Parasitic diseases

Diseases caused by helminthes

Lung worms

*Dictyocaulus viviparous* is a lung worm in cattle causing verminous pneumonia or bronchitis, husk or hooze. Mature lung worms live in the bronchi. During coughing the eggs are swallowed by the host. Hatching of eggs take place in air passages or the digestive tract. Larvae are passed in the faeces. These will survive and develop on the ground if moist and at moderate temperatures they will become invasive in 3 – 7 days. Larvae are resistant to the cold, although their maturation will be delayed.

Upon ingestion by the primary host larvae migrate through the intestinal wall to the mesenteric lymph nodes. From the mesenteric lymph nodes they pass via the lymphatic to the venous circulation and to the heart. From the heart they reach the lung alveoli. Three to six weeks after infection they migrate to bronchi where they mature and lay eggs. They survive 7 weeks in bronchi where they terminate their life cycle.

**Ante mortem findings:**

1. Elevated temperature (40 - 41 °C)
2. Rapid shallow breathing which in later stages becomes laboured breathing
3. Nasal discharge
4. Grunting
5. Cyanosis and recumbence.

**Post mortem findings:**

1. Hemorrhagic inflammation of bronchi with froth
2. Lung edema and emphysema
3. Consolidation of lung parenchyma
4. Lung worms
5. Enlarged lung lymph nodes
Judgment: Carcass of animal affected with lung worms is approved if infestation is slight and no secondary changes are observed. The lungs are condemned. The carcass is condemned if lung worm infestation has caused pneumonia which is accompanied with emaciation or anemia.

Differential diagnosis: Bacterial bronchopneumonia, abscess, necrobacillosis, tuberculosis, actinobacillosis, hydatid disease and atelectasis

Fascioliasis:

Fascioliasis is caused by different liver flukes. Fasciola hepatica is the most widespread in distribution. Fasciola gigantica in Africa and some parts of South East Asia and Fasciola magna found namely in North America including Canada and Europe. In Zimbabwe between 30–70 % of cattle slaughtered are infested with flukes. Usually the liver needs to be trimmed or condemned.

Fasciola hepatica is the most common of liver flukes. It is leaf shaped and measures 2.5 cm to 5 cm by 1.3 cm. It lives in the bile ducts of ruminants and other mammals.

Fasciola magna is one of the largest of flukes (10 cm by 2.5 cm) noted in the liver and rarely in the lungs of cattle, sheep, deer, moose, elk and other cervidae in Canada. It is found in North America. It may differentiate from Fasciola hepatica by the absence of an anterior cone like projection.

Fasciola gigantica is two or three times larger then Fasciola hepatica. It causes severe economic losses in cattle of Africa.

The term “fascioliasis” is commonly used to cover all liver flukes”.

Life cycle: Each adult is hermaphroditic and produces fertilized eggs which are passed.
in the bile and faeces onto pasture. The eggs hatch, in the presence of water or moisture into larvae called *miracidia*. If the *miracidia* find a suitable intermediate host, which is usually the aquatic snail *Limnea truncata* it will develop into *sporocysts*. In different parts of the world different snails act as intermediate hosts.

The sporocysts divide to form rediae. The *rediae* transform into *cercariae* which are the final larval stage of the cycle. They leave the snail and encysted into a *metacercaria*. After ingestion by a herbivorous animal, the cyst wall is digested in the duodenum and the *larva* crosses the small intestine wall and peritoneal space to the liver. It penetrates the liver and makes its way to the bile ducts and matures within a few weeks. The complete cycle of this fluke takes 3 – 4 months in favorable conditions.

**Ante mortem findings:**

1. Weight loss and emaciation
2. Fall in milk production
3. Anemia
4. Chronic diarrhea
5. Swelling in the mandible area

**Postmortem findings:**

1. Emaciated, anemic or edematous carcass in severe chronic infestations
2. Presence of flukes in enlarged and thickened bile ducts and in the liver parenchyma
3. Hepatic abscesses and secondary bacterial infection
4. Calcification of bile ducts
5. Black parasitic material (excrement) in the liver, lungs, diaphragm and peritoneum
6. Hemorrhagic tracts of migratory immature flukes in the lungs and liver in an acute infestation
7. Black lymph nodes of the lungs and liver due to fluke excrement
8. Icterus due to liver damage

Acute hemorrhagic tract in the bovine liver.

**Judgment:** Judgment depends on the extent of the fluke lesions and the condition of the carcass. Severe infestation with associated emaciation or edema would necessitate *total condemnation* of the carcass. Mild, moderate and heavy infestation without emaciation may have a *favorable judgment*. If the parasitic lesions in the liver are clearly circumscribed, the liver may be *salvaged* after trimming of affected tissue. Otherwise it is *condemned*. 
Differential diagnosis: Melanosis, melanoma, Dicrocelium dendriticum and Gigantocotyle explanatum infections in South East Asia

Dicrocoelium dendriticum infection

_Dicrocoelium dendriticum_ (the lancet fluke) is the smallest of the four mentioned flukes in the liver.

**Life cycle:** Two intermediate hosts are required for its complete cycle. The eggs excreted with faeces by the final host are ingested by a _land snail_. Many species of land snail can act as intermediate hosts where they develop into _sporocysts_ and _cercariae_. _Cionella lubrica_ is the principal first intermediate host in North America.

The cercariae are expelled by the snail in mucus and are deposited on plants. They are further ingested by _ants_ of the genus _Formica_ where they develop into _metacercariae_. Several species of this genus can act as second intermediate hosts. In North America _Formica fusca_ is the second intermediate host.

Ruminants, while grazing, may ingest these ants. The cyst wall of the metacercariae is digested and _larvae_ then migrate to the bile ducts where they mature. _Dicrocoelium dendriticum_ is only slightly pathogenic and does not produce clinical symptoms in the animal.

**Postmortem findings:** In cattle, sheep and swine, the lancet fluke causes moderate thickening of the bile ducts, with slight damage to liver parenchyma. Upon close examination, the parasites can be seen in the bile ducts.

**Judgment and differential diagnosis:** see Fascioliasis

Oesophagostomiasis (Pimply gut, Nodular worms)

Oesophagostomiasis is a parasitic disease of ruminants and swine. _Oesophagostomum radiatum_ is found in cattle, _Oesophagostomum Columbiana_ in sheep and _Oesophagostomum dentate_ in swine. The larvae in these species are found in the intestine, caecum and colon. In some Southern African the parasite may affect 5 – 10% of cattle, sheep and pigs.

**Life cycle:** The larvae develop to the infective stage on pasture. They are sensitive to cold, dryness and temperature changes. The infected _larvae_ penetrate the intestinal mucosa and many of them become encysted. The larvae which penetrate into deeper mucosal layers provoke an inflammatory reaction and nodules of “pimply gut”. Further stages of development occur in the intestinal wall. It is believed that many larvae are killed by the reaction they provoke in the intestine. When the larvae leave nodules due to malnutrition or lower resistance of the animal, they reach the colon. In the colon they become _adults_ and attach themselves to the colonic mucosa where they lay _eggs_. A great number of nodules disappear as gross lesions after the departure of larvae. With repeated parasitic exposure, the host becomes
immune and resistant to these larvae and local intestinal reaction becomes glaucomatous. The nodules which surround dead larvae and those which calcify after caseation, are persistent and they protrude from the intestinal wall. This may explain why nodules are present in adult animals and why no adult worms are observed in the intestinal lumen. In young animals which have no immunity, adult worms are present in the lumen of the intestine and nodules are lacking. There are some adults with both, nodules and adult worms in the intestine. O. Columbiana in sheep may cause extensive formation of nodules which may become supportive and may rupture. This further lead to inflammation of the peritoneum and adhesions.

**Ante mortem findings:**

1. Diarrhea with black-green faeces which may be mixed with mucus and blood
2. Loss of condition and emaciation
3. Stiff gait
4. Young calves may show loss of appetite, diarrhea, emaciation and anemia.

**Postmortem findings:**

1. Grayish white nodules ranging in size from a pinhead to a pea. The nodules may contain a greenish pasty material in younger lesions or a yellow - brown crumbly material in older lesions.
2. Thickening of the intestinal wall
3. Local peritonitis
4. Mild inflammation of intestine in the acute stage
5. Chronic inflammation of colon in the chronic stage

**Judgment:** Intestines affected with nodular worms are condemned. The carcass is also condemned, if severe infestation of this parasite is associated with emaciation and edema. Mild, moderate and heavy infestation without emaciation may have a favorable judgment. However, intestines should always be condemned as they cannot be used for sausage manufacture.

Oesophagostomiasis. Parasitic nodules on the intestinal mucosa (top) and serosa (bottom) in a young bovine animal.

**Cysticercoids**

Bovine cysticercoids are caused by *Cysticercus bovis*, which is the cystic form of the human tapeworm *Tania saginata*. 
**Life cycle:** *Cysticercus bovis* is the larval stage of *Tania saginata*. *Tania saginata* may grow from 3 – 7 m in length and lives in the intestine of man. It consists of a suckered head called scolex which is attached to the intestine. It also consist of a neck and hundreds of proglotid segments. Mature proglotids are filled with eggs. The proglotids break off and are excreted in the faeces where they fragment and release the oncosphere. Cattle become infected by grazing on ground and by the digestion of foodstuff contaminated with human faeces. The oncosphere liberated in the intestine from the egg penetrates the intestinal wall and through the lymphatic and blood stream reaches the skeletal muscles and heart. In the muscles the oncosphere develops into the intermediate or cysticercus stage containing a scolex. The sites of predilection are the *masseter muscles, tongue, heart* and *diaphragm*. In some countries in Africa the cysticercoids appear to show uniform distribution in the musculature. If ingested by man, the final or definite host, the scolex attaches itself to the intestinal wall and tapeworms then develop and mature.

**Transmission:** Infection in man occurs following consumption of raw or undercooked beef containing viable cisticerci. Cattle become infected by ingestion of feedstuff containing ova passed from infected humans. Cattle raised on free range become often infected through contamination of grazing with human faeces. Infected farm workers may contaminate hay, silage, other feeds or sewage effluent. Intrauterine infection of a bovine fetus was also recorded.

**Ante mortem findings:**

Heavy infestation in cattle may show:

1. Muscle stiffness
2. Rarely fever ……. 

By Dr. khaled Fujairah Municipality
**Postmortem findings:**

1. Small white lesions (cysticercoids 2 – 3 weeks after infection) in muscle tissue
2. Clear transparent bladders $5 \times 10$ mm (infective cysticercoids, 12 – 15 weeks after infection,
3. Opaque and pearl like (over 15 weeks of infection)
4. Degeneration, caseation and calcification (after 12 months or more after infection)
5. Degenerative myocarditis

**Judgment:** Carcass and viscera of an infested animal should be differentiated with those with “heavy infestation” and those with “light infestation.” Carcass and viscera of heavily infested animals are condemned and those with light infestation should be treated either by boiling or freezing. The extent of “heavy infestation” is prescribed by the controlling authority. An animal is commonly considered heavily infected, if lesions are discovered in two of the usual inspection sites including the masseter muscles, tongue, esophagus, heart, diaphragm or exposed musculature and in two sites during incisions into the shoulder and into the rounds. Generalized infection according to Canadian regulations means 2 or 3 cysts found on each cut into the muscles of mastication, heart, diaphragm and its pillars, and also if 2 or 3 cysts are found in muscles exposed during dressing procedures. In moderate or light infestation consisting of a small number of dead or degenerated cysticercoids, the carcass is held depending on the existing country regulations for approximately 10 days at -10° C.

**Differential diagnosis:** Hypoderma species (migration to heart), nerve sheath tumor, eosinophilic myositis, abscess and granuloma caused by injections

Caseous cysticercus. Numerous clear transparent cysts on the heart surface. 0.6 mm in diameter in the heart muscle.

**Hydatid disease (Hydatidosis, Echinococciosis)**

Hydatid disease in cattle is caused by the larval stages of the 2–7 mm long tape worm *Echinococcus granulose*, which lives in the intestines of dogs and other carnivores. Several strains of E. granulose exist; the cattle/dog strain is primarily responsible for hydatid disease in cattle. In Africa hydatid disease is reported more commonly in cattle that are communally owned or are raised on free range, and which associate more intimately with the domestic dogs. Hydatidosis in domestic ruminants inclicts enormous economic damage due to the condemnation of affected organs and lowering of the meat, milk and wool production.

**Life cycle:** The infective eggs containing the oncosphere passed in the faeces are accidentally ingested by cattle, sheep, pigs, other animals or humans which act as intermediate hosts. After the infective eggs are ingested by these intermediate hosts, the oncospheres in the eggs penetrate the intestine and reach the liver, lungs and other organs including the brain and muscles to develop into hydatid cysts at the end of about five months. These cysts measure commonly 5 – 10 cm and contain fluid. Some may reach up to 50 cm in diameter. Others may produce daughter’s cysts. The diagnostic features of a
hydatid cysts are a concentrically laminated thick outer layer within which is a germinal layer. In fertile hydatids the germinal layer is granular and has brood capsules each containing protoscoleces. When brood capsules become detached and float free in the cysts fluid they are referred to as hydatid sand. In some animals a fair proportion of hydatid may be sterile. The life cycle is completed when a fertile hydatid cyst is eaten by a definitive host, the dog or the appropriate carnivore. Cattle and majority of intermediate hosts show no clinical evidence of infection. However, in humans hydatid cysts can cause serious disease.

Ante mortem findings: None of significance

Postmortem findings:

1. Liver, heart, lungs, spleen, kidneys
2. Muscle and brain
3. Any tissue including bone

Judgment: Carcass showing emaciation, edema and muscular involvement is condemned and destroyed. Affected viscera and any other tissue are also condemned. Burying of carcass is not sufficient, since dogs may retrieve the affected organs.

Differential diagnosis: Retention cysts in kidneys, cysts in liver, glaucomatous lesions, Cysticercus...
**Life cycle:** The adult worm lives in the nodules and the fertilized females liberate *microfilaria* into the tissue lymph spaces from where they are taken up by an insect vector which act as an intermediate host. The common vectors are the midges of the genus *Culicoides*. Other biting flies can act as intermediate hosts. The larvae develop to the infective stage in these insect vectors. Infection of cattle occurs when these biting flies with the infective larvae feed on them.

**Ante mortem findings:** Careful palpation reveals sub-cutaneous nodules in the brisket and buttock regions.

**Postmortem findings:**

1. Firm fibrous nodules (0.5 cm - 5 cm in diameter) singly or in clusters in the regions of brisket, buttocks and thighs.
2. The nodules have tightly coiled worms.
3. The worms may be dead or calcified in order nodules.

**Judgment:** The affected carcasses can be passed after the nodules have been removed. In heavy infestations the affected briskets are removed, and the tissue and the fascia around the stifle and the brisket are stripped off before the carcasses are passed.

**Differential diagnosis:** Abscesses, neurofibromatosis, cysticercoids, eosinophilic myositis

Firm fibrous nodules of Onchocerca gibsoni in the brisket of an ox.

**Parafilariasis**

*Parafilaria bovicola* is a filarial parasite of cattle which causes focal cutaneous hemorrhage and subcutaneous lesions which are observed as bruising on a dressed carcass. The parasite occurs world wide in countries such as France, Canada, Sweden, South Africa and Zimbabwe, as well as other parts of Africa.

**Transmission:** The parasite is spread by several haematophagus species of the fly Musca.

**Life cycle:** During the 7 - 10 month's life cycle of the worm, the fly picks up the egg off the skin surface of infected cattle. The larvae then develop in the fly, and are transmitted to the bovine through the saliva where they migrate subcutaneously and cause the lesions. The lesions appear like a bruise, hence the
pseudonym “false bruising”. They have a greenish tinge due to the presence of a large number of eosinophils. The adult filaria pierces the skin and lays eggs around the periphery of the pierced hole.

**Judgment:** Lesions vary from mild and localized to severe and extensive. Mild and localized lesions require trimming of the affected portions and extensive lesions may warrant a total condemnation of the carcass.

**Diseases caused by protozoa**

**Trypanosomiasis**

This is a protozoan disease of animals and humans caused by parasites of the genus Trypanosoma, which are found in blood plasma, various body tissues and fluids.

**Transmission:** Trypanosoma are transmitted primarily by the Glossina spp., tsetse fly, Stomoxys, tabanid and reduviid bugs, and by venereal contact. Trypanosoma species in the insect vector undergo one or two cycles of development.

**Ante mortem findings:**

1. Intermittent fever
2. Anemia
3. Weight loss and weakness
4. Edema, particularly observed in the face and legs
5. Enlarged body lymph nodes
6. Hemorrhage
7. Opacity of the cornea, keratitis and photophobia

Chronic form of trypanosomiasis is sometimes manifested by progressive weakness, despite absent parasitemia, and death.

**Postmortem findings:**

1. Enlarged lymph nodes
2. The enlargement of spleen, liver and kidney may also occur.
3. Edematous and emaciated carcass
4. Mild icterus

**Judgment:** The carcass affected with trypanosomiasis or any other protozoan diseases is **condemned** if an acute condition is associated with systemic body changes. Heat treatment may be recommended in some cases if economically feasible. The carcass of recovered and reactor animals may be **approved** if generalized lesions are lacking. Carcass showing borderline emaciation or slight edema should be examined after the 24 - 48 hours in the chiller. A satisfactory setting would lead to a **favorable judgment** of the carcass. The affected parts of the carcass and organs are **condemned**.

**Differential diagnosis:**

Helminthiasis, malnutrition and other chronic wasting diseases, equine infectious anemia, heart water, babesiosis and anaplasmosis
Trypanosomiasis. This animal shows icteric mucous membranes, weakness in leg muscles and emaciation.

An impression smear of the trypanosomes and the RBC in the capillaries.

Trypanosoma vivax in blood smears.

**Theileriosis (East coast fever)**

East coast fever is a sub acute haemoproteozoan disease of cattle caused by *Theileria parva*. Theileriosis is characterized by fever, enlarged lymph nodes, dyspnea and death. In chronic cases loss of condition, emaciation, diarrhea, blindness, etc. can be seen.

**Transmission:** Vectors are ixodid ticks of the species *Rhipicephalus*.

**Ante mortem findings:**

1. Mortality up to 90 %
2. High temperature (up to 41 °C)
3. Difficult breathing and coughing
4. Nasal discharge, salivation and watery eyes
5. Swelling of the lymph nodes draining the area where the infected tick fed
6. Cerebral signs manifested by circling to one side, convulsions and death

East Coast fever (Theileriosis). Enlarged body lymph nodes.

**Postmortem findings:**

1. Froth in nostrils and bronchi associated with pulmonary edema and emphysema
2. Swollen, edematous lungs and interstitial pneumonia
3. Enlarged and hemorrhagic lymph nodes and splenic lymphoid hypertrophy
4. Enlarged and mottled liver
5. Infarcts, thrombosis and lymphoid hypertrophy in spleen
6. White spots of lymphoid aggregates in a kidneys
7. Brownish coloration of fat
8. Hemorrhagic and rarely ulcerative enteritis

Confirmation of diagnosis is only made through detection of parasites in a Giemsa stained lymph node biopsy smear and/or blood smear.

**Judgment:** Carcass and viscera of an animal affected with febrile chronic theileriosis and without systemic lesions are approved. Carcass is condemned, if acute febrile theileriosis is accompanied with fever and generalized lesions. The affected organs are also condemned.

**Differential diagnosis:** Hemorrhagic septicemia, babesiosis, malignant catarrhal fever, trypanosomiasis, Rift Valley fever, heart water and bovine leucosis

Theileriosis. Swollen edematous lungs and interstitial pneumonia.
Theileriosis. Infarcts, thrombosis and lymphoid hyperplasia in spleen.

**Besnoitiosis**

Besnoitiosis is a chronic debilitating protozoan disease of cattle and horses. It also occurs in wild animals such as antelope, wildebeest (gnu) in Africa and caribou in Canada. The causative agent in cattle is *Besnoitia besnoiti* and *Besnoitia benetti* in horses.

The organism is closely related the genus *Toxoplasmosis*. The mode of transmission is still unknown. It is believed that tabanids are mechanical vectors.

**Ante mortem findings:**

1. Elevated temperature
2. Increased respiration
3. Nasal discharge and lacrimation
4. Diarrhea
5. Cysts in the skin and subcutaneous tissue and loss of hair
6. Swollen body lymph nodes
7. Severe generalized edema of the head, neck, ventral abdomen and legs
8. Chronic skin lesions show in folding and cracking
9. Decreased milk production
10. Inflammation of the testicles

**Postmortem findings:**

1. Inflammation of the pharynx, larynx and trachea
2. Sand-like granules and cysts in the turbinate and nostrils
3. Sand-like granules in the endothelium of large vessels
4. Dermatitis

**Judgment:** The carcass is **approved** if the lesions are localized with no systemic involvement. Carcass is **condemned** if disseminated, generalized lesions are accompanied with emaciation.

**Differential diagnosis:** Lumpy skin disease, sweating sickness and ectoparasitism (mites, ticks, fungi)
Besnoitiosis. Sand like granules and cysts in the nostrils of an antelope.

**Anaplasmosis (gall sickness)**

Anaplasmosis is a rickettsial disease characterized by severe debility, emaciation, anemia and jaundice and is caused by *Ana plasma* spp... They are obligate intracellular parasites. *Ana plasma marginal* is the causative agent in cattle and wild ruminants.

**Transmission:** Boophilus species of ticks transmit anaplasmosis. Mosquitoes and the horsefly are mechanical transmitters. Transmission is also possible through injection needles.

**Ante mortem findings:**

Acute infection with *A. marginal*

1. High fever
2. Jaundice and anemia demonstrated by pale mucous membranes
3. Frequent urination and constipation Chronic infection
4. Emaciation

**Postmortem findings:**

1. Enlarged and congested spleen (splenomegaly) showing soft pulp
2. Distended gall bladder with dark tarry bile
3. Thin, watery blood, which clots poorly
4. Enlarged, icteric liver, deep orange in color and distended bile ducts
5. Lemon yellow carcass and connective tissue of the sclera of the eye, tendons, pleura, peritoneum, and attachments of diaphragm.

Diagnosis can only be confirmed by detecting parasites in a blood smear stained with Giemsa.

**Judgment:** Carcass of an animal showing acute infection should be condemned. Recovered and “suspect” animals manifesting inconclusive signs of anaplasmosis are approved if otherwise healthy. A mildly yellow discolored carcass may be chilled and assessed after setting. If the discoloration has disappeared, the carcass is approved. Animals affected with anaplasmosis could be treated under the supervision of a government official. Guidelines for the withdrawal period for therapeutic agents should be followed if the animals are being shipped for the slaughter.

**Differential diagnosis:** Icterus and anemia of different causes, anthrax, leptospirosis, emaciation caused by parasitism and malignant lymphoma, babesiosis.
Remarks: The access of biting insects to contaminated fresh blood should be prevented. Blood from suspicious carcasses should not be salvaged.

Anaplasmosis. Ox liver affected with disease showing distended bile ducts.

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

Babesiosis of cattle, horses, sheep and swine is a febrile, tick borne disease caused by various species of the protozoan genus *Babesia*.

Transmission: Different species of ticks in the family Ixodidae serve as vectors in different locations. The Babesia parasites can be transmitted transstadially and transovarially within a tick species.

Ante mortem findings:

1. Incubation 7–10 days
2. Mortality up to 50 % or over depending on age, breed, etc.
3. High fever (41.5° C)
4. Dark reddish brown urine in the terminal stage
5. Reddened and injected mucous membranes at the early stages and later, anemic mucous membranes
6. Clinical signs may resemble rabies in cerebral form of babesiosis.

Postmortem findings:

1. Edema and congested lungs
2. Enlarged and yellow liver and distended gall bladder with thick dark green bile.
3. Enlarged spleen
4. Anemia and pale muscles
5. Jaundice particularly noted in the connective tissue
6. Edematous and hemorrhagic lymph nodes
7. Yellowish-orange color of musculature (mild cases)
8. Occasionally dark kidneys with no other findings
9. Pink hemorrhage of a bovine brain

Diagnosis can only be confirmed by identification of parasite in the peripheral blood smear stained with Giemsa
**Judgment:** Carcass of an animal in acute form of the disease, with associated icterus, is *condemned*. An emaciated, jaundiced carcass showing yellow gelatinous fat also requires total condemnation. A mild form of this disease showing yellow orange coloration of carcass not associated with icterus may be *approved*. The satisfactory setting of the carcass in the chiller must be considered in this approval.

**Differential diagnosis:** Anaplasmosis, trypanosomiasis, theileriosis, leptospirosis and bacillary haemoglobinuria.

Pink hemorrhage. Cerebral form of babesiosis caused by B. bovis. It is characterized by formation of thrombi and emboli in brain capillaries.

Babesia bigemina in American bison blood.

**Sarcocystosis (Sarcosporidiosis)**

Sarcocystosis is caused by the various species of the protozoan genus *Sarcocystis*. This is one of the most common parasitic conditions in domestic food animals and a high percentage of cattle in various parts of the world are infested with these parasites which are usually host specific. In cattle three species have been recognized. They are listed in Table 1. Cattle are the intermediate hosts of *Sarcocystis* spp. All *Sarcocystis* spp. in the intermediate hosts, the food animals, are characterized by the formation of cysts in the muscles.
### Table 1: Sarcocystis spp. in Cattle

<table>
<thead>
<tr>
<th>Species</th>
<th>Distribution</th>
<th>Definitive Host/s</th>
<th>Size of cyst</th>
<th>Pathogenicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. cruzi</td>
<td>World-wide</td>
<td>Dog, coyote, red fox, racoon and wolf</td>
<td>Microscopic, less than 0.5mm long.</td>
<td>Most pathogenic species in cattle it can cause fever, anemia, abortion neurological signs and even death.</td>
</tr>
<tr>
<td>S. hirsuta</td>
<td>Probably world-wide</td>
<td>Cat</td>
<td>Macroscopic, up to 8mm long and 1mm wide, fusiform in shape</td>
<td>Mildly pathogenic</td>
</tr>
<tr>
<td>S. hominis</td>
<td>Europe</td>
<td>Humans and some primates</td>
<td>Microscopic</td>
<td>Mildly pathogenic to cattle</td>
</tr>
</tbody>
</table>

**Life cycle**: All Sarcocystis species require two hosts and a pre-predator cycle to complete their life-cycle. A herbivore, the prey, and a carnivore or omnivore, the predator are involved. Sexual development occurs in the predator which is therefore a *definitive host*, and asexual development occurs in the prey which is the *intermediate host*.

Two species, one in cattle (S. hominis) and one in pigs (S. suihominis) use humans as definitive hosts and therefore these infections in animals are zoonoses. Generally speaking dog transmitted Sarcocystis are pathogenic and whereas cat transmitted ones are not.

The most important species in cattle is *S. cruzi* which has world-wide distribution and uses the dog as the definitive host. Sexual development takes place in the dog after which infective sporocysts are passed in the faeces. The details of development in cattle are illustrated in

The buffalo is the intermediate host for two species: *S. levinei* which forms microscopic cysts and uses the dog as the definitive host, and *S. fusiformis* which forms macroscopic spindle or globular shaped cysts measuring 3.2cm × 8 mm and uses the cat as the definitive host. *S. fusiformis* cysts are seen in the esophagus and the skeletal musculature and are common parasite of the water buffalo in many parts of the world.

**Transmission**: Cattle acquire infection by ingesting sporocysts contaminating feed, pasture or water. After several generations of asexual reproduction by schizogony they form cysts in muscles. *S. cruzi*, the most pathogenic species for cattle forms microscopic cysts. The definitive host, including humans acquires the infection when they eat bovine tissues containing the viable Sarcocystis cysts. The data provided below are for *S. cruzi* infestations, unless specified otherwise.

**Life cycle of Sarcocystis hominis (cattle) and Sarcocystis suihominis (pigs) in final host (man)**

**Life cycle of Sarococystis cruzi in the bovine and canine (prey-predator cycle)**
Ante mortem findings:

By Dr. Khaled Fujairah Municipality
1. Incubation period 5 - 11 weeks
2. Fever
3. Loss of appetite
4. Excessive salivation
5. Anemia
6. Abortion
7. Loss of hair especially at the tip of the tail

Postmortem findings:

1. The cysts are microscopic and therefore are not detected on routine postmortem inspection. They cause little tissue reaction.
2. In some cases the cysts may be associated with eosinophilic myositis.
3. A histological section of bovine muscle affected with eosinophilic myositis showing massive accumulation of eosinophiles and two micro cysts of S. cruzi.
4. S. hirsuta cysts may be seen as fusiform objects 8 mm × 1 mm in the esophagus, diaphragm and skeletal muscles of older animals especially bulls.
5. Macroscopic cysts of S. fusiformis in the skeletal muscle of buffalo.

Judgment: Judgment should be made on macroscopic presence of cysts. In heavy and widespread infestations with the visible cysts the whole carcass is condemned. In lighter infestations those parts of the carcass which are not affected are passed for human consumption. Microscopic examination of muscle may show as much as 70 % infestation in animals worldwide.

Differential diagnosis: Cysticercoids, toxoplasmosis, neurofibromatosis, eosinophilic myositis.

Sarcocystosis. Eosinophilic myositis.

Histological section showing accumulation of eosinophiles and two micro cysts of S. Cruzi. There is no tissue reaction.
S. Fusiformis in the skeletal musculature of buffalo.

**Diseases caused by arthropod parasites**

**Hypoderma bovis infestation**

There are two warble flies in cattle, Hypoderma bovis and Hypoderma lineatum. They have similar cycles. During the summer the adult fly lays its eggs on the leg hair and occasionally on the body of cattle. Within a week the larvae hatch and burrow into the skin and, for several months they travel through the body. Hypoderma bovis migrates into the thoracic and abdominal cavities towards the spinal canal before moving under the skin of the back. Hypoderma lineatum migrates to the esophageal area before reaching the dorsal area of the animal. In spring (February-May), the larvae reach the area of the back. They burrow a breathing hole and increase in size to approximately 8 mm × 25 mm. They are visible for a month. After this cycle, maggots fall to the ground where they develop into flies and start the whole cycle once again.

**Ante mortem findings:**

1. Swelling or eroded skin on the back
2. Larvae protruding from the skin of the back
3. Cattle may violently rush and kick the abdomen with hind legs.
4. Erected tail
5. Paralysis of the lower body and legs if the spinal cord is involved.

**Postmortem findings:**

1. Inflamed area of subcutaneous tissue, red, green or yellow in color, around the maggot or at the site where the maggot lodged
2. Inflammation of the esophagus may cause rumen bloat due to obstruction
3. Hypoderma bovis larvae
Hypodermal bovis. Life cycle of warble fly in cattle

**Judgment:** Carcass of an animal affected with Hypoderma bovis is approved. Subcutaneous lesions are removed.

**Differential diagnosis:** Cysticercus bovis cysts in esophagus

| Hypodermal bovis. Larvae protruding from back in a 2 year old steer. | Hypodermal bovis larvae. |

**Screwworm myiasis**
Screwworm Myiasis caused by larvae of the flies Cochliomyia hominironux (New World Screwworm - NWS) and Chrysomya bezziana (Old world screwworm - OWS), is characterized by larvae feeding on living tissues in open wounds of any warm blooded host including humans, resulting in weight loss, other signs of morbidity and sometimes death. The NWS is found in Central and South America including the Caribbean region. The OWS is located in India, Southeast Asia, and Tropical Africa and in the Persian Gulf area.

**Life Cycle:** In the preferred temperature range (20 – 30°C) it is about 21 days. The female, which mates only once, lays one or more batches of up to 300 eggs at the edge of any wound or break in the skin in any warm blooded animal. Skin breaks as small as tick bites, as well as natural orifices can be sites of oviposition. The larvae develop within 24 hours, and burrow into the living flesh, creating large, deep, open wounds which attract further egg laying females. If unattended, these wounds are often fatal, particularly in newborn animals where the oviposition site is usually the navel.

**Ante mortem Findings:** A serosanguinous discharge often exudes from the infested wounds, and a distinct odor may be detected. In some cases, the openings in the skin may be small with extensive pockets of screwworm larvae beneath. In dogs, screwworm larvae commonly tunnel under the skin. Screwworm infestations in anal, vaginal, and nasal orifices may be difficult to detect, even in the later stages.

**Postmortem Findings:** After 5 – 7 days of infestation, a wound may be expanded to 3 cm or more in diameter and 5 – 20 cm deep with larvae from a single screwworm egg mass. Usually by this stage, additional screwworm flies have deposited eggs, resulting in a multiple infestation. However, after the death the larvae leave the body due to the temperature reduction and some third stage instar larvae may pupate in the body.

**Judgment:** The affected carcasses can be passed after the wounds tissues have been removed and incinerated.

**Differential diagnosis:** Other blow flies as C. Macellaria and Sarcophagidae spp.
Screwworm. Life cycle

Typical pocket like wound from screwworm larvae.

Mange skin thickening & hair loss from vulva to the udder (psoroptic mange)

Circular area of alopecia in which the skin is thickened & encrusted
Diseases caused by Rickettsia and Mycoplasma spp.

Heart water (Hydro pericardium)

“Black dung” when affecting African cattle and buffalo

“Sheep fever” when seen in sheep

Heart water is an acute, non contagious disease of cattle, sheep, goats, antelopes and wild ruminants. It is caused by the rickettsial organism Cowdria (Rickettsia) ruminantium.

Transmission: Heart water is transmitted by various species of Amblyomma ticks. Transstadial transmission of the organism occurs in vector ticks.

Ante mortem findings:

Per acute form

1. Incubation 14 – 28 days
2. Fever
3. Diarrhea
4. Convulsions and death

Acute form

5. Fever up to 41.7°C
6. Rapid breathing
7. Lack of appetite, depression and listlessness

Nervous signs include
8. Twitching of the eyelids
9. Protrusion of the tongue
10. Champing of the jaw
11. Walking in circles
12. Paddling with legs in recumbent animals
13. Opisthotonos and convulsions

Postmortem findings:

1. Hydro pericardium
2. Hydrothorax
3. Pulmonary edema and ascites
4. Hemorrhagic gastroenteritis
5. Enlarged liver, spleen and lymph nodes
6. Hemorrhage in the abomasums and intestine
7. Edema and hemorrhage of the brain

Judgment: Carcass of an animal affected with heart water is condemned in the acute stage of the disease. In a chronic case, the carcass may be approved if adequately bled and muscles are wholesome in color and texture. The affected organs are condemned.

Differential diagnosis: Per acute form of heart water should be differentiated from anthrax. The acute nervous form of the disease is differentiated from tetanus, rabies, cerebral trypanosomiasis, strychnine poisoning, piroplasmosis, theileriosis, lead and organophosphate poisoning, parasitism, arsenical poisoning and poisoning with certain plants.

Heart water Cowdria ruminantium in bovine brain smear (arrow).

Q fever (Queensland fever, Nine mile fever, American Q fever, Australian Q fever)

Q fever is a disease of cattle, sheep, goats, donkeys, camels, fowl, dogs, cats, pigeons and humans. It is caused by *Coxiella burnetii*. Q fever is an occupational disease of livestock personnel. Farmers and laboratory personnel.

Transmission: Ticks spread infection to cattle which develop mild disease. The faeces deposited on animal hide by ticks may be the source of infection for humans. Q fever is also transmitted by inhalation or dust contaminated with infected animal secreta or excreta. Healthy animals may serve as a carrier.
and shed the organism in milk, urine, faeces, placenta and fetal fluids. They harbour the infection and no clinical signs are observed. Contaminated meat and water are further means of infection read.

Field cases there are no clinical signs of this disease. In the disease produced by the inoculation of cows via the udder the clinical signs may include:

1. Acute mastitis
2. Loss of appetite and depression
3. Serous nasal and lacrimal discharge

Coxiella burnetii is highly resistant and was isolated from farm soil 6 months after the removal of animals. It may persist in the udder up to 3 years. The temperatures of milk pasteurization (in bulk at 63°C for 30 minutes or the common method at 72°C for 15 seconds) kill this agent in milk.

Vaccination will reduce shedding of organisms in milk.

This disease in humans has a sudden onset and is characterized by loss of appetite, weakness and generalized malaise lasting from 1 – 2 weeks. Pneumonia may also be present. Death may be caused by endocarditis in older people. More severe symptoms of Q fever are noticed.

Contagious bovine pleuropneumonia. Straw colored fluid in the thorax and partial lung hepatization.

Contagious bovine pleuropneumonia. Lobar pneumonia with red hepatization and marbled appearance of lung lobules.
Contagious bovine pleuropneumonia (CBPP): posture of an affected cow with stretching neck to breath easier

Contagious bovine pleuropneumonia (CBPP): pleuritis with unilateral infection of the left lung

Contagious bovine pleuropneumonia (CBPP): sub-epicidal hemorrhage

Contagious bovine pleuropneumonia (CBPP): excessive synovial fluid & bits of fibrin in a joint

This is an acute, sub acute or chronic highly infectious disease of cattle caused by *Mycoplasma mycoides* var, *mycoides*.

**Transmission:** Aerosol and droplet infection from the infected animals. The recovered animal called “lungers” act as carriers and shedders, especially under stress.

**Ante mortem findings:**

1. Incubation: acute 10 – 14 days, chronic 3 – 6 months
2. Morbidity: 90 % in susceptible cattle
3. Mortality: 10 – 50 %
4. Fever
5. Depression
6. Lack of appetite and loss of weight
7. Coughing on exercise
8. Shallow rapid respiration, grunting and gurgling
9. Extended neck, lowered head and open mouth
10. Arched back and outward rotated elbow
11. Arthritis in young animals

**Postmortem findings:**

1. Fibrinous inflammation of the pleura (pleuritis)
2. Straw colored fluid in the thorax
3. Lobar pneumonia with red hepatization, marbled appearance of lung lobules due to thickening of interlobular septae and interlobular pulmonary edema
4. Enlarged mediastinal lymph nodes
5. Walled-off sequestrate formation in chronic cases
6. Hemorrhage in the heart
7. Arthritis and tenosynovitis

**Judgment:** Carcass of an animal affected with contagious bovine pleuropneumonia is *condemned* if the disease is associated with fever, inadequate bleeding of carcass, serous infiltration of the brisket and emaciation. Recovered animals showing no generalized signs of the disease are *approved* and the affected organs are *condemned*.

**Differential diagnosis:** Shipping fever (Pasteurellosis). East coast fever, foreign body pneumonia, IBR, tuberculosis, Chlamydia infections and lungworms

**Metritis**

Metritis is inflammation of the uterus. This condition is of *bacterial origin*. It occurs as a result of calving problems such as retention of placenta, abortion, twin births, abnormal labour and traumatic lesions of the uterus cervix and vagina.

**Ante mortem findings:**

1. High fever and depression
2. Muscular weakness
3. Placental retention
4. Listlessness
5. Reddish fetid discharge from the vulva

**Postmortem findings:**

1. Enlarged flaccid uterus showing “paint-brush” hemorrhages on the serosal surface
2. Inflammation of the uterus with light-brown foul smelling uterine exudates
3. Enlarged uterus containing greenish-yellow purulent exudates (Pyometra)
4. Inflamed peritoneum at the entrance of the pelvic cavity
5. The iliac, lumbar and sacral lymph nodes are enlarged
6. Degeneration of the liver, kidney and heart muscles may be present
7. Congested musculature of the carcass
8. Necrosis of abdominal fat

**Judgment:** Carcass of an animal affected with acute metritis is *condemned* if it is associated with septicemia or toxemia. In chronic cases, when toxemic signs are lacking, the carcass may be *approved* if no antibiotic residues are found.

**Differential diagnosis:** Recent calving
Metritis. Necrotizing inflammation of the uterus with grayish-brown foul smelling uterine exudates.

Pyometra. Enlarged uterus containing greenish-yellow exudates.

**Mastitis**

Mastitis is inflammation of the udder caused by *bacteria, fungi and yeasts*. Depending on the virulence of the agent and resistance of the udder, mastitis is manifested in acute or chronic forms.

**Ante mortem findings:**

1. Variable temperature depending on stage of condition
2. Swollen warm, painful udder or hard enlargement involving one or all quarters
3. Depression, loss of appetite and dehydration
4. Abnormal gait caused by rubbing of the hind leg against inflamed quarter
5. Animal tends to lie down. Purulent or bloody exudates from teats or watery pale fluid in chronic cases

**Postmortem findings:**
1. Pale yellow granular appearance of the udder parenchyma
2. Light brown edematous udder parenchyma
3. Enlarged supramammary, iliac and lumbar lymph nodes.
4. Injection sites

**Judgment:** Carcass and viscera are *condemned* if acute or gangrenous mastitis is associated with systemic changes. If infection has spread from the supramammary lymph nodes via the iliac lymph nodes to the lumbar lymph nodes, this can be taken as evidence of spread of infection from its primary location. The condemnation of the carcass may then be warranted. A localized condition of the udder has a *favorable judgment* of the carcass.

**Differential diagnosis:** Edema, haematoma and rupture of the suspensory ligament

Chronic mastitis. Enlarged, firm udder. Incision into the udder parenchyma shows normal milk and pale yellow granular appearance of the udder parenchyma.

Brown red edematous udder parenchyma. The udder culture resulted in a heavy growth of *Staphylococcus aureus*.

**Endocarditis**
Endocarditis is inflammation of the endocardium of the heart. The lesion is most commonly seen in the valves. It may be the result of bacterium caused by infection in some remote organs such as the udder, uterus or other sites.

In cattle, the organisms most commonly associated with endocarditis are *Actinomyces pyogenes* and *Streptococcus spp...* Strains of *Escherichia coli* are also frequently found. The lesion is most commonly found on the valves. Portions of vegetation may become detached and released into the blood stream as emboli which may lodge in other organs. They may be septic or aseptic. The latter contain thrombosis material. Emboli, brought from the right heart to the lungs by blood vessels may cause pulmonary abscesses, or pulmonary thrombosis and the emboli brought from the left heart to the spleen and kidneys may cause septic or aseptic infarcts in these organs. Abscesses in the heart may also be observed.

**Ante mortem findings:**

1. Moderate fever
2. Breathing with accompanied grunt
3. Pallor of mucosa
4. Loss of condition and muscle weakness
5. Temporary fall in milk production in lactating animals
6. Jaundice and death

**Postmortem findings:**

1. Large cauliflower-like lesions in the endocardium
2. Small wart-like and varicose lesions in the endocardium
3. Embolic lesions in other organs including the lungs, spleen, kidneys etc.
Heart cow: aortic stenosis – vegetative endocarditis myocardial infection

**Judgment:** Carcass of a debilitated animal is *condemned* for varicose endocarditis if it is associated with lesions in lungs, liver or kidneys. Carcass affected with ulcerative or varicose endocarditis with no signs of systemic changes and negative bacteriological result may be *approved after heat treatment* is applied. Endocarditis showing scar tissue is *approved*. The heart is *condemned*.

**Differential diagnosis:** Pneumonia, pericarditis, pulmonary edema, emphysema, pleuritis, lymphoma, high altitude disease, congenital heart disease, congenital valvular heart cysts or deformities especially in calves.

**Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis)**

TRP is caused from the perforation of the reticulum by a metallic foreign body. It is mostly seen in adult dairy cattle and can occur in beef cattle.

**Ante mortem findings:**

1. Sudden drop in milk production
2. Depression, loss of appetite and weight loss
3. Stretched head and neck
4. Reluctance to walk, arched back and tucked up abdomen
5. Scant, hard faeces, rarely covered with mucus.
6. Mild rumen bloat
7. Audible “grunt” in early stages

If mild *septicemia* develops the animal shows:

8. Elevated temperature (39.2 ° C - 40° C)
9. Increased heart rate

In chronic localized peritonitis, acute signs and pain lessen, temperature falls and stomach reticulo-rumen motility may return.

**Postmortem findings:**
1. Adhesions of rumen, reticulum and peritoneum and abscessation
2. Acute or chronic peritonitis
3. Splenic abscessation
4. Traumatic pericarditis
5. Metallic objects such as nails, pieces of wire, magnets etc. in the reticulum
6. Lung abscessation or pneumonia
7. Septic pleuritis
8. Edema of the chest

**Judgment**: Viscera and carcass are **condemned** - a) if the animal is affected with acute diffuse peritonitis or acute infectious pericarditis associated with septicemia; b) carcass with traumatic pericarditis associated with fever, large accumulation of exudates, circulatory disturbances, degenerative changes in organs, or abnormal odor. c) Carcass with chronic traumatic reticulo-peritonitis and/or purulent pericarditis with associated pleuritis, abscessation and edema of the chest.

Chronic adhesive localized peritonitis and chronic pericarditis without systemic changes in well nourished animals allow a **favorable judgment** of the carcass. The affected parts of the carcass and organs are **condemned**.

A carcass affected with infectious exudative pericarditis in a sub acute stage may be **conditionally approved** pending heat treatment, if bacteriological and antibiotic residue findings are negative.

**Differential diagnosis**: Uterine or vaginal trauma, abomasal ulceration with perforation, liver abscessation, Pyelonephritis, ketosis, abomasal displacement and volvulus, and “grain overload”.

TRP. Cross section of the heart reveals thick Fibrinous deposits that encircled heart. Rusty nail has penetrated through the wall of the reticulum into the pericardium in this case

By Dr. Khaled Fujairah Municipality
Lodgment of wire in the reticulum

**BEEF CUT**