

# SPECIFIC DISEASES OF SHEEP AND GOATS

## Diseases caused by viruses

### Contagious ecthyma (contagious pustular dermatitis, orf)

A highly infectious pox virus disease of sheep and goats manifested by the occurrence of the pustular and scabby lesions on the lips, muzzle and udder.

**Transmission** : Direct contact between animals. Indirect contact with dry scabs in pens. The virus is resistant to drying and may be viable in scabs for months and years in empty feedlots and pens. Farm workers may disseminate the virus among animals of different pens with contaminated equipment, feed and farm vehicles.

#### Antemortem findings :

1. Incubation: 2 – 3 days
2. Pustular and scabby lesions on the muzzle (Fig. 151), lips and eyes.
3. Lesions on the udder and teats and the coronary band
4. The invasion of lesions by larvae of the screw worm fly and secondary bacterial infection with *Fusobacterium necrophorum*
5. Lambs and kids are unable to suckle or graze due to lip lesions.
6. Uncomplicated cases may heal within one month.
7. Emaciation
8. Pneumonia in feeder lambs

#### Postmortem findings :

1. Pustular and scabby lesions on the head, udder and feet
2. Ulcerative lesions in the nasal cavity and erosions in the mucosa of the oesophagus and upper respiratory tract.
3. Inflammation of the reticulum, omasum and intestine
4. Necrotic lesions in the lungs, pleura and liver

**Judgement** : The carcass is *condemned* if the disease is accompanied with inflammation of the stomachs and intestines, and with bronchopneumonia. Otherwise, it is *approved*.

**Differential diagnosis** : Bluetongue, sheep and goat pox, ulcerative dermatosis, cutaneous anthrax and vesicular diseases



**Fig. 151:** Contagious ecthyma. Close up view of a proliferative muzzle lesion.

## Sheep and goat pox

Sheep and goat pox is a contagious viral disease of sheep and goats manifested by papular and pustular eruptions on the skin and in generalized conditions with haemorrhagic inflammation of the respiratory tract.

**Transmission :** Direct contact with infected animals, aerosols of nasal secretions and saliva and dried scabs. Indirectly by fomites and transportation vehicles.

### Antemortem findings :

1. Incubation 6 – 8 days
2. Fever
3. Laboured breathing
4. Depression
5. Lacrimation and salivation
6. Lesions on the muzzle and lips (Fig. 155)
7. Skin lesions may vary from macules, papules, vesicles, pustules to pocks and scabs.
8. Necrosis and coalescing of the lesions and loss of wool (Fig. 156)
9. Clinical signs of goat pox are less severe than in sheep pox. The benign form of sheep pox is commonly found in adult sheep and the malignant form in lambs.

