral infections and toxins, such as mycotoxins, cause immunosuppression and predispose the birds to colibacillosis.

Postmortem Findings

1. A layer of white fibrin covers heart (pericarditis) and liver (perihepatitis). **This is typical of blood infection by *E. coli*** (Fig. 51, 52, 53, 54, 55, 56, 57, 58, 59, 60).
2. Infected airsacs are thickened and usually contain cheese-like material.
3. Rarely, in certain cases, liver presents a **corn meal appearance**. That is, it appears as if ground maize has been sprinkled on its surface (Fig. 61).

3. Egg Peritonitis

Egg peritonitis is inflammation of the peritoneum caused by the presence of a broken egg in the abdominal cavity. Postmortem findings include the presence of scattered pieces of yolk, thickened yolk, cheese-like material, or **milky fluid in the abdominal cavity** (Fig. 62). Almost always a profuse growth of ***E. coli*** can be isolated from the caseous inspissated material in such cases. There is also inflammation of the ovaries and oviduct. A complete, or partially formed egg, may be found impacted in the oviduct.

Diagnosis

All the three conditions, namely**, yolk sac infection, colisepticaemia,** and **egg peritonitis** can be easily diagnosed from the **characteristic postmortem findings**.

Treatment

Any treatment must begin with a **complete sanitation programme** as most ***E. coli*** infections start with dirty surroundings and various environmental stresses. Broad-spectrum antibiotics (chloramphenicol, chlortetracycline, neomycin, nitrofurans, gentamicin, oxytetracycline, enrofloxacin, and others) may be helpful in treating the conditions. An antibiotic sensitivity test is essential in identifying the best drug to use in a treatment programme.

Control

1. Maintain the highest standards of management and sanitation.
2. Avoid overcrowding.
3. Avoid dry, dusty conditions.
4. Provide adequate ventilation in the poultry house.
5. Avoid build-up of ammonia.
6. Maintain proper litter conditions.
7. Protect the birds against extremes of temperature, heat or cold.
8. Ensure supply of clean water. Also use **water sanitizers**. Contaminated water can contain organisms in high numbers.
9. To prevent yolk sac infection and early chick mortality, provide the best possible brooding conditions.
10. Provide well-balanced diet to avoid mineral deficiencies and malnutrition. Ensure a supply of high protein, vitamin A, vitamin E, vitamin C, and zinc.
11. Provide probiotic in the very first drinking water given to chicks and continue to use it for at least a week, and thereafter in the feed.

Fig.46. **Yolk sac infection** in **two 3-day-old broiler chicks**. Note yolk sac is markedly distended and hyperaemic That is, it shows prominent vessels.

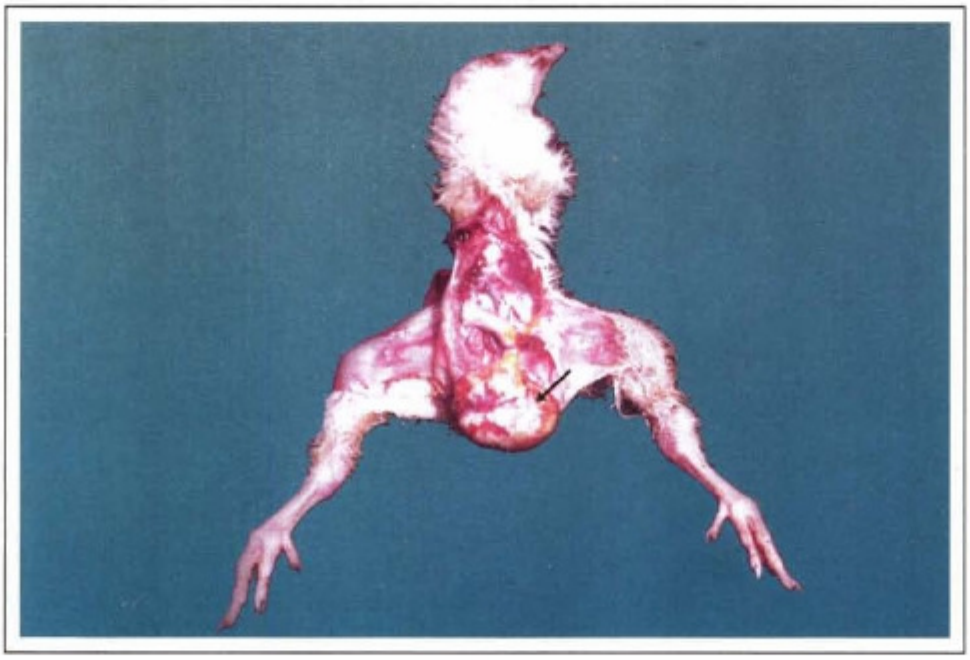

Fig.47. **Yolk sac infection** in a **10-day-old broiler chicken**. Note markedly infected yolk sac (arrow).

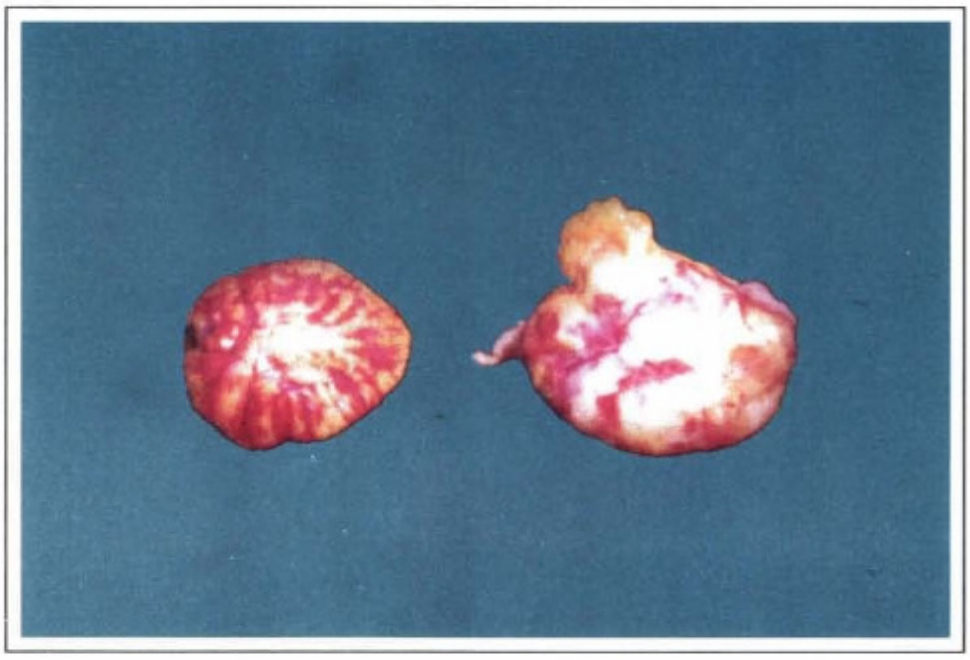

Fig. 48. **Two infected yolk sacs** from **two different 10-day-old broiler chickens.**

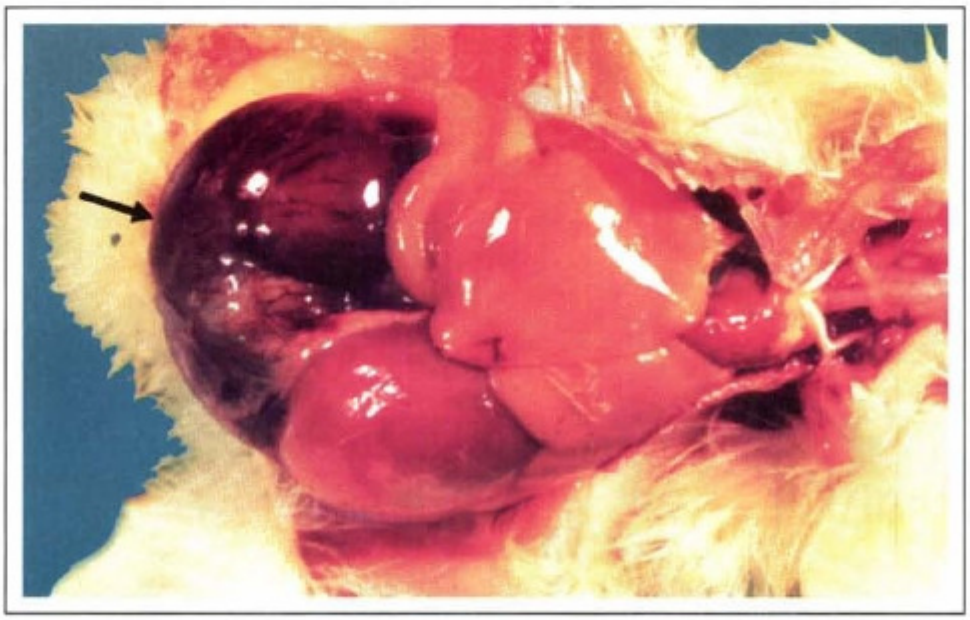

Fig. 49. **Yolk sac infection** in a **4-day-old chick**. Note yolk sac is distended, hyperaemic (showing prominent vessels), and filled with abnormal brown watery contents (arrow).

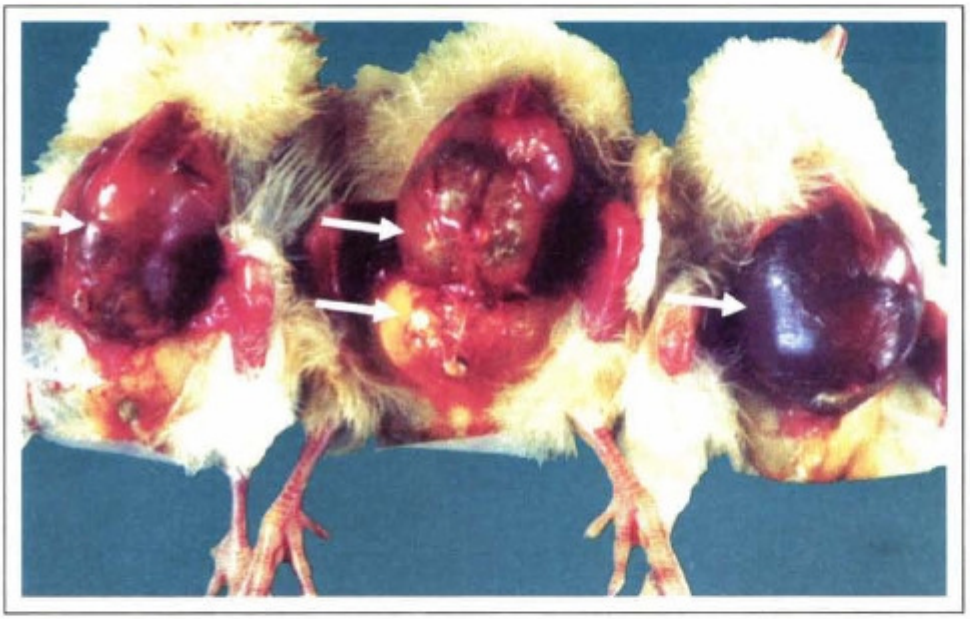

Fig. 50. **Omphalitis** (inflammation of navel) **and yolk sac infection** in **three 3-day-old chicks**. Navels are inflamed and yolk sacs distended with abnormal contents (arrows).

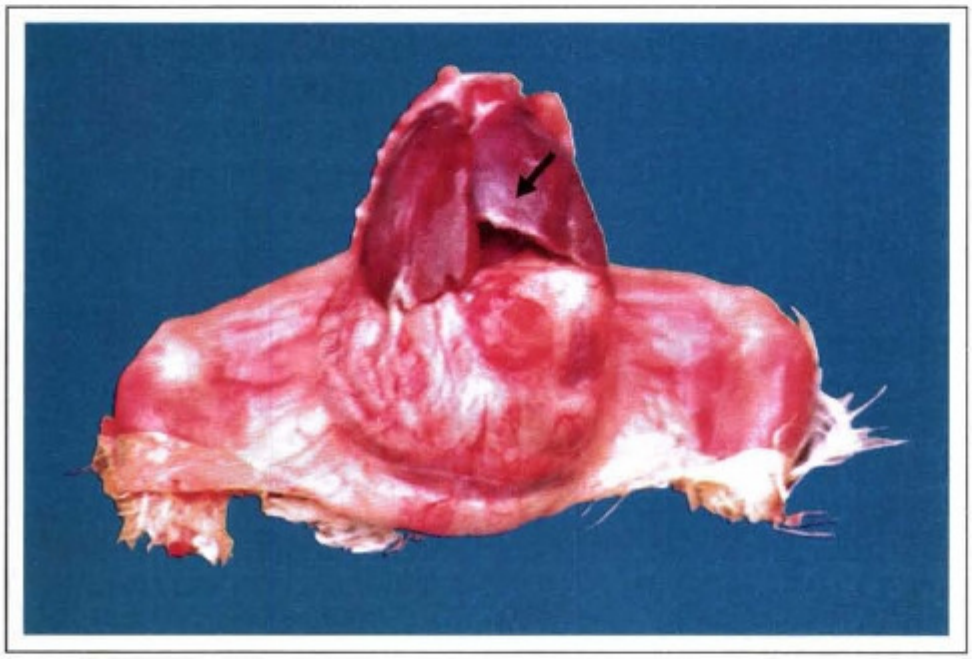

Fig. 51. ***E. coli* infection (Colisepticaemia)** in a **7-day-old broiler chick**. Note a thin layer of white fibrin covers the liver (arrow). **This is characteristic of *E. coli* infection.**

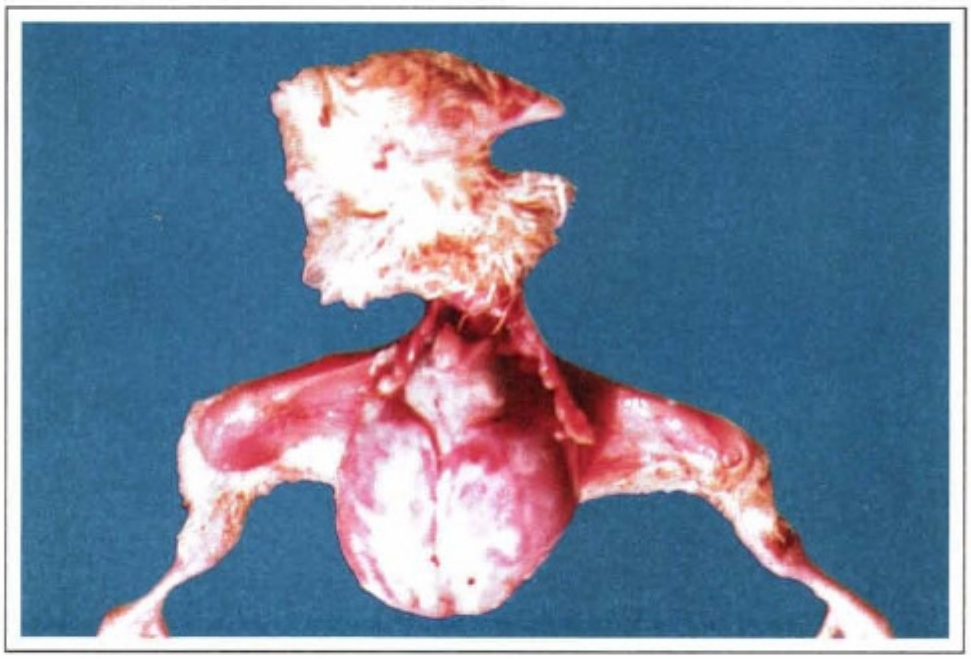

Fig. 52. ***E. coli* infection (Colisepticaemia)** in another **7-day-old broiler chick**. Note that the layer of fibrin in this chick is thicker and covers both heart and liver, **typical of *E. coli* infection**.

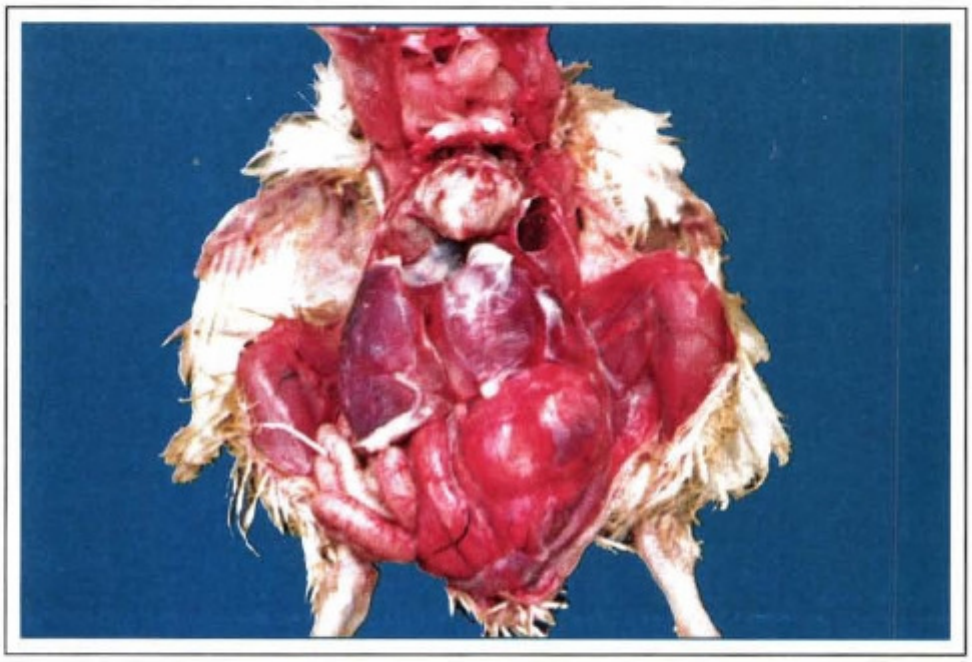

Fig. 53. ***E. coli* infection (Colisepticaemia)** in a **30-day-old broiler chicken**. A thick layer of white fibrin covers heart and liver. **This is typical of *E. coli* infection.**

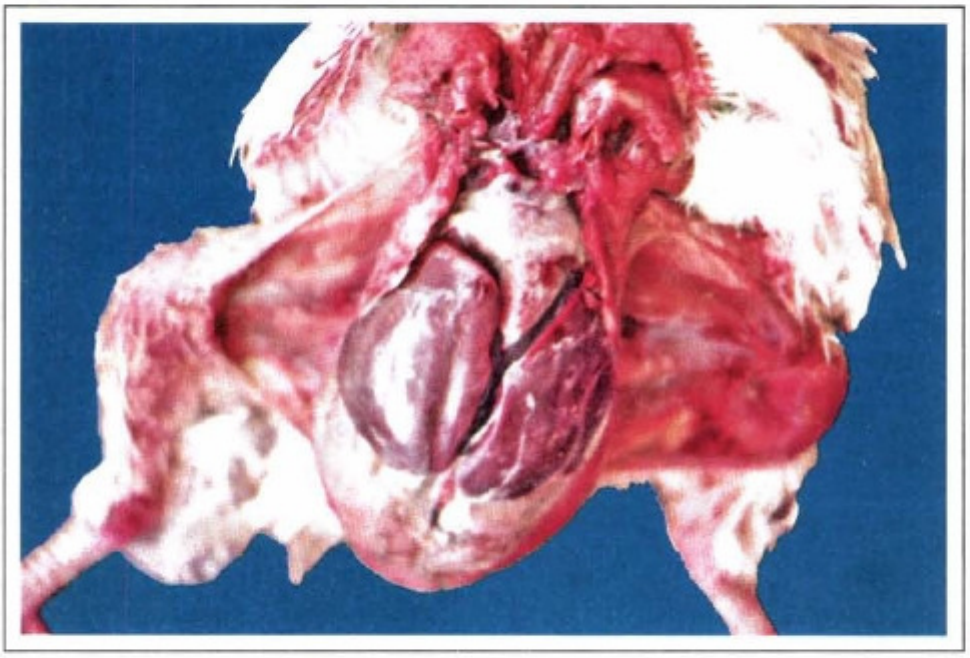

Fig. 54. ***E. coli* infection (Colisepticaemia)** in **another 30-day-old broiler chicken**. A thick layer of white fibrin covers heart and liver. **This is characteristic of *E. coli* infection.**

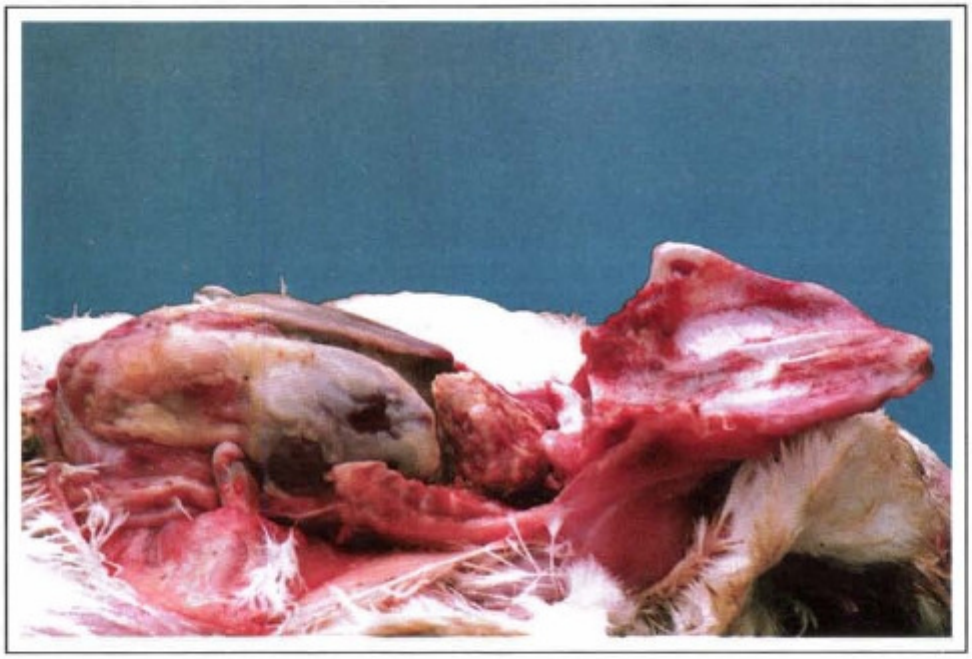

Fig. 55. ***E. coli* infection (Colisepticaemia)** in yet another **30-day-old broiler chicken**. The fibrin layer on the liver in this bird is so thick that it appears as a yellowish, opaque, adherent, caseous (cheese-like) deposition. **This is an advanced stage of *E. coli* infection.**

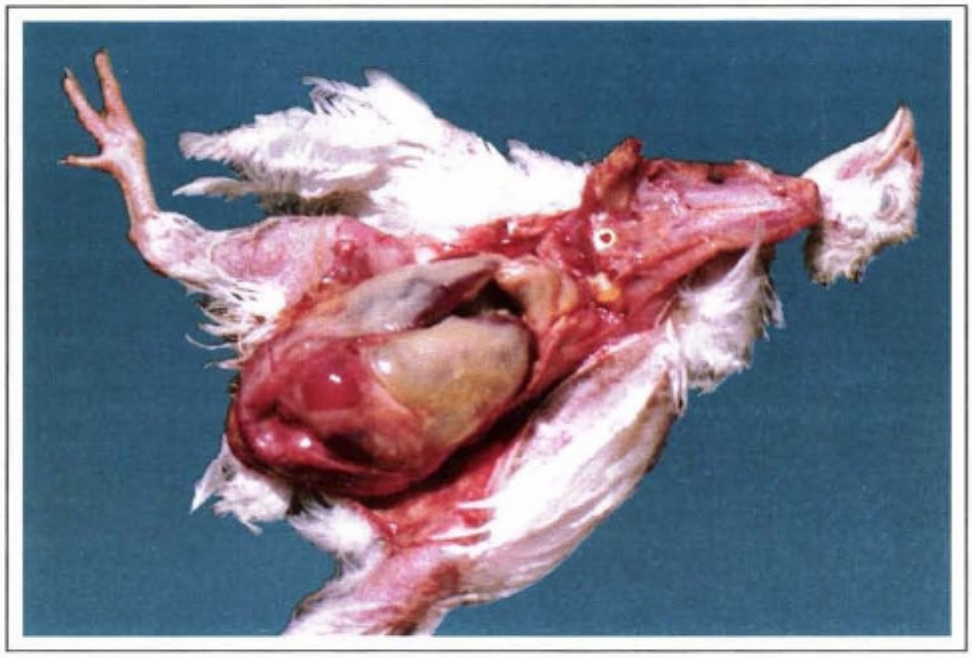

Fig. 56. ***E. coli* infection** (**Colisepticaemia**) in a **34-day-old broiler chicken**. Note thick, yellowish, adherent, caseous fibrinous material on the liver. This is characteristic of the advanced stage of ***E. coli*** infection.

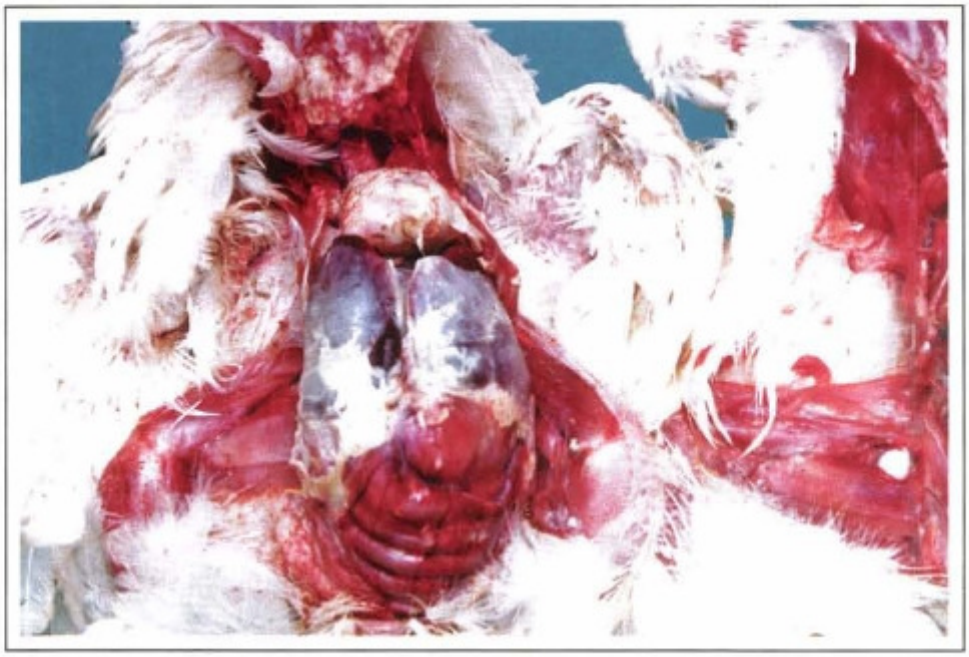

Fig. 57. ***E. coli* infection (Colisepticaemia)** in another **34-day-old broiler chicken**. Note thick white fibrin layer covering the heart and liver, typical of advanced ***E. coli*** infection.

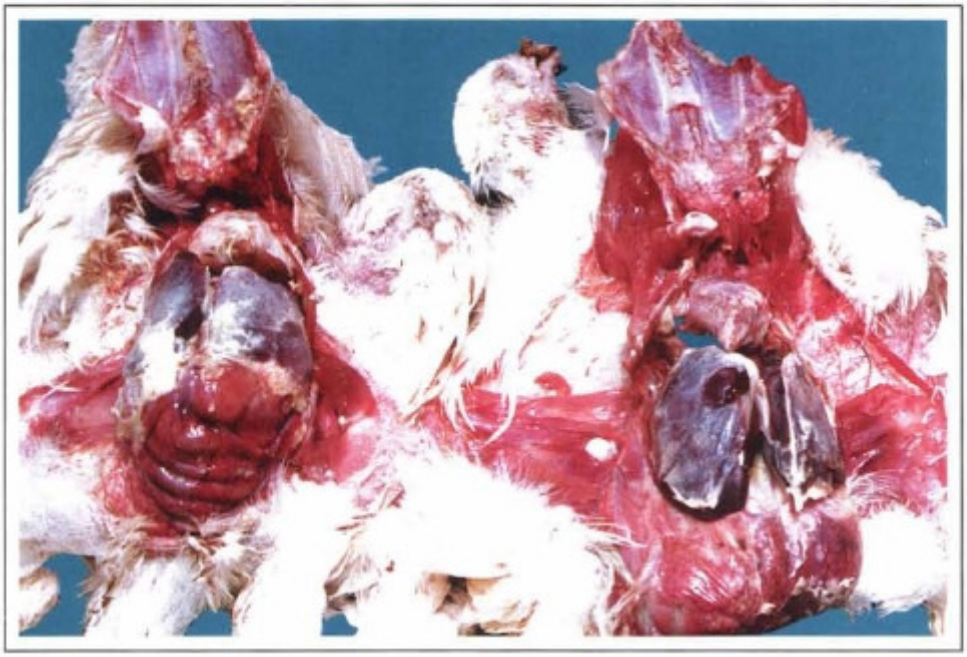

Fig. 58. ***E. coli* infection (Colisepticaemia)** in yet **two other 34-day-old broiler chickens** showing thick white fibrin layer on the liver and heart, characteristic of advanced ***E. coli*** infection.

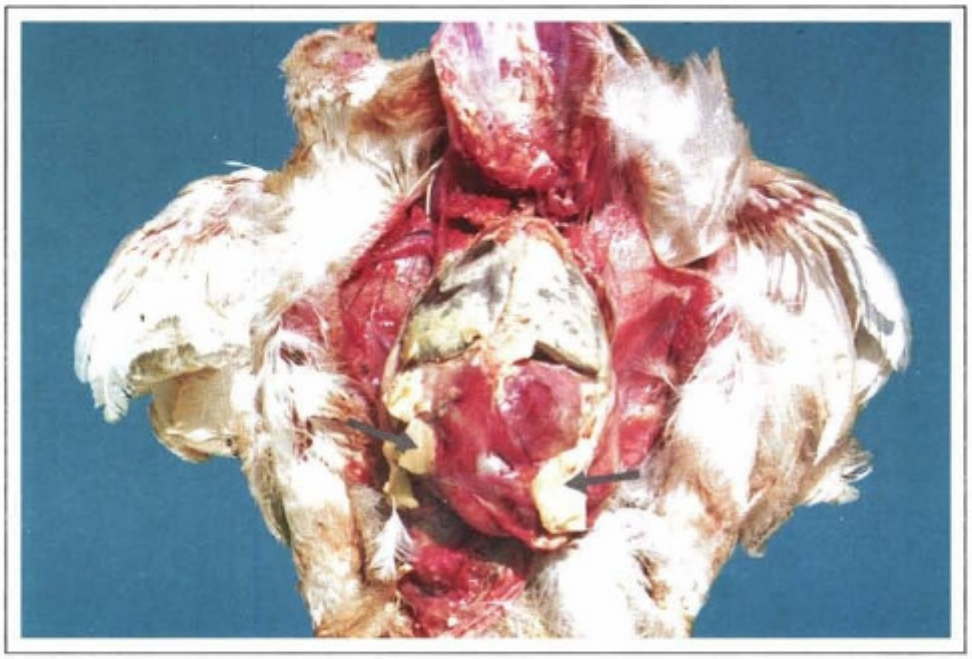

Fig. 59. ***E. coli* infection** (**Colisepticaemia**) in a **35-day-old broiler chicken**. Note fibrin deposition is now so massive that it is distinctly yellow and cheesy. At the lower end, fibrin deposits are seen as small lumps (arrows). **This is the most advanced stage of *E. coli* infection.**

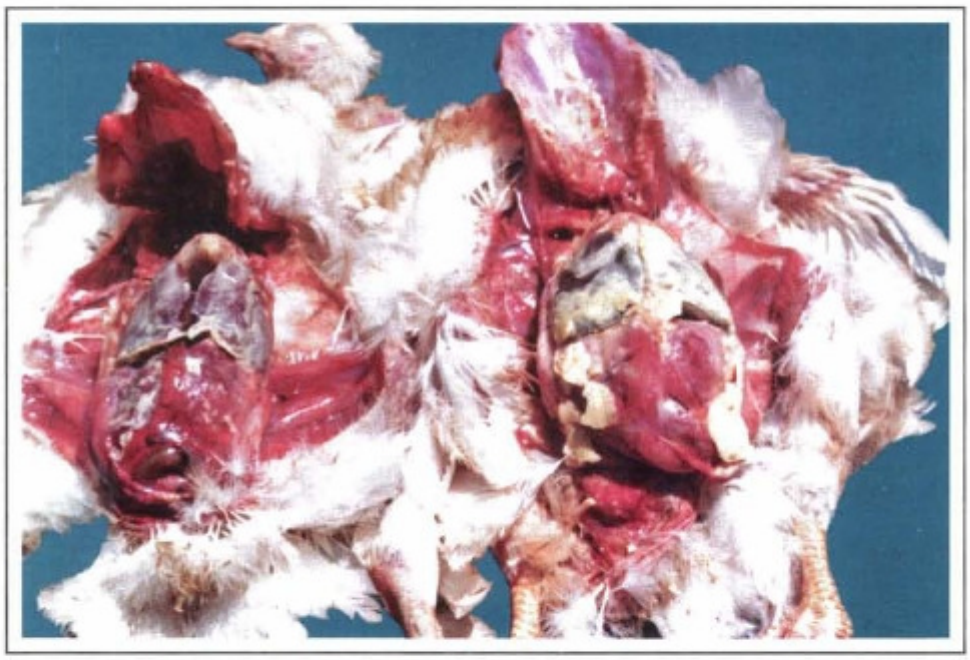

Fig. 60. ***E. coli* infection** (**Colisepticaemia**) in **two 35-day-old broiler chickens.** Note that fibrin deposition on the liver and heart in the right bird is more pronounced compared to the left. Therefore, in the right bird fibrinous material is yellow whereas in the left it is whitish.

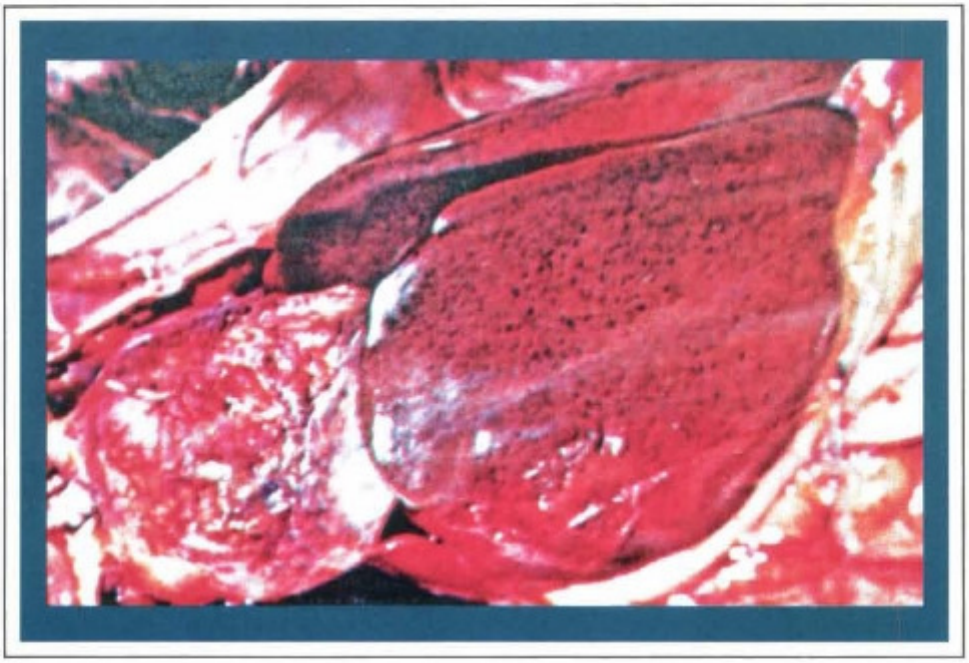

Fig. 61. ***E. coli* infection** (**Colisepticaemia**) in a **chicken**. Liver is enlarged, congested, and contains areas of dead tissue. Note its characteristic **corn meal appearance**. The liver appears as if sprinkled by ground maize. This appearance is rare and is seen only in certain severe cases.

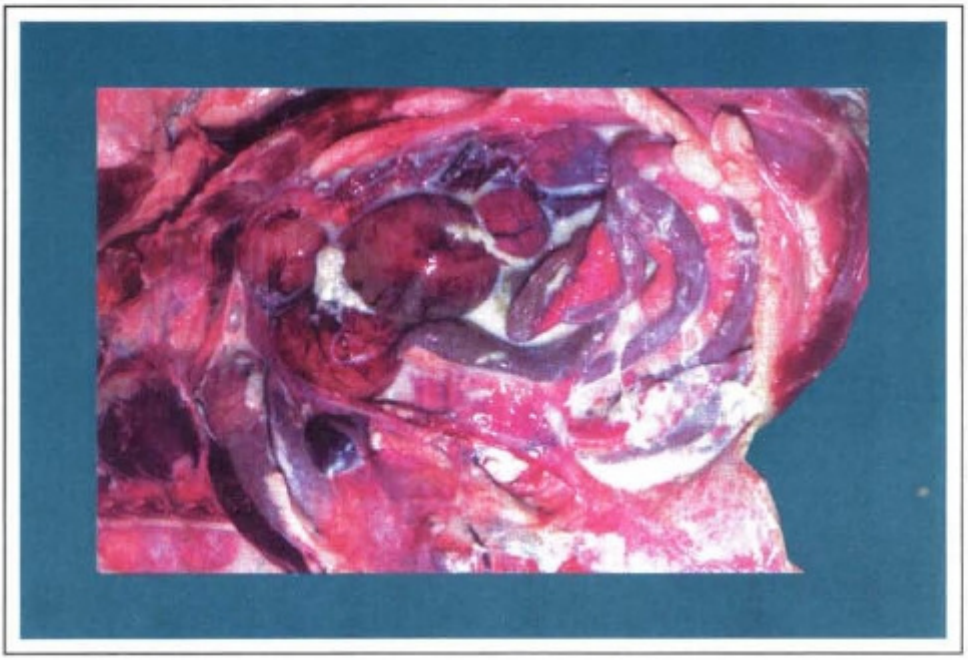

Fig. 62. **Egg peritonitis** in a **hen**. Note the presence of whitish-pale yolk in the abdominal cavity. Almost always egg peritonitis is caused by ***E. coli*** infection.

NECROTIC ENTERITIS

Necrotic enteritis is a bacterial disease of the intestinal tract of the chicken. **It is an important disease**, and is usually seen **in birds of 4 weeks of age or older**. **It is quite a common disease**. There are sudden deaths that are caused by severe necrosis (death) of the inner intestinal lining. Mortality in untreated flocks can reach 10% or more, **and is most common in broilers**. The disease is often established after an outbreak of coccidiosis. The author has come across **severe necrotic enteritis in the caeca of young broiler chicks** in cases of caecal coccidiosis, caused by ***Eimeria tenella*** (see 'caecal coccidiosis').

Cause

Necrotic enteritis is caused by the growth of a bacterium called ***Clostridium perfringens*** (type A and C) in the caeca and large intestine. Afterwards, this organism migrates to the small intestine where it produces toxins. These toxins cause death of cells lining the intestine and are responsible for the characteristic changes of necrotic enteritis. **Contaminated feed and litter act as sources of infection.**

Predisposing Factors

These are those factors which make the bird easily prone to infection. Necrotic enteritis is produced when there is damage to the inner intestinal lining. This allows the bacteria to grow and produce toxins which damage the intestine and cause death. **The predisposing factors of necrotic enteritis include:**

1. **Outbreaks of coccidiosis, especially mild and subclinical.**
2. Partial impaction of the lower intestine due to litter and/or grain ingested without the provision of insoluble grit.
3. Rapid changes in feed components.
4. Removal of the antibiotic growth promoters from the feed results in a significant increase in the incidence of necrotic enteritis. This is because of their direct effect on control of the causative organism ***Clostridium perfringens.***

However, the **most important predisposing cause** of necrotic enteritis, both in broilers and layers, appears to be **coccidiosis**. Therefore, every outbreak of necrotic enteritis must be examined for coccidiosis, and if present, must be treated. Likewise, in every outbreak of coccidiosis, necrotic enteritis must be ruled out, and if present, must be treated.

Symptoms

1. There may be no symptoms. Clinical disease is very short, and often, birds are just found dead.
2. The birds may show depression (Fig. 63), loss of appetite, unwillingness to move, diarrhoea, ruffled feathers, and increasing mortality.

Postmortem Findings

1. Postmortem findings are usually confined to the **middle part of the small intestine**. The intestine is distended and filled with a foul-smelling brown fluid. The inner intestinal lining is covered by a brownish membrane (Fig. 64, 65).
2. Similar changes in the caeca may also occur (66, 67, 68, 69).
3. The small intestine is greatly thickened due to extensive velvet-like (looking like silk) necrosis (death) of the intestinal lining (mucosal lining) (Fig. 64, 65).

4. The intestinal lining (mucosa) is covered by a loose to tight yellow or green layer (Fig. 64, 65) that may be deeply cracked. Spots or small patches of blood may occur, but haemorrhage is not a feature.

Diagnosis

Diagnosis can be made from the characteristic postmortem findings.

Treatment

A number of antibiotics (amoxycillin, ampicillin, chloramphenicol, ciprofloxacin, doxycycline, neomycin, erythromycin, furazolidone) given in drinking water may be helpful in treating the condition. Others (lincomycin, tylosin, bacitracin, and furazolidone) in feed reduce the number of organisms shed in the faeces.

Control

1. Identify the predisposing factor and make sure that it is controlled. Give particular attention to coccidiosis and Gumboro disease.
2. Drugs which have an effect on the causative organism such as lincomycin, ampicillin, penicillin, virginiamycin, bacitracin, furazolidone and others may be included in the feed.
3. Probiotics reduce the severity of necrotic enteritis.
4. Anticoccidial drugs, e.g., the ionophores, in the feed are also helpful.
5. Good management and sanitation practices should be followed.

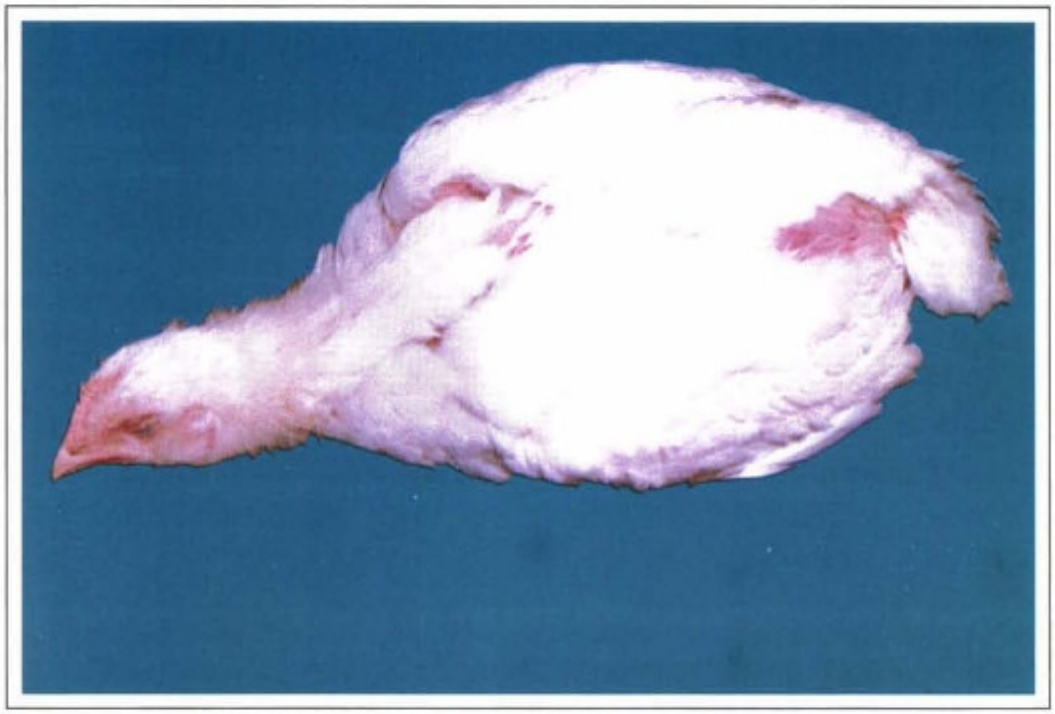

Fig. 63. **Necrotic enteritis**. A **30-day-old broiler chicken** suffering from necrotic enteritis. The bird is depressed, eyes are closed, neck is extended, and head drooping.

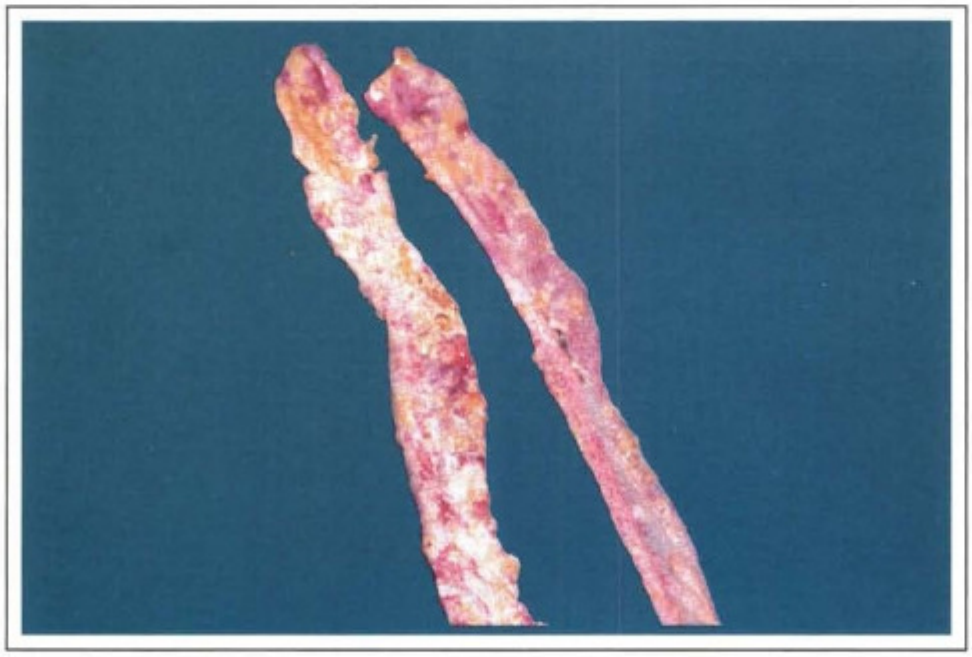

Fig. 64. **Necrotic enteritis** in a **50-day-old broiler chicken**. The picture presents **middle portion of the small intestine where the changes are most marked**. The intestines are cut open. Note that their walls are greatly thickened and the inner surface is lined by a loose to tightly attached yellow layer (a false layer). Areas of small haemorrhages are present. There was also concurrent mild coccidiosis.

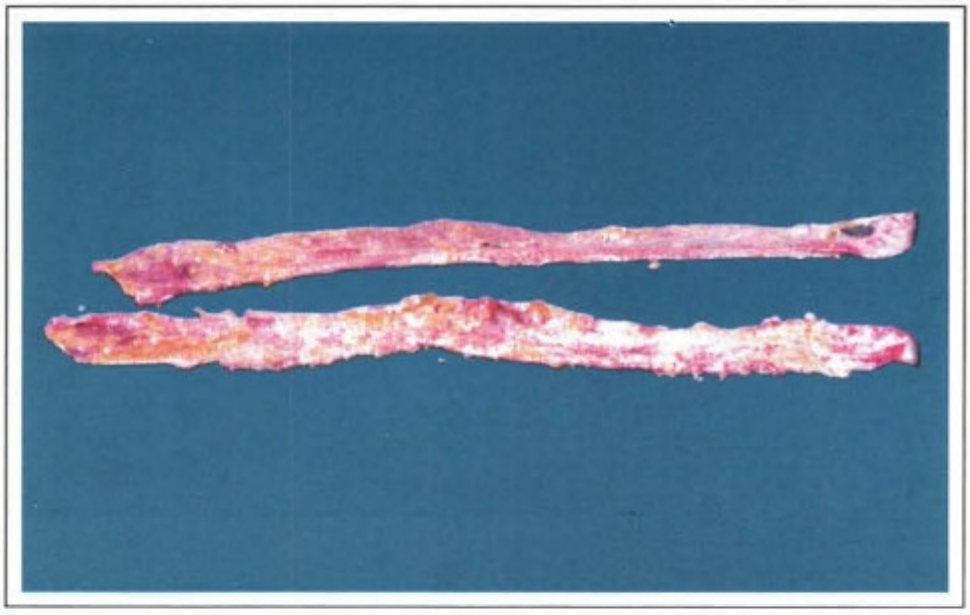

Fig. 65. **Necrotic enteritis** in the small intestine of the same **50-day-old broiler chicken** shown in Fig. 64. Intestines in this photograph are presented horizontally.

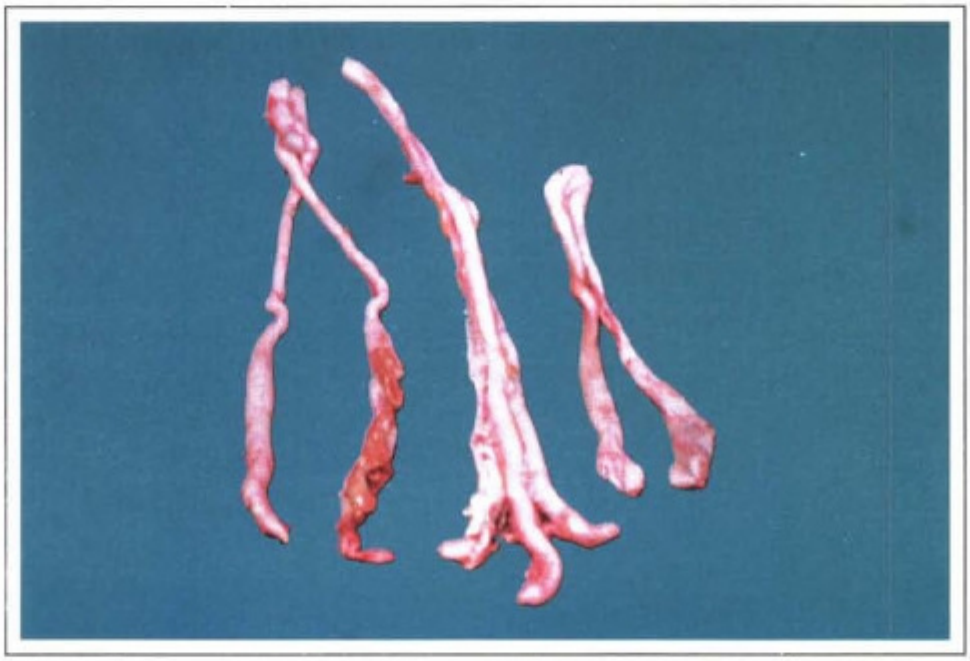

Fig. 66. **Necrotic enteritis** in the **caeca** of **three 18-day-old broiler chickens**. Most of the caeca are unopened. The next figure (No. 67) reveals their internal changes.

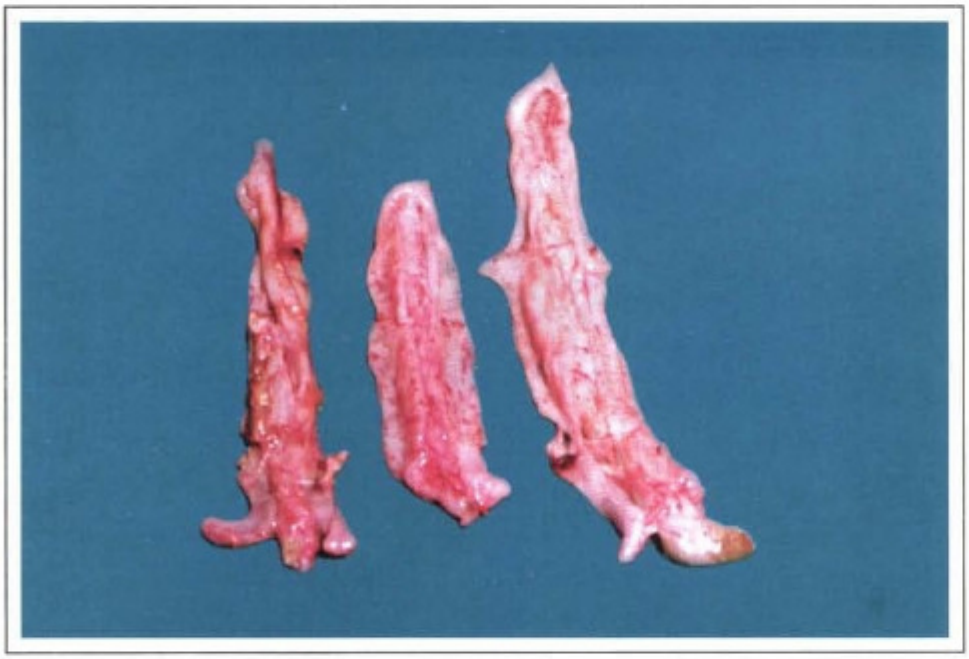

Fig. 67. **Necrotic enteritis** in the **caeca** of **three 18-day-old broiler chickens** shown in Fig. 66. The caeca are cut open. Their closer view reveals that they are greatly thickened and their internal surfaces are lined by a loose to tightly attached yellow layer (a false membrane). Areas of small haemorrhages and patches of blood are usually present.

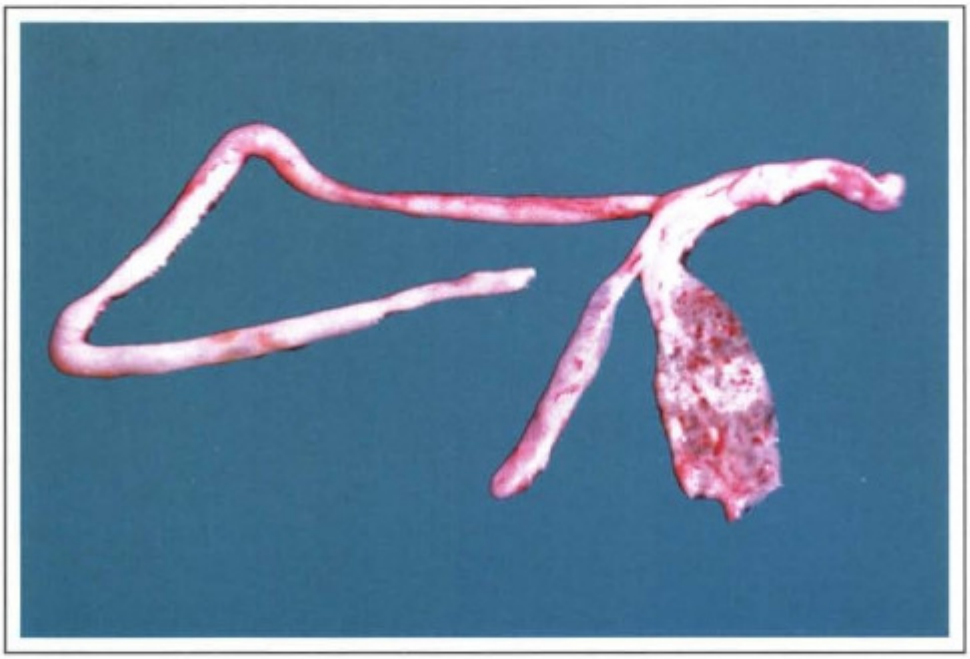

Fig. 68. **Necrotic enteritis** in the **caeca** of a **30-day-old broiler chicken**. The right caecum is cut open. It shows necrotic lesions and minute haemorrhages. The caecum also had mild coccidiosis.

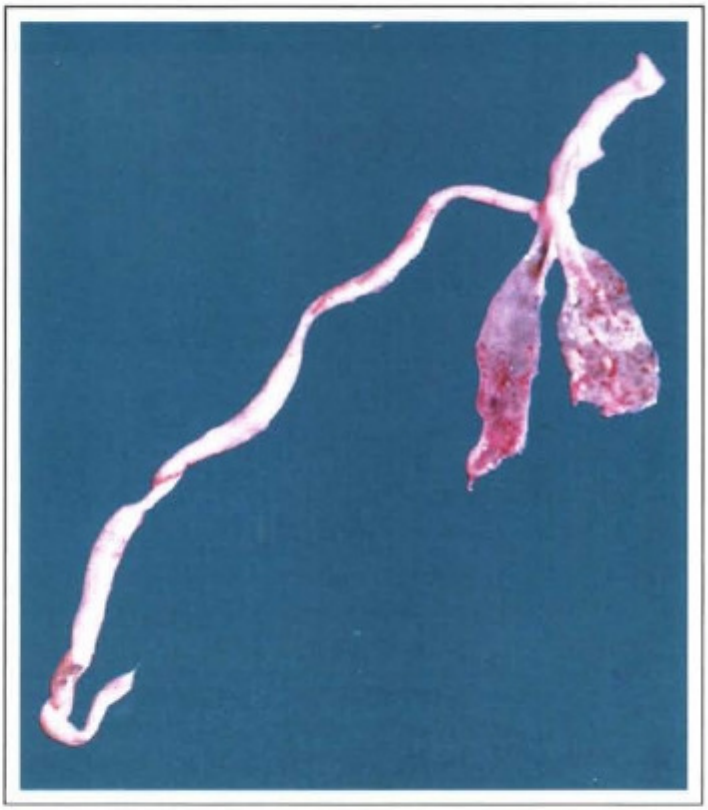

Fig. 69. **Necrotic enteritis** in the **caeca** of the same **30-day-old broiler chicken** shown in Fig. 68. Both the caeca are cut open. They reveal necrotic lesions and minute haemorrhages.

GANGRENOUS DERMATITIS

Gangrenous dermatitis is characterized by areas of death and putrefaction in the skin and underlying tissues, including muscle. **It is quite a common disease**. Also known as **"wing rot", gangrenous dermatitis is a serious disease and usually results in death**. It is mainly a disease of the **broiler** over 4 weeks of age, but is also quite common **in grower between16 and 19 weeks of age**. Its occurrence is more common in warm, humid conditions. Mortality ranges from 1 to 60%.

Causes

1. Bacteria known as clostridia, staphylococci, and ***E. coli***, either singly or in combination, are involved. **Combined infections are more severe.**
2. Gangrenous dermatitis is more likely to occur in the presence of those diseases which produce immunosuppression, such as Gumboro disease. Immunosuppression is suppression of the natural immune responses that produce immunity.
3. Other predisposing factors are unhygienic management (poor sanitation), overcrowding, mycotoxins, infectious anaemia virus, and reovirus.

Development of the Disease

1. Most cases in broilers occur between 4 and 6 weeks and in layers between 6 and 20 weeks.
2. **Clostridial organisms** are present in soil, faeces, dust, contaminated litter or feed, and in the intestinal contents (mainly caeca) of healthy birds. **Staphylococci** exist everywhere and are **common inhabitant of skin** and linings of the digestive, respiratory, and urinary tracts.
3. In many cases, gangrenous dermatitis follows diseases caused by other infectious agents, such as Gumboro virus, chicken infectious anaemia virus, reticuloendotheliosis virus, and avian adenovirus infections, including inclusion body hepatitis.
4. **Gangrenous dermatitis usually occurs secondarily following skin injury**. This is because all the infectious agents mentioned above produce **immunosuppression**, and a damaged immune system is the underlying predisposing factor that allows gangrenous dermatitis to develop.

Symptoms

1. There is increased mortality in the flock. Affected birds are depressed, show loss of appetite, leg weakness, incoordination of movements, and **die within a few hours.**
2. The period of illness is very short, **usually less than 24 hours**. Mortality ranges from 1 to 60%.

Postmortem Findings

1. The skin and underlying tissues under the wings, between the thighs, and over the ribs and flanks (i.e., area between ribs and hips) of the birds are **very dark, moist, usually devoid of feathers**, foul-smelling, and **show putrefaction** (Fig. 70, 71, 72, 73).
2. Deep red and swollen areas are found on the feet, legs, and sometimes around the feather follicles of the wings (Fig. 73). There is a blood-tinged watery fluid under the skin.
3. The surrounding muscle is usually coloured and contains fluid (oedematous). If clostridial organisms are involved, **gas** is produced in the muscle.

Diagnosis

The postmortem picture is diagnostic.

Treatment

1. No treatment has proved completely successful. This is because of the nature of tissue changes. **Dead tissue cannot be treated.** Moreover, many kinds of causes are involved in the production of disease.
2. However, penicillins, tetracycline, chlortetracycline, oxytetracycline, erythromycin, or bacitracin may be given in the drinking water.
3. Gentamicin injections have proved helpful in controlling the disease. But this antibiotic could be very damaging to the kidneys.
4. Follow up should be done by the addition of antibiotics in the feed.
5. Water acidification, with citric and propionic acid, has been used to reduce but not eliminate mortality in flocks.
6. Control of symptoms and lesions has also been achieved by using water acidifiers and acidification of litter.

Control

1. Avoid overcrowding to prevent skin damage.
2. Maintain good standards of hygiene in management. Improve litter condition, reduce moisture and bacterial levels in the environment, and minimize trauma (injury) to the bird.
3. Protect the birds against immunosuppressive diseases, like Gumboro disease and others, by carrying out proper and timely vaccinations.
4. Remove dead birds daily as quickly and as often as possible.
5. House sanitation should be carefully controlled. Feed, water, and litter management are very important.
6. Good nutrition and management of stress are also helpful.

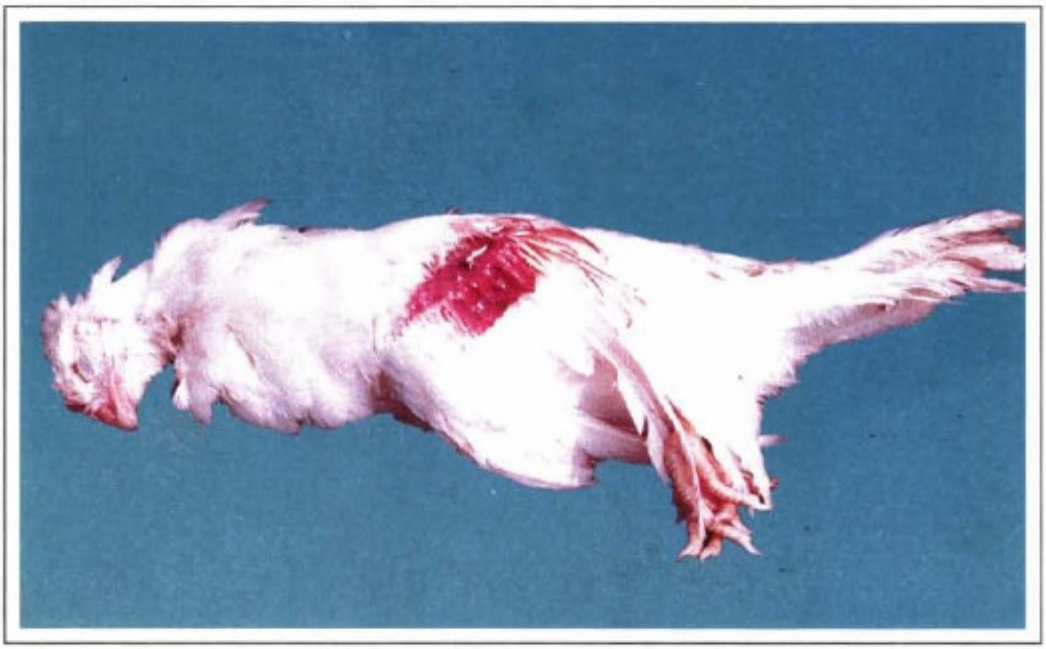

Fig. 70. **Gangrenous dermatitis** in a **9-week-old grower chicken**. Note skin and tissues under the wings and over the ribs are deep red, moist, and devoid of feathers.

Fig. 71. **Gangrenous dermatitis** in **two 10-week-old grower chickens.** Note the affected area in the left bird, besides being red, is moist and swollen.

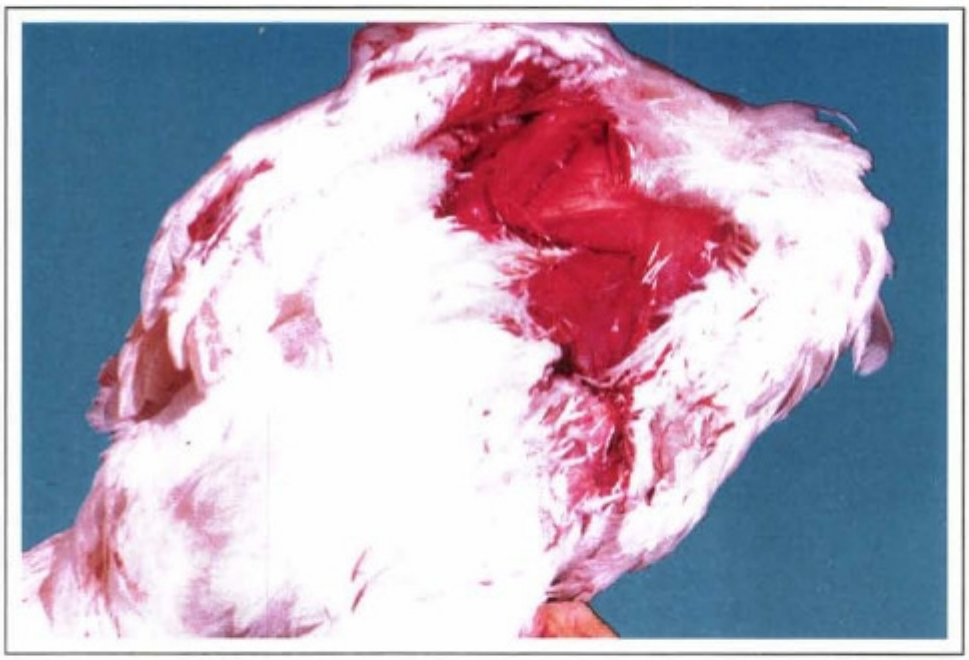

Fig. 72. A more advanced case of **gangrenous dermatitis** in another **10-week-old grower chicken.** Note the affected area is now intense red, moist, swollen, and devoid of feathers. The skin beneath contains blood-tinged watery fluid.

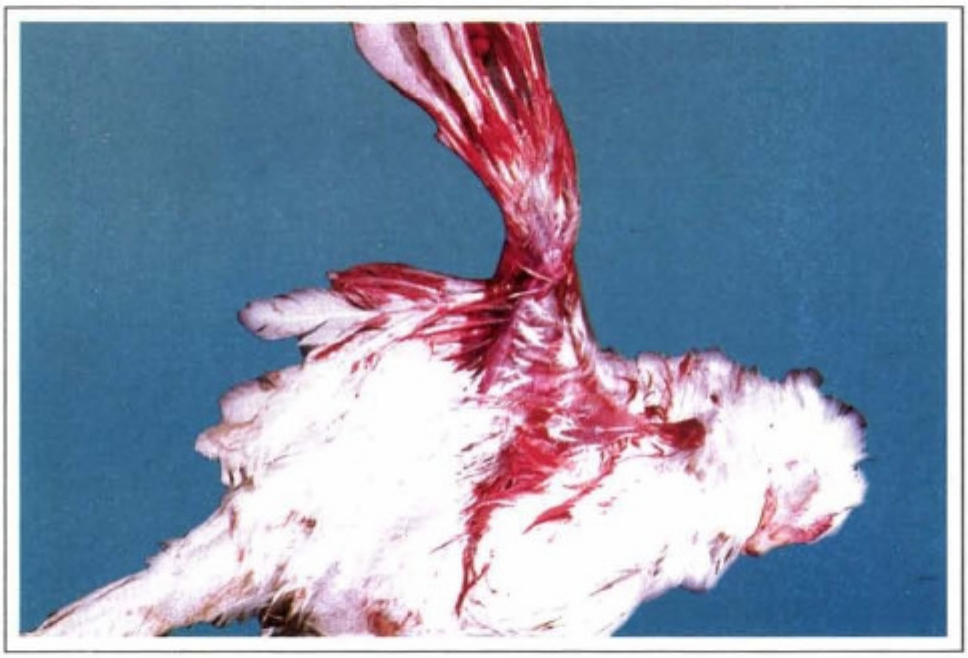

Fig. 73. **Gangrenous dermatitis** under the wings in another **10-week-old grower chicken. This site is commonly involved.** The changes become more pronounced with progression.

INFECTIOUS CORYZA

Also known as **'fowl coryza'**, infectious coryza is a sudden and severe respiratory disease of chickens. The disease is characterized by **swelling of the face and discharges from the eyes and nostrils** (Fig. 74, 75). The greatest economic losses occur from poor growth in growing birds, and a marked drop in egg production (10-40%) in layers.

The disease is extremely common both in broilers and layers. Chickens of all ages are susceptible, but older birds suffer more severely.

Cause

A bacterium called ***Haemophilus paragallinarum***. It is a delicate bacterium and dies quickly outside the bird. Survival outside the body under farm conditions is not more than 48 hours.

Spread

1. The infection can be spread by **drinking water** contaminated with discharges from the nose.
2. Infection can also be spread over a short distance **by the air.**

3. Lateral spread occurs readily by **direct contact**.
4. Spread between cages with nipple drinkers occurs more slowly.

Symptoms

1. The first symptoms include sneezing, mucus-like discharge from the openings of the nose and eyes, and swelling on the face (Fig. 74, 75). In severe cases, marked conjunctivitis (inflammation of conjunctiva) with closed eyes, swollen wattles, and difficulty in breathing can be seen.
2. Feed and water consumption is usually decreased. This results in a drop in egg production, or an increase in the culling (removal) of inferior, weaker, and worthless birds.

Postmortem Findings

1. Swelling of the face and wattle due to accumulation of fluid under the skin is marked (Fig. 75).
2. There is inflammation of the nasal passages and the space present below the eye.
3. The upper trachea may be involved but the lungs and airsacs are affected only in chronic, complicated cases.

Diagnosis

1. Marked swelling on the face and discharges from the eyes and nostrils are quite diagnostic of the disease.
2. Postmortem findings are helpful in making the diagnosis.

Treatment

Various sulpha drugs and antibiotics are useful in treating the disease. The combination of sulpha drug trimethoprim, tetracyclines, and enrofloxacin may be used. Erythromycin and oxytetracycline are also commonly used antibiotics.

Control

1. Improved management practices, such as culling of birds, good sanitation, traffic control, and avoiding birds of multiple ages help in breaking the disease cycle. Keep only birds of the same age on the farm.
2. Because of the difficulty of controlling infectious coryza by sanitary measures, drug therapy and/or vaccination are used. Vaccination with a killed virus can protect chickens against the disease.
3. To eliminate the agent from the farm, it is necessary to dispose of the infected or recovered birds because such birds remain as a reservoir of the bacterial agent. After cleaning and disinfection, the farm should be allowed to remain vacant for 2-3 weeks before restocking with clean birds.

Fig. 74. **Sudden onset of discharge from the eye of a chicken**. This may be of **infectious coryza** or infectious bronchitis.

Fig. 75. **Infectious coryza**. Note typical **swelling on the face of a chicken** (facial oedema).

PULLORUM DISEASE

Pullorum disease is a highly contagious, egg-transmitted disease. **It is mainly a blood infection of chickens.** The disease is characterized by **white diarrhoea in young chicks.**

Cause

A bacterium called ***Salmonella pullorum***. Mortality from pullorum disease is usually confined to the **first 2-3 weeks of age.**

Spread

1. The most important method of spread is **vertical transmission**. That is, from an infected hen through the eggs to the newly hatched chicks.
2. Such newly hatched chicks spread the infection to other chicks in hatcheries through their soft feathers which are heavily contaminated by ***S. pullorum***.
3. Affected chicks shed ***S. pullorum*** through the faeces. This is the major method of spread among chicks.
4. Contaminated feed, water and litter can also be a source of ***S. pullorum*** infection.
5. Attendants, visitors, feed dealers, and chicken buyers who move from house to house and from farm to farm, may carry infection.

Symptoms

1. Pullorum disease is **mainly a disease of chicks**. It is seen in chicks below 3 weeks of age. The first indication is usually a large number of dead-in-shell chicks, and deaths soon after hatching.
2. **Affected chicks** show depression with a tendency to crowd together in a small space, respiratory distress, loss of appetite and white thick and sticky droppings that adhere to the feathers around the vent. That is, **white diarrhoea**. The chicks breathe with difficulty and death may soon follow. The peak mortality usually occurs during the 2nd and 3rd week. Mortality varies greatly and in extreme cases can be 100%.
3. **Older birds** may appear depressed and have pale shrunken combs with ruffled feathers. Low egg production may be the only symptom of the disease when adult birds are affected. The condition, however, is rare in adult birds.

Postmortem Findings

1. **Chicks** may have an inflamed, unabsorbed yolk sac. The lungs may be congested and the liver dark and swollen with haemorrhages visible on surface.
2. **Chicks** that die after showing signs for 1 or 2 days show inflammation of the caeca. The caeca are enlarged and distended with hard, dry, necrotic (dead) material. Distinct, small, white, necrotic foci (area of dead tissue) are also usually found in the **liver** (Fig. 76), lungs, heart, and gizzard wall.
3. **In adult birds**, the characteristic lesion is an abnormal ovary. The ova are irregular, cystic, deformed and pedunculated (attached) with prominent thickened stalks (Fig. 77). In some infected adult hens the ovary is inactive, the ova being small, pale and undeveloped.

Diagnosis

1. The symptoms and postmortem findings may vary and are not quite characteristic to make a firm diagnosis. The disease is diagnosed in the chick by isolating ***S. pullorum*** from liver or lungs.
2. Infection in older birds can be detected by demonstrating ***S. pullorum*** antibodies in blood samples by agglutination test.

Treatment

A number of antibiotics reduce the mortality.

Control

1. Test repeatedly the blood of breeding flocks (parents) and remove reactors. This must be combined with high management standards and hatchery discipline.
2. Replacement birds must be purchased only from flocks known to be free of the disease.
3. Eggs from pullorum-free flocks should be incubated and hatched only in hatcheries receiving eggs exclusively from clean flocks.

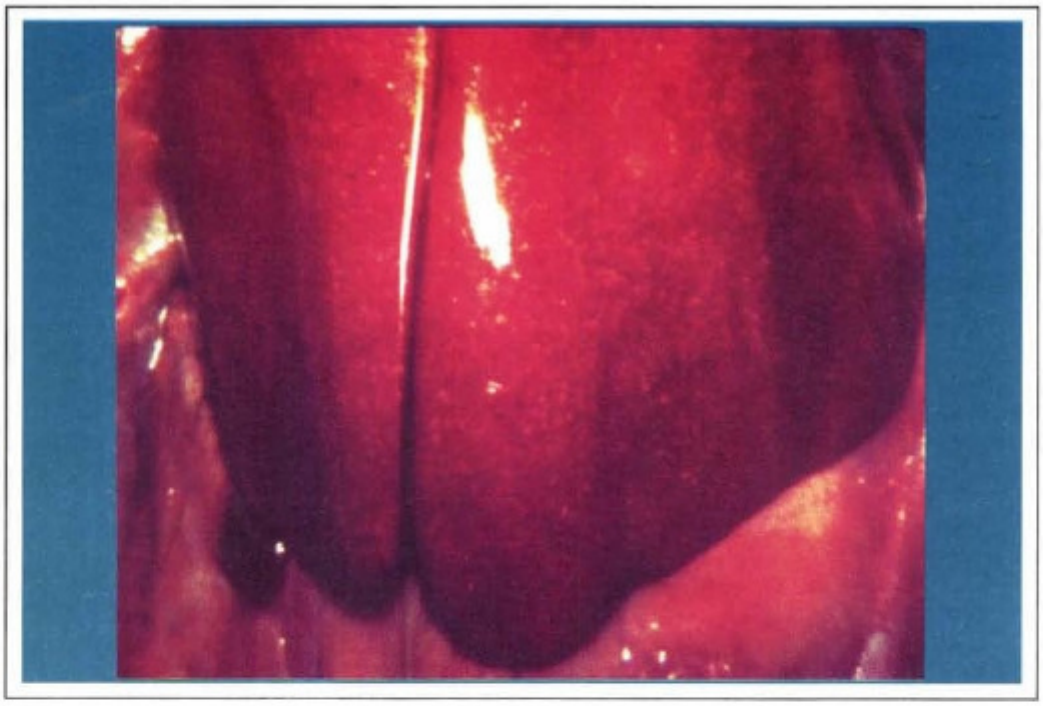

Fig. 76. **Pullorum disease** in a **chicken**. Note **liver** is enlarged, congested, and shows **small necrotic foci** (minute white spots). Such necrotic foci are highly suggestive of pullorum disease.

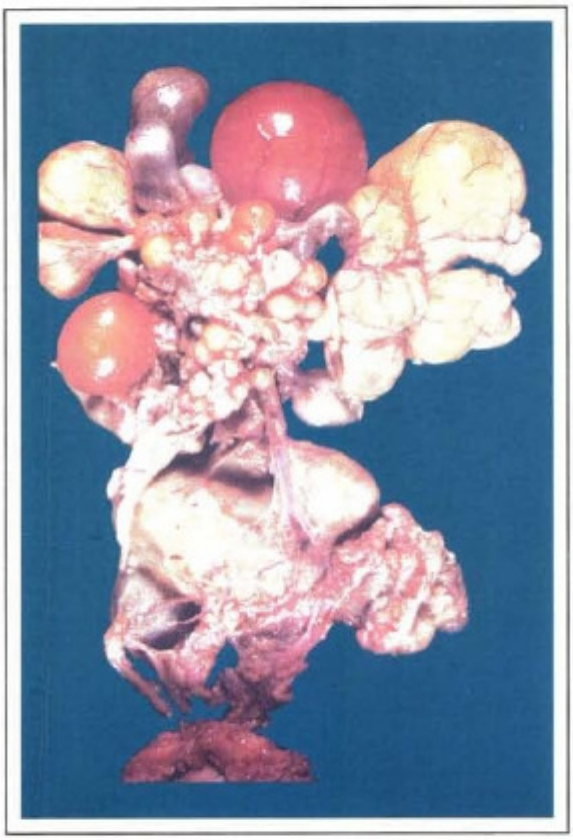

Fig. 77. **Pullorum disease** in a mature **hen** showing **lesions** (changes) **in the ovary**. Note ova are irregular, cystic, deformed and pedunculated with prominent thickened stalks.

FOWL CHOLERA

Fowl cholera is a septicaemic disease (blood infection) of chickens. In its severest form, fowl cholera is one of the most harmful and highly contagious diseases, inflicting heavy mortality, but less severe (chronic) and harmless conditions also occur.

Losses from fowl cholera usually occur in **laying flocks**. Chickens less than 16 weeks of age are usually quite resistant. Mortality ranges from 0 to 2%, but greater losses have been reported. Reduced egg production and **persistent localized infection** usually occur.

Cause

A bacterium called ***Pasteurella multocida***. Strains of ***P. multocida*** vary in their disease-producing power (virulence). Some are most harmful, others moderately so, and a certain number harmless.

Spread

1. Spread of ***P. multocida*** within a flock is mainly by excretions from mouth, nose, and conjunctiva of diseased birds. These excretions contaminate their environment, particularly feed and water. Thus, spread is **through contaminated feed and water**. Birds are infected through mouth, nose and eye, and through wounds.
2. Spread can be also by people, clothing, or their footwear.

3. Spread through the air does occur between pens. However, **spread through water and feed is more common.**
4. The disease is not egg-transmitted.

Symptoms

1. **The disease occurs in several forms**. In the **severest form**, there are no preceding symptoms and a large number of birds in a flock are found dead, in good bodily condition. 50% or more may die. Birds between12 and 18 weeks of age are most susceptible.
2. In the **less severe form**, marked depression, loss of appetite, mucus discharges from the openings, ruffled feathers, bluish discoloration of comb and wattles, and foul-smelling greenish coloured diarrhoea may be seen.
3. The **chronic form** is seen in birds which survive the severe disease. Symptoms are usually due to **localized infection**. Symptoms include depression, difficult breathing, and later lameness, twisting of the neck to one side, and **swelling of the wattles** (see Fig. 79). One or both the wattles may be swollen containing cheesy, hard deposit. Chronically infected birds may die, remain infected for long periods, or recover.

Postmortem Findings

1. Postmortem findings in the severe form include marked congestion of the carcass, pinpoint haemorrhages throughout the internal organs, and **multiple necrotic areas** (areas of dead tissue) **in the liver** (Fig. 78). The liver may be enlarged and also show very small haemorrhages on the surface.
2. In the laying hens free yolk may be present in the abdominal cavity.
3. In the less severe disease, oedema of the lungs (i.e., accumulation of fluid) and pneumonia (inflammation of lungs) are seen.
4. In **chronic cases**, changes include arthritis (inflammation) of the hock and foot joints, and **swelling of one or both wattles** (Fig. 79).

Diagnosis

The history of the disease, symptoms and postmortem findings are helpful, but all forms of the disease can be confused with other infections. Demonstration of ***P. multocida*** confirms the diagnosis.

Treatment

1. The severest form of fowl cholera is so rapid that treatment is rarely of value.
2. In the less severe form, a number of drugs have proved effective. They include sulphonamides and antibiotics. In antibiotics, penicillin, streptomycin, oxytetracycline, chlortetracycline, and erythromycin have been used successfully.

Control

1. Dispose of all birds and clean and disinfect the buildings thoroughly. Good management practices with emphasis on sanitation are the best means of preventing fowl cholera.
2. The main source of infection is the sick bird, or those that have recovered but still carry the organism. Only young birds should be introduced as new stock. They should be raised in a clean environment completely isolated from other birds.
3. Vaccination should be considered in areas where fowl cholera is prevalent, but it should not be substituted for good sanitary practice.

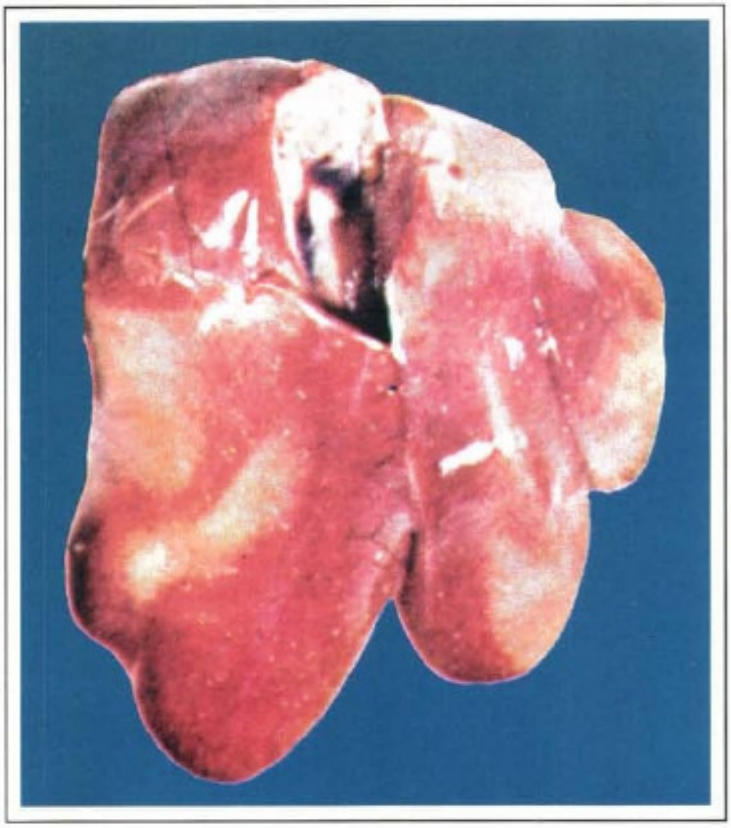

Fig. 78. **Fowl cholera** in a **chicken** (**acute case**). Note liver shows tiny necrotic areas (minute white spots) on the surface. Note also streaks (lines) of light areas.

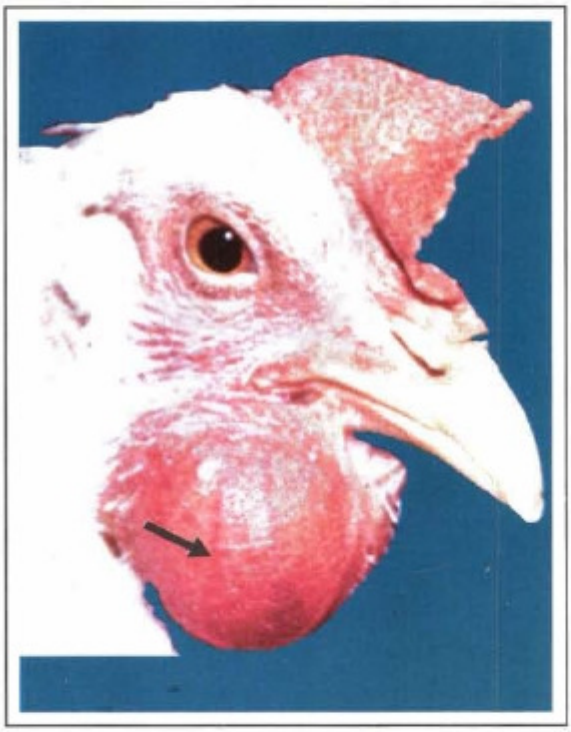

Fig. 79. **Fowl cholera** in a **chicken** (**chronic case**). Note **swollen wattle** (arrow) resulting from **localized infection**. **This is characteristic of chronic cases of fowl cholera.**

STAPHYLOCOCCOSIS

Staphylococcosis is caused by the bacterium ***Staphylococcus aureus. S. aureus*** infections are common in poultry. **A wide variety of conditions are associated with** the disease-producing strains of ***S. aureus***. Among these the important conditions are yolk sac infection, gangrenous dermatitis, bumble foot, and staphylococcal septicaemia (i.e., blood infection). ***S. aureus*** produces a number of toxins and enzymes which influence its disease-producing power.

Spread

1. **For infection to occur, injury to the skin or mucous membranes is essential**. ***S aureus*** enters through the broken barriers and travels to internal locations, where a focus of infection is established.
2. **In newly hatched chicks**, the open navel provides a route of entry leading to omphalitis (inflammation of navel) and other types of infection.

Disease

1. Yolk sac Infection

See **'yolk sac infection'** under **'colibacillosis'** (see also Figs. 46, 47, 48, 49, 50).

2. Gangrenous Dermatitis

This occurs in birds of all ages, but is most common in **broilers**, and in **growers between 15 to 20 weeks of age**. The wing tips and the dorsal pelvic region are the sites affected. The affected sites are dark, moist, and gangrenous (decaying) in appearance (see Fig. 70, 71, 72, 73) with a crackling sound. Staphylococci are usually associated with ***Clostridium perfringens***, which may be the main disease-producing organism.

3. Bumble Foot

Bumble foot is an abscess in the foot pad (see '**bumble foot**'). It usually occurs in mature birds. The abscess formation is accompanied by swelling, heat, and usually some pain. This leads to **lameness**. The undersurface of the foot is first affected, and the lesion may then spread to invade the whole foot (Fig. 144, 145).

4. Staphylococcal Septicaemia (Blood Infection)

Staphylococcal infection is the invasion of the blood by staphylococci. It usually occurs from a local seat of infection. It is relatively rare and may result in sudden death.

Symptoms

Early symptoms include ruffled feathers, limping on one leg, hanging downwards of one or both wings, and fever. This may be followed by severe depression and death.

Postmortem Findings

Postmortem findings of yolk sac infection, gangrenous dermatitis and bumble foot have been described under their descriptions at other places. Postmortem findings in case of **staphylococcal septicaemia** include appearance of multiple foci of necrosis (white spots of dead tissue) in the liver (Fig. 80), lungs, kidneys, spleen, and heart.

Diagnosis

The history, symptoms, if any, and postmortem findings may be helpful, but it is necessary to isolate and identify ***S. aureus*** to confirm diagnosis.

Treatment

S. aureus infection can be successfully treated, but sensitivity tests should always be performed because antibiotic resistance is common. Drugs used successfully for treatment include penicillin, tetracycline, streptomycin, erythromycin, lincomycin, and sulphonamides.

Control

1. Because wounds are a route of entry for ***S. aureus*** into the body, anything reducing the chances of injury will help prevent infection.
2. Maintenance of good litter quality will reduce foot pad ulceration.

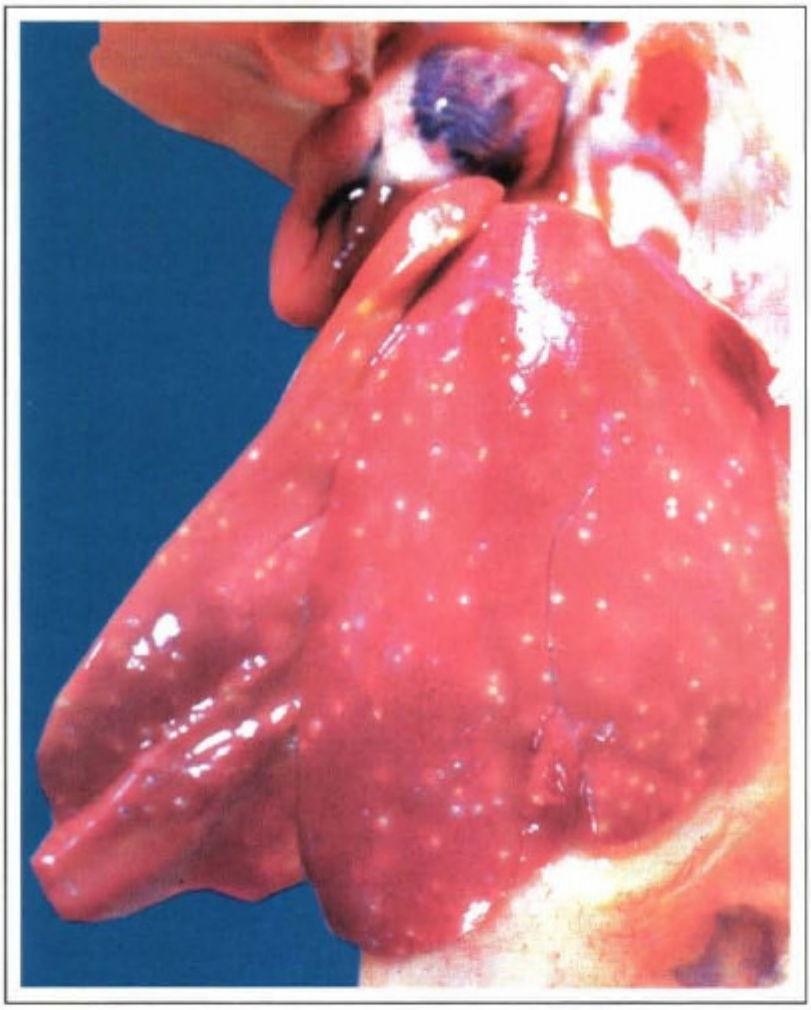

Fig. 80. **Staphylococcosis** in a **20-week-old hen**. Note multiple foci of necrosis (white spots of dead tissue) in the liver following blood infection with staphylococcal bacteria.

3 MYCOPLASMAL DISEASES

MYCOPLASMOSIS

Mycoplasma gallisepticum Infection

M. gallisepticum causes a respiratory disease, affecting the entire respiratory tract, particularly the **airsacs**, where it localizes. All the airsacs may be involved, become cloudy in appearance, and filled with mucus. In the later stages, mucus develops a yellow colour and cheesy consistency.

Commonly known as **'chronic respiratory disease'** or '**CRD**', ***M. gallisepticum*** infection is extremely important both in broilers and layers. While not a great killer, the disease is of great economic importance. Affected laying flocks have been shown to produce as many as 20 fewer eggs per year than normal flocks. **Also, it is an important egg-transmitted disease**. ***M. gallisepticum*** infection is a serious problem in our country.

The disease is characterized by abnormal respiratory sounds, coughing, and nasal discharge. Symptoms are usually slow to develop and the disease has a long course. '**Complicated CRD** (**CCRD**)', also known as **'airsac disease'** is a severe airsacculitis (inflammation of airsac), which occurs when ***M. gallisepticum*** infection gets complicated by ***E. coli*** and some respiratory virus infections.

Spread

1. **Infection is usually transmitted through the hatching egg. This is the major means of spread.**
2. **Carrier birds** (i.e., birds which carry the infection without showing symptoms) are responsible for transmitting the disease. Direct contact of susceptible birds with the infected carrier chickens causes outbreaks of the disease.
3. Spread may also occur **by contaminated dust, droplets**, or **feathers carried through the air.**
4. **People are important carriers.**

Symptoms

1. **In adult flocks** symptoms include abnormal respiratory sounds, nasal discharge, sneezing, coughing, and breathing through the open beak. Feed consumption is reduced and birds lose weight.
2. **In laying flocks**, egg production decreases, and the disease is usually more severe during winter.
3. **In young chicks** there is rattling, sneezing, and sniffing, all indicative of a respiratory difficulty.
4. **In broilers**, most outbreaks occur between 4 and 8 weeks of age.
5. However, the appearance of disease depends, as already mentioned, on the presence at the same time of other disease-producing organisms, or stress factors. **Uncomplicated infections usually cause no symptoms, or cause mortality only in the very young.**

Postmortem Findings

1. Presence of inflammatory material (exudate) in trachea, bronchi, and airsacs (Fig. 81, 82). Airsacs usually contain cheese-like inflammatory material. Some degree of pneumonia (inflammation of the lungs) may be seen.

2. In severe cases of airsac disease, **thin or thick white layer of fibrin covers heart, liver, and airsacs**. These are cases of CCRD.

Diagnosis

1. There are no symptoms or postmortem findings which are characteristic of ***M. gallisepticum*** infection in chickens.
2. Demonstration of the organism is the most certain method of confirming the infection.

Treatment

M. gallisepticum is susceptible to several antibiotics. These include streptomycin, oxytetracycline, chlortetracycline, tiamulin, neomycin, gentamicin, **tylosin**, erythromycin, lincomycin, enrofloxacin, and others.

Control

1. Treatment is only a temporary solution and is usually quite expensive. Removal of infection is the most satisfactory means of control.
2. Since ***M. gallisepticum*** is transmitted through eggs, maintaining chicken flocks free of ***M. gallisepticum*** is only possible by obtaining replacement flocks that are known to be free of the infection, and rearing them in strict isolation to avoid introduction of the disease.

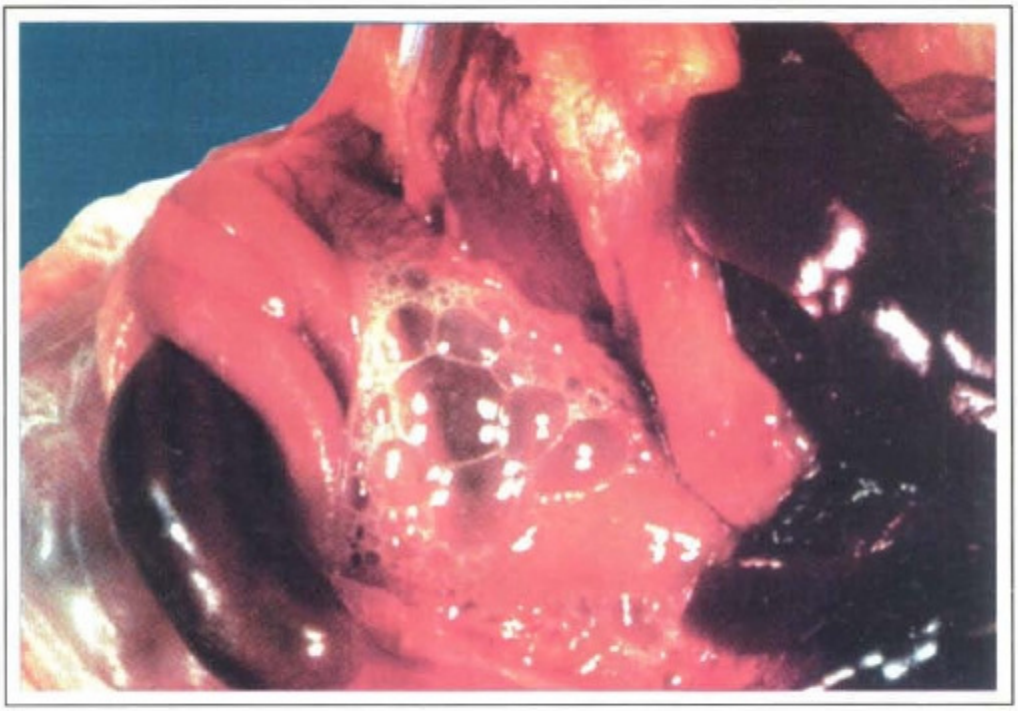

Fig. 81. **Mycoplasmosis** in a **chicken** showing foamy airsacculitis (inflammation of airsac) caused by ***Mycoplasma gallisepticum*** infection. Note the presence of foamy air bubbles.

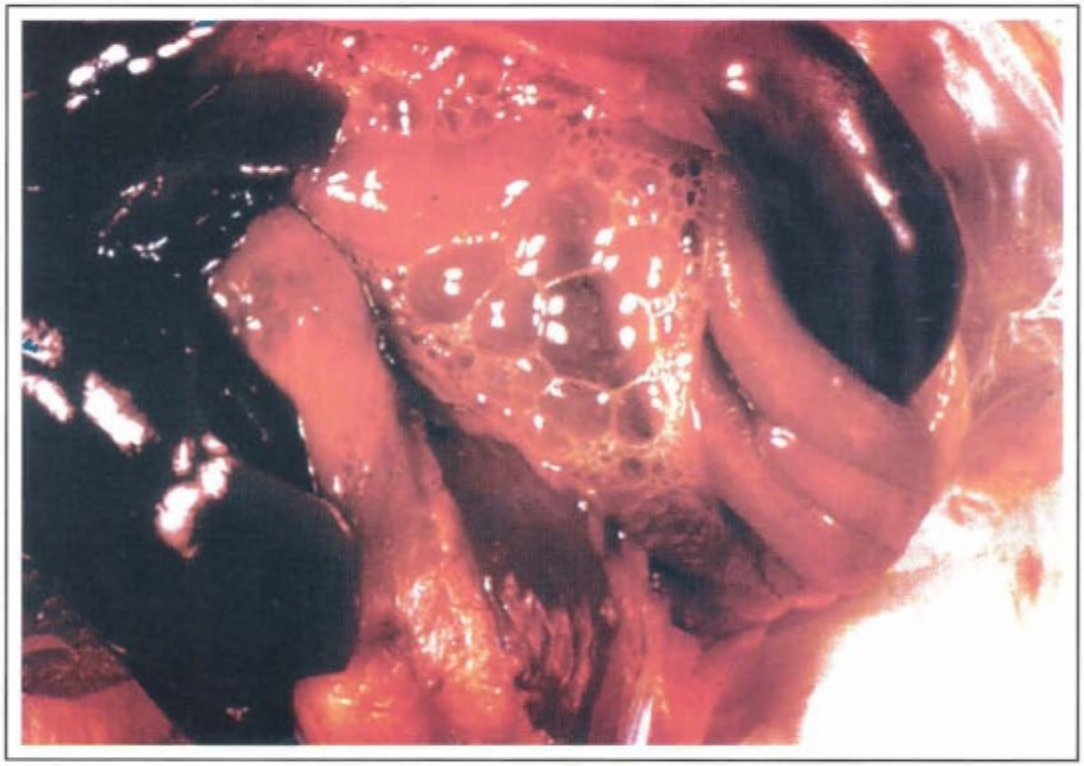

Fig. 82. **Mycoplasmosis**. Photograph of the same bird shown in Fig. 81 from a different angle, showing foamy airsacculitis caused by ***Mycoplasma gallisepticum*** infection. Note the presence of foamy air bubbles.

4 FUNGAL DISEASES

ASPERGILLOSIS

Also known as **'brooder pneumonia'**, aspergillosis is **a disease of very young chicks**. It is a disease of the respiratory system, and usually occurs when there are poor sanitary practices on the farm. Newly hatched chicks are highly susceptible to infection. Stress of cold, high ammonia, and dusty environments increase incidence and severity of the disease.

Cause

The disease is caused by a fungus called ***Aspergillus fumigatus***. Contaminated poultry litter is usually the source of infection. Chicks become infected during hatching or during the first day or two in the brooder house, hence the name '**brooder pneumonia**'.

Spread

Infection occurs by inhalation of spores from contaminated feed or litter. A spore is that form of fungus which is very tough and resistant, and is therefore difficult to destroy. Fungal growth in wet litter produces large numbers of spores. These spores spread as suspended particles in air as wet litter dries.

Symptoms

The affected chicks may stop eating and show symptoms of gasping or laboured breathing. They breathe with an open mouth due to obstruction of the airway.

Postmortem Findings

The **lungs** are the main organ affected. They show **small nodules** that are **hard** and **yellow** (Fig. 83, 84, 85, 86, 87). In some cases nodules are only a few, in others there may be hundreds. Nodules are also seen in the trachea and airsac.

Diagnosis

Aspergillosis is usually diagnosed at postmortem examination. Examination of the trachea or a **cut lung** will show nodules. This forms a basis for the diagnosis.

Treatment

Apart from providing adequate ventilation, there is no treatment for birds that are affected.

Control

1. A thorough cleaning of the brooding premises will eliminate the source of infection for future flocks.
2. Any mouldy feed should be removed, feed containers cleaned, and old litter removed from the house and replaced with new.
3. Drinkers and feeders should be cleaned and disinfected.
4. Since ***M. gallisepticum*** is transmitted through eggs, maintaining chicken flocks free of ***M. gallisepticum*** is only possible by obtaining replacement flocks that are known to be free of the infection, and rearing them in strict isolation to avoid introduction of the disease.

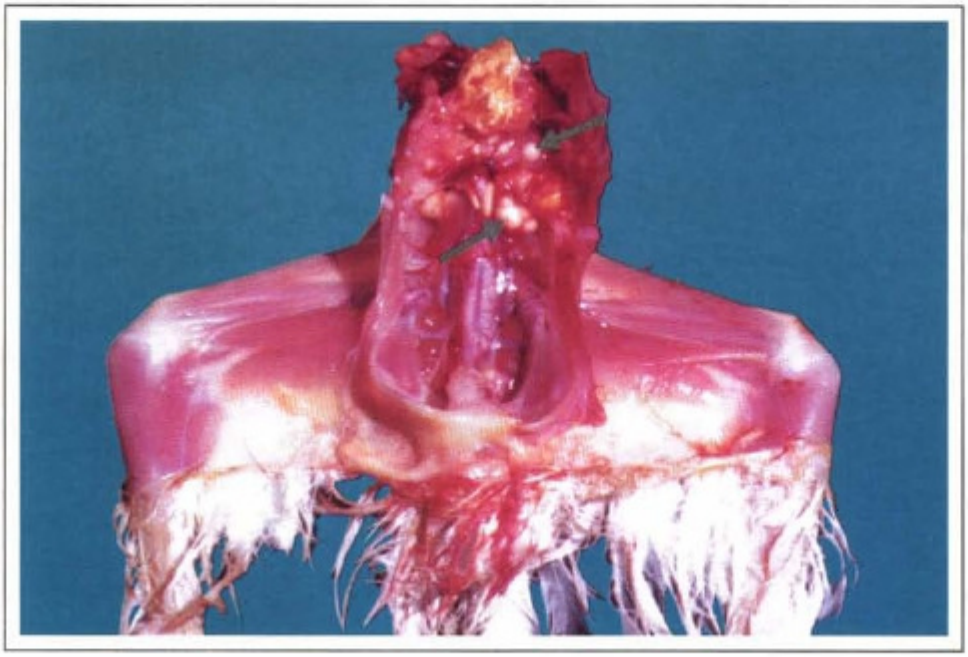

Fig. 83. **Aspergillosis** in a **42-day-old cockerel**. Note lungs show the presence of yellowish-white small nodules (arrows).

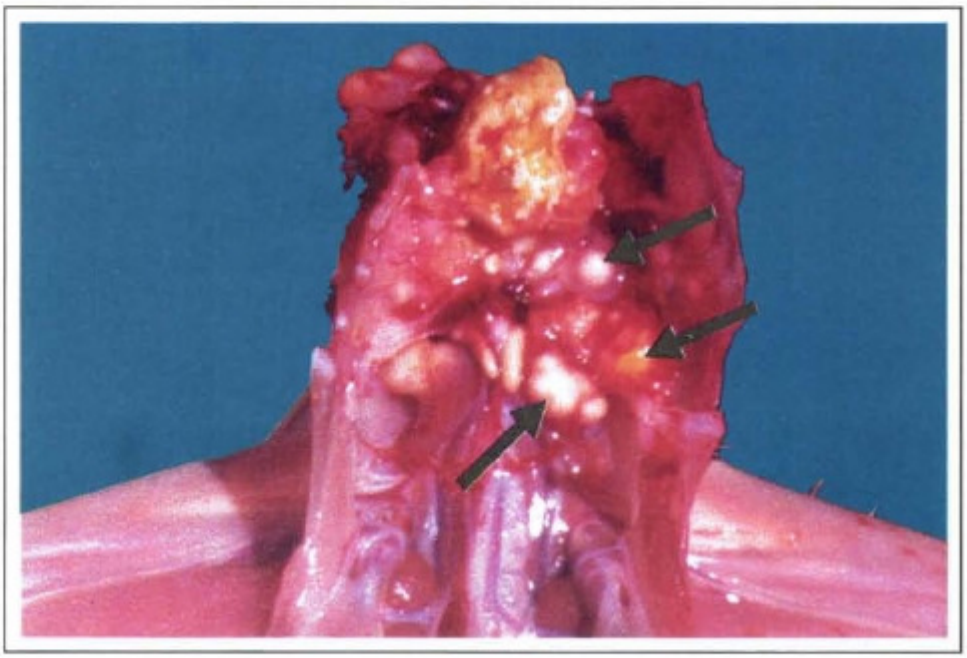

Fig. 84. **Aspergillosis**. A close-up picture of the same **42-day-old cockerel** shown in Fig. 83. The nodules are now prominent (arrows).

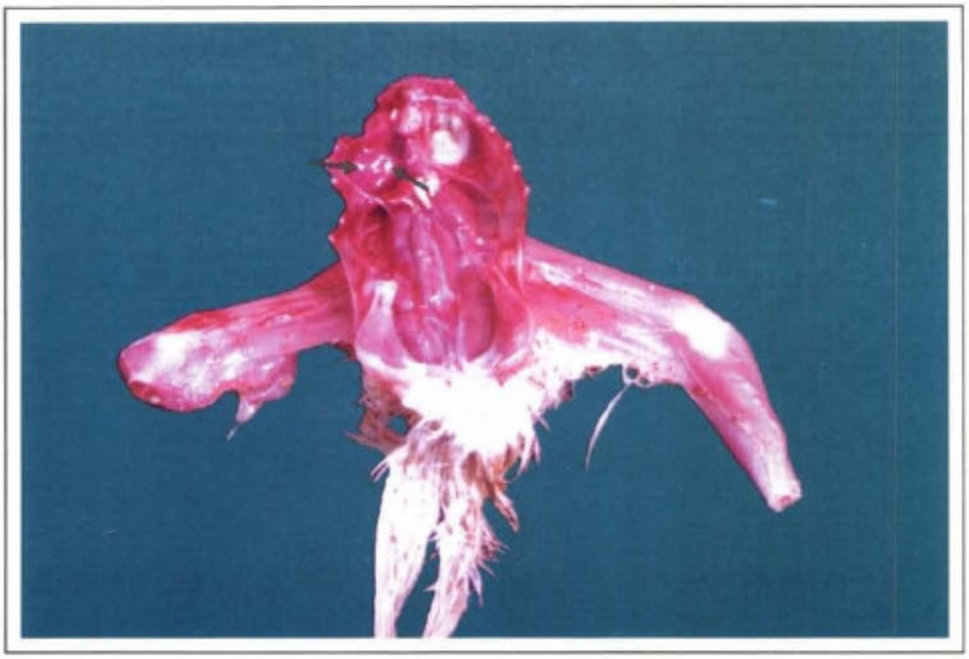

Fig. 85. **Aspergillosis** in a **45-day-old cockerel**. Note lungs contain yellowish-white small nodules (arrows).

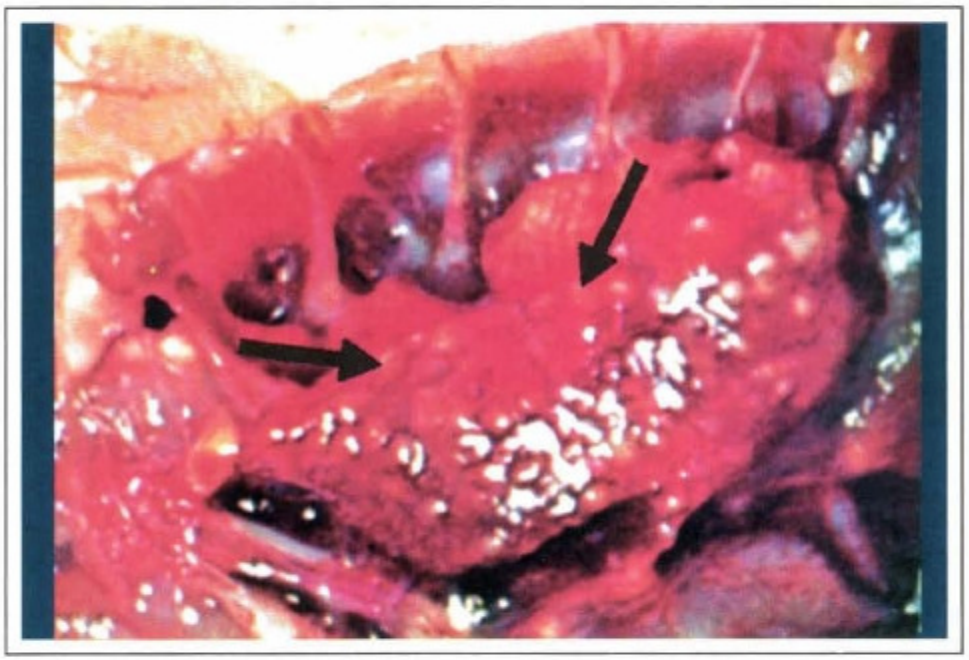

Fig. 86. **Aspergillosis** in a **chick**. Note chick's lungs are filled with yellowish-white small nodules (arrows). This is typical of aspergillosis.

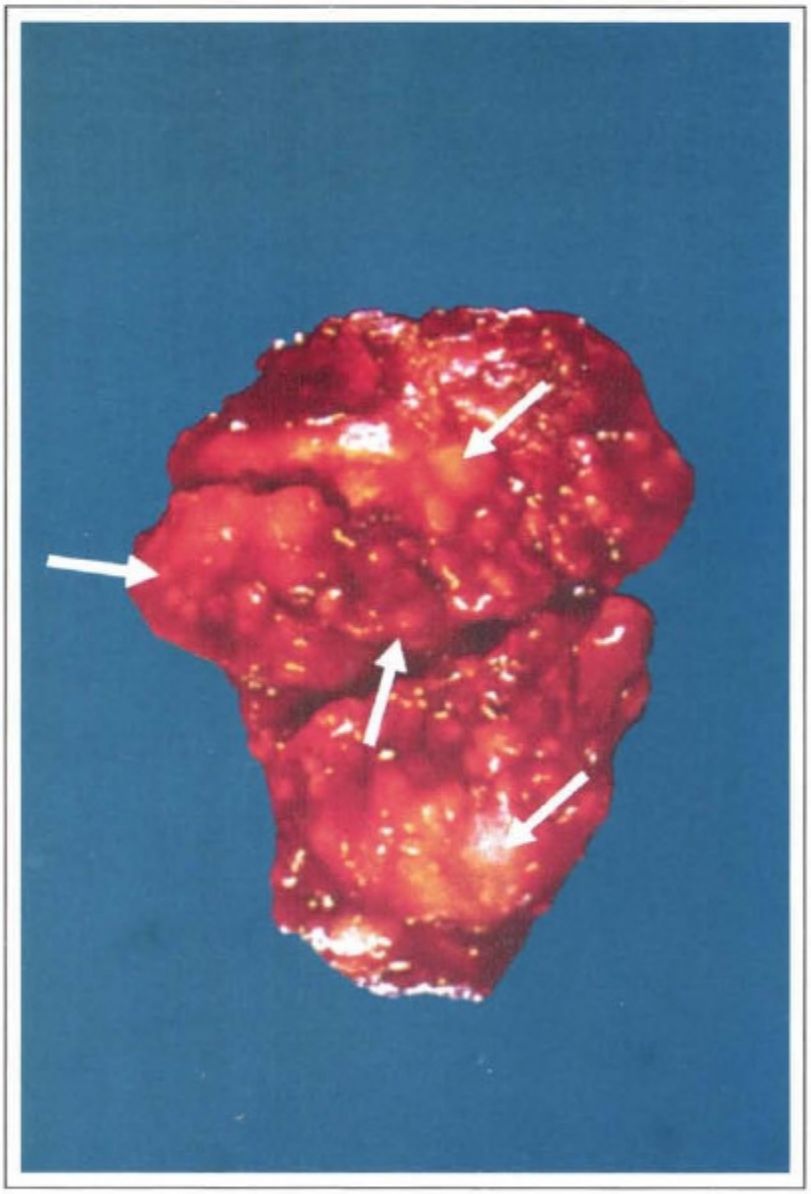

Fig. 87. **Aspergillosis** in a chick-showing severe lung infection. Note the presence of numerous nodular lesions (arrows). They tend to merge together.

FUNGAL INFECTION (UNIDENTIFIED)

(Author's observations)

Once, a few years ago, when the cost of soybean had gone exorbitantly high, certain farmers at Jabalpur tried blood meal as an alternative source of cheaper protein. This happened to be in the rainy season. When the blood meal was fed to layer birds, it resulted in heavy mortality in 8-week-old growers.

The **symptoms** included mainly diarrhoea and mortality. In less severe cases, symptoms were those of depression, loss of appetite, and chicks lying down listlessly.

The **postmortem examination** revealed massive growth of fungus in the internal organs of the abdominal cavity (Fig. 88, 89, 90). The fungus, however, could not be identified.

It is concluded that during storage, the blood meal may have gone mouldy, that is, covered with fungus; and when fed, this may have led to the fungal infection.

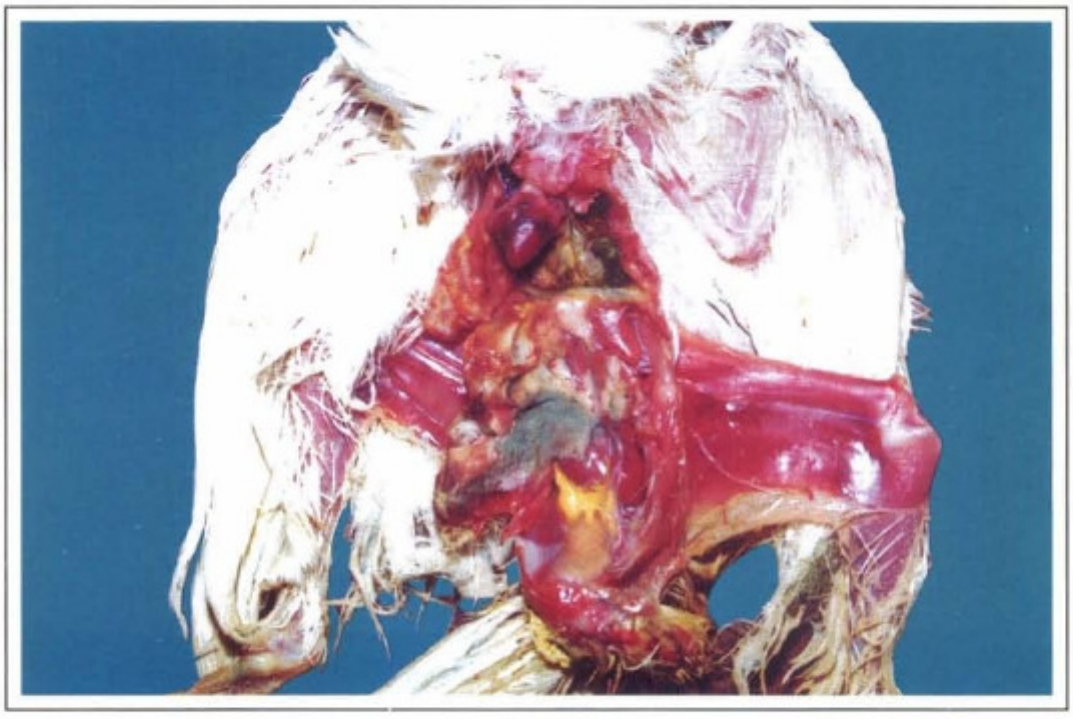

Fig. 88. **Fungal infection** (unidentified) in an **8-week-old grower chicken**. Note the presence of massive bluish fungus growth in the abdominal cavity.

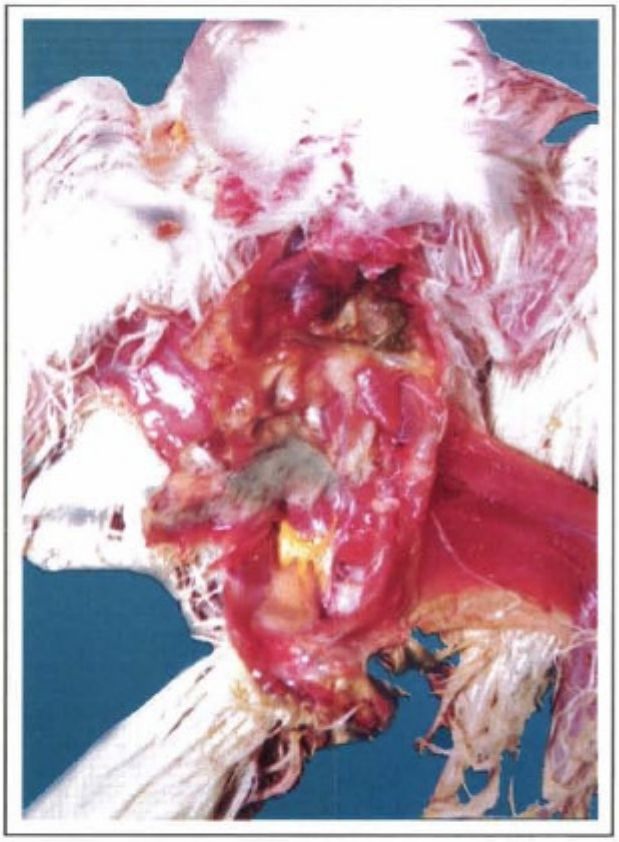

Fig. 89. **Fungal infection** from the same bird shown in Fig. 88. A close-up picture reveals fungal infection in various abdominal organs.

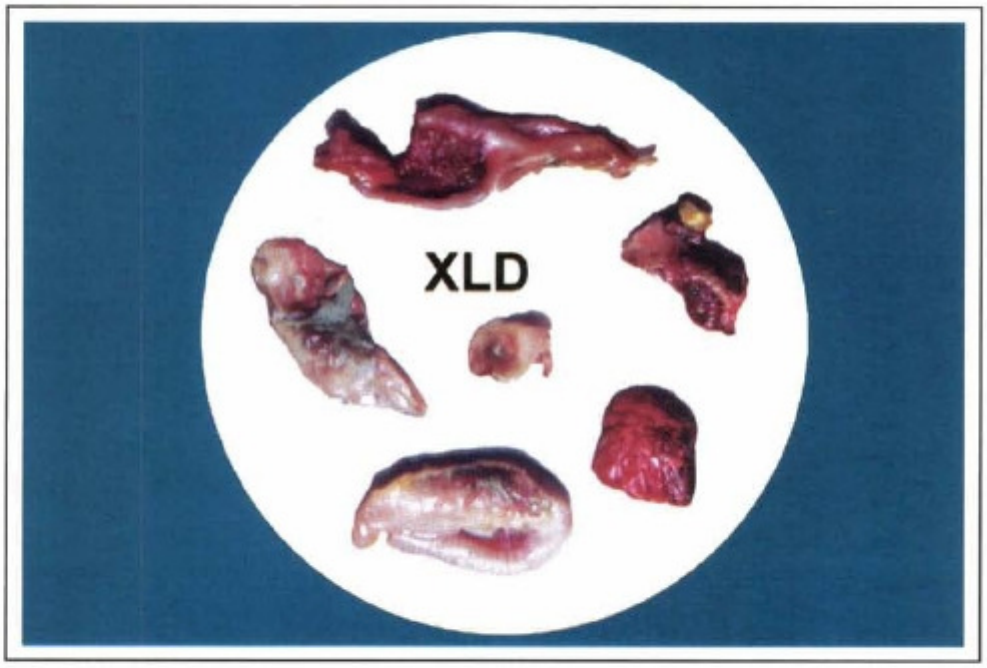

Fig. 90. Fungal infection in the same chicken shown in Fig. 88, showing various internal organs. Note the bluish tinge indicates the presence of fungus.

AFLATOXICOSIS

Growing fungi (moulds) produce a large number of chemicals as by-products and secrete them into surrounding substances. Some are toxic to birds. These toxic by-products are called **'mycotoxins'** or **'fungal toxins'.** Among them, **aflatoxin** is the most common and also the most important mycotoxin likely to be consumed by poultry.

Aflatoxins are highly toxic mycotoxins produced by various species of fungus **Aspergillus**. The fungus produces aflatoxin in warm, high humidity conditions, such as rainy season. Aflatoxins can withstand extreme environmental conditions and are very heat stable. Aflatoxin contamination is therefore more common in grains in a tropical country like ours. **Young birds are more sensitive to aflatoxin than adults.**

Harmful Effects of Aflatoxin

1. Affects weight gain
2. Affects feed intake
3. Affects feed conversion efficiency
4. **Affects egg production**
5. Affects male and female reproductive performance
6. Increases susceptibility to infectious diseases due to immunosuppression. That is, suppression of the immune response.
7. Egg size, yolk weight, and yolk as percentage of total egg size, are decreased.
8. Even less than 100 ppb (parts per billion) in broilers, can result in poor feed conversion and reduced weight gain, which may be due to liver damage and reduced nutrient absorption.

Symptoms

1. In growing birds, there is decreased growth and poor feed conversion.
2. There is also marked decrease in the resistance of birds to infections, such as coccidiosis and Gumboro disease, due to immunosuppression.
3. Affected hens have decreased egg production. Also, the hatchability of eggs is reduced.

Postmortem Findings

1. Liver is greatly enlarged, **yellow** and friable (easily broken) (Fig. 91).
2. Small haemorrhages may occur due to increased fragility of minute blood vessels. This leads to a condition known as **'bloody thigh syndrome'.**

Diagnosis

Symptoms and postmortem findings would indicate aflatoxicosis. Confirmation requires identification of the level of toxin present in the feed.

Treatment

1. Toxic feed should be removed and replaced with uncontaminated feed.
2. Increase the dietary levels of protein. Increase also the vitamin supplementation.
3. Supply of methionine and other sulphur-containing amino acids should be increased.
4. Poor management, if present, should be improved.
5. Liver tonics may be given.
6. Increase also the supplementation of **vitamin E** and **selenium**.

Control

1. Purchase a clean feedstuff. Check the ingredients before purchase.
2. Monitor feed ingredients for levels of mycotoxins.
3. Discard the grains suspected of contamination. That is, mouldy and caked feed.
4. Keep the moisture of grains less than 12%.
5. **Sun drying is the best method to prevent mould growth. However, it does not destroy the toxin.**
6. Store the feed and ingredients in well ventilated dry place.
7. Avoid storage of feed for more than a week.
8. Withdraw toxin contaminated feed immediately.
9. **The most practical way to control aflatoxicosis is through the effective use of mould inhibitors and broad-spectrum toxin binders.**

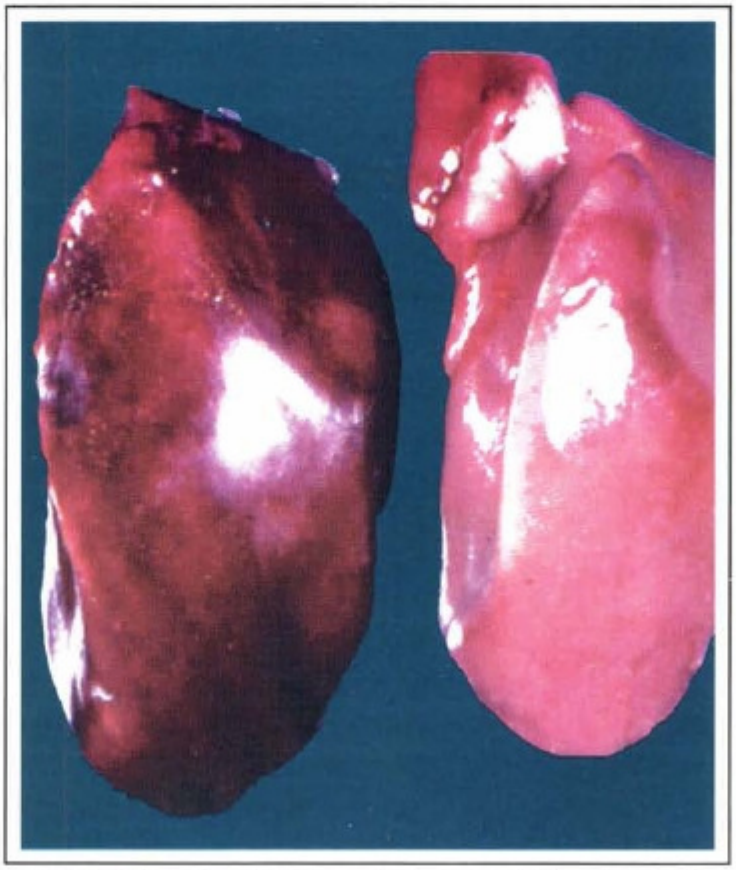

Fig. 91. **Aflatoxicosis**. Note pale liver of a chicken (on right) receiving 200 ppb aflatoxin in feed. Compare it with the normal liver on the left.

Fig. 92. **Toxicity (suspected). Three 80-day-old cockerels** suffering from some toxicity, may be fungal mycotoxicity. The exact cause of death, however, remained undiagnosed

5 PARASITIC DISEASES

ROUNDWORMS

Roundworms are the most important worms of poultry. Among them, the large roundworm ***Ascaridia galli*** is the most common. **Ascaridia** are the largest roundworms of birds. The adults live in the lumen of the small intestine. The larval stages invade the intestine.

Harmful Effects

1. ***A. galli*** infection causes poor bodily condition and weight loss. The degree of effect is related to the number of worms present.
2. **In severe infections intestinal blockage can occur**. This may lead to deaths, especially in younger birds. Chickens infected with a large number of worms suffer from loss of blood, retarded growth, enteritis (inflammation of the intestine), and greatly increased mortality.

Diagnosis

The worms are easily seen on postmortem examination in the small intestine (Fig. 93, 94).

Treatment

1. Piperazine compounds have been widely used for treatment against **Ascaridia** infection, since they are non-toxic. Piperazine may be given to chickens in the feed, water, or as a single treatment.
2. Piperazine in drinking water is the best practical method of giving piperazine for commercial flocks.
3. Fenbendazole is also effective.

Control

1. Roundworms, in large part, can be prevented by raising birds in cages.
2. Poultry should receive a diet which is adequate, especially in vitamin A and vitamin B complex compounds. **Lack of these vitamins makes poultry more susceptible to worm infections.**
3. Proper management of litter. Keep the litter as dry as possible by frequent stirring. Change of litter can reduce infections. Litter may also be treated with suitable insecticides.
4. Avoid overcrowding. Overcrowding produces more favourable conditions for worm development by increasing the chances of infection. Overcrowding provides more warmth and moisture, both of which favour the development of worm eggs.
5. Extreme care should be taken to ensure that feed and water are not contaminated.

Fig. 93. **Large roundworms** (**Ascaridiasis**) in the small intestine of a **chicken**. **This is the most common worm of poultry**, and is usually found in the middle portion of the small intestine.

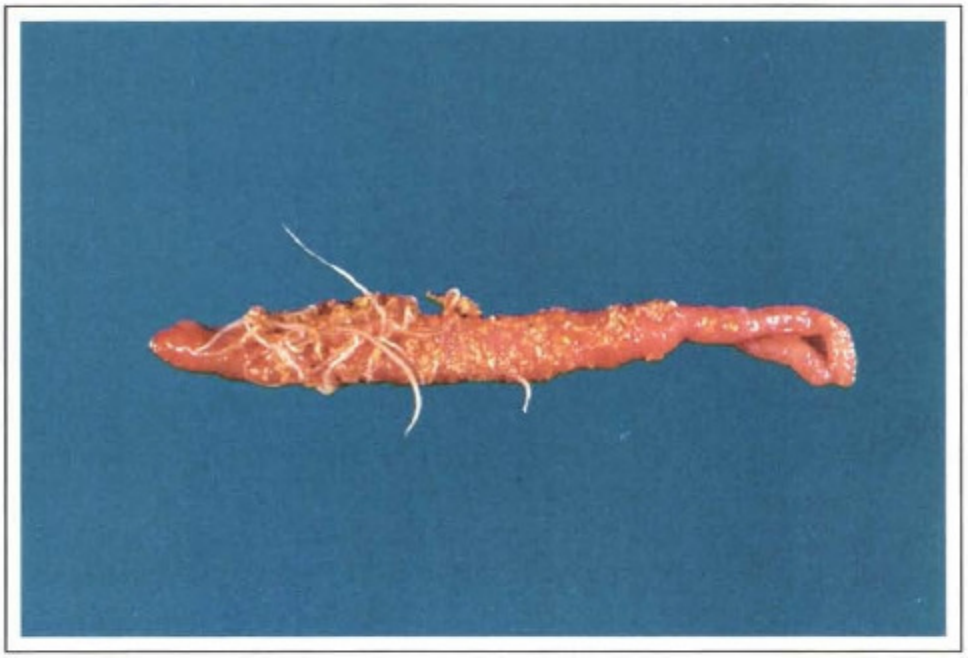

Fig. 94. **Large roundworms** in the small intestine of a **chicken**. Severe infestation may cause obstruction of the intestinal tract.

COCCIDIOSIS

Coccidiosis is one of the most important diseases of poultry worldwide. **In our country, it is a serious problem and one of the biggest causes of economic losses.** Coccidiosis mostly occurs in rainy season, and is characterized by bloody diarrhoea and high mortality. **It is mostly a disease of young birds** because immunity quickly develops after exposure and gives protection against later outbreaks. Coccidiosis inflicts heavy mortality in broilers and growers raised on deep litter. Outbreaks are common between 3-6 weeks of age and rare in flocks of less than three weeks. However, author has observed coccidiosis in **7-day-old broiler chicks**. Coccidiosis rarely occurs in layers because of prior exposure of coccidia resulting in immunity. It mainly occurs under conditions of overcrowding.

Spread

1. **Ingestion of the infective form of oocysts** (sporulated oocysts) is the only method of spread.
2. **Oocysts can be spread mechanically** by animals, insects, contaminated equipment, wild birds, and dust. They are resistant to environmental extremes and to disinfection.
3. Spread from one farm to another is facilitated **by movement of people and equipment between farms**, which may spread the oocysts mechanically.

Symptoms

Coccidiosis occurs in **two forms**: (1) **caecal coccidiosis**, and (2) **intestinal coccidiosis**. Symptoms vary accordingly.

Caecal coccidiosis is caused by ***Eimeria tenella***. It is a severe disease associated with bloody droppings, high mortality, reduced weight gain, and emaciation (Fig. 92). **The losses caused make this species one of the most harmful in chickens**. Most of the mortality occurs between 5th and 6th day following infection.

Intestinal coccidiosis is caused mainly by ***E. necatrix***. It is associated with severe weight loss, morbidity (number of birds affected in a flock), and mortality. Intestinal coccidiosis occurs in relatively older birds. Droppings of affected birds usually contain blood. **Like *E. tenella*, *E. necatrix* is also most harmful.** It has caused more than 25% mortality in commercial flocks.

Postmortem Findings

In caecal coccidiosis, caeca may be greatly enlarged and distended with clotted blood (Fig. 95, 96, 97, 98, 99, 100, 101, 102, 103).

In intestinal coccidiosis, the **middle portion of the small intestine** is usually distended to twice its normal size (ballooning) and the lumen may be filled with blood (Fig. 104, 105, 106, 107, 108, 109). Changes may extend throughout the small intestine in severe infections.

Diagnosis

1. The presence of faeces with blood, dysentery, or diarrhoea suggests coccidiosis.
2. **However, postmortem examination is necessary to confirm diagnosis**. Greatly enlarged caeca distended with clotted blood confirm **caecal coccidiosis**, whereas small intestine distended with blood confirms **intestinal coccidiosis**.

Treatment

Outbreaks of coccidiosis are usually treated with water-soluble drugs, such as sulphonamides, amprolium, diclazuril and toltrazuril. Water medication is convenient and can be rapidly given.

Control

1. **Use anticoccidial drugs for prevention**. Most anticoccidials are formulated as feed additives, and broiler feed almost always contains an anticoccidial agent. The most widely used drugs are the ionophores, such as maduramicin, salinomycin, and monensin.
2. **Maintain good hygiene**. Good hygiene can greatly reduce the numbers of oocysts contaminating the environment. Moreover, good hygiene ensures that litter is kept dry so that it does not provide good sporulation condition.

Fig. 95. **Caecal coccidiosis** in a **45-day-old cockerel**. Note the bird is emaciated and anaemic, as evidenced by the yellowish discoloration of its beak, comb, and legs.

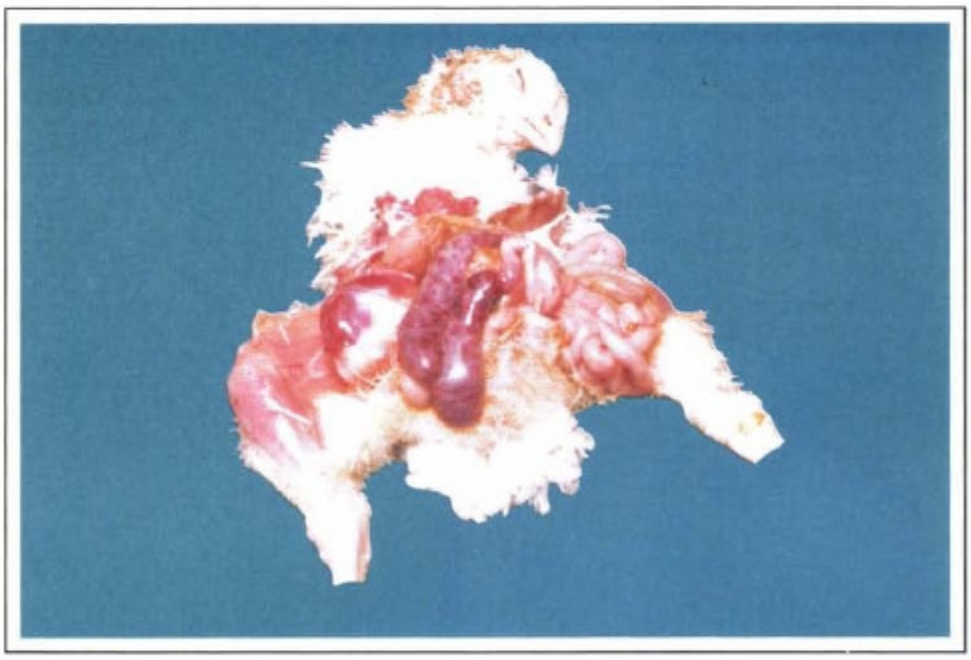

Fig. 96. **Caecal coccidiosis** in a **21-day-old broiler chicken** caused by ***Eimeria tenella***. Note caeca are enlarged and filled with blood.

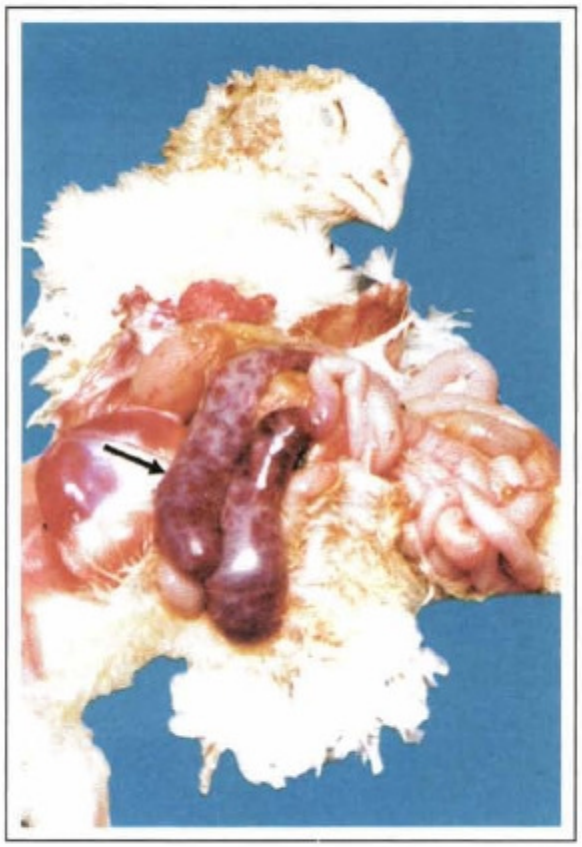

Fig. 97. **Caecal coccidiosis** from the same **21-day-old broiler chicken** shown in Fig. 96. A close-up picture shows that caeca are greatly enlarged and markedly distended with blood (arrow).

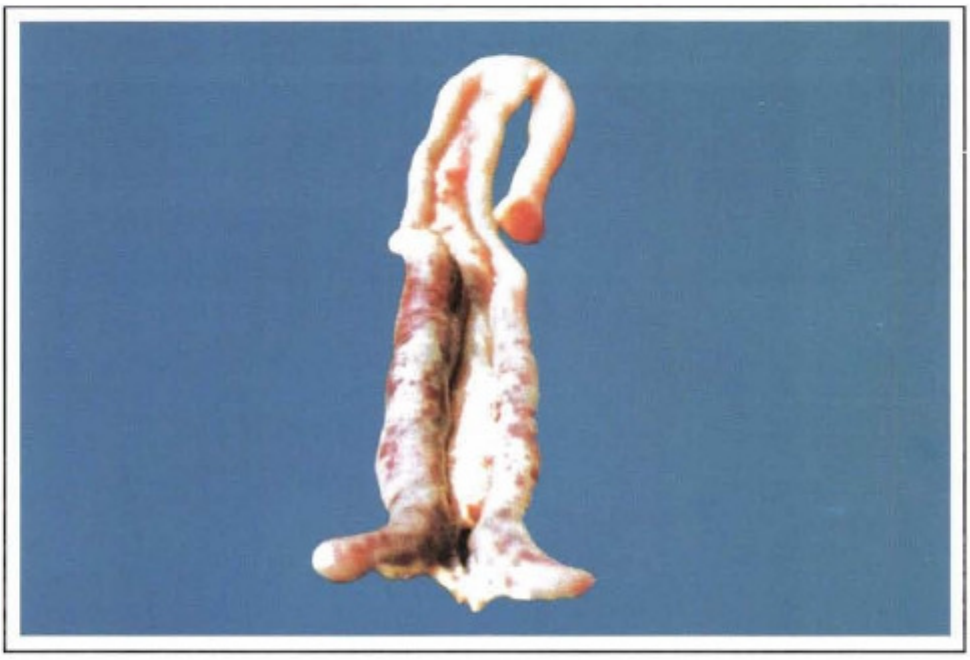

Fig. 98. **Caecal coccidiosis** in a **28-day-old broiler chicken** caused by ***Eimeria tenella***. Note caeca are markedly enlarged and distended with blood.

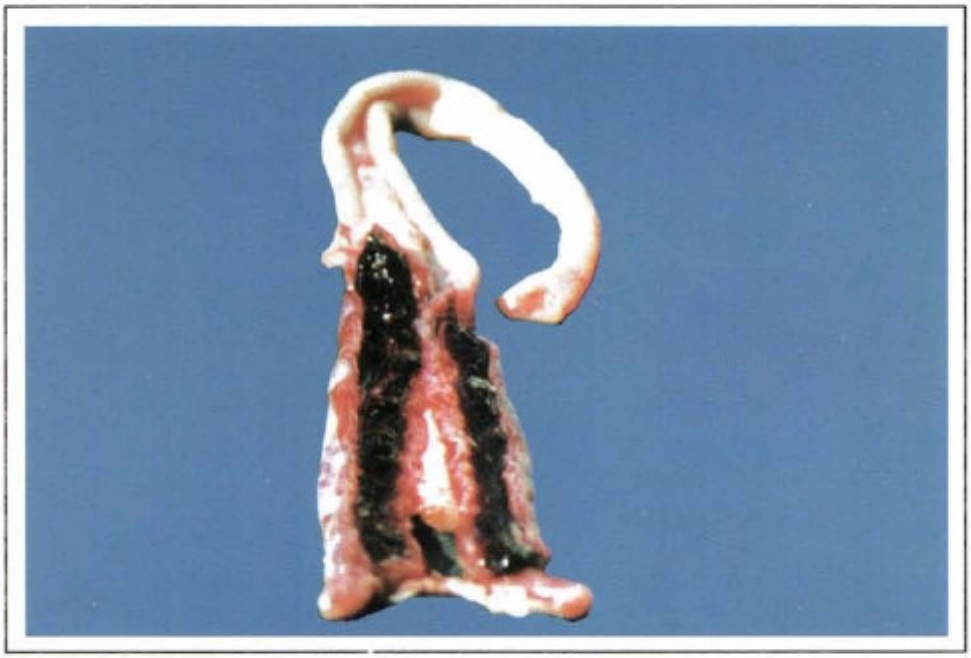

Fig. 99. **Caecal coccidiosis** in the same **28-day-old broiler chicken** shown in Fig. 98. Caeca are now cut open to reveal the presence of clotted blood.

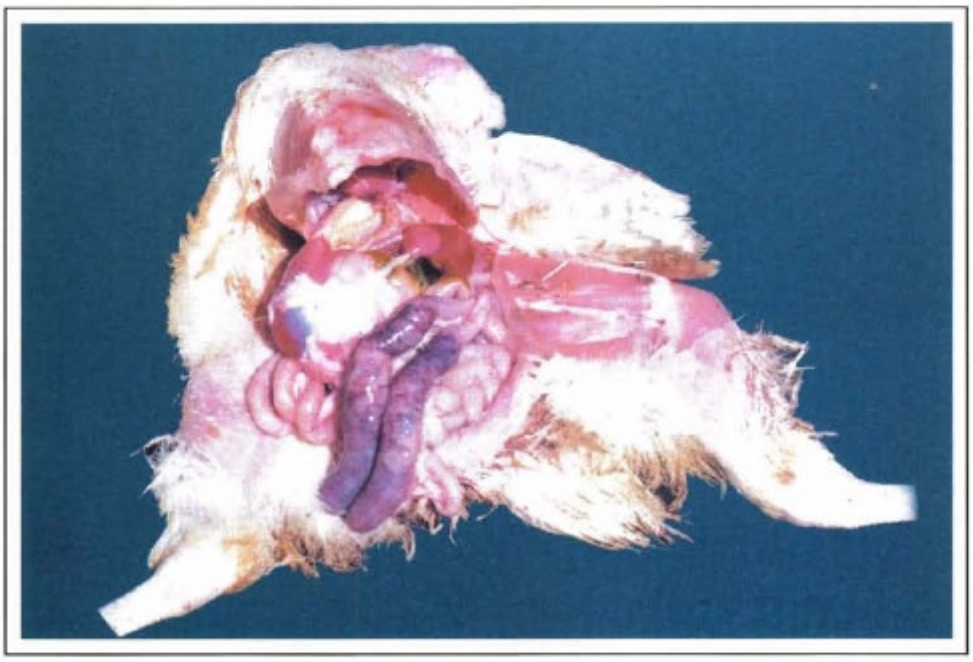

Fig. 100. **Caecal coccidiosis** in a **34-day-old broiler chicken** caused by ***Eimeria tenella***. Note caeca are greatly enlarged and distended with blood.

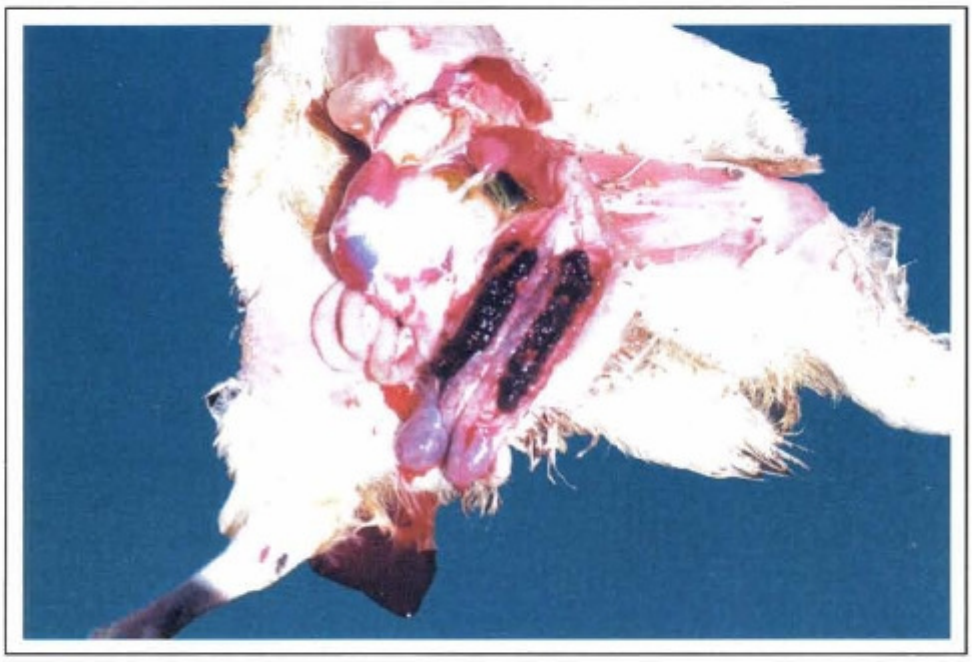

Fig. 101. **Caecal coccidiosis** from the same **34-day-old broiler chicken** shown in Fig. 100. The caeca are now cut open and reveal the clotted blood.

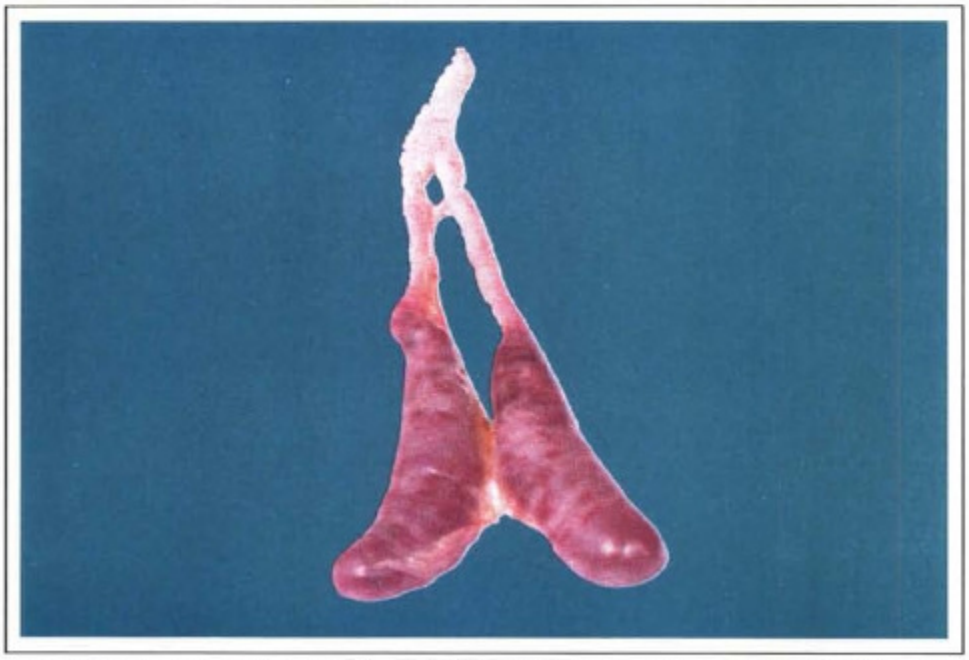

Fig. 102. **Caecal coccidiosis** in a **14-day-old broiler chicken** caused by ***Eimeria tenella***. Note caeca are greatly enlarged and distended with blood.

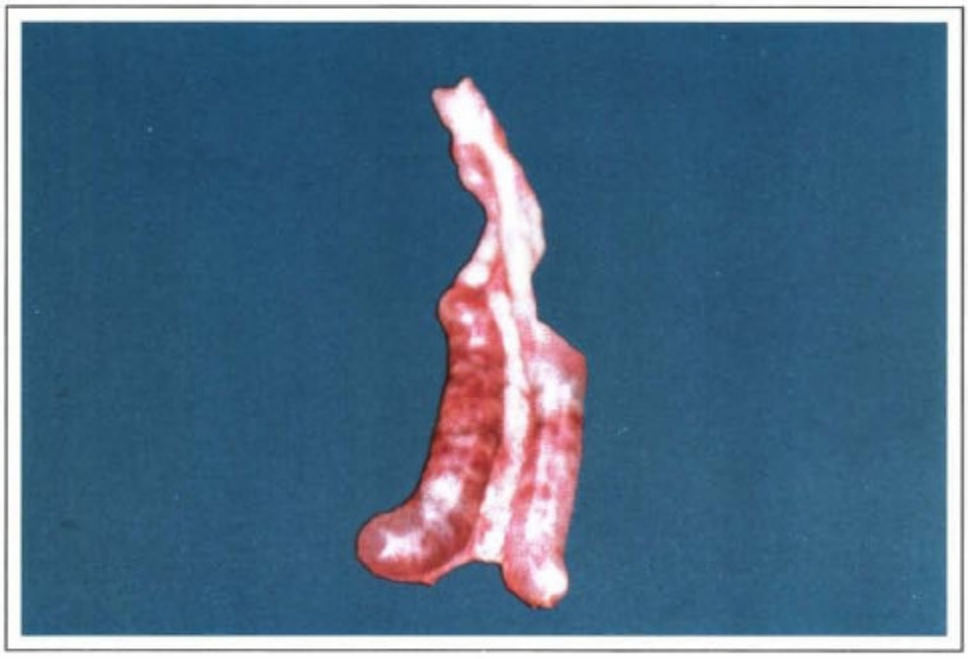

Fig. 103. **Caecal coccidiosis** in a **35-day-old cockerel** caused by ***Eimeria tenella***. The caeca are greatly distended with blood.

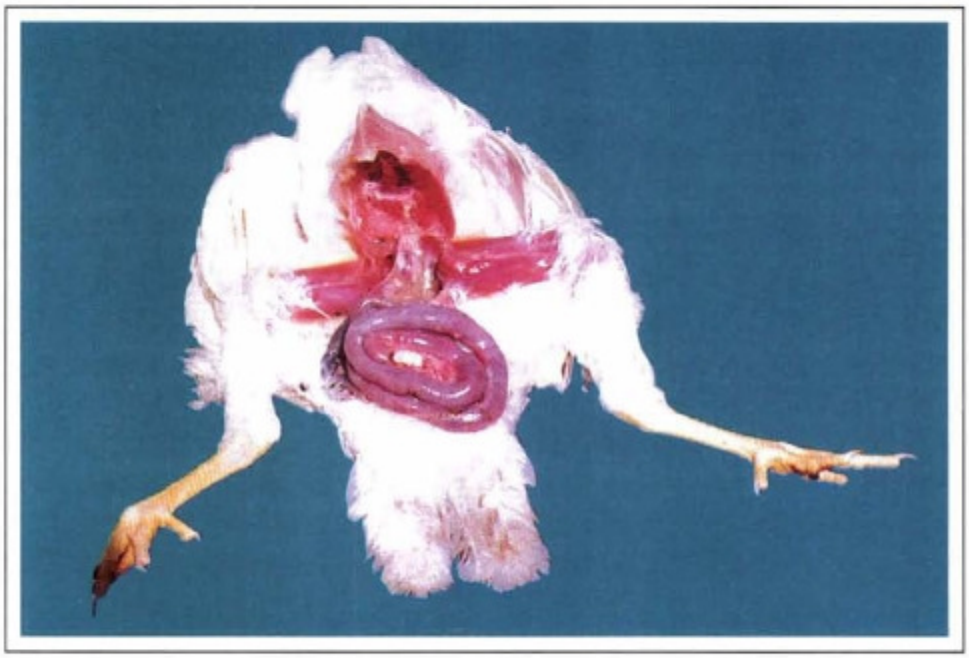

Fig. 104. **Intestinal coccidiosis** in a **10-week-old grower chicken** caused by ***Eimeria necatrix***. Note small intestine is greatly enlarged and distended with blood.

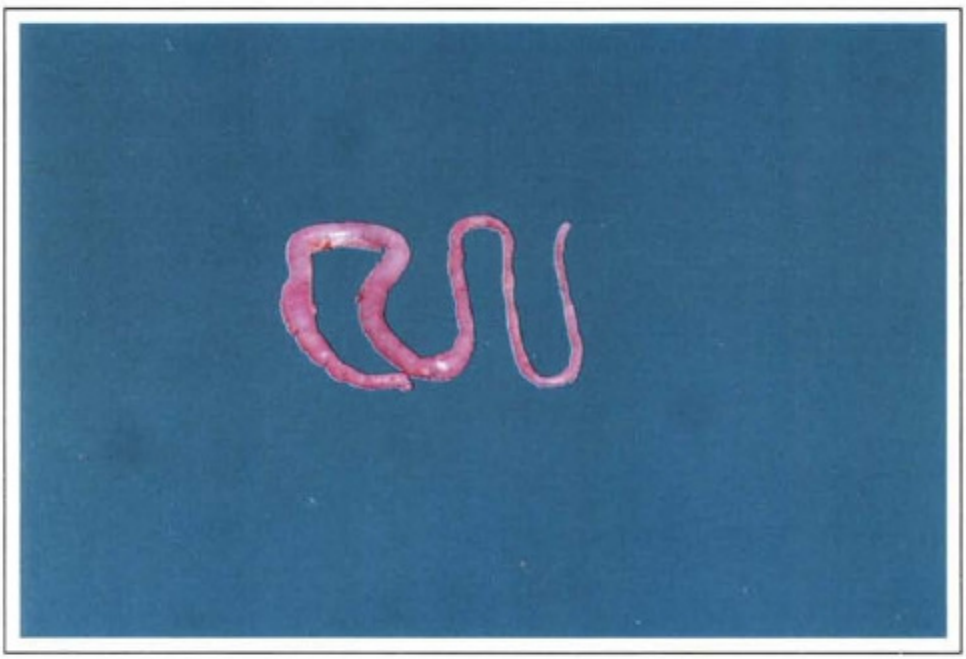

Fig. 105. **Intestinal coccidiosis** from the same **10-week-old grower chicken** shown in Fig. 104. A part of the small intestine has been spread. Note affected portion on the left is distended. Compare it with the relatively unaffected portion on the right.

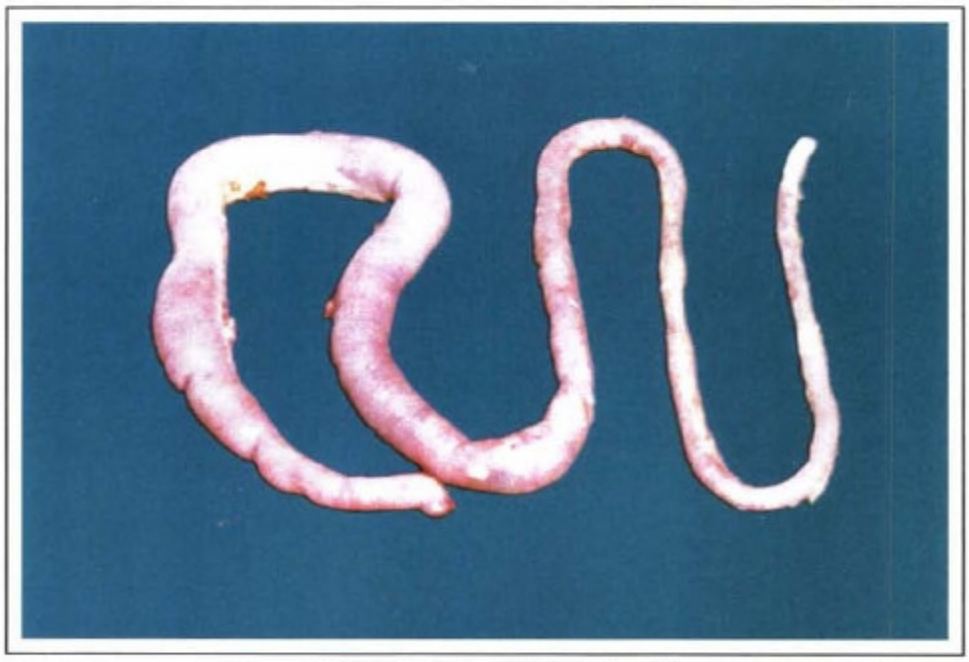

Fig. 106. **Intestinal coccidiosis** of the same **10-week-old grower chicken** shown in Fig. 105. A close-up picture shows that the affected portion of the small intestine on the left is distended twice its normal size (**ballooning**) and filled with blood. **This ballooning is typical of the intestinal coccidiosis caused by *Eimeria necatrix*.**

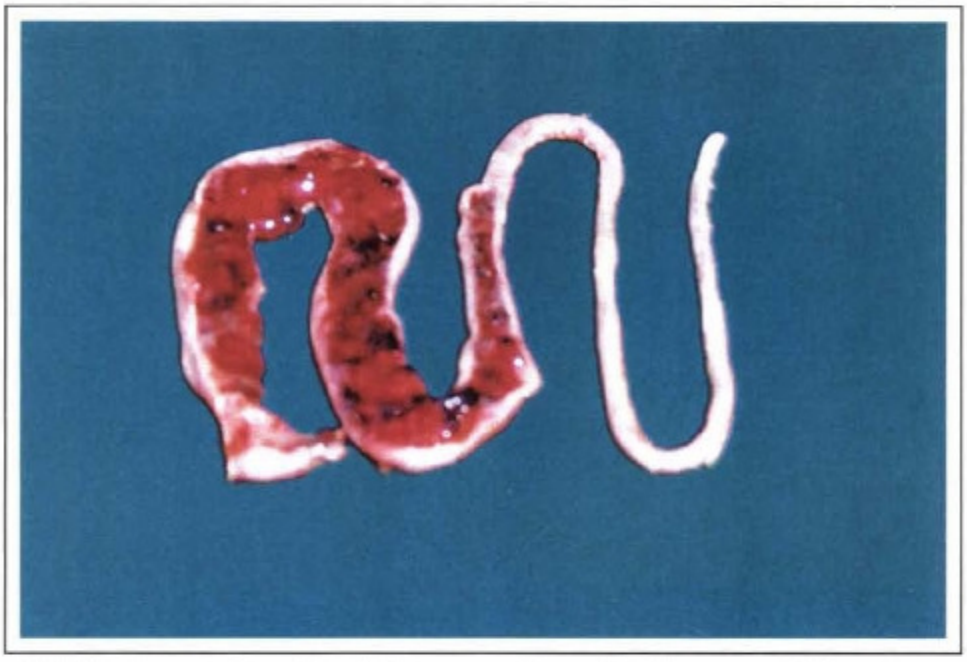

Fig. 107. **Intestinal coccidiosis** in the same **10-week-old grower chicken** shown in Fig. 106. The affected portion of the small intestine on the left is cut open to reveal the presence of clotted blood.

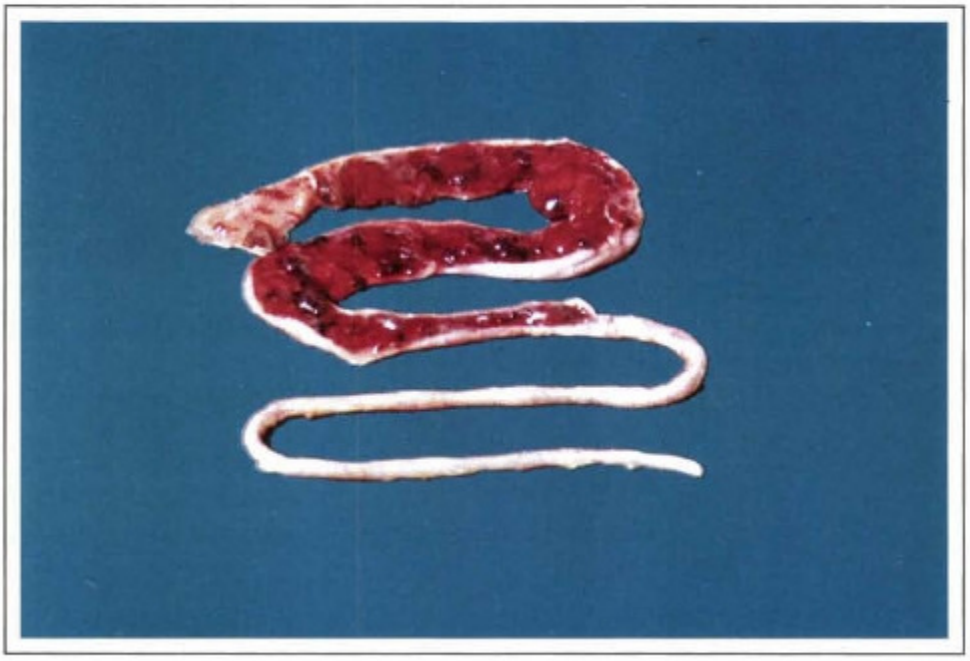

Fig. 108. **Intestinal coccidiosis** in the same **10-week-old grower chicken** shown in Fig. 107. Affected portion of the small intestine revealing clotted blood is shown differently.

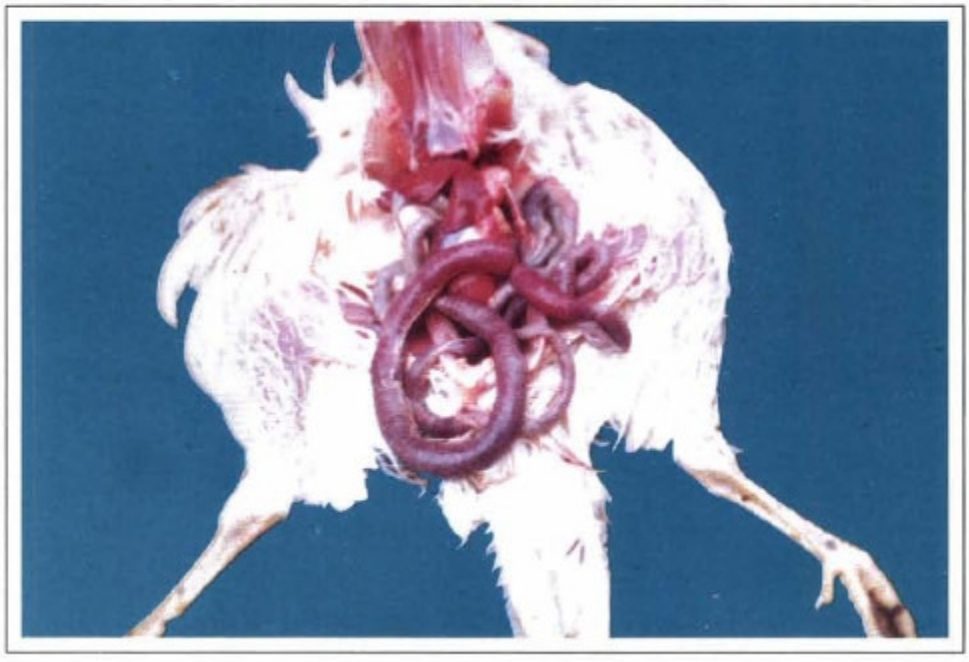

Fig. 109. **Intestinal coccidiosis** in a **6-week-old grower chicken** caused by ***Eimeria necatrix.*** Note intestine is greatly enlarged and distended with blood.

6 NUTRITIONAL DISEASES

VITAMIN A DEFICIENCY

Vitamin A is essential in poultry diets for growth, good vision, and integrity of mucous membranes. Mucous membranes are the inner linings of the digestive, respiratory, urinary, and genital systems in which changes of vitamin A deficiency are mostly observed. From a nutritional point of view, vitamin A is the most challenging, because it is most likely to be deficient in poultry.

Causes of Deficiency

1. Low levels of vitamin A in the feed
2. Oxidation of vitamin A in the feed
3. Errors in mixing
4. Intercurrent disease, for example, coccidiosis, worm infestation

Deficiency Symptoms

In chicks, symptoms include loss of appetite, poor growth, ruffled feathers, sleepiness, and weakness. Deficient chicks are very susceptible to infections, due to inadequate antibody production.

In adult chickens, changes of vitamin A deficiency first appear in the pharynx and oesophagus. These changes block the ducts of mucous glands (present in the mucous membranes). As a result, their contents lead to the development of **small white pustules** (raised spots). That is, the blocked ducts of the mucous glands cause distension of the glands with secretions and dead materials. **Pustules** are easily seen and are the characteristic lesions of vitamin A deficiency on postmortem examination (Fig. 110, 111). Egg production decreases sharply and hatchability is reduced.

Diagnosis

This is based on the characteristic postmortem findings in the oesophagus.

Postmortem Findings

1. Inner lining of the **oesophagus shows tiny white raised granules** (Fig. 107, 108). These are distended, impacted mucous glands.
2. Kidneys may present gout.

Treatment of Deficiency

Administer a stabilized vitamin A preparation at a level of about 10,000 IU vitamin A per kg of feed.

Prevention

1. Use stabilized vitamin A preparation in the feed.
2. Add adequate levels of antioxidants in the feed.
3. Provide adequate storage for feed and feed ingredients.

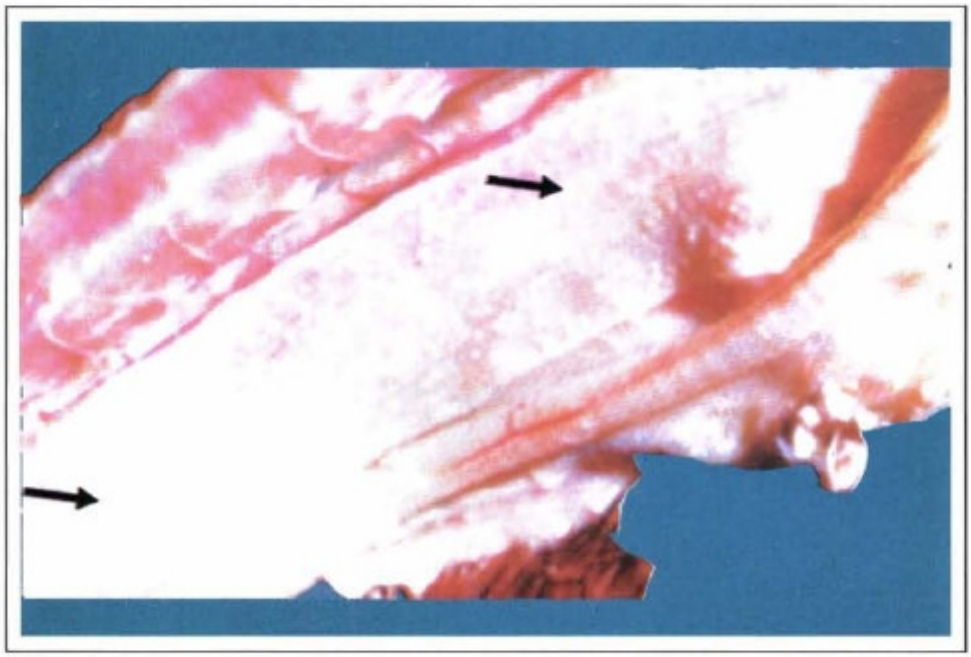

Fig. 110. **Vitamin A deficiency** in a **chicken**. Note distended, impacted mucosal glands in the oesophagus (tiny white raised granules, see arrows). **This is characteristic of vitamin A deficiency.**

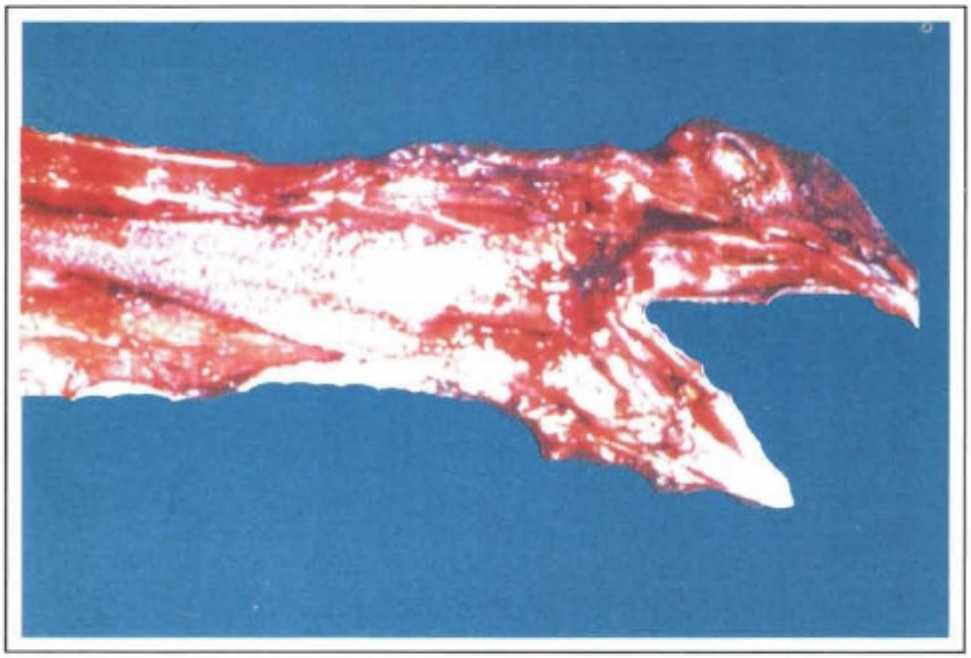

Fig. 111. **Vitamin A deficiency** in another **chicken**. Note nodular lesions in the mucosa (inner lining) of the oesophagus. **This is characteristic of vitamin A deficiency.**

VITAMIN E DEFICIENCY

The most important function of vitamin E is that it acts as an **antioxidant**. An antioxidant is something that is added to a product to prevent or delay its deterioration by the oxygen in the air. That is, a substance that prevents oxidation. Vitamin E prevents oxidation of unsaturated lipids within cells. **Vitamin E can be called a naturally occurring antioxidant**. In addition, vitamin E prevents degenerative changes in muscles and liver.

Deficiency Symptoms

Vitamin E deficiency gives rise to **three disease conditions**. These include: **(1)** encephalomalacia (crazy chick disease), **(2)** exudative diathesis, and **(3)** nutritional muscular dystrophy. Since **encephalomalacia** is the most important among these, it will be described briefly.

Encephalomalacia is a nervous disorder. It is often seen in **birds between 2 and 3 weeks of age**. Symptoms include muscular weakness, muscular incoordination with frequent falling, rapid contraction and relaxation of the legs, paralysis, and death.

Postmortem Findings

In the brain, there are **petechial (small) haemorrhages on the surface of the cerebellum** (a part of the brain) (Fig. 112, 113). Such gross changes in cerebellum in association with appropriate symptoms **are almost diagnostic**.

Treatment

The usual treatment is administration of vitamin E through drinking water.

Prevention

The condition is prevented by ensuring the availability of adequate vitamin E. Selenium also has some preventive effect.

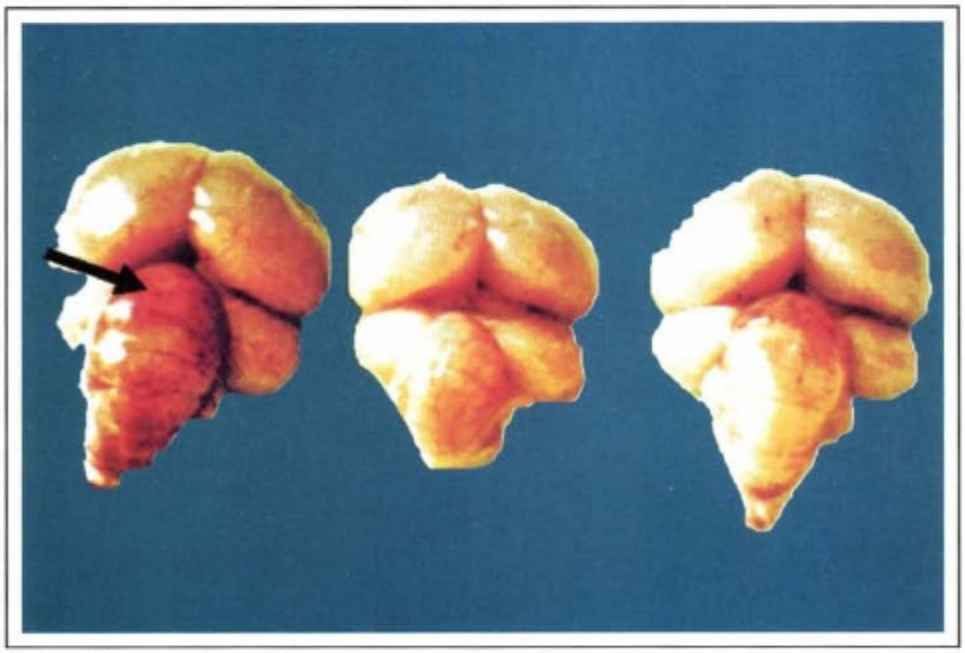

Fig. 112. **Vitamin E deficiency** (**nutritional encephalomalacia**) in **chickens**. Note haemorrhages on the cerebellum part of three brains. Haemorrhages are most marked on the cerebellum of left brain (arrow). **Such haemorrhages on the cerebellum are characteristic of vitamin E deficiency.**

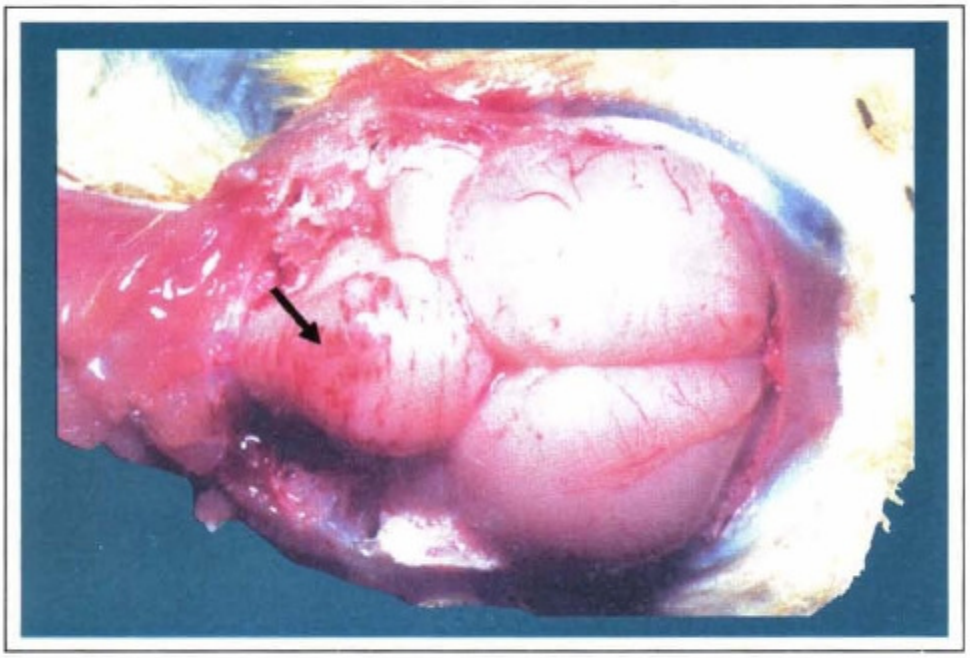

Fig. 113. **Vitamin E deficiency (nutritional encephalomalacia)** in another **chicken.** Note haemorrhages on the cerebellum portion of the brain (arrow). Such haemorrhages are characteristic of vitamin E deficiency.

7 METABOLIC DISEASES

GOUT

Gout is a metabolic disease characterized by **deposition of urates** on the surfaces of various internal organs, or various joints. **It is a very common finding** during postmortem examination of poultry, and can be a source of great economic loss to the farmer. Birds usually die from kidney failure. Both in broiler and layer chicks, gout can be seen from 4th day onward and can be a serious problem between 2nd and 3rd week. In layers, though relatively rare, gout can be seen even up to 50th week, and is mostly of nutritional origin (see 'urolithiasis).

Gout occurs in **two forms**: 1. **Visceral**, and 2. **Articular.**

Causes

1. Lack of water, or inadequate supply of water (**dehydration**).
2. Inadequate number of drinkers, or even just overcrowding.
3. Excess of dietary calcium, or calcium:phosphorus imbalance, or low intake of phosphorus in relation to calcium.
4. Increased intake of protein.
5. Vitamin A deficiency.
6. Excessive amount of salt (sodium chloride).
7. Infection with certain kidney damaging strains of infectious bronchitis virus in young chickens.
8. Fungal toxins (mycotoxins), such as oosporein and ochratoxin.
9. An electrolyte excess or deficiency.
10. Treatment with sodium bicarbonate.

Postmortem Findings

1. Presence of white chalky deposits on the surfaces of heart, liver, kidneys, proventriculus, and lungs. These deposits are seen as **white chalky coating** (Fig.114, 115).
2. The chalky deposits first begin on the heart and then spread (Fig. 116, 117).
3. Kidneys are swollen, congested, and typically gouty (greyish white in colour) (see Fig. 121).
4. One or both ureters may be distended with white material.
5. In the **articular gout**, when joints are opened, the tissue surrounding the joints is white due to urate deposition (Fig. 118).

Diagnosis

Can be done easily from the typical postmortem findings.

Treatment and prevention

1. Give plenty of water containing electrolytes. Make sure that there are at least 30 drinkers per 1000 birds. Correct overcrowding, if any.

2. Reduce the high level of protein, increase maize, and formulate the feed accordingly for a few days to get over the problem.
3. In the laying hens, avoid feeding high levels of calcium before sexual maturity. No more than 1% calcium should be fed to Leghorn birds before maturity.
4. Use urine acidifiers, such as methionine hydroxyl analogue (MHA), or even supplemental DL-methionine.

Fig. 114. **Gout** (**visceral**) in **three 11-day-old broiler chicks**. Note extensive deposition of urate on the surfaces of heart, liver, and other organs. The deposits appear as **white chalky coating**. This is characteristic of the visceral gout.

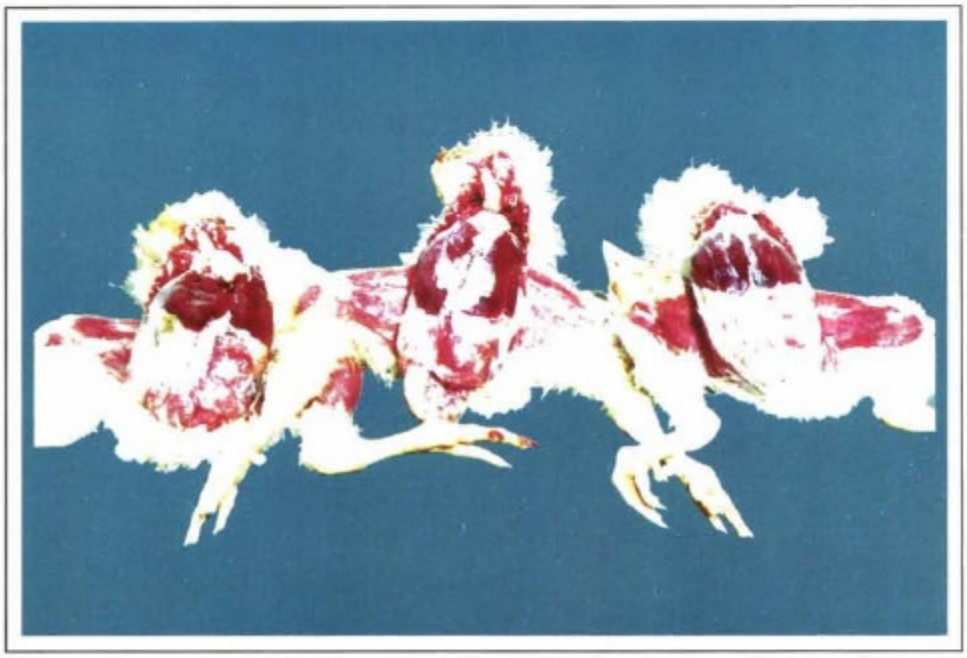

Fig. 115. **Gout (visceral)** in another set of **three 11-day-old broiler chicks**. Note urate deposits on the heart, liver and other organs are more extensive.

Fig. 116. **Gout (visceral)** in a **63-week-old layer chicken**. Note surface of the heart is covered by urate deposits. Liver and other organs are free of the deposition. **Urate deposition usually begins at the heart.**

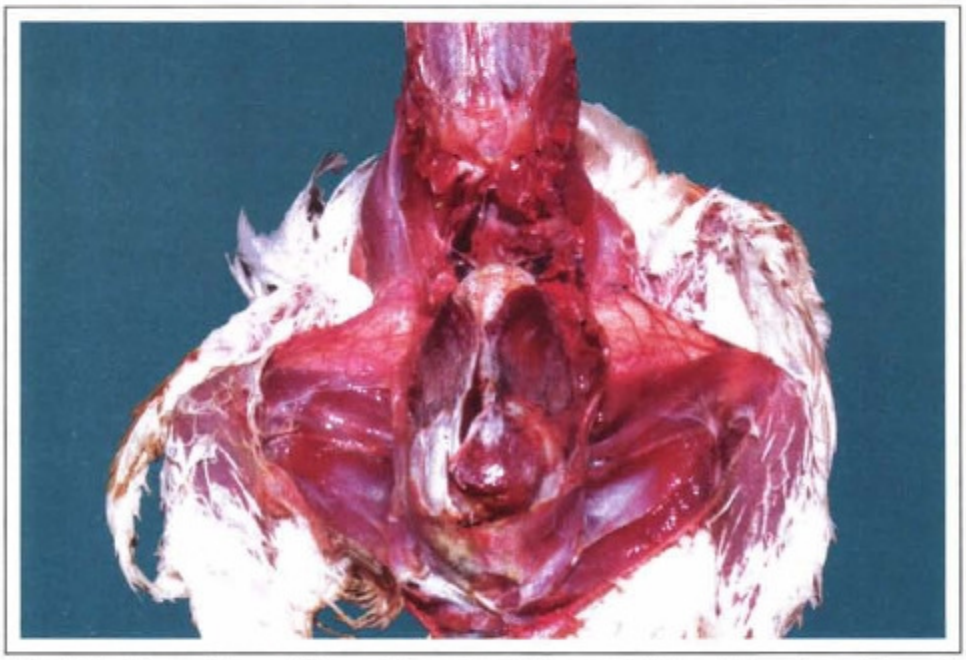

Fig.117. **Gout (visceral)** in a **65-week-old layer chicken**. Note deposition of urate on the surface of heart, liver, and other organs.

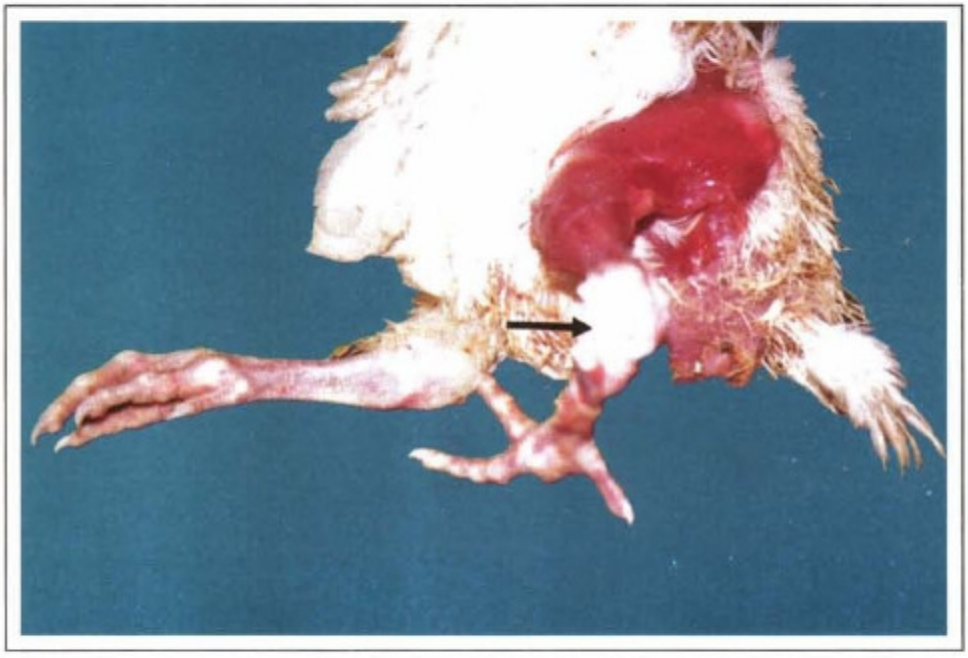

Fig. 118. **Gout** (**articular**) in a **14-day-old broiler chicken**. Note massive deposits of urates around the left hock joint (arrow). (The joint is cut open). As a result, the joint is enlarged and foot deformed. Further note that the other small joints of both the feet also show deposition of urates. Such joints have turned white.

UROLITHIASIS

Urolithiasis in chickens is **formation of urinary calculi in the urinary tract, mainly kidneys**. The condition is characterized by **gout in the kidneys** and abdominal organs, marked reduction in the size of one or both kidneys (atrophy) and distended ureters, often containing calculi (uroliths). In recent years, outbreaks of mortality in the laying hens have been caused by urolithiasis. Urinary calculi may cause sudden death by plugging ureters. However, this is secondary to kidney damage.

Urolithiasis is primarily a disease of laying flocks.

Causes

1. Kidney damage.
2. Excess dietary calcium, particularly if associated with low available dietary phosphorus fed to growers.
3. Infection with infectious bronchitis virus (nephrotropic strains).
4. Water deprivation.
5. Certain mycotoxins which damage kidneys, such as ochratoxin and oosporein.

Symptoms

Mortality in affected flocks may exceed 20% for several months. In severe cases, it may exceed 50%. Laying hens die suddenly and may be in good condition and in full lay. Or, they may have small pale combs, and white pasting on feathers surrounding cloaca.

Postmortem Findings

1. Kidneys show diffuse urate deposits, and may be greatly swollen (Fig. 119, 120, 121).
2. Kidneys may be atrophied and ureters dilated.

The dilated ureters often contain white irregular urinary calculi. These calculi are composed of compact masses to fine crystals of **calcium sodium urate**.

Diagnosis

Urolithiasis can be diagnosed from the typical postmortem finding of renal gout (kidney gout).

Treatment

Dietary acidification with ammonium chloride, ammonium sulphate, or methionine decreases incidence of urinary calculi and improves condition of the kidneys. However, use of ammonia chloride is not a practical treatment because it causes increased water consumption, urine flow, and faeces moisture. Ammonium sulphate is more effective than two forms of methionine.

Control

1. Ensure availability of adequate water.
2. Control mycotoxicosis by adding toxin binders in the feed.

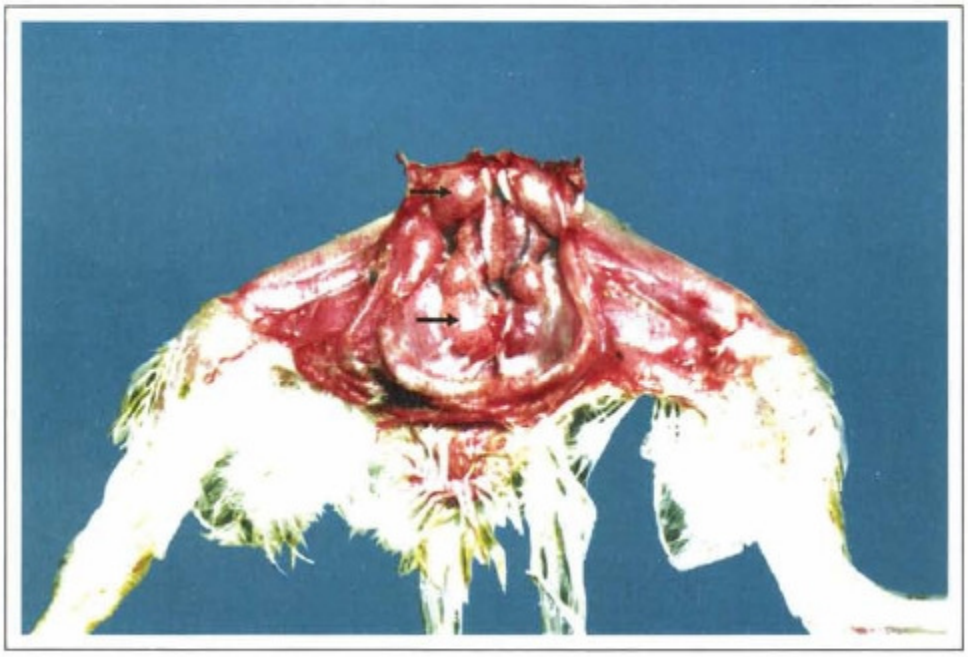

Fig. 119. **Urolithiasis** in a **33-day-old broiler chicken**. Note kidney lobes are enlarged and gouty from urate deposition (arrow). This is renal gout (i.e., kidney gout).

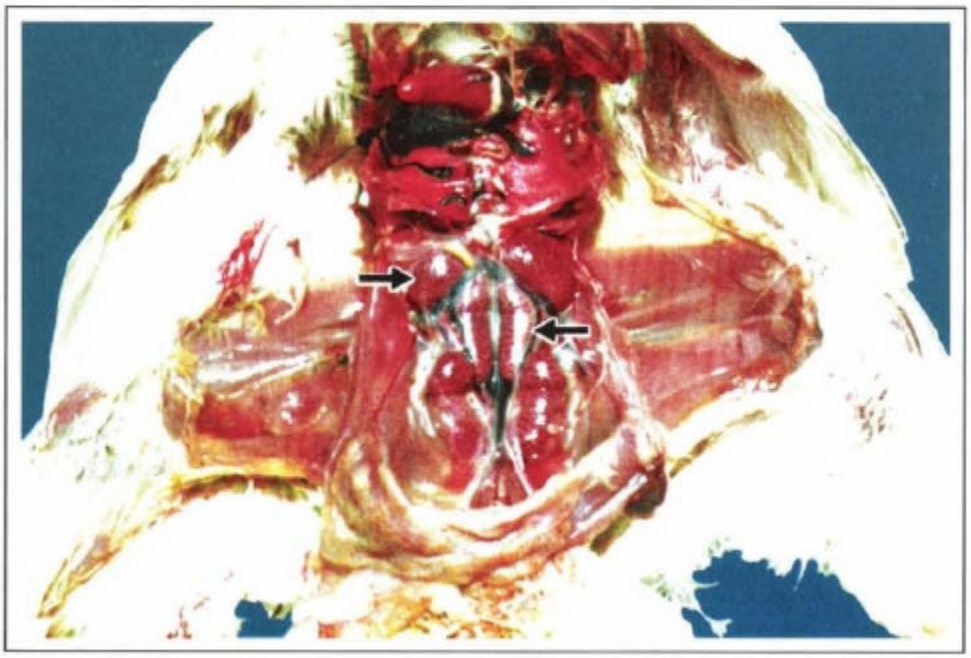

Fig. 120. **Urolithiasis** in a **44-day-old broiler chicken**. Note kidney lobes are enlarged and gouty from urate deposition (arrows). This is renal gout (kidney gout).

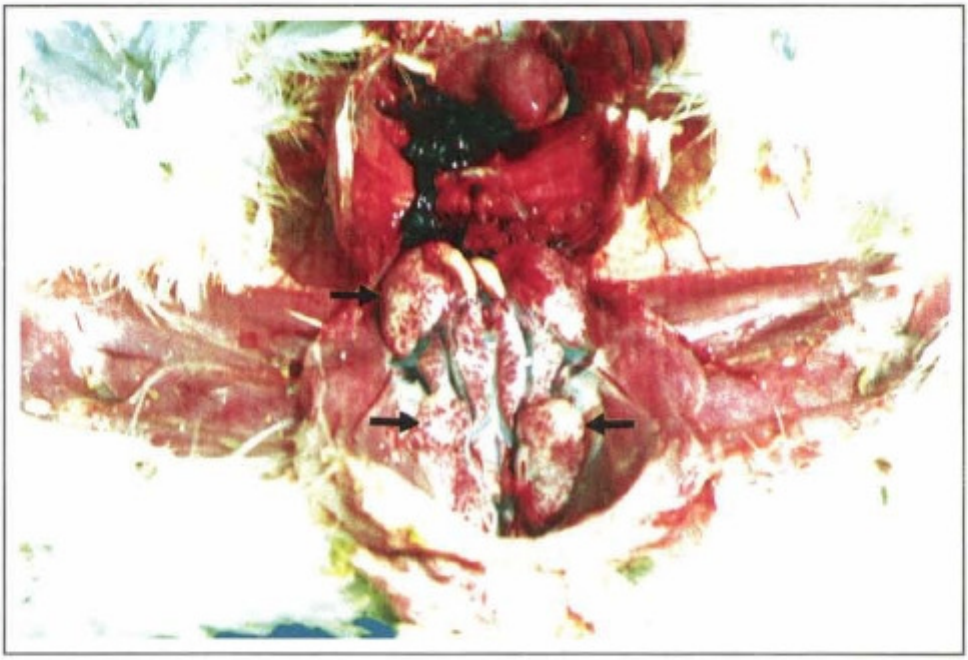

Fig. 121. **Urolithiasis** in a **63-week-old layer chicken**. Note kidney lobes are enlarged, pale, and gouty from accumulation of urates (arrows). This is renal gout (kidney gout).

ASCITES (Water Belly)

Ascites is an accumulation of fluid in the abdominal cavity of chickens. It is not an infection, but is caused by a series of complex events that affect the supply of oxygen for the tissues. **Ascites has emerged as a major source of economic loss in the broiler industry**. In extreme situations, up to 25% mortality is seen. **Ascites is confined to broilers.**

The modern broiler chicken is susceptible to ascites because of its rapid growth rate, high feed efficiency, and a large breast muscle mass, which require a high demand for oxygen. The metabolic rate of fast-growing broiler chicken is very high. Thus, an imbalance between oxygen supply and the oxygen required to sustain rapid growth rates and high food efficiencies, causes ascites in broiler chickens.

Causes

1. Rapid growth rate
2. Poor ventilation
3. Cold. It causes an increased demand for oxygen.
4. High energy diets
5. Overcrowding

6. Faulty brooding
7. Ammonia formation and dust
8. Sodium toxicity
9. Vitamin E/selenium deficiency
10. Mycotoxicosis
11. Stress

Symptoms

1. Sudden death.
2. Mortality is greatest after 28 days. The peak incidence of ascites occurs between 35 and 42 days of the growing period. However, cases can occur from as early as 15 days and onward.
3. Affected birds are smaller than normal and depressed, with ruffled feathers. This is because the growth stops.
4. They have a pale head and a shrunken comb. Birds may be reluctant to move, show difficult breathing, even panting.
5. **Severely affected birds show abdominal distension.**

Diagnosis

Diagnosis can be easily made from the typical postmortem finding of **abnormally distended abdomen**, containing fluid.

Postmortem Findings

1. **The abdomen is markedly distended with fluid** (Fig. 122, 123, 124, 125, 126, 127).
2. On opening, the abdominal cavity shows the presence of a large amount of clear yellow fluid. More than 300 ml of fluid, with or without fibrin clots, may be present.
3. The heart is greatly enlarged.
4. There may be accumulation of fluid under the covering of the heart (pericardium).
5. The liver may be swollen and congested, or firm and shrunken.
6. The lungs are extremely congested and oedematous. The intestines are severely congested.

Note: **Some birds may die before ascites develops**. That is, not all birds that die from ascites show accumulation of fluid in the abdominal cavity.

Treatment

1. There is no effective treatment. Once birds show symptoms, death occurs fairly quickly.
2. Frusemide, a drug that increases the flow of urine, reduces mortality in ascites.
3. Vitamin C, vitamin E and organic selenium are effective in reducing mortality from ascites.

Control

1. Ascites can be prevented through feed restriction, or through the use of very low energy feed.
2. Ensure adequate ventilation.
3. In winter, prevent chilling, that is, sudden and severe exposure to cold.
4. Avoid dust and ammonia.
5. Minimize toxin contamination of the feed.
6. Increased vitamin E and organic selenium in the diet reduce mortality in ascites.

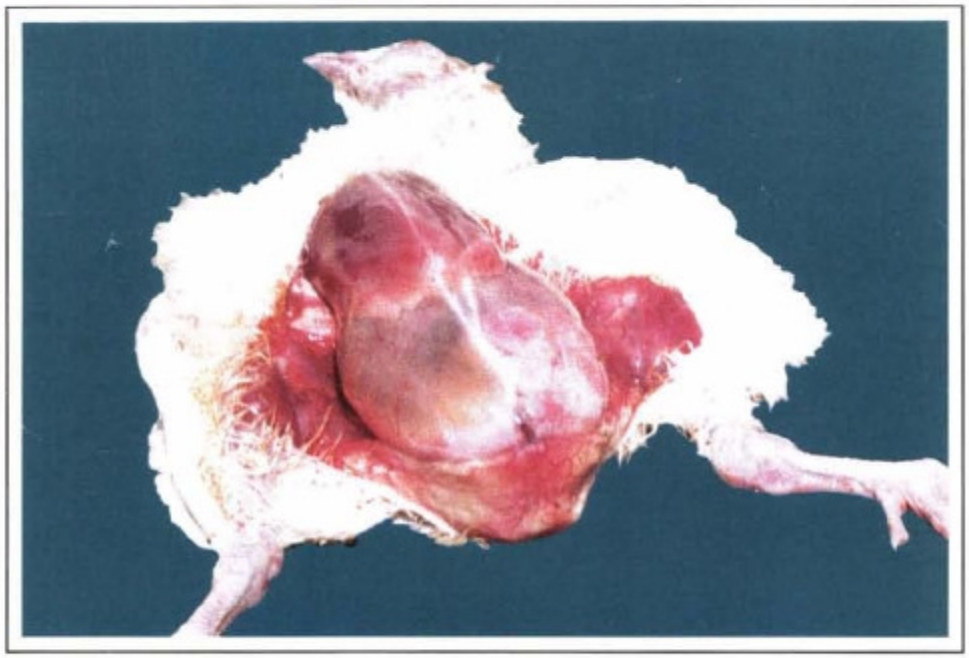

Fig. 122. **Ascites** in an **18-day-old broiler chicken**. Note abdomen is markedly distended with fluid.

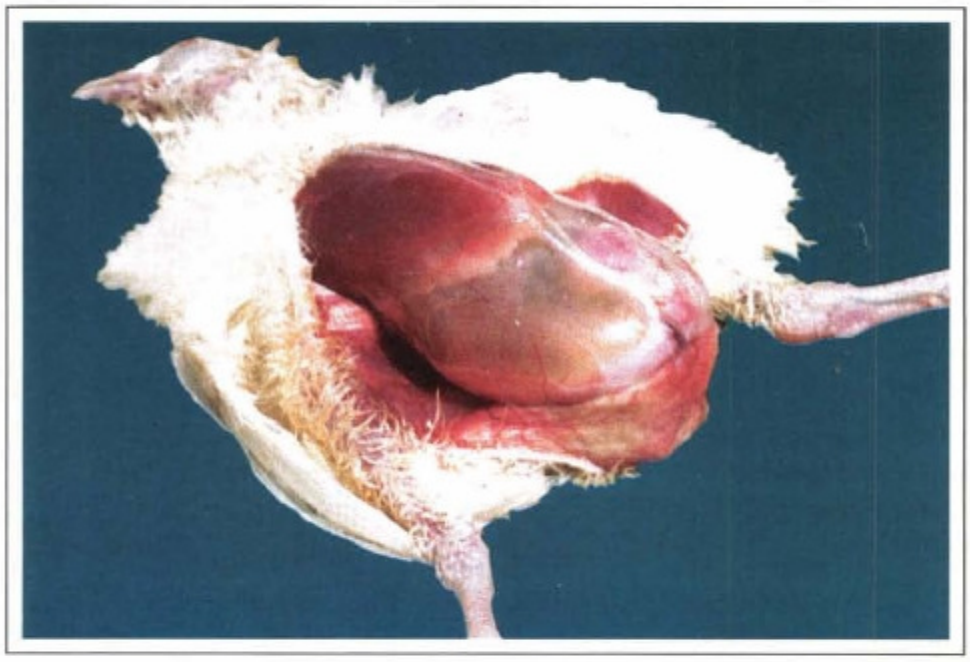

Fig. 123. **Ascites**. Side view of the **18-day-old broiler chicken** shown in Fig. 122. Note the swollen abdomen.

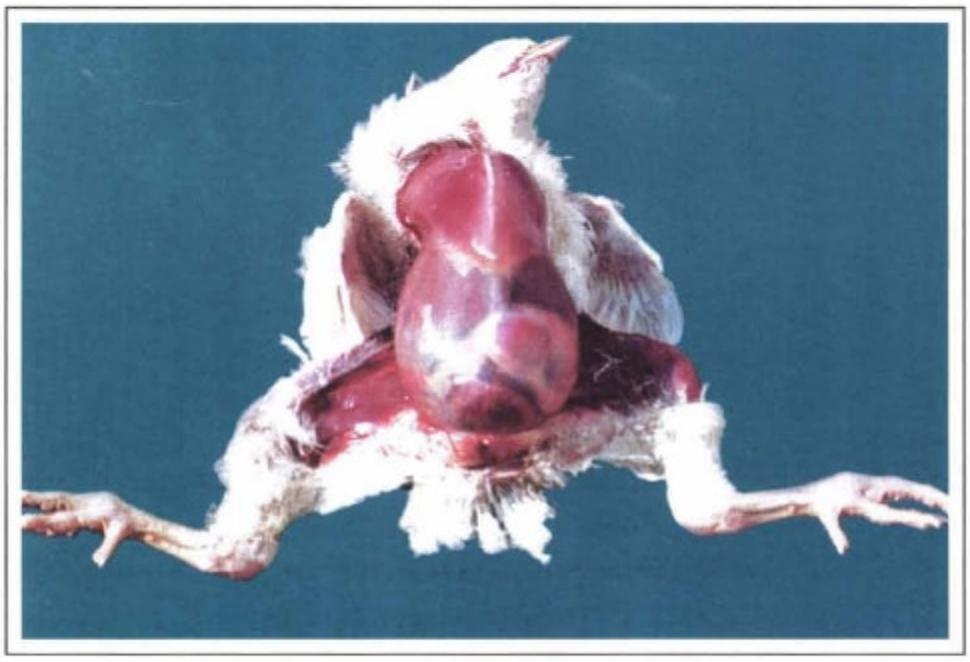

Fig. 124. **Ascites** in a **21-day-old broiler chicken**. Note swollen abdomen and somewhat congested blood vessels.

Fig. 125. **Ascites.** A close-up side view of the **21-day-old broiler chicken** shown in Fig. 124. Note the swollen abdomen and distinctly visible congested blood vessels.

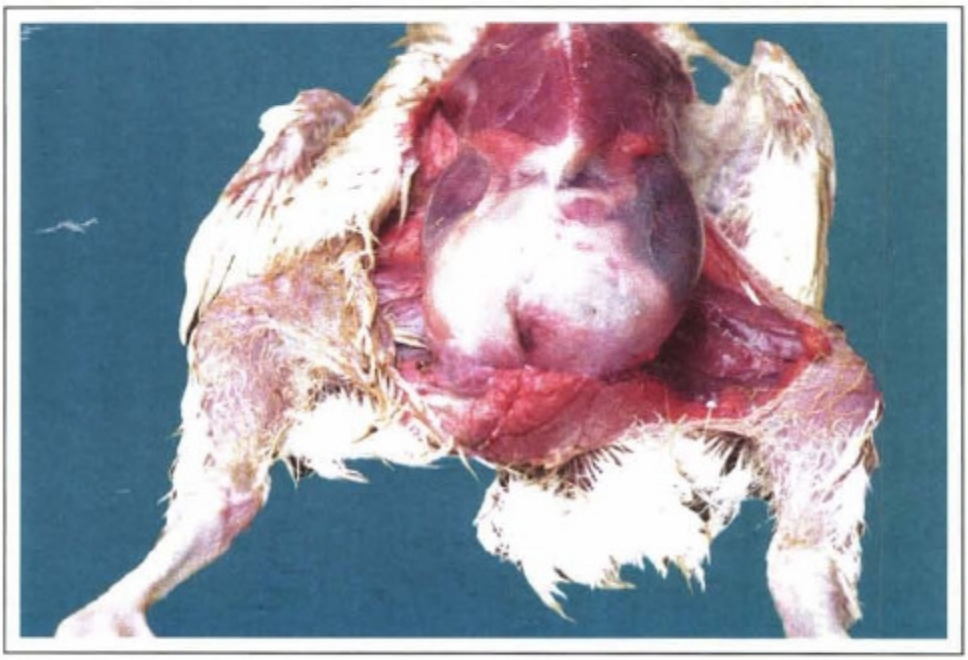

Fig. 126. **Ascites** in a **28-day-old broiler chicken**. Abdomen is markedly distended with fluid.

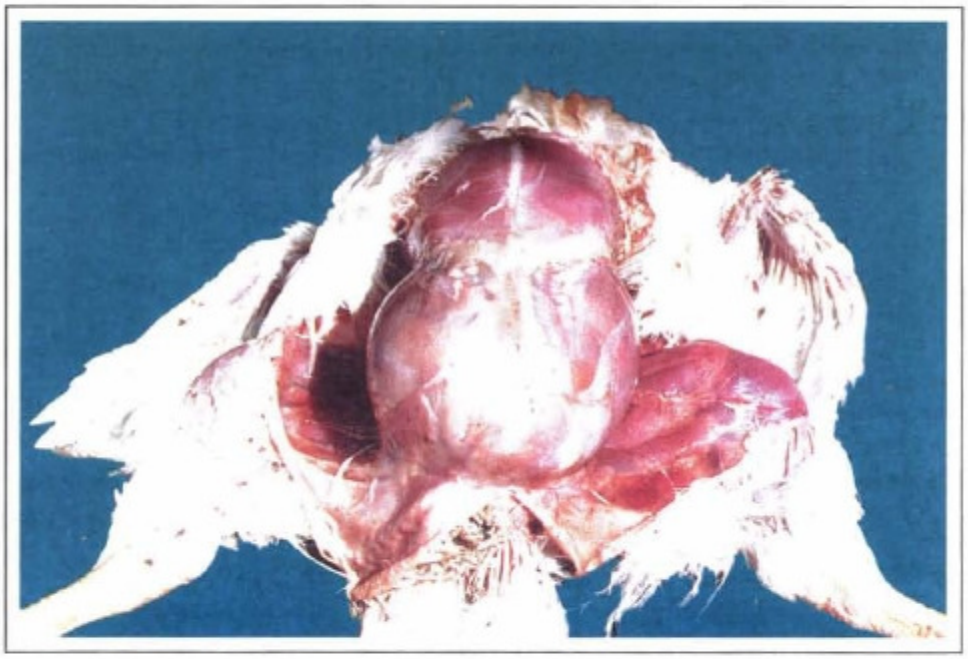

Fig. 127. **Ascites** in a **36-day-old broiler chicken** showing swollen abdomen.

RICKETS

Rickets is a condition in which there is a **failure or inadequate deposition of minerals in the bones**, that is, **calcium** and **phosphorus**. This results in the abnormalities of shape and structure of bone. Rickets causes increased bone flexibility and deformity, resulting in **lameness**. It is usually seen in rapidly growing young chicks. **Birds under four weeks of age** are most susceptible, but the condition may occur at any age.

Causes

1. Calcium, phosphorus and/or vitamin D3 deficiency, or imbalances.
2. Faulty mixing of feed ingredients.
3. Intercurrent diseases influencing vitamin D3 and mineral deposition.

Symptoms

1. Affected chickens show poor growth, symptoms of muscular incoordination, stiff-legged gait (walking), and **progressive lameness** (Fig. 128, 129, 130).
2. The hock joints are enlarged. The beak and shanks (leg bones) in young chicks are soft and flexible.
3. As the condition progresses, feathers become ruffled, and there is loss of condition. Morbidity (number of birds affected) in an untreated flock may reach 100%, and mortality (deaths) up to 50% has been observed.

Postmortem Findings

1. The bones do not break with a sudden sharp sound, because they become **soft and rubbery** from poor mineralization (Fig. 131). The beak and shanks are soft and pliable.
2. In birds with severe rickets, the keel bones are not straight, but twisted, bent, or curved, **ribs are 'beaded'.** That is, heads of the ribs are enlarged as nodules (Fig.132, 133).
3. The legs are bent, and ends of the long bones enlarged.

Diagnosis

Typical postmortem findings are usually enough for field diagnosis.

Treatment

1. Administer vitamin D3 in the recommended dose 2-3 times through drinking water.
2. Replace the suspected feed with the known well balanced and properly mixed feed.

Control

1. Ensure adequate supply of vitamin D3 and calcium and phosphorus in 2:1 ratio in the feed.
2. Mineral premixes should be properly mixed for even distribution.

Fig. 128. **Rickets. 7-day-old chick** suffering from calcium deficiency. The chick was lame and unable to stand.

Fig. 129. **Rickets. Two 42-day-old broiler chickens** suffering from severe calcium deficiency. The birds are unable to stand, toes are curled inward, and bones are rubbery and easily bent.

Fig. 130. **Rickets**. A close-up picture of **one of the 42-day-old broiler chickens** (right) shown in Fig. 129. The bird has been suffering from severe calcium deficiency.

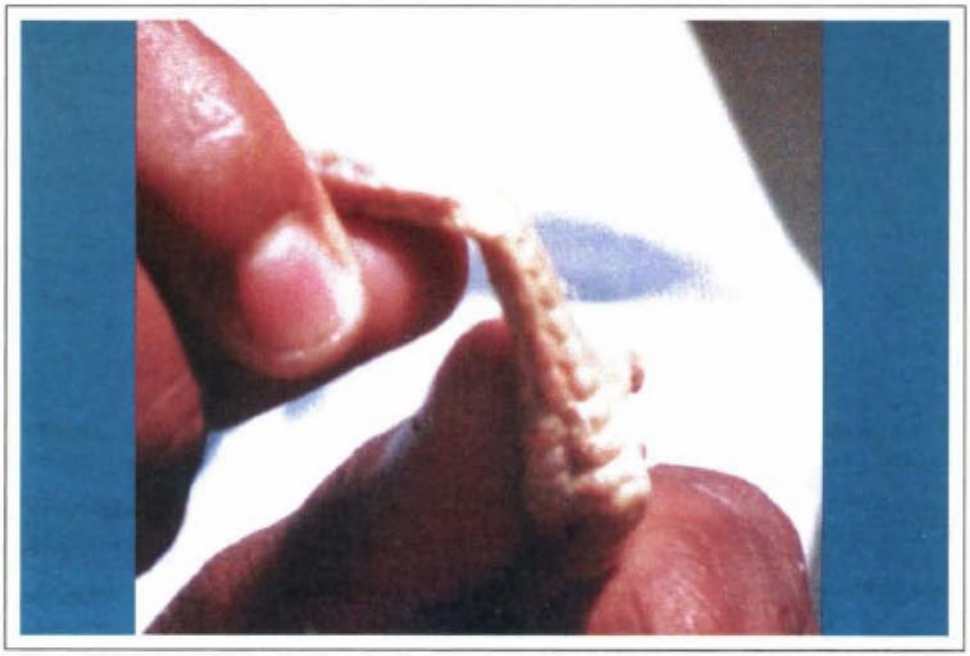

Fig. 131. **Rickets** in an **11-week-old grower chicken.** Note that the leg bone is rubbery and easily bent.

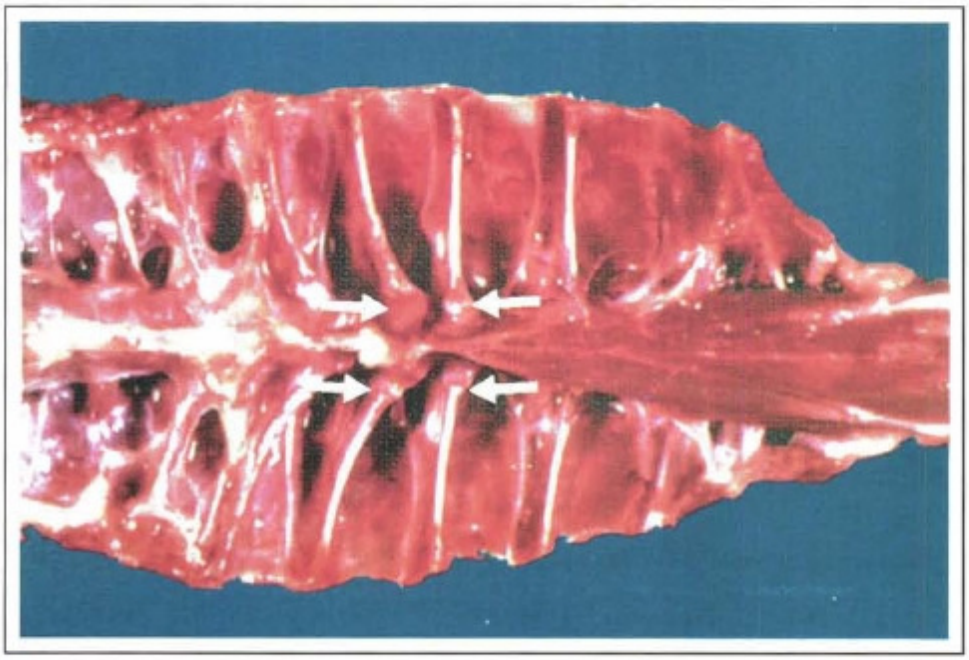

Fig. 132. **Rickets** in a **chicken**. Note enlargement at junctions of ribs with vertebrae (arrows), and also distortion of the ribs.

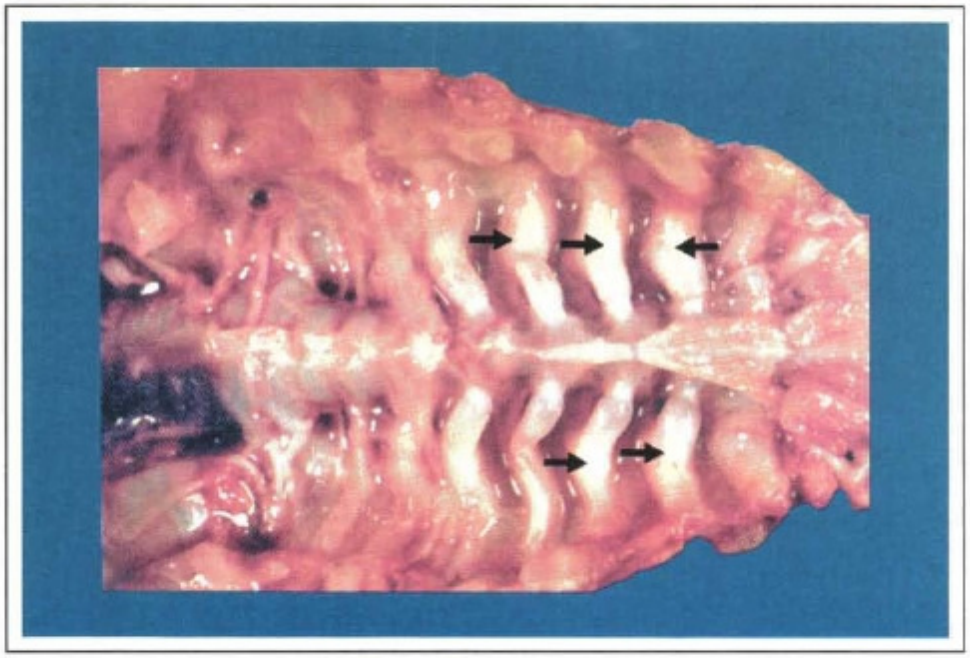

Fig. 133. **Rickets. 8-day-old broiler chick**. Note enlargement of the ribs to spine (costo-chondral) junctions (arrows). Ribs are short and thick.

FATTY LIVER-HAEMORRHAGIC SYNDROME

Fatty liver haemorrhagic syndrome (FLHS) is a metabolic disorder characterized by a **very fatty liver**, accompanied by **haemorrhages**. The condition is seen sometimes, particularly in hot weather, in older laying hens kept in cages. There is usually a drop in egg production in the affected flock. Death occurs occasionally and is due to massive liver haemorrhage.

Causes

1. High energy diets to increase egg production. Excessive consumption of high-energy diets in birds, whose exercise is restricted in cages, leads to fattiness. This is aggravated by hot weather.
2. Nutritional imbalances.
3. High temperature.
4. Deficiency of nutrients that mobilize fat from the liver, such as choline.
5. Stress, toxins, high egg production.

Symptoms

1. The condition is seen in good-laying flocks. Most birds appear in good physical condition.
2. There is a sudden drop in egg production, or the flock fails to reach a high peak of production.
3. Hens may be overweight, with large pale combs and wattles. Body weight increases from 20 to 25%.
4. There is usually an increase in mortality, and birds in full production are found dead with pale heads. Birds die specially following stress, such as hot weather, handling, transportation, or sudden fear or alarm. Mortality varies from 2 to 10%.

Postmortem Findings

1. Dead birds have large blood clots in the abdomen (Fig. 134). Usually the clots partly cover the liver and also originate from it.
2. **The liver is enlarged, fatty, is of light greyish brown to yellow colour** (Fig. 134), **and very friable.**
3. There is an increased fat content. The fat content is more than 70%. The normal range is between 25 to 50%. Death occurs only occasionally and is due to massive liver haemorrhage.

Diagnosis

Flock history and postmortem findings are usually adequate for field diagnosis.

Treatment

1. Various nutritional supplements have been tried with mixed results.
2. Addition of vitamin E, choline chloride, vitamin B12 and inositol in the feed has been shown to significantly improve the condition in some laying flocks.

Control

1. Reduce energy intake, either by feed restriction, or by lowering the metabolizable energy.
2. Addition of choline and inositol to the feed has given variable results.
3. Avoid use of mouldy feed or feed ingredients in the poultry diets.

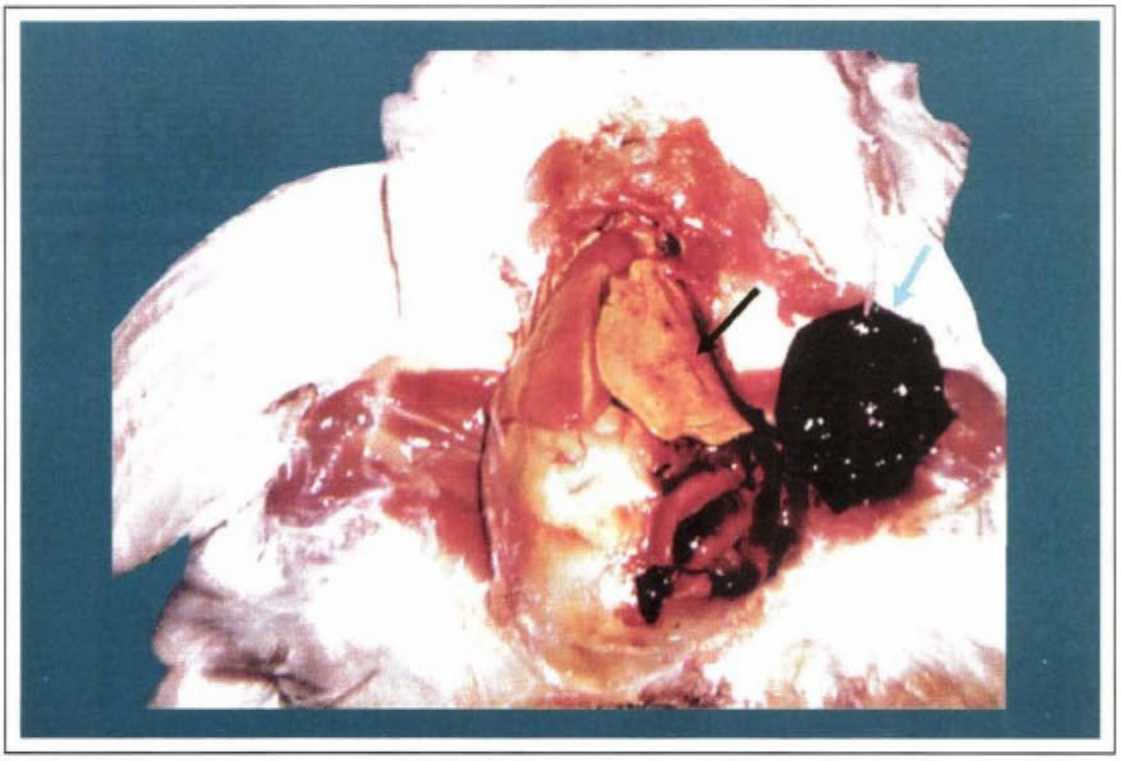

Fig. 134. **Fatty liver-haemorrhagic syndrome** (FLHS) in a **laying hen**. Note liver is pale (black arrow), has also ruptured, resulting in massive haemorrhage into the abdominal cavity. Blue arrow points to clotted blood.

8 MISCELLANEOUS DISEASES/ CONDITIONS

HEAT STRESS (Heat Stroke, Heat Prostration)

Heat stress is a condition in which the body temperature is so high that it interferes with normal body functions, and leads to extreme physical exhaustion and death. Heat stress is an important cause of mortality in our country. Summer season, associated with thick layer of feathers on the body of the chickens and absence of sweat glands, make them highly prone to heat stress.

Birds in production are particularly susceptible to high temperatures, when accompanied by increased humidity. As birds lack sweat glands, their only method of cooling is rapid respirations with open mouth. If the body temperature rises, birds become exhausted, and die from respiratory, circulatory or electrolyte imbalances.

Causes

1. High environmental temperature and hot dry winds, as in summer
2. Inadequate water supply
3. Inadequate ventilation
4. Overcrowding
5. Very low ceiling of the poultry house

Symptoms

1. Panting (open-mouth breathing)
2. Increased thirst
3. Reduced appetite
4. Fall in egg production
5. Reduced growth in broilers
6. Prostration (lying down with exhaustion), and death

Postmortem Findings

1. The carcass is severely dehydrated and congested.
2. The breast muscles are particularly affected. They lose their normal red colour (Fig. 135) and become pale to white, and present a **'cooked meat appearance'** (Fig. 136, 137, 138, 139, 140). This is characteristic of heat stress.

Diagnosis

Diagnosis is based on the pale to white 'cooked meat appearance' of the breast muscles.

Treatment

1. Immediately make available adequate drinking water.
2. Air movements, in the form of ventilation and fans, or sprinklers and foggers, facilitate heat loss.

3. Give vitamins in increased amounts, particularly vitamin E and C.
4. Give also electrolytes, as heat stress creates electrolyte imbalances.
5. Every effort should be made to increase circulation of air by running ventilation equipment, such as fans, at full capacity.

Control

1. Water sprinklers may be provided for the roof.
2. Foggers may be provided for inside cooling, without the birds themselves becoming wet.
3. The birds should be provided with adequate cool water in the summer and a ration with a reduced protein:energy ratio. Water increases bird's resistance to heat stress.
4. Avoid overcrowding. The stocking density should be reduced both on floors and cages to about 80% of that tolerated in cooler conditions.

Remarks: It is very important to control heat stress. Otherwise, apart from the direct losses, such a flock may also suffer from a number of infectious diseases on account of the **lowered resistance from heat**. This, in turn, will further increase the losses and ruin farmer's economy.

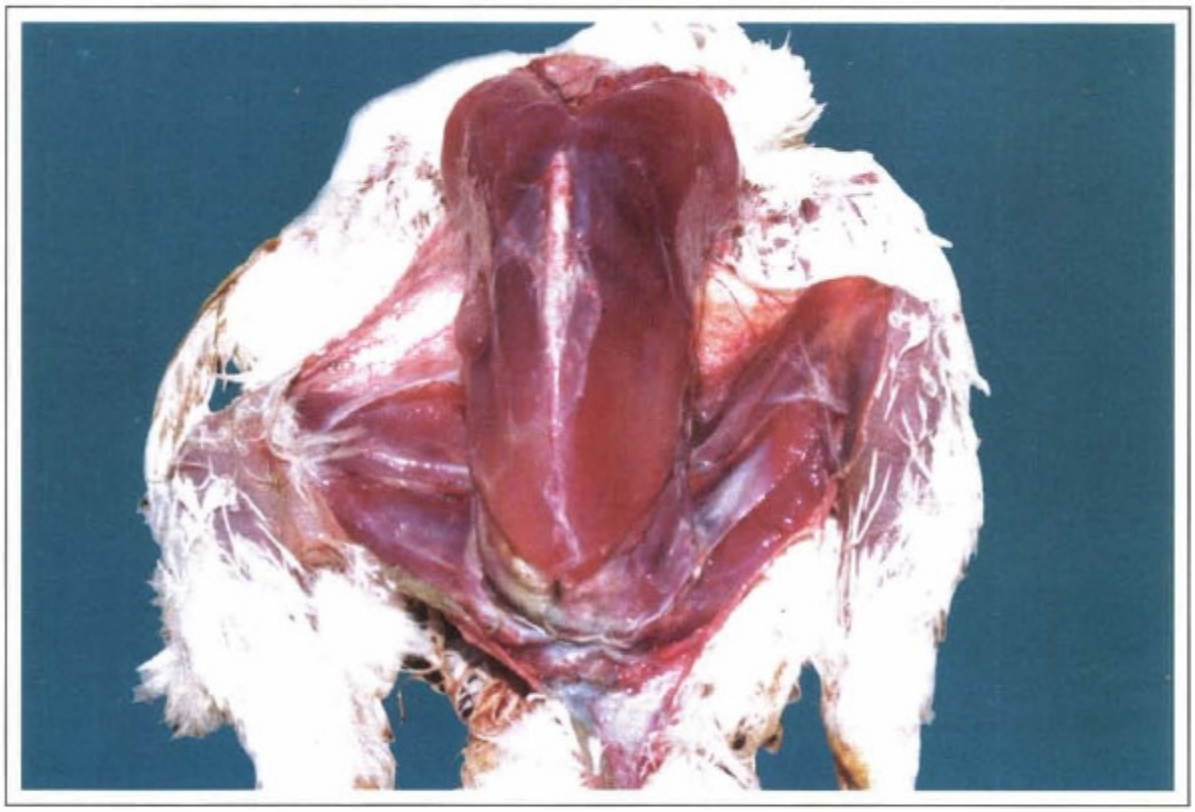

Fig. 135. **Normal breast muscle** in a **45-day-old broiler chicken**. Note **normal red colour** of the breast muscle and compare it with those affected with heat stroke in figures 136, 137, 138, 139, and 140.

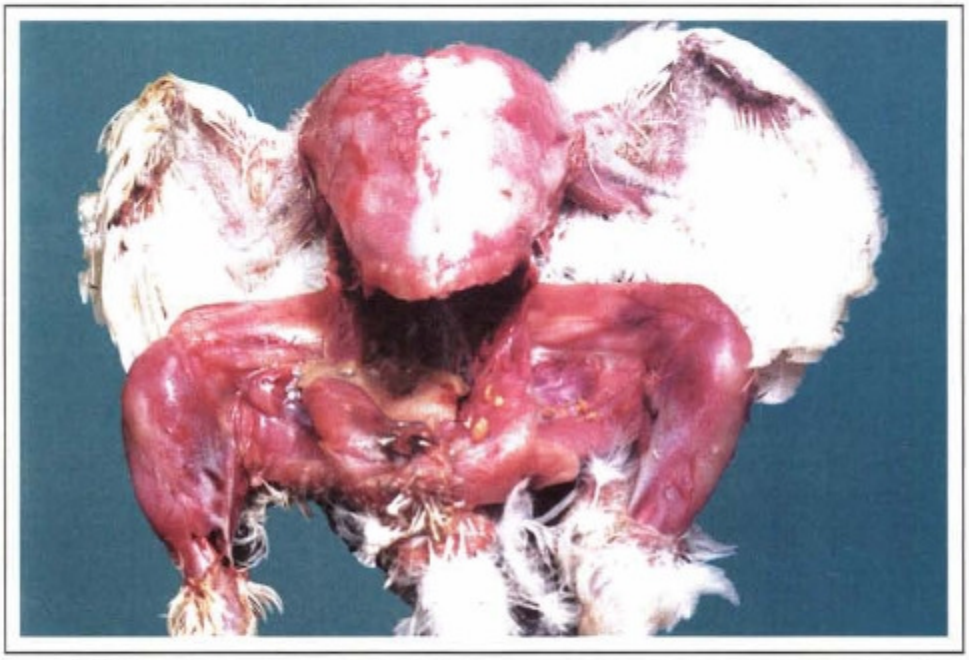

Fig. 136. **Heat stress** in a **36-day-old broiler chicken**. Breast muscles bear the maximum brunt of heat. Muscles on the left are relatively less affected; those on the right have turned white. Note their **cooked-meat appearance. This is characteristic of heat stress.**

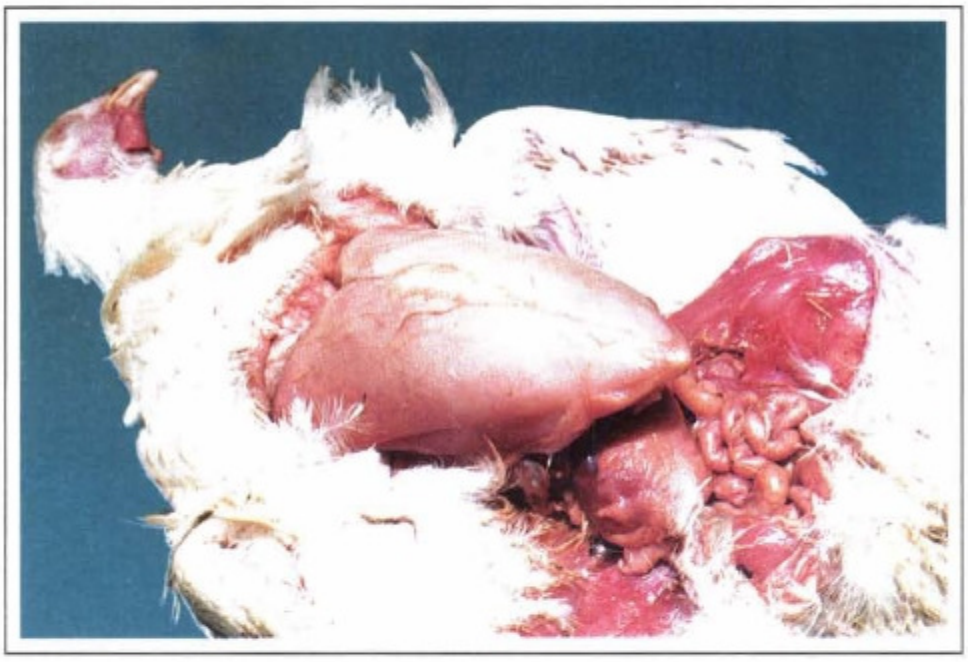

Fig. 137. **Heat stress** in a **44-day-old broiler chicken**. Note the entire breast muscle mass has turned white from severe heat stress, and appears like cooked meat.

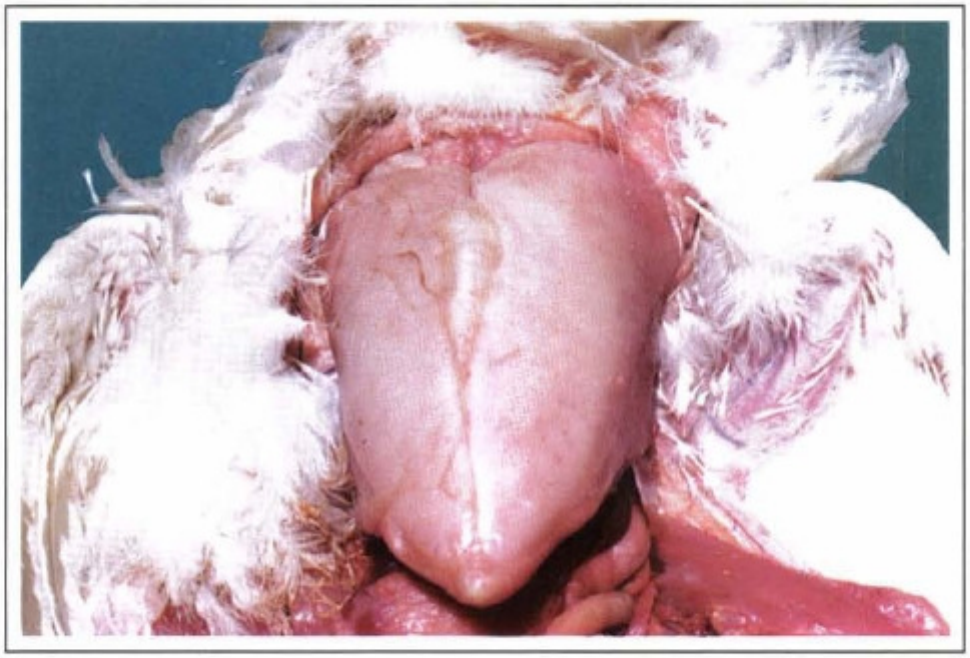

Fig. 138. **Heat stress** in a **47-day-old broiler chicken**. The entire muscle mass has turned white from heat stress.

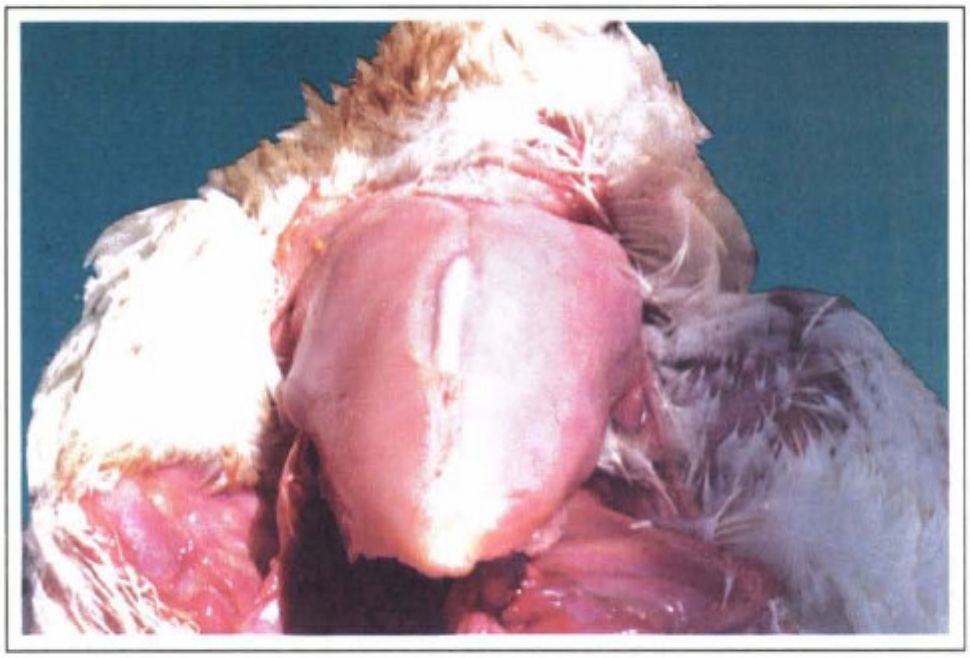

Fig. 139. **Heat stress** in another **47-day-old broiler chicken**. The breast muscle mass has turned white and exhibits a cooked meat appearance.

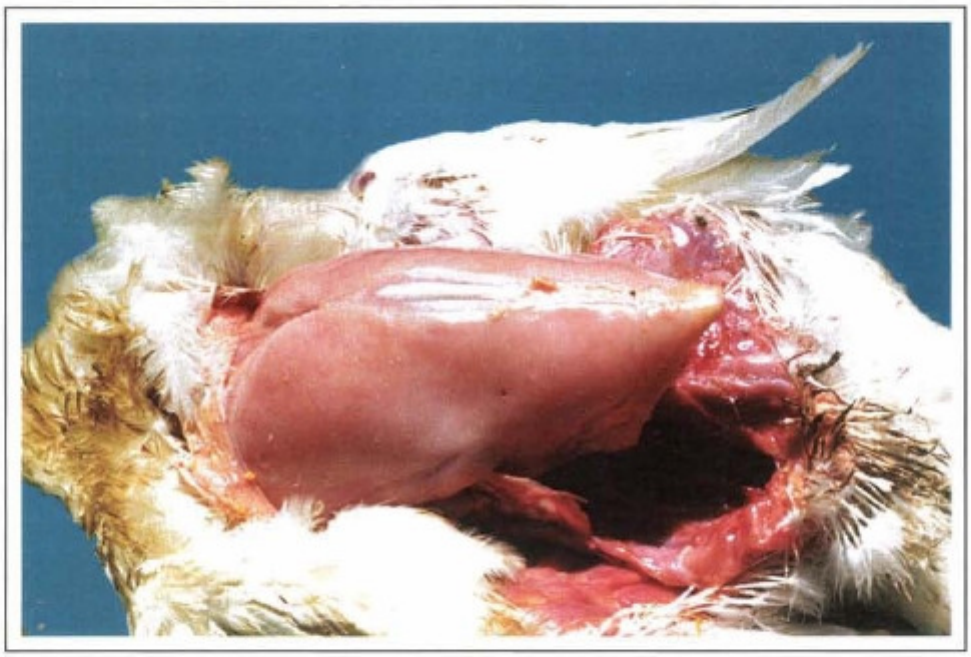

Fig. 140. **Heat stress**. Lateral view of the same **47-day-old broiler chicken** shown in Fig.139. The entire breast muscle mass has turned white and presents a cooked meat appearance.

FEMORAL HEAD NECROSIS

Disintegration (decomposition) of the head of the femur (long bone of the leg) in broilers is a very common postmortem finding, and an important cause of **lameness**. It also occurs in layers. The condition is also known as **'proximal femoral degeneration'.**

Causes

1. Femoral head necrosis is usually the result of a **bacterial infection**. The most commonly involved bacteria include **staphylococci**. Staphylococci are most commonly present in the poultry environment. They may be found in the litter, on feather, and on particles in the air of poultry houses. The other organisms involved include ***Escherichia coli*. Salmonella** may also be involved, though this is rare. Besides these three types of bacteria, viruses known as '**reoviruses**' may also sometimes be involved mainly in broilers between 4 and 8 weeks of age.
2. **Trauma** or **injury** may provide a focus for bacterial infection.

Symptoms

Femoral head necrosis occurs **in broilers** usually between 25 and 50 days of age. The affected birds show a characteristic trembling gait (way of walking). They often use a wing for support while moving and also while sitting down.

Postmortem Findings

The femoral head usually separates from the shaft by a fracture through the neck when hip joints are separated. **Both head and proximal portion of the femur show marked degeneration** (Fig. 141, 142, 143).

Diagnosis

The condition can be easily diagnosed from the characteristic postmortem findings.

Treatment/Control

It is advisable to mix an effective broad-spectrum antibiotic in the feed.

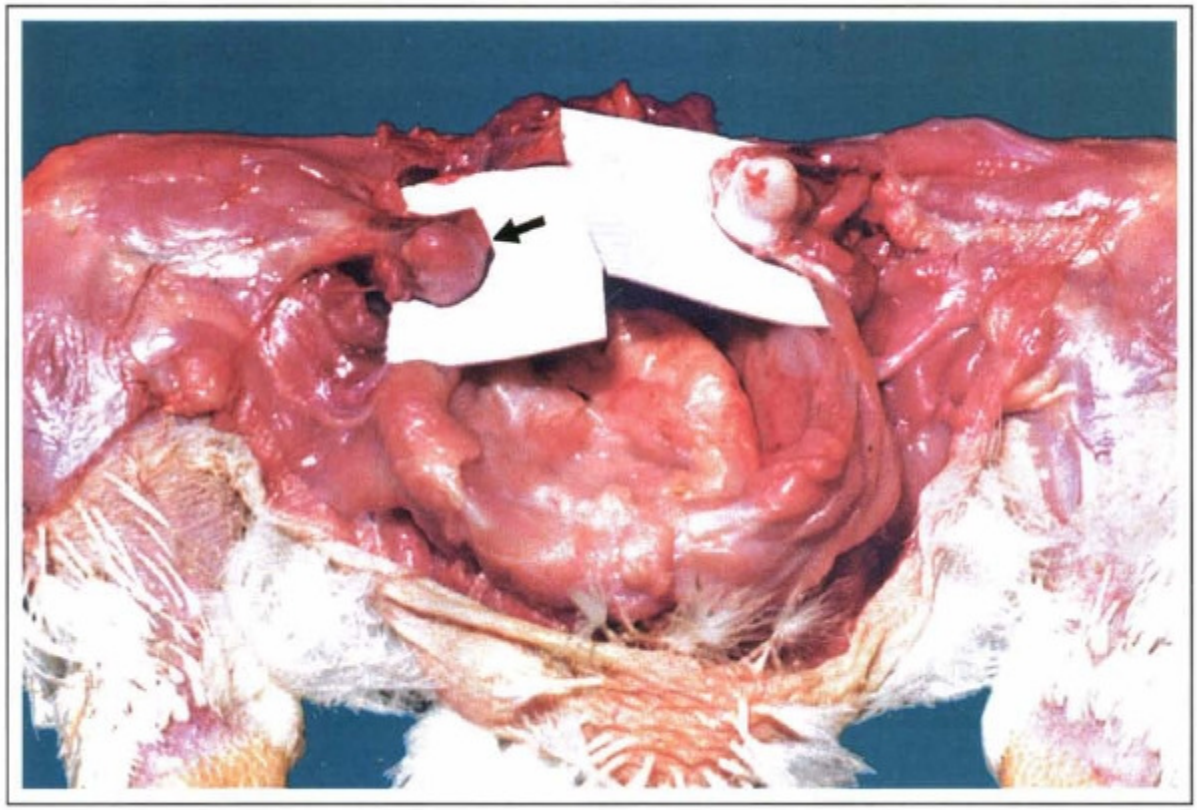

Fig.141. **Femoral head necrosis** in a **35-day-old broiler chicken**. Note that left head and proximal portion of the femur show some degeneration (arrow). **This is an early stage**. The right head is normal.

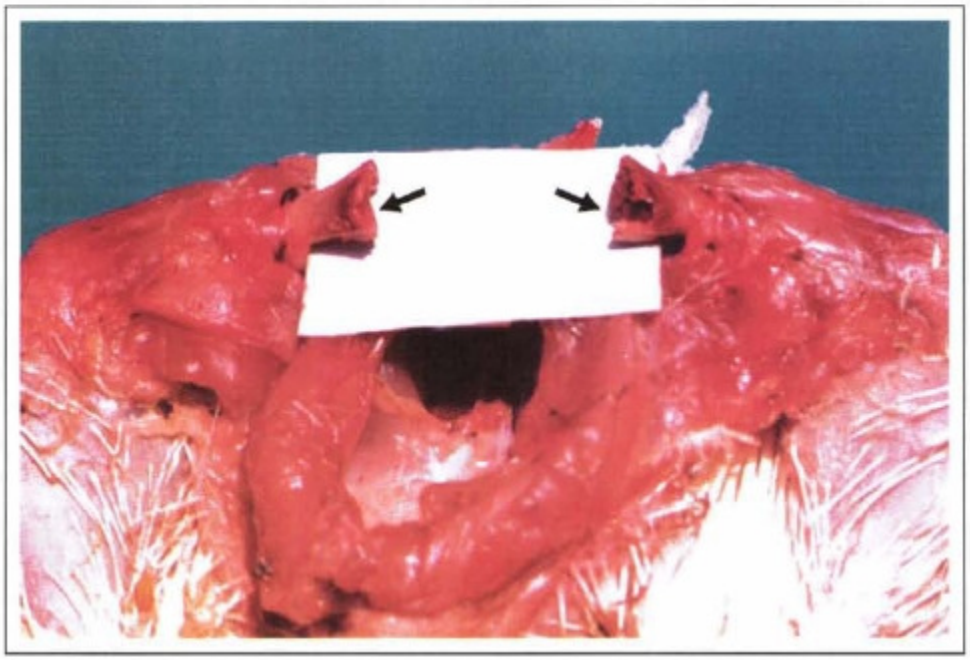

Fig. 142. **Femoral head necrosis** in another **35-day-old broiler chicken**. Note that both heads, and also the proximal portions of the femurs, **show marked degeneration** (arrow). As a result, when the legs were spread, the heads had separated from the shaft.

Fig. 143. **Femoral head necrosis** in an **11-week-old grower chicken**. Note head of the femur shows marked degeneration.

BUMBLE FOOT

Bumble foot is an abscess in the foot pad. It is a common infection in mature chickens. Bumble foot is caused by staphylococcal bacteria. The undersurface of the foot is first affected (Fig. 144), and the lesion may then spread to involve the whole foot. Bumble foot then leads to massive swelling of the foot (Fig. 145), and causes lameness.

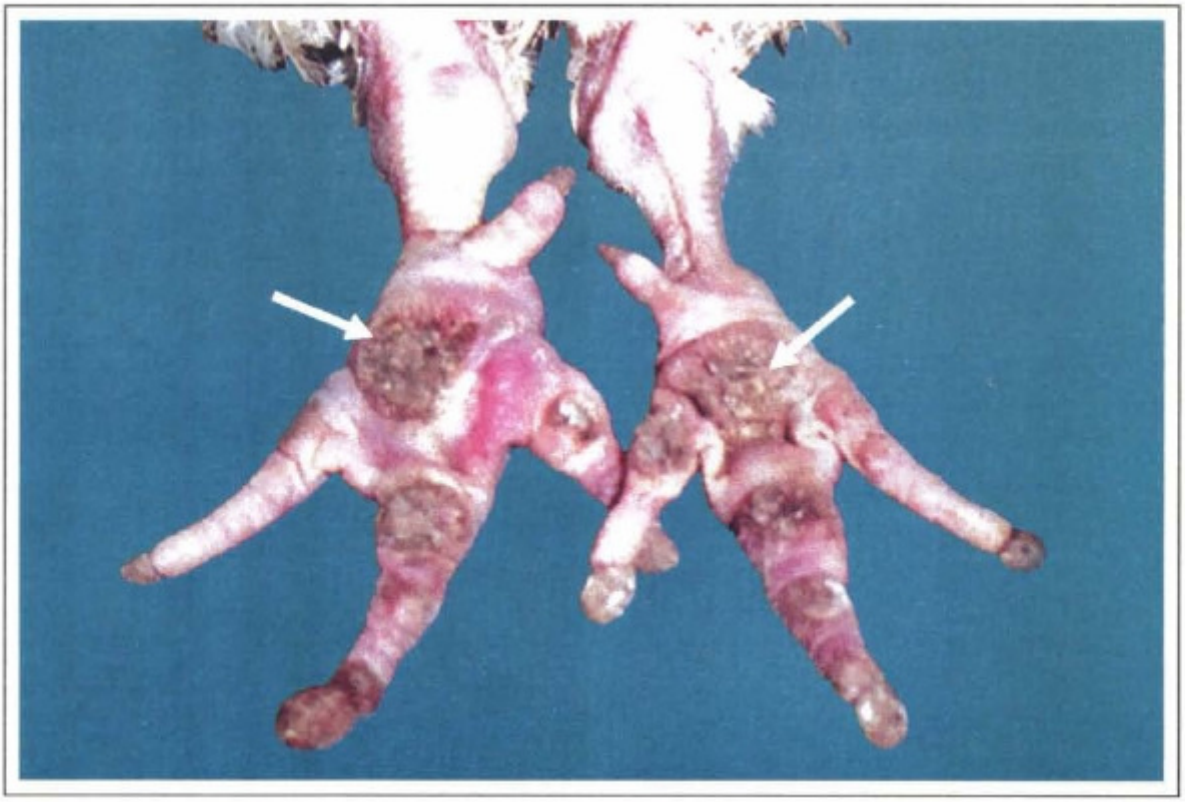

Fig. 144. **Bumble foot** in a **34-day-old broiler chicken**. Note small abscess-like lesions in both the legs, mainly in the centre of foot pads (arrows).

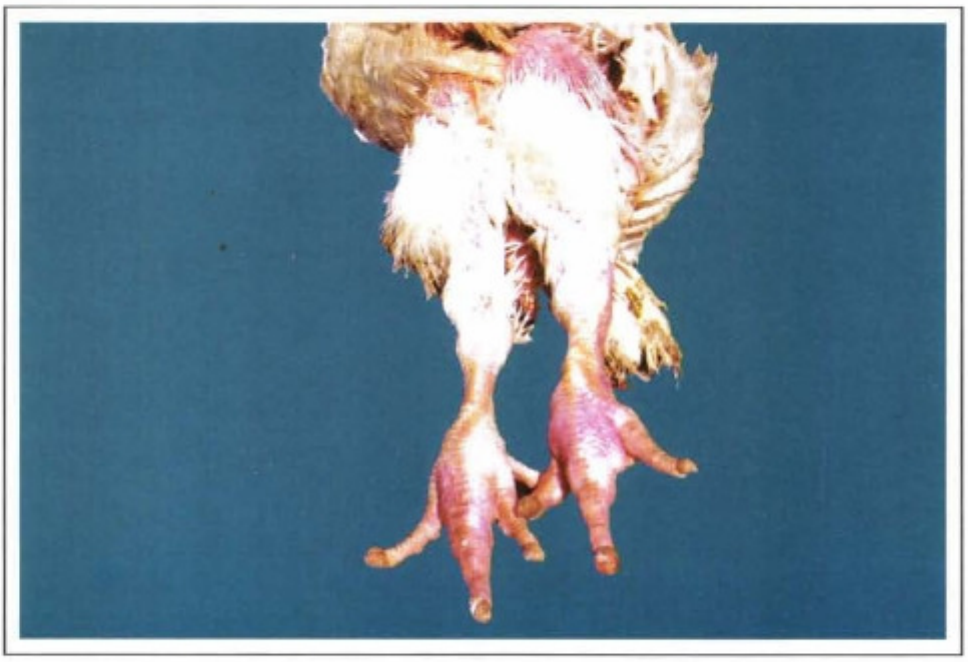

Fig. 145. **Bumble foot** in the same **34-day-old broiler chicken** shown in Fig. 144. Note that changes have spread to involve the whole foot. Both feet are inflamed, red, and markedly swollen.

EGG-BOUND CONDITION

This is a condition in which an egg is lodged in the cloaca/oviduct, **but cannot be laid**. Cloaca, in birds, is a common chamber at the end of the alimentary tract into which the digestive, urinary, and reproductive tracts open.

Causes

1. Inflammation of the oviduct.
2. Partial paralysis of the muscles of the oviduct, or
3. Production of an egg so large that it is physically impossible for it to be laid.

However, the exact cause of the condition is not known. Young hens laying an unusually large egg are more prone to the problem.

Postmortem Findings

At postmortem, an egg is found lodged in the cloaca/oviduct and fails to be laid (Fig 146, 147). This leads to bird's death. The egg is usually large.

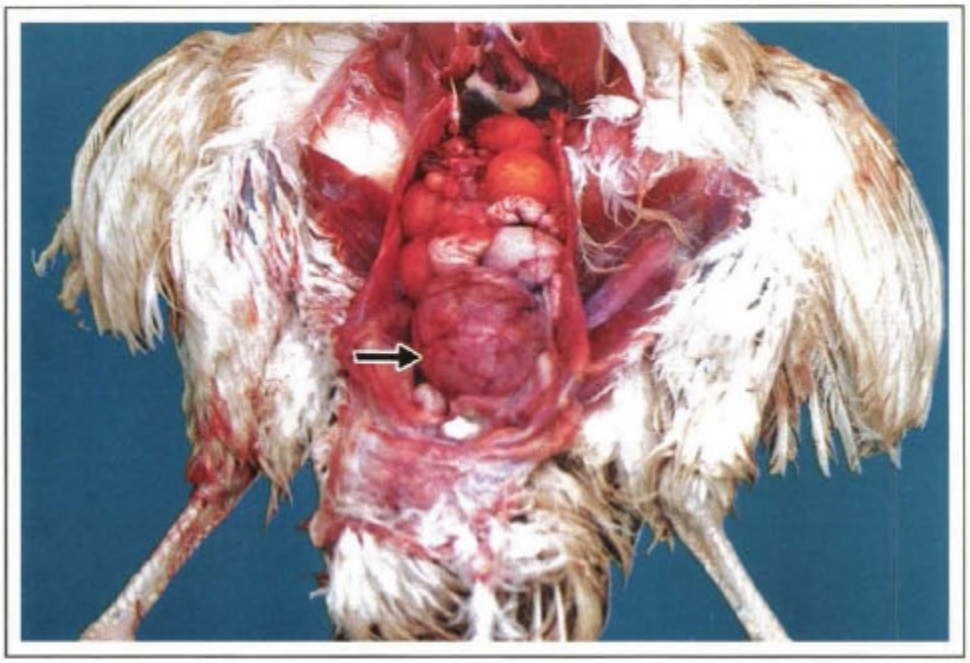

Fig. 146. **Egg-bound condition** in a **65-week-old layer chicken**. Note that egg is lodged in the oviduct/cloaca, but could not be laid (arrow).

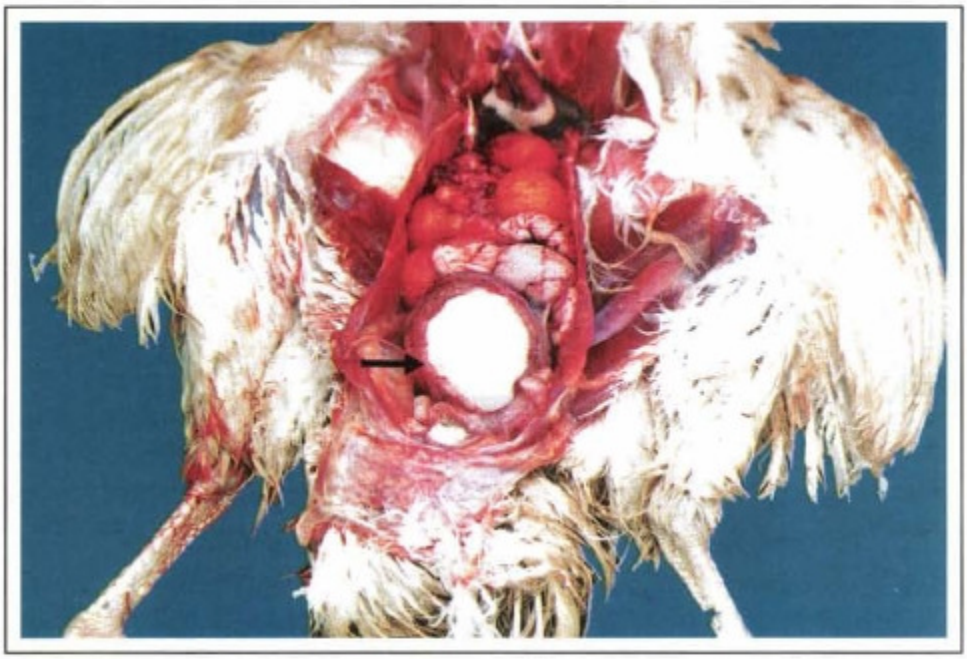

Fig. 147. **Egg-bound condition** in the same **65-w. eek-old chicken** shown in Fig. 146. The oviduct/cloaca has been cut open to reveal the eggshell (arrow).

AMMONIA EXPOSURE

Ammonia is a gas. It is **highly soluble in water** when it comes in contact with the inner lining (mucous membrane) of the respiratory tract following inhalation, or in contact with the eyes. It dissolves in the mucus (liquid produced by the mucous membrane) and produces a highly irritating chemical substance **ammonium hydroxide**, which produces very damaging effects.

Ammonia production in a poultry shed depends on:

1. **Inadequate ventilation**. Improper ventilation can lead to wet litter. Ammonia fumes develop in wet litter and droppings.
2. **Wet litter (increased moisture).** When litter moisture is between 20 - 25%, ammonia is usually not produced. Ammonia production starts when moisture exceeds 30% and increases further as the temperature rises. **Wet litter exposes to more coccidiosis.**

Harmful Effects of Ammonia

1. Ammonia damages the respiratory system and thus predisposes to infections, such as ***E. coli*** infection, infectious coryza, and Ranikhet disease.
2. Decreases growth rate.
3. Ammonia also reduces body weight and feed efficiency.
4. Ammonia reduces feed consumption and growth rate. Egg production could also be affected.

Ammonia, in concentration of 150 ppm and above, sometimes causes **'ammonia blindness'** in broilers, also known as **'ammonia burn'** (Fig. 148). It is caused by ammonia fumes coming out from poorly managed litter in an ill-ventilated house.

Symptoms

Affected birds keep their eyes closed, stand depressingly with ruffled feathers, and are reluctant to move. The eyelids are swollen (Fig. 148). The condition usually affects both eyes. Affected birds do not eat and become weak.

Prevention

1. Ensure proper ventilation.
2. Ensure proper litter management.

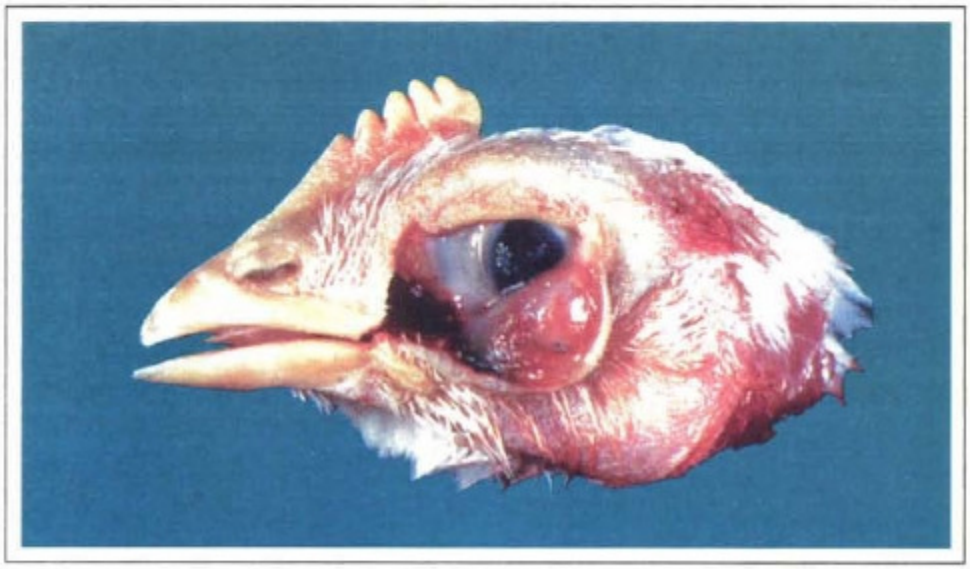

Fig. 148. **Ammonia exposure**. Note inflammation of the eye ('keratitis' of cornea and 'conjunctivitis' of conjunctiva) following exposure to high levels of atmospheric ammonia.

SWOLLEN HEAD SYNDROME

Swollen head is a condition that affects chickens of all types, but mainly **broilers**. It involves the eyes and the surrounding tissues of the head.

Causes

The main cause appears to be ***Escherichia coli***. It infects tissues beneath the skin following upper respiratory viral infections by avian pneumovirus or infectious bronchitis virus. Ammonia aggravates the disease. The route of entry is the conjunctiva or inflamed mucous membranes of the sinuses or nasal cavity. From here bacteria gain access to tissues underneath the skin.

Symptoms

The main symptom is **swelling of the head**. It gives the face a swollen appearance and is caused by accumulation of inflammatory fluid under the skin around eyes in response to bacteria, usually ***E. coli*** (Fig. 149, 150). The swelling extends over the head, and below between the jaw and wattles. Respiratory symptoms include coughing and sneezing.

Postmortem Findings

Swelling of the skin is seen over the head. Gelatinous fluid and thickened pus are observed under the skin. Removal of the skin over the head shows yellow, oedematous subcutaneous tissue.

Treatment and Control

1. Administration of broad-spectrum antibiotics through water, followed by medication in the feed to control secondary bacterial infection, is helpful.
2. Control of other disease-producing organisms, particularly infectious bronchitis virus and Ranikhet disease virus, which predispose to swollen head syndrome, is essential.
3. Good ventilation is essential to minimize the amount of ammonia and dust in the air which predispose to secondary ***E. coli*** infection.

Fig. 149. **Swollen head syndrome** in a **broiler chick**. Note conjunctival inflammation and swelling surrounding the eye. It may be from exposure to high ammonia level, or infection with ***E. coli*** and infectious bronchitis virus.

Fig. 150. **Swollen head syndrome** in a broiler chicken showing inflammation of the face and head, with closed eyes.

DEHYDRATION

"To dehydrate" means to deprive the body or tissues of water. **Nearly 80% of the newly hatched chick is water.** Chicks can survive several days without water, but will die from the 4th or 5th day. Mortality reaches its peak during the 5th or 6th day and stops suddenly if water is provided.

Causes

1. Complete lack of water
2. Inadequate number of drinkers
3. Failure of birds to find water
4. Inability of birds to reach water
5. Intercurrent disease
6. Unreachable drinkers

Symptoms

Symptoms include inadequate weight of chicks for size and age, and dehydrated and wrinkled skin on the shanks. They appear weak with sunken eyes.

Postmortem Findings

1. The chickens are emaciated and dehydrated.
2. The kidneys are gouty (see Fig. 121). That is, they are swollen and congested and greyish white in colour with a soft consistency.

Control

Ensure adequate supply and distribution of fresh clean water.