

COURSE CODE:	VPT 404
COURSE TITLE:	Special and Avian Pathology
NUMBER OF UNITS:	2 Units
COURSE DURATION:	Two hours per week

COURSE DETAILS:

Course Coordinator:	Dr. S.O. Omotainse. <i>DVM, MVSc., PhD</i>
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Other Lecturers:	Dr. Olaniyi, Moshood Olajire, <i>DVM, MVSC</i> & Dr. Ajayi, Olusola Lawrence, <i>DVM, MVSC</i>

COURSE CONTENT:

Gross and microscopic structure of avian tissues in various diseases of domestic animals and birds in the tropics including trypanosomosis, tuberculosis, Contagious Bovine Pleuropneumonia, Equine encephalitis, African Swine Fever, Canine Distemper, Rabies, Canine Parvovirus, Newcastle Disease, Infectious Bursal Disease, Marek's Disease, Pox and related viral infections.

COURSE REQUIREMENTS:

This is a compulsory course for all DVM students and attendance of at least 75% is required to write the examination.

READING LIST:

1. McGavin, M. Donald and Zachary, F. James. 2007. Pathologic basis of veterinary disease, 4th Ed. Mosby Elsevier.
2. Jubb, K. V. F., Kenedy, P. C. and Perma, M. 1985. Pathology of domestic animals. 3rd Ed. Academic Press, New York.

LECTURE NOTES

TRYPANASOMIASIS

DEFINITION

Trypanosomes are extracellular flagellated protozoan blood and tissue parasites of man and animals transmitted by haematophagous insects from hosts to hosts.

Trypanosomes cause sleeping sickness in human and nagana in animals

Clinical signs:

Intermittent fever, anaemia, emaciation, subcutaneous oedema, weakness, infertility, enlarged lymph nodes, corneal opacity, keratitis, photophobia

Transmission:

Mainly by a) vector: *Glossina spp.* and

(b) Mechanical transmitters: *Tabanids* and *Stomoxys*

Pathogenesis: Some of the species are host-specific while some have wide range of hosts. Some have ability to invade the extravascular spaces and organs eg. *T. brucei* and *T. vivax*.

In the blood they cause anaemia which is the most prominent feature of the disease. Extravascularly, they cause damages in the organs

Histopathology:

- aggregates of parasites (*T. vivax*, *T. congolense*) in capillaries of the blood vessels (coronary, skeletal and brain: -polio-encephalomalacia).
- Necrosis in tissues, organs and surfaces (Pericardial and serosal surfaces of organs).
- Erythrophagocytosis by the mononuclear phagocytic system throughout the reticulo-endothelial system.

Gross lesions:

- Emaciation, pale mucous membrane
- Oedema, congestion, necrosis and enlargement of viscera and lymph nodes.
- Bone marrow may (red BM) or may not expand, depends on the stage of the disease.
- Brain (Nervous): non-suppurative meningitis and encephalitis.

Differential Diagnosis:

- Helminthiasis, Mal nutrition, babesiosis, anaplasmosis,

HEPATOZOONOSIS:

- Protozoan parasite. Notably: *Hepatozoon canis* (*Hepatozoon americanum*) infects dogs, cats (domestic and wild).

Transmission – By host's ingestion of infected ticks.

Clinical signs:-Fever, anaemia, splenomegaly, emaciation, pain, abnormal gait and paralysis.

- Death in 4 – 8 weeks after the onset of clinical signs.

Lesions: - Related to anaemia

- Suppurative/ granulomatous inflammation within invaded tissues
- Parasites in the monocytes and Peripheral neutrophils.

Diagnosis:- By demonstration of parasites in the WBC or in the affected tissues.

BABESIOSIS

☒ Tick fever ☒ red water ☒ Piroplasmosis

This is a tick-borne disease of animals caused by protozoan of the genus *Babesia*.

Clinical signs:

Incubation-1–10days.

- High fever (up to 41.50c), dehydration, jaundice.
- dark reddish brown urine terminally
- reddened membrane early stages.
- Nervous sign (ataxia, coma)
- Mortality – up to 50% depending on age and breed
 - due to circulating failure.
- Pale carcass,
- Oedematous and enlarged lungs,
- Haemolytic anaemic (severe anaemia) depend on severity.
- Haemorrhages in internal organs
- splenomegaly in *B. bovis* and *B. bigemina* infectious.
- Icterus of all serous membrane.
- Hepatomegaly and yellowish-brown liver.
- Enlarged kidney and lymph nodes.
- Cherry-pink grey matter of the cerebrum and cerebellum in cattle.

Histological:-

1. degeneration and necrosis of the renal tubular and accumulation of hyaline or granular casts in tubular lumen.
2. Centrilobular hydropic/fatty degeneration of hepatocytes; centrilobular and midzonal hepatic necrosis and bile stasis.
3. Congestion of the splenic sinusoids.
4. Oedema, congestion of the lymph nodes and depletion of the lymphocytes in the germinal centers.
5. Congestion and distension of brain vessels with rbc.
6. Myocardial haemorrhages and hyaline degeneration
7. Skeletal muscle degeneration.

Diagnosis: by Clinical signs, pathology and serology (eg. Radio-immuno assay, ELISA).

Note – a brain crush preparation will often demonstrate parasitized rbc in the brain capillaries.

- Blood smear may also demonstrate the parasites in rbc.

Differential diagnosis: -- Haemoglobinuria by *Cl. haemolyticum*

- Anthrax

- Pasteurellosis

CANINE EHRLICHIOSIS

- an infectious tick-borne disease of dogs characterized by recurrent fever and nervous symptoms.

Aetiology: *Ehrlichia canis*

Transmission: By tick, *Rhipicephalus sanguineus*

Pathogenesis

The organism multiplies in the reticulo-endothelial cell, lymphocytes and monocytes.

Clinical signs: The disease is usually mild except in puppies or with concurrent sign like Babesiosis. Puppies, signs include fever (recurrent), serous nasal discharge, photophobia, vomiting, splenomegaly and sign of nervous involvement (stable mental status)

Pathology

Microscopically: reduction in mature lymphocytes and pronounced hyperplasia of the immature lymphocytes and reticulo-endothelial cells of the spleen and lymph nodes, presence of increased numbers of lymphoreticular cells in organs. Perivascular cuffing of the CNS vessels, focal hepatic necrosis and interstitial pneumonia. Organisms can be demonstrated in the cytoplasm of monocytes.

Gross: Splenomegaly ± enlarged lymph nodes, haemorrhages on serosal and mucosal surfaces.

HEART WATER DISEASE

- a tick borne disease of ruminants, characterized by the presence of hydropericardium.

Aetiology: *Cowdria ruminantium*, an intracellular organism transmitted by ticks.

Transmission: by ticks = *Amblyomma*

The organism is seen in epithelial cells of the jugular vein, vena cava, renal glomerula capillaries and the cerebral gray matter.

Clinical signs: High fever, nervous sign after a 1 – 4wks incubation period. Chewing movement, twitching of muscles and eyes, trembling charge at trees or fences, diarrhea in cattle, sudden drop in temperature before death.

Pathology: Respiratory and haemocardiopathy, lymphadenopathy, haemorrhagic or catarrhal enteritis.

Differential diagnosis – Anthrax, rabies, babesiosis, listeriosis.

LUNG WORMS of cattle

Dictyocaulus viviparus - lungworm of cattle characterized by bronchitis (husk or hoose). Also affects pigs, mouse and deer.

Transmission and Pathogenesis

Larvae ingestion and migration from the intestine through vessels and organs. By 3 – 6wks of ingestion, they reach the lung where they get mature and lay eggs.

Clinical signs: - Elevated temp. (40 – 41⁰C)

- Shallow rapid breathing (tachypnoea) which later leads to
- dyspnoea: hacking cough, especially in the young animals.
- Nasal discharge, grunting,
- Cyanosis and recumbency.

Gross pathology:- Haemorrhagic inflammation of bronchi with froth.

- Lung oedema and emphysema in unconsolidated part.,
- Consolidation of the lung parenchyma.
- Presence of lungworms (nodules in mucosal)
- Enlargement of the lung lymph nodes

Histology:- Haemorrhages in the alveoli of affected parts

- Occlusion of the bronchioles/alveoli with leucocytes
- Secondary bacterial infection leading to purulent inflammation (pneumonia)

Other lungworm

Dictyocaulus filaria - ruminants

Aelurostrongylus abstrusus – cat

Filaroides osleri - Dog

Dictyocaulus amfieldi – Horse, donkey

Capillaria aerophila - cats

Metastrongylus apri -Swine

M. pudendotectus

M. salmi - swine

Protostrongylus sps - Rabbit

Crenosoma sps – cat and dogs.

THEILERIOSIS ☑ East Coast fever.

- A protozoan tick borne disease of cattle characterized by pyrexia, dyspnoea and enlarged lymph nodes.

Aetiology: *Theileria parva*

Transmission: By both the nymphs and adults of *Rhipicephalus* and *Hyalomma sps* of ticks.

Pathogenesis: Parasites divide in the rbc (as tiny rod or comma or ring-shaped); reproduce in lymphocytes or histiocytes in lymph nodes, spleen and liver.

These cytoplasmic aggregates in these cells (called **Koch's bodies**) are characteristic of the disease.

Clinical signs: Incubation period - about 15 days post exposure to tick, loss of appetite and rumination. Reduction in milk production, rough hair coat, dry muzzle and enlarged superficial lymph nodes, dyspnea and death.

Gross Pathology: Most common-generalized enlarged lymphnodes, pulmonary oedema, emphysema, submucosal and intramuscular oedema, spleen may be normal, hydrothorax and hydropericardium, hepatomegaly with yellowish and mottled appearance of the liver.

Histology: Proliferation of lymphocytic cells (lymphoid aggregates white spots) in the lymphnodes, spleen, Payer's patches, demonstration of Koch's bodies in tissues may be present, responsive bone marrow-due to anaemia.

Diagnosis by clinical signs + pathology + demonstration of KOCH's bodies.

Differential Diagnosis – Babesiosis.

LIVER FLUKE (FASCIOLIASIS)

Occasionally may wander into and produce lesion like abscess in the lung and other tissues. Primarily, the immature fluke produce lesions in the liver where they spend up to 6 – 7 weeks wandering and destroying the parenchyma.

Lesions:- Necrosis and inflammatory reactions and growth disturbances around the parasites/eggs in organs; depressed haemopoiesis (Normocytic, normochromic anaemia).

CESTODIASIS (CYSTICERCOSIS)

Development of connective tissue around the bladder worm in the heart muscle, voluntary muscles, oesophagus and rarely the lungs of sheep and goats in addition, nervous tissues of dogs (*Coenurus cerebralis*, the larval stage of *Multiceps multiceps* of dogs), liver, mesentery and omentum (ruminants and rodents)

ASCARIASIS

(*Ascaris columnaris*, *Toxocara canis*, *Toxascaris leonina*)

Lesion produced are determined by the migratory pattern the larvae follow:

1. Tracheal migration: Penetration of the infective larvae through the intestine to the liver, lung, (alveoli wall) to the bronchi, trachea and swallowed into the intestine.
Eg. *Ascaris lumbricoides*, *A. I. suis*, *Parascaris equorum* and *toxocara canis*.
2. Penetration of larvae through the intestinal wall through somatic tissues e.g. *Toxocara canis*.
Neoascaris vitulorum (cattle).
3. Larvae penetration of the intestinal wall and there they develop to adult worm before returning to the lumen of the intestine. Eg. *Toxascaris leonine* and *Toxocara cati*.

Therefore, apart from the intestinal wall, liver, and lung ascarid larvae can penetrate various tissues of the host, where they may cause tissue damage.

PM: Gross: Bile duct obstruction, icterus e.g. in pork; obstruction of the intestinal lumen particularly in young animals, migratory (necrotic) tracks of larvae in organs.

Histology: In the liver, area of necrosis mostly affected is the portal areas, where the larvae may be identified with severe inflammatory responses involving neutrophils, eosinophils, lymphocytes with a central mass of caseous necrotic tissue. Similar but with less inflammation response could be seen in the lungs; here there could be loss of bronchiolar epithelium and leucocytic infiltration. Other sites in the body would lead to granulomatous reaction (nodules) including the eyes and nervous tissues.

OESOPHAGOSTOMUM

The larvae of *Oesophagostomum radiatum* and *Oes. columbianum* penetrate the intestinal mucosae to form cysts in the lamina propria. There they produce inflammatory reaction before molting to adult and re-enter the intestinal lumen. This brings about accumulation of large numbers of neutrophils, eosinophils, lymphocytes and macrophages in the submucosa and adjacent mucosa. The centre with dead larvae can be caseated and or calcified.

The entire lesion is encapsulated to preserve nodular features. With secondary infection, it could lead to peritonitis.

OTHER WORMS OF IMPORTANCE

1. *Dirofilaria immitis* – cardiovascular system.
(Heart worm disease of dogs) – hepatomegaly, cardiac enlargement.
 - Aneurysm of the aorta.
 - Haemorrhages, congestive heart failure.
 - Hyperplasia of the heart muscles and hypertrophy of veins/arterioles, obstruction and thrombi-vena cava, hepatic circulation.
2. Spirocercosis
3. Capillariasis
4. Filariasis – Poll evil and fistulous wither: *Onchocerca armillata*, *O. gutturosa* and *O. gibsoni*.

FOOT AND MOUTH DISEASE (FMD)

By Dr. S. O. Omotainse

FMD -A contagious and an epitheliotropic viral disease of ruminants, and swine.

Aetiology: Picorna virus :- A, O, C, SAT-1, SAT-2, SAT -3 and Asia-1. They all cross react with each other. Transmission is by oral ingestion.

Clinical signs: - Excessive salivation, anorexia, smacking of the lips and tongue, erosion of the skin and tongue, lameness, presence of lesions on the less hairy parts of the body (udder, vulva), conjunctivitis. In young animals, acute gastroenteritis and myocarditis, high mortality which may not be more than 5% in adults.

Pathogenesis: Oral epithelium – blood stream – epithelial tissues all over the body –form vesicles.

Pathology:

Grossly:-

Oral mucosa -1/3rd dorsum of the tongue affected as well as the hoof and brisket; vesicle formation leading to bullae

- raw red haemorrhagic surfaces (erosion)
- myocardial degeneration and necrosis
- “**Tiger Heart**” appearance of the cardiac muscle
- similarly seen in skeletal muscles

Histology :

- balloonic degeneration of the stratum spinosum
- Oedema intracellularly
- hyaline degeneration with lymphocytic infiltration
- leucocytic infiltration with liquefaction
- Dissociated epithelial cells with pyknotic nuclei

Differential diagnosis:-

Vesicular exanthema

Vesicular stomatitis

RINDERPEST

-Also known as Cattle plague

-a pantropic viral disease of ruminants (Cloven hooved)

-characterized by fever, necrotic stomatitis, gastroenteritis, lymphoid necrosis and high mortality

Aetiology:- Morbillivirus closely related to PPR, Canine distemper and measles. The virus is shed in body secretions including the nasal and rectal.

Transmission:- via nasopharyngeal to the regional lymph nodes where they proliferate and enter the blood stream and spread to the mucosae of the GIT and upper respiratory tract. The virus causes cytological lesions

Clinical signs:- fever after 3-15 days incubation period,

-anorexia

-Depression

-naso-ocular or oculo-nasal discharge

-leucopenis

-constipation

-pin-point necrosis coalescing to form cheesy plaque on the gum, buccal cavity mucosa, ventral part of the tongue, the hard and soft palates, dry and cracked muzzle, diarrhoea, abdominal pain, dyspnoea and dehydration.

-death could range between 25%-90% (average of about 50%).

Pathology:-

Grossly: necrosis and erosion, congestion and haemorrhages on the GI tract (necrotic stomatitis and oesophagitis, ulcerative haemorrhagic enterocolitis) and respiratory tract mucosae; **"Zebra-striping"** in the rectum; enlarged and oedematous lymph nodes, necrotic foci on the Peyer's patches.

Histology: lymphoid and epithelial necrosis; necrosis of the stratum germinatum with pyknotic nuclei and eosinophilic cytoplasm. Giant cells with intracytoplasmic inclusion bodies with or without INIB especially in the lymph nodes; -No vesicle formation; -Rare ulceration, -submucosal oedema and congestion of abomasum and intestines.

Diagnosis:- Unfrozen blood/ serum and body fluid (lymph node aspirate) on wet ice to the laboratory for viral isolation

- Spleen, lymph nodes and tonsil for histology in 10% formalin

Control:- Vaccination of herd

-slaughter of infected animals and burn or bury deeply, quarantine during outbreaks.

Differential diagnosis:- FMD, CBPP, PPR, Mucosal disease, bovine viral diarrhoea, malignant catarrhal fever.

KATA: Peste des petite ruminant, PPR (Pneumoenteritis complex)

Morbillivirus- in goats and sheep mainly; by inhalation and ingestion

Post Mortem Lesions

• Inflammatory and necrotic lesions

-nasal cavity/ Oral cavity-oedema and erosion

- Throughout GI tract

-diarrhoea

• Emaciation of the carcass

• "Zebra stripe" lesions of congestion in large intestine

Post mortem lesions are similar to rinderpest, with inflammatory and necrotic lesions in the oral cavity and throughout the GI tract.

• In severe cases the hard palate, pharynx and upper esophagus also have lesions.

- Bronchopneumonia and other respiratory lesions with consolidation and atelectasis occurs frequently (consolidation of the anterior lobe, pulmonary abscess, and adhesion to the ribs
 - Contracted spleen
 - Pale friable liver
- The lymph nodes are generally congested, enlarged and edematous.
- Lesions similar to Rinderpest

The most severe lesions are seen in the large intestine, with congestion and

“zebra stripes” of congestion on the mucosal folds of the posterior colon including the rectum.

Erosive lesions may also occur in the vulva and vaginal mucous membranes.

Congestion and enlargement of the spleen may be seen.

Histology:

Acute:

-necrosis and ballooning degeneration of the stratified squamous epithelium

-ulceration

Chronic:

-proliferation of the labial

-Hyperkeratosis

-fibroblast and macrophage infiltration

-presence of giant cells with intranuclear inclusion bodies in the epithelial cells of the tongue of the animals.

-degeneration and necrosis of the glandular cells of the small intestines

-thickened epithelium and glandular degeneration and presence of polymorphonuclear cells and eosinophilic intranuclear inclusion bodies in the glandular cells.

-necrosis of the tracheal wall with necrotic debris; vacuolation and hyperplasia of the epithelial cells with INIB and ICIB

-giant cell pneumonia, hyperplasia of the bronchial and bronchiolar epith

-accumulation of desquamated necrotic debris with PNM and MQs in the bronchial lumen, with INIB and ICIB in the epith. Cells

-Thickened alveolar wall with PNM and MQs, lymphocytes, plasma cells and giant cells.

-congestion of the spleen

-multifocal coagulative necrosis with vacuolation of hepatocytes

-mild congestion of the brain

Differential Diagnosis

- Rinderpest
- Contagious caprine pleuropneumonia
- Bluetongue
- Pasteurellosis
- Contagious ecthyma
- Foot and mouth disease
- Heartwater
- Coccidiosis
- Bacterial pneumonia
- Nairobi sheep disease
- Mineral poisoning

The case history, geographic location and the combination of clinical signs can help differentiate some of these diseases.

CANINE DISTEMPER

A multisystemic (pantropic) viral disease of dogs and dog's Family (*Canidae*- Wolves,

ferrets, etc)

- Clinical signs**:- Diphasic fever curve: acute- after 7-8 days of incubation lasting for about 3-4 days; second phase takes place in 11th -12th days
- dullness and redness of the eyes (conjunctivitis)
- Discharge from the nose
- vomiting and diarrhoea
- Cough
- Shivering
- loss of appetite (anorexia) and energy
- weight loss
- seizures or paralysis or ataxia
- thickened foot pads
- tooth enamel hypoplasia
- photophobia

Transmission is by air (aerogenous) or oral

Lesions:-

Respiratory:- purulent or catarrhal exudate on the nasal and pharyngeal mucosae. Microscopically there is characteristic eosinophilic intracytoplasmic and intranuclear inclusion bodies in the exudative cells

-pulmonary congestion and consolidation leading to focal pneumonitis: purulent bronchopneumonia with the bronchi and alveoli filled with PMN, mucin and tissue debris. In acute case the exudate may be haemorrhagic.

Microscopically, the alveoli may be filled with exudates. The bronchial lining and alveolar septae may be with multinucleated giant cells.

The Skin:- vesicular and pustular dermatitis confined to the malpighian layer of the epidermis, congestion of the dermis with lymphocytic infiltration.

-ICIB and INIB may be present within the epithelial cells eg. Of the sebaceous gland.

- proliferation of the keratin layer of the dermis especially of the foot pad leading to "hard"-pad

Urinary epithelium (Renal pelvis and bladder) may show vascular congestion; INIB in the epithelial cells, microscopically.

Stomach and intestines: grossly, catarrhal exudates, congestion. Microscopically: Inclusion bodies in epith. Lining; lymphocytic infiltration of the lamina propria.

Spleen:- grossly, enlarged; microscopically, necrosis of the lymphoid cells in the spleen follicles

Blood:- Inclusion bodies in circulating PMNs and lymphocytes,

Eyes:- Inclusion bodies in conjunctiva cells

Liver:- Inclusion bodies in the biliary epithelium

CNS:- affinity for the myelinated portions of the brain and spinal cord. The neurons are not primarily affected. Mostly affected are cerebellum and the white columns of the spinal cord.

Microscopically, spongy appearance (status spongiosa) with microglia and astrocyte infiltration, perivascular cuffing around the Virchow-Robin spaces. Inclusion bodies in the astrocytes and microglia cells, neuronal necrosis and degeneration, gliosis, non-suppurative leptomeningitis.

Differential diagnosis:-

Leptospirosis

Infectious canine hepatitis

Toxoplasmosis

Coccidiosis

Organophosphate poisoning

Diagnosis:- Clinical signs, Postmortem, Immunofluorescence,

SPECIAL PATHOLOGY (BACTERIAL DISEASES)

- ANTHRAX
- DERMATOPHILOSIS
- CLOSTRIDIAL DISEASES
- TUBERCULOSIS
- SALMONELLOSIS
- LEPTOSPIROSIS
- JOHNE'S DISEASE
- MYCOPLASMOSIS(CBPP & CCPP)
- PASTEURELLOSIS
- BRUCELLOSIS
- LISTERIOSIS
- COLIBACILLOSIS

ANTHRAX

Synonyms: Woollsorters' Disease, Cumberland Disease, Charbon, Malignant Pustule, Malignant Carbuncle, Milzbrand, Splenic Fever

Etiology: Anthrax results from infection by *Bacillus anthracis*, a spore forming, Gram positive aerobic rod (family Bacillaceae).

Geographic Distribution: Anthrax can be found worldwide; it is particularly common in parts of Africa, Asia and the Middle East.

Species Affected: Many species can develop anthrax but susceptibility varies: dogs, rats and chickens are resistant to disease while sheep, cattle and horses are very susceptible. Anthrax has been seen in pigs, mink, cats and dogs fed contaminated meat.

Transmission

In animals, transmission is usually by ingestion. Herbivores usually become infected when they ingest spores on plants in pastures. Outbreaks typically occur in neutral or alkaline calcareous soil and are often associated with heavy rainfall, flood or drought; under optimal levels of moisture, temperature and other conditions, spores in the soil can revert to the vegetative form and grow to infectious levels. Contaminated bone meal and other feed can also spread this disease. Carnivores usually become infected after eating contaminated meat. Vultures and flies may spread anthrax after feeding on carcasses. In infected animals, large numbers of bacteria are present in the hemorrhagic exudates from the mouth, nose and anus; when they are exposed to oxygen, these bacteria develop endospores and contaminate the soil. Sporulation requires oxygen and does not occur inside a closed carcass; opening an infected carcass for necropsy should be avoided. Anthrax spores can remain viable for decades in the soil or animal products such as dried or processed hides and wool. Spores can also survive for 2 years in water, 10 years in milk and up to 71 years on silk threads. Vegetative organisms are thought to be destroyed within a few days during the decomposition of unopened carcasses.

Humans usually develop the cutaneous form of anthrax after skin contact with infected animal tissues such as hides, wool, bone meal and blood. Biting flies that feed on infected animals or carcasses may also be able to transmit this form. Inhalation anthrax is seen after inhalation of spores from contaminated dust or animal products. Intestinal anthrax results from the ingestion of contaminated meat containing viable spores.

Large numbers of bacteria are present in the carcass and in bloody discharges from body openings. Tissues including skin and wool can contain spores, which remain viable for long periods of time. Anthrax spores are resistant to heat, sunlight, drying and many disinfectants. Spores can be killed with 2% glutaraldehyde formaldehyde or 5% formalin; soaking overnight is recommended. A 10 % NaOH or 52 % formaldehyde solution can be used for stockyards, pens and other equipment. Sterilization is also possible by heating to 121°C for at least 30 min. Blowtorches can be used to disinfect buildings. Exposed arms and hands can be washed with soap and hot water then immersed for one minute in a disinfectant such as an organic iodine solution or 1 ppm. solution of mercuric perchloride.

Clinical Signs

In ruminants, sudden death may be the only sign. Staggering, trembling and dyspnoea may be seen in some animals, followed by rapid collapse, terminal convulsions and death. In the acute form, clinical signs are apparent for up to 2 days before death. Fever and excitement may be followed by depression, stupor, disorientation, muscle tremors, dyspnoea, abortion, congested mucous membranes and bloody discharges from the nose, mouth and anus. Chronic infections, characterized by subcutaneous edematous swellings, are also seen; the ventral neck, thorax and shoulders are most often involved. This swelling may be widespread.

In horses, common symptoms include fever, chills, anorexia, depression and severe colic with bloody diarrhea. Swellings may be seen in the neck, sternum, lower abdomen and external genitalia. Affected animals usually die within 1 to 3 days but some animals can survive up to a week.

In Pigs, Sudden death may be seen in pigs. Many pigs have mild chronic infections characterized by localized swelling, fever and enlarged lymph nodes, with eventual recovery. Some animals develop progressive swelling of the throat, with dyspnoea and difficulty swallowing; these animals may suffocate. Intestinal involvement, with anorexia, vomiting, diarrhea or constipation, is less common. Recovered, asymptomatic animals may have signs of localized infections in the tonsils and cervical lymph nodes at slaughter.

Clinically apparent anthrax in dogs, cats and wild carnivores resembles the disease in pigs.

Post-Mortem lesions

Rigor mortis is usually absent or incomplete and the carcass is typically bloated and decomposes rapidly. Dark, tarry blood may ooze from the body orifices. Edema may be noted, particularly around the throat and neck, in horses. **Necropsies should generally be avoided, to prevent contamination of the surrounding area with spores.**

If the carcass is opened, signs of septicemia will be evident. The blood is dark, thick and does not clot readily. Edematous, blood-tinged effusions may be seen in the subcutaneous tissues, between skeletal muscles and under the serosa of organs. Hemorrhages, petechia and ecchymoses are often noted in the lymph nodes, abdomen and thorax; hemorrhages and ulcers are also common in the intestinal mucosa. Peritonitis and excessive peritoneal fluid may be seen. The spleen is usually enlarged and has a 'blackberry jam' consistency. The lymph nodes, liver and kidneys may be swollen and congested. Pigs with chronic anthrax usually have lesions only in the pharyngeal area. The tonsils and cervical lymph nodes are typically enlarged and a mottled salmon to brick-red color on cut surface. The tonsils may be covered by diphtheritic membranes or ulcers. The surrounding area is usually edematous and gelatinous. Some pigs may have a chronic intestinal form, with inflammation and lesions in the mesenteric lymph nodes.

Diagnosis

A presumptive diagnosis is often made by examining blood or other tissues for the characteristic bacteria. Blood clots poorly in anthrax cases and sampling may be done post-mortem. In pigs, bacteremia is rare and a small piece of aseptically collected lymphatic tissue is often used. *Bacillus anthracis* is a large Gram positive rod that may occur singly, in pairs or in chains; endospores are not formed inside the body but may be found under certain culture conditions. Other diagnostic methods include polymerase chain reaction to detect bacterial nucleic acids, immunofluorescence for bacteria in blood or tissues, or a chromatographic assay to detect antigens in the blood.

DERMATOPHILOSIS

Also known as Streptothricosis and "Kirchi"(Local name)

Predominantly a disease of cattle, sheep and horses. Dogs, cats pigs and goats may also be infected.

Aetiology: *Dermatophilus congolensis*

The condition is more common in tropical countries especially during wet weathers. Lesions are usually found around the dorsum of the back and distal extremities.

Pathogenesis: Infection may accompany epidermal irritation from ectoparasites trauma or prolonged wetting of the skin. It is important to note the lesions of dermatophilosis are strictly cutaneous usually involving mainly the epidermis. The organisms proliferate in the outer root sheath of the hair follicle and the superficial epidermis. The filamentous branching organisms multiply and stimulate an acute inflammatory response in which neutrophils migrate into the skin forming micro abscesses. . Repeated cycles of bacterial growth, inflammation and epidermal regeneration result in the formation of multi-laminated pustular crusts.

Pathology: What is observed grossly is a combination of papules, pustules, and thick crusts which may coalesce and mat the hair together. The crusts are easily removable leaving a raw surface. The crust is rich in the invading organisms. Microscopic lesions are those of a hyperplastic superficial dermatitis with presence of multi-laminated crusts of alternating layers of keratin and inflammatory cells.

Diagnosis: Usually based on a combination of the observation o the characteristic lesions disease epidemiology and demonstration of the filamentous branching organisms.

CLOSTRIDIAL DISEASES

A group of diseases caused by various species of Gram positive spore forming bacilli called *Clostridium*. Clostridial organisms are ubiquitous and abundant in the soil. They also form part of the gastro-intestinal micro flora of most domestic animals. In certain conditions especially when the anaerobic condition which enables germination of spores is provided, the organisms proliferate and elaborate toxins which may precipitate clinical disease.

Clostridium perfringens Type A enterotoxins have been associated with gas production in "Gastric dilation and volvulus syndrome" in dogs and also "Gastric dilatation" in Horses. Other specific diseases in which various strains and species of Clostridia have been incriminated include:

Blackleg

Caused by *Clostridium chauvoei*. It is a common disease of beef cattle. The most common presentation is acute death without premonitory signs due to severe toxemia

C. Chauvoei spores are capable of crossing the intestinal mucosa after ingestion to localize in skeletal muscles. The spores lie dormant until localized trauma to the muscle results in local hypoxia which enables the spores to germinate and elaborate their toxins. The toxins cause capillary necrosis, hemorrhage and edema and necrosis of adjacent muscle fibers. The lesions are usually locally extensive with crepitus due to gas bubbles in affected muscles, fascia and overlying subcutaneous tissue. Necrotic muscles appear dark red and may be wet and exudative initially, and later, may become dry. Cardiac muscles may also be involved and in such cases, the organisms cause hemorrhagic myocarditis.

Malignant Edema

This condition is usually observed in horses and less frequently cattle. It varies due to contamination of deep penetrating wounds by Clostridial organisms especially *C. perfringens*, *C. septicum* and *C chauvoei*.

Clinical signs of heat, swelling and pain within a muscle group are usually observed at the onset. Fever, depression, dehydration and anorexia may later follow.

The organisms in the anaerobic environment proliferate and elaborate toxins which damage blood vessels causing edema and hemorrhage. The toxins also cause necrosis of adjacent muscle fibers. The affected muscles are usually swollen, necrotic, edematous and hemorrhagic with gas production. Bubbles due to gas production may be present.

Bacillary Haemoglobinuria

An acute and highly fatal disease of cattle and sheep, commonly observed in areas where liver fluke is endemic. This condition is caused by *Clostridium haemolyticum*.

The spores of the organisms, when ingested by the animals, reside within Kupffer cells where they remain until the enabling environment for their germination is created. The low oxygen tension required for sporulation may be created by migrating liver fluke, other parasites or local necrosis from liver biopsy. The bacteria subsequently proliferate and elaborate toxins, the most important of which is **phospholipase C**. This toxin induces severe hepatocellular necrosis and intravascular hemolysis. Affected animals manifest icterus, hemoglobinuria and hemoglobinemia. Focal areas of hepatic necrosis are observed in the liver. Excessive accumulations of straw colored or blood tinged, fibrin-laden fluid are also usually observed in serous cavities.

Infectious Necrotic Hepatitis

Also known as 'Black disease'. More common in sheep and cattle, but also observed in swine and Horses. The pathogenesis is similar to that of Bacillary hemoglobinuria i.e associated with germination of dormant spores of *Clostridium novyi* Type B in areas of lowered oxygen tension and subsequent elaboration of toxins.

The toxins in this case cause one or more discrete areas of coagulative necrosis in the liver seen as discrete pale areas of variable sizes surrounded by a zone of intense hyperemia. Migratory tracts are also seen within the liver. Fluid accumulations within pericardial, pleural and peritoneal cavities may also be observed.

Tyzzer's Disease

Caused by *Clostridium piliformis*. A disease of foals, cats, calves and dogs. Seen in very young and immuno-compromised animals.

Characterized by enlarged edematous and hemorrhagic abdominal lymph nodes. Enlarged and necrotic liver. Randomly distributed pale foci of necrosis are usually observed in the liver. The characteristic elongated bacilli may be demonstrated within hepatocytes or at the margin of the necrotic foci.

Botulism

Condition is not as a result of infection with *Clostridium botulinum* but rather due to the effect of its toxins which may be ingested whole without the organisms or produced in the gut or contaminated wounds. Horses are extremely sensitive to Botulinum toxins. The hallmark of botulism is generalized flaccid paralysis. Toxins produced by *C.botulinum* bind irreversibly to the pre-synaptic nerve terminals preventing release of acetyl choline thus causing flaccid paralysis.

No specific gross or histopathologic findings are associated with the condition although aspiration pneumonia due to dysphagia may be observed.

Tetanus

Also known as Lockjaw. It is an acute, infectious disease resulting from an intoxication of the nervous system with the exotoxin of *Clostridium tetani*, and characterized by spasmodic contractions of the entire body musculature or single groups of muscles without impairment of consciousness

Pathogenesis; Spores enter wounds especially puncture wounds the organisms in the anaerobic microenvironment multiply rapidly and elaborate an exotoxin (tetanospasmin). The toxin action resembles those of strychnine. It suppresses all types of synaptic inhibitions.

The animals, when agitated by movement or noise immediately go into a state of continuous muscular rigidity.

The incubation period may be as short as 24hrs and may extend up to 2weeks.

No Gross CNS lesions are seen.

Death is usually due to asphyxiation due to interference with respiratory and cardiac functions.

CLOSTRIDIAL ENTERITIS

Clostridium perfringens is a Gram positive anaerobic rod that is a normal inhabitant of the gastrointestinal tract.

<i>C perfringens</i>	Type A	Alpha toxins
Type B		Alpha Beta and Epsilon Toxins
Type C		Alpha and beta toxins
Type D		Epsilon toxins

Type E

Alpha and iota toxins

All of these toxins are exotoxins, proteins, some of which are pro-enzymes and some actually have enzymatic activities.

Enterotoxigenic strains of *C. perfringens* are responsible for clostridia food poisoning. All clostridia enteritides are enterotoxaemias.

Enterotoxaemia

Enterotoxaemia is produced by one of the five toxigenic *C. perfringens* types. Condition usually affects young fat animals. Outbreaks usually follow a change in feed or an increase in the carbohydrate content of feed. A change in feed or overfeeding precipitates an alteration in the balance in the bacterial flora of the intestine. An overgrowth of *C. perfringens* produces abundant toxins.

Clinical signs of enterotoxaemia include;

Diarrhea with dark brown or black bloody faeces, anorexia, lethargy tachycardia, dilated atonic abdomen, dehydration, prostration and death

Lesions seen in the small intestine include petechiae, ecchymoses and paintbrush hemorrhages of the serosa and mucosa. The intestines are flaccid and thin walled, dilated and often gas-filled.

Gastric hyperemia, excessive pleural and peritoneal fluid. Are also observed in affected animals Cooked appearance of skeletal musculature with enlarged and pulpy spleen may also be observed.

Pulpy Kidney Disease

Also known as Over-eating disease or *Clostridium* Typed D Encephalopathy

Caused by *C. perfringens* Type D. Affects fattening sheep, goats and calves It is diet- related and associated with grain overload or overeating. The disease is often characterized by unexpected death, sometimes preceded by central nervous system signs or blind staggers.

Clostridium perfringens Type D enterotoxaemia associated with epsilon toxin production is a disease of sheep, goats and cattle. But only sheep commonly exhibit the neurological form of the disease. Gross lesions are usually absent in per-acute cases.

In acute cases, focal, bilaterally symmetrical yellow-gray to red areas of encephalomalacia may be seen in the internal capsule, basal nuclei, thalamus, hippocampus, midbrain, cerebellar peduncles and pons.

Lesions in other organs include pulmonary congestion and edema, serous pericardial effusions petechiations, and soft (pulpy) kidneys.

Microscopically, acute lesions are those of vascular damage such as perivascular edema, hyalinization of walls of arterioles, peri-capillary hemorrhages especially in the brain. Other changes include acute necrosis of neurons, accumulation of neutrophils and foamy macrophages with lymphocytic perivascular cuffing in the brain.

TUBERCULOSIS

A chronic communicable disease of domestic animals and man.

Aetiology:

Mycobacterium tuberculosis

primarily human host

Mycobacterium bovis

primarily bovine host

Mycobacterium avium-intracellulare

primarily avian host

Tuberculosis is an ancient disease of worldwide importance. It used to be a major problem in underdeveloped countries. But now due to the scourge of AIDS and HIV, infection is on the rise in developed countries. The disease is of zoonotic importance as cross-species infections do occur among the etiological groups. All the three species are infective to cattle and man.

Transmission: Animals can be infected with any of the agents through several routes.

Two routes are prominent i.e

- Ingestion of infected milk (common in young animals)
- Inhalation (common in adults).

Clinical signs: The clinical signs are non specific and are mainly the signs of general debilitation. Other signs include weight loss, emaciation, reduced milk production, and generalized lymph node

enlargement. Organ dysfunction is also noticed depending on the affected organs. In the pulmonary form, a chronic moist cough is usually observed

Pathogenesis: Respiratory infection starts with inhalation of bacilli and subsequent phagocytosis by alveolar macrophages. Infection may be abolished at this stage, if the macrophages succeed in killing the bacterial organisms. On the other hand, the organisms may multiply intracellularly, kill the macrophages and initiate infection. The bacilli spread throughout the lungs and airways. The lymph vessels aid the spread of the bacilli to the regional lymph nodes (i.e the tracheobronchial and mediastinal lymph nodes).

The extent of spread of infection thus far is known as “the primary (Ghon’s) complex of tuberculosis”. If the infection is not contained within the primary complex, dissemination to distant organs and other lymph nodes occur through the lymphatics, or hematogenously. Sudden massive dissemination of bacilli may lead to development of small foci of infection referred to as millitary tuberculosis.

Pathology: The basic pathological manifestation of tuberculosis is the presence of typical granulomatous lesions in various organs. All the organs of the body are available for invasion by the organisms. Presence of a few / many caseated granulomas which may some times be hard and calcified, eliciting gritty sound on cutting.

Histopathology: The young tubercles without any cell mediated immune response typically present with non-caseated tubercles of various sizes. Sometimes the tubercles may appear confluent. The typical tubercle is made up of definite arrangement of mononuclear cells (epithelioid cells and Langhan’s giant cells are at the center surrounded by lymphocytes, plasma cells and macrophages).

Later, after the cell-mediated immune response, the center becomes necrotic with presence of thick fibrous connective tissue at the periphery.

The necrotic center may later on become calcified. Acid-fast organisms are easily demonstrable at the center of the lesion.

Diagnosis: usually based on a combination of clinical signs and typical lesions. The organisms can be cultured using Lowensstein – Jensen medium. Organisms can be demonstrated in smear with Ziehl Niehlsen stain.

SALMONELLOSIS

This disease is a significant cause of acute and chronic diarrhea and deaths in numerous animal species and human beings. There are numerous species of Salmonella organisms, but those of disease significance are:

S. typhimurium, S. enteritidis, S.dublin, S. cholerasuis and S. typhosa.

Salmonella organisms are Gram negative, motile bacilli. They are aerobes or facultative anaerobes.

In carrier animals, organisms reside in gall bladder, intestinal tract and mesenteric lymph nodes.

Pathogenesis:

Infection is usually acquired via ingestion or contamination of feed and water. Tonsils and Peyer’s patches are portals of entry for some species of salmonella while others either colonize the intestines or invade and enter the epithelium and subsequently become