### Postmortem findings :

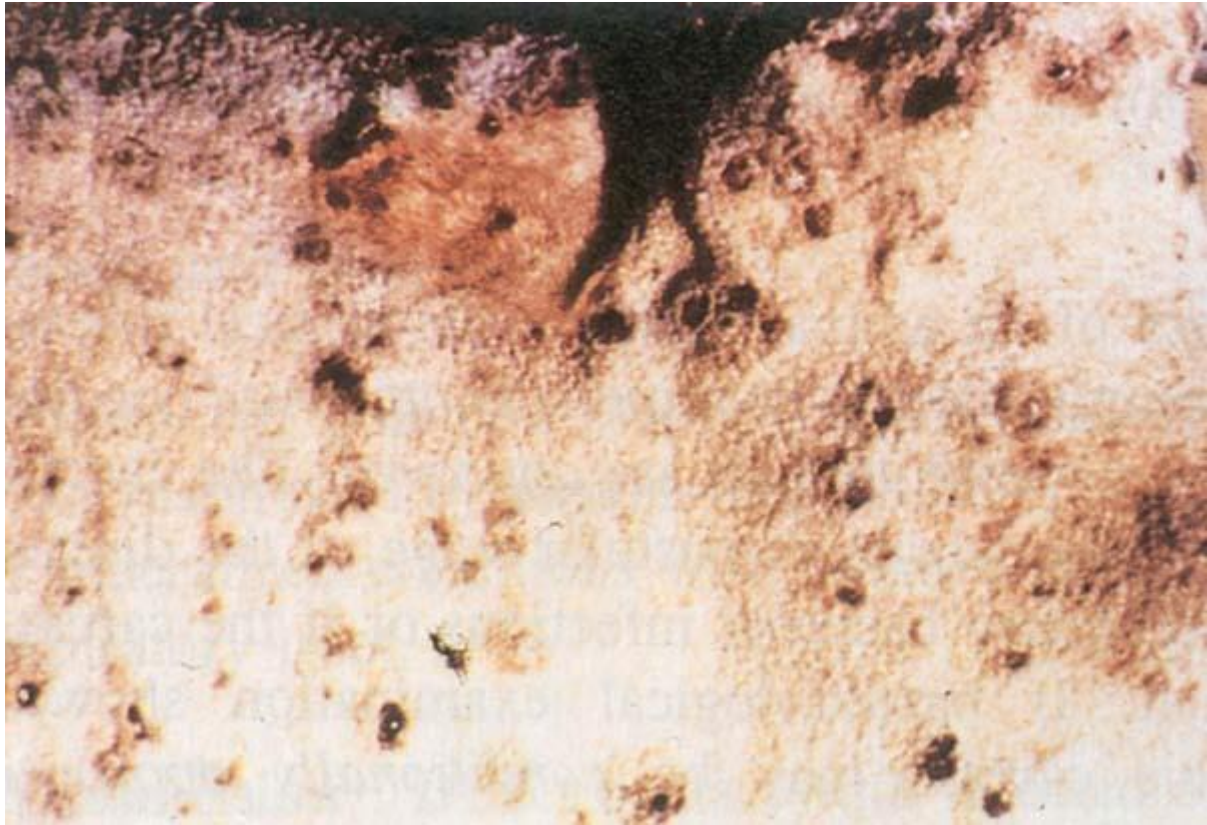
1. Reddish to whitish firm nodules in the mucosa of the pharynx and trachea
2. Reddish to whitish nodules in the lungs (Fig. 157). Rarely pneumonia
3. In malignant form: inflammation of the respiratory and digestive tract

**Judgement** : Carcass of an animal showing the clinical disease without secondary complications is conditionally *approved* pending heat treatment. The recovered animals are *approved*. The carcass is *condemned* if the acute febrile or pustular stage of the disease is associated with secondary bacterial infections or if the carcass is inadequately bled. If bacteriological examination showed negative results, this carcass may be *conditionally approved* pending heat treatment.

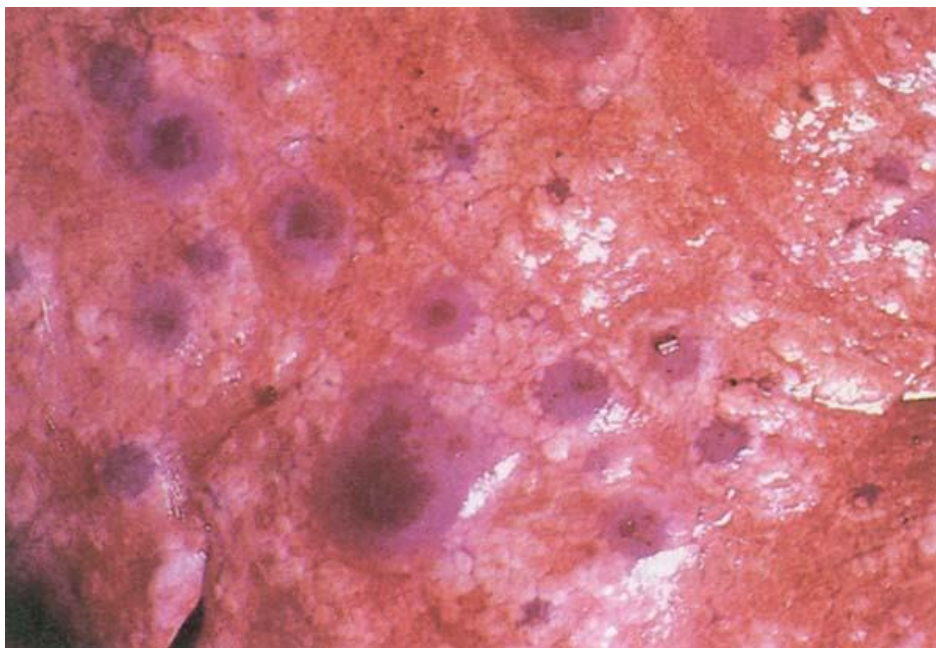
**Differential diagnosis** : Contagious ecthyma, scabies, eczema. ulcerative dermatitis and peste des petits ruminants.



**Fig. 155:** Sheep pox. Lesions on the muzzle and lips.



**Fig. 156:** Sheep pox. Necrosis and coalescing of the lesions and loss of wool.



**Fig. 157:** Sheep pox. Reddish to whitish nodules in the lungs.

**Diseases caused by *Mycoplasma* spp.**

**Diseases caused by bacteria**

**Black quarter (Black leg)**

Black quarter is an acute infectious disease of sheep and cattle manifested by inflammation of the muscles, toxæmia and high mortality. It is caused by *Clostridium chauvoei*.

**Transmission** : Contaminated soil. The organisms enter into the digestive tract with feed and through cuts which occur during the shearing, docking, and castration, and via naval infection during birth. Infection of the vulva and vagina of the ewes during lambing may cause serious outbreak of the disease. Black leg is worldwide in distribution. Well nourished and grass fed animals are more often affected.

**Antemortem findings :**

1. Fever
2. Loss of appetite
3. Depression
4. Stiff gait and reluctance to move due to lameness
5. Subcutaneous edema is not common.
6. Gaseous crepitation occurs before death.
7. Head lesions associated with edema and nose bleeding

**Postmortem findings :**

1. Subcutaneous edema particularly noted around head.
2. Affected muscle is dark brown, dry and sponge like or moist. A pungent odour is noted. Less gas is formed than in cattle.
3. Tongue, heart muscle and/or diaphragm may be blackish red. Marked abdominal extension if fetus is infected.
4. Genital tract lesions in the walls of the vagina and occasionally uterus
5. Serosanguineous and haemorrhagic fluid in body cavities and pericardial sac
6. Edema of lungs

**Judgement** : *Total condemnation* of the carcass and viscera of an animal affected with black leg. It is prohibited to slaughter and dress the animal diagnosed with this disease on antemortem examination.

**Differential diagnosis** : Other acute Clostridial infections, lightning strike, anthrax, bacillary haemoglobinuria, malignant edema, extensive haemorrhage, acute lead poisoning and lactation tetany

**Enterotoxaemia (Pulpy kidney)**

This disease is a fatal toxæmia in lambs, sheep, goats, calves and seldom in adult cattle. The disease is manifested by diarrhoea, involuntary contraction of muscles, paralysis and sudden death. It occurs after a sudden change to a better, more nutritious diet. The disease is often noted in sheep that have been fed heavy grain, and in animals which graze on lush growing pastures. *Clostridium perfringens* multiplies in abomasum and intestine and produces toxin which paralyses the vital centres in brain and damages endothelium of blood vessels. The disease occurs extensively in particular in Southern Africa but is well controlled by vaccination.

**Antemortem findings :**

1. Short course of the illness (2 – 12 hours) in lambs and longer course (24 hours) in sheep
2. Animal found dead without previous sign of the disease
3. Dullness and depression
4. Rapid shallow respiration
5. Loss of appetite and frothing
6. Muscular contractions
7. Green pasty diarrhoea
8. Grinding of the teeth and muscular tremor
9. Logging behind the flock
10. Staggering and recumbency

**Postmortem findings :**

1. No lesions in peracute cases
2. Large amount of clear, straw coloured pericardial fluid
3. Petechial haemorrhages of the heart muscle
4. Congestion of the abomasal and intestinal mucosa (Fig. 163) and liver
5. Soft pulpy kidneys a few hours after death is characteristic of this disease
6. Overload of the rumen and abomasum with concentrate
7. Haemorrhage and edema in sheep brain
8. Rapid decomposition of the carcass

**Judgement :** Carcass of an animal affected with enterotoxaemia is *condemned*.

**Differential diagnosis :** Sudden death in lambs: pasteurellosis, hypocalcemia and hypomagnesemia (reduced blood calcium and

magnesium), polioencephalomalacia (less acute form), acute rumen impaction (no convulsions are present and the course is longer) and other septicemias. Adult sheep: rabies, acute lead poisoning, pregnancy toxemia and louping-ill



**Fig. 163:** Enterotoxaemia (pulpy kidney). Dilated intestine showing a patchy congestion. Note also congestion of mesenteric lymph nodes.

### **Infectious necrotic hepatitis (Black disease)**

Black disease causes acute necrotic hepatitis in sheep and cattle and rarely in pigs. It is caused by bacterium *Clostridium novyi* in association with immature fluke invasion of the liver.

#### **Antemortem findings :**

1. Fever (40 – 42°C)
2. Rapid and shallow respiration
3. Sheep may be found dead without clinical signs.
4. Sick animal usually segregates from the rest of the flock.
5. Depression and incoordination
6. Recumbency

#### **Postmortem findings :**

1. Dark brown swollen liver showing necrotic areas surrounded by a zone of hyperaemia (Fig. 164)
2. Evidence of recent infestation of liver flukes
3. Darkened and cyanotic subcutaneous tissue due to small blood vessel engorgement (dark appearance of the skin). The name “Black disease” was derived from this.
4. Clear straw coloured fluid in the abdominal and thoracic cavities and in the pericardial sac

*Clostridium novyi* is an endemic environmental contaminant and remains latent in the liver, spleen and bone marrow. Immature liver flukes, by migrating through the liver, cause liver necrosis. This initiates *Cl. novyi* spores to germinate and proliferate. Necrotizing and haemolytic toxins are produced which cause generalized toxæmia and haemolysis of the blood.

**Judgement** : Carcass and viscera affected with black disease are *condemned*.

**Differential diagnosis** : Fascioliasis, enterotoxaemia, blackleg, malignant edema anthrax



**Fig. 164:** Black disease. Dark brown swollen liver showing necrotic areas (1–2 cm) in diameter surrounded by a zone of hyperaemia.

## **Anthrax**

Anthrax is a peracute disease of ruminants manifested with septicemia, sudden death and tarry blood from the body openings of the cadaver. It is caused by *Bacillus anthracis*.

**Transmission:** Man may contract anthrax by inhalation, ingestion and through a wound in the skin. Biting flies have been shown to be transmitters.

### **Antemortem findings:**

The peracute and acute forms in cattle and sheep are without clinical signs. Death may follow in the acute form after 1 – 2 hours of illness. The acute form lasts about 48 hours.

In pigs and horses this disease is usually localized and chronic and is often characterized by swelling around the throat and head.

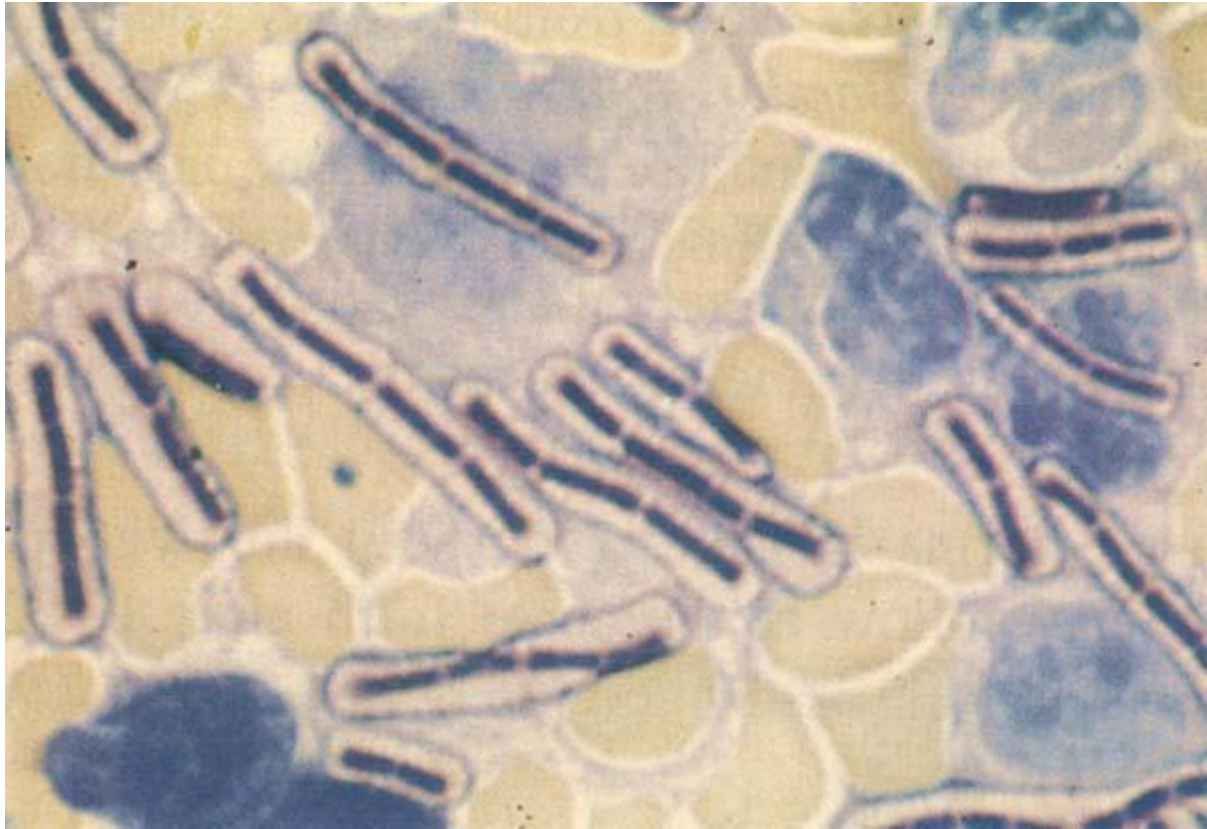
Antemortem findings in pigs:

1. Incubation 1 – 2 weeks
2. Edematous swelling of the throat and neck
3. Swallowing and breathing difficulties
4. Death due to choking or toxemia
5. Septicemia is not observed.

### **Postmortem findings:**

1. Dark-tarry blood discharge from body orifices
2. Absence of rigor mortis
3. Haemorrhage of the mucous and serous membranes, lymph nodes and subcutaneous tissue
4. Enlarged spleen
5. Severe haemorrhagic enteritis
6. Degeneration of the liver and kidneys
7. Bloating and rapid decomposition of carcass
8. Localized lesions in the intestine of pigs (dysentery)

*Diagnosis* of anthrax is carried out by direct microscopic examination of tissues and fluids (Fig. 73).



**Fig. 73:** Anthrax. Toluidine blue stain. *Bacillus anthracis* in a bovine spleen. Anthrax bacilli in tissue seen in short chains surrounded by a common capsule.

**Judgement:** *Condemnation* of the carcass and its parts by burning or burial. If disposed by burial, the carcass should be buried at least 6 feet below ground. The site should be surrounded by a foot thick layer of quicklime.

**Differential diagnosis:** Peracute blackquarter and septicaemic form of other diseases. In splenic enlargement as seen in babesiosis, anaplasmosis and leucosis, spleen consistency is firm. In anthrax, the spleen is soft and upon incision the pulp exudes like thick blackish-red blood.

## **Diseases caused by protozoa**

### **Babesiosis (Piroplasmosis, Texas fever, Red water, Tick fever)**

Babesiosis is a protozoan parasitic febrile disease of cattle, horses, sheep and swine caused by *Babesia* spp..

In sheep and goats, babesiosis is caused by *Babesia motasi* and *Babesia ovis*. Acute signs of the disease are characterized with fever, anaemia, parasitemia and haemoglobinuria. *B. ovis* usually causes a milder form of the disease than does *B. motasi*. The parasite grows and multiplies in the blood corpuscles (erythrocytes) of sheep and goats and causes haemoglobin (constituent of erythrocytes) elimination in urine (haemoglobinuria).

**Transmission** : Different species of ticks in the family Ixodidae serve as vectors of infection. *Babesia ovis* infection transmitted experimentally in sheep has caused acute signs of disease, parasitemia and lasting immunity similar with babesiosis in cattle.

**Antemortem findings :**

1. Incubation 7 – 10 days
2. High fever (41.5°C)
3. Difficult breathing
4. Anaemia
5. Loss of appetite
6. Dark reddish brown urine
7. Recovered animals may be emaciated, have reduced milk production, and some may also abort.

There are no characteristic signs in the chronic disease.

**Postmortem findings :**

1. Enlarged, yellow liver and distended gall bladder containing thick dark bile. The bladder mucosa is edematous and yellow.
2. Subcutaneous tissue and connective tissue in the muscles are edematous and jaundiced.
3. Thin watery blood and red urine in the bladder
4. Enlarged spleen
5. Edematous and haemorrhagic lymph nodes

**Judgement** : Carcass of an animal in the subclinical form of the disease or in the chronic stage may have a *favourable judgement* providing the carcass is adequately set and icterus is not present. An animal carcass showing acute form of the disease accompanied with fever, marked anaemia and haemoglobinuria and/or emaciation is *condemned*.

**Differential diagnosis** : Trypanosomiasis, theileriosis, haemobartenellosis, leptospirosis, bacillary haemoglobinuria and anaplasmosis

## **Toxoplasmosis**

Toxoplasmosis is a contagious disease of animals and man caused by protozoon *Toxoplasma gondii*. It is found most frequently in pigs and sheep. Toxoplasma in sheep is manifested with abortion and stillbirths in ewes.

**Life cycle** : see Fig. 147

### **Antemortem findings:**

1. Abortion and stillbirths in ewes
2. Fever
3. Generalized tremor
4. Difficult breathing

The systemic disease is seldom found in sheep.

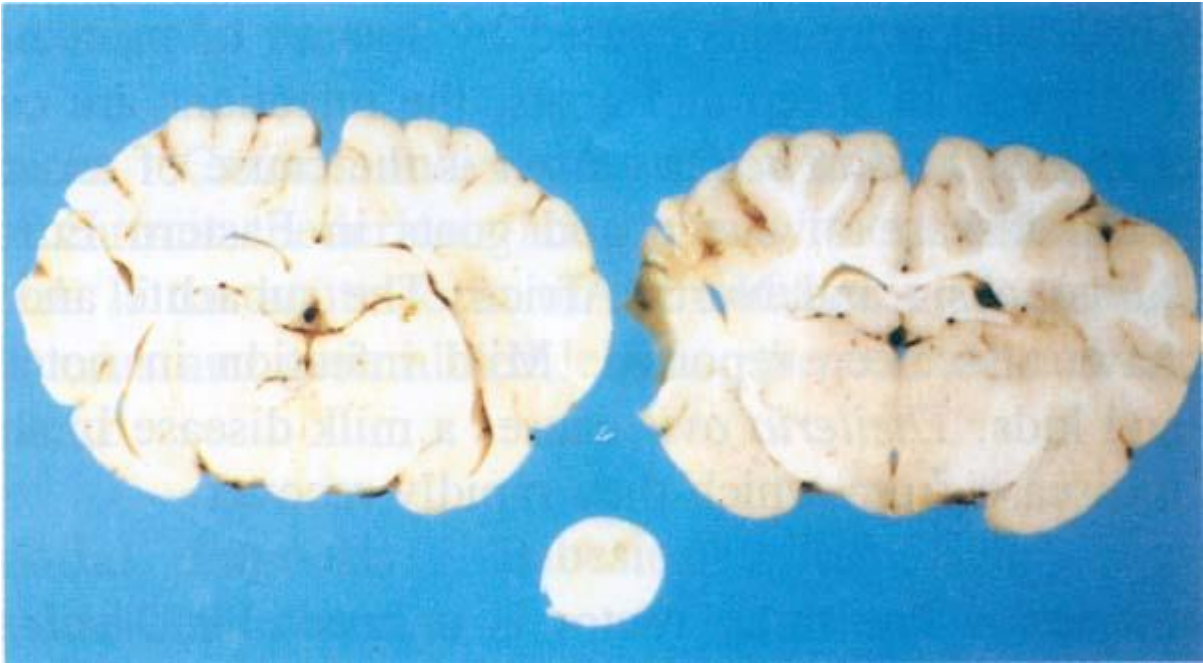
### **Postmortem findings:**

1. Multiple granulomatous lesion in the lungs
2. Hydrothorax
3. Ascites
4. Intestinal ulceration
5. Necrosis in the liver, spleen and kidneys
6. Necrosis of placenta
7. Brain haemorrhage, edema and ventricular dilatation (Fig. 176)
8. Inflammation of the brain (Fig. 177)

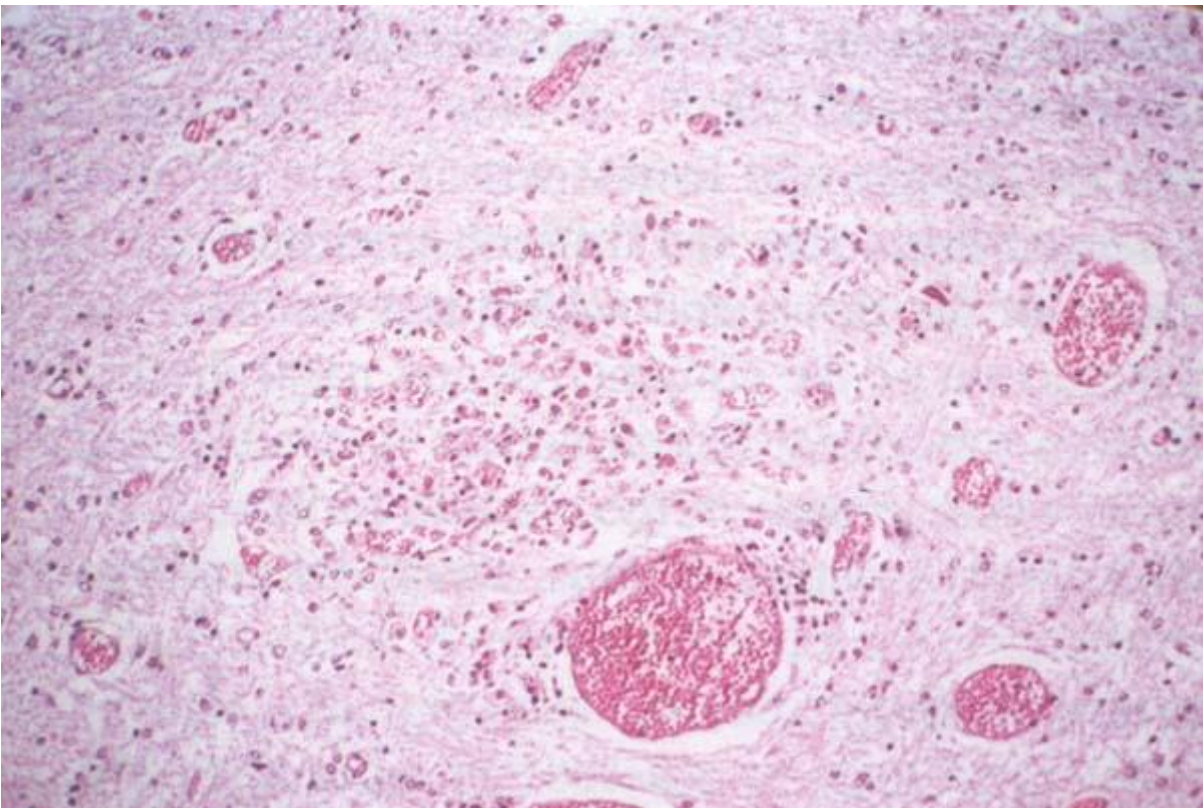
**Judgement:** Carcass of an animal showing clinical signs of acute disease is *condemned*. Recovered and reactor animals are *approved*.

### **Differential diagnosis:**

Abortion in ewes: brucellosis, campylobacteriosis, listeriosis, salmonellosis and Rift Valley fever  
Brain lesions: salt poisoning, chlorinated hydrocarbons, lead, mercury, Vitamin A deficiency, hypoglycaemia, encephalomalacia, meningitis, rabies and scrapie



**Fig. 176:** Toxoplasmosis. Brain haemorrhage, edema and ventricular dilatation. The specimen was fixed in 10% formalin solution.



**Fig. 177:** Toxoplasmosis. Inflammation of the brain (encephalitis). Tachyzoites are distributed throughout the brain where they encyst and produce bradyzoites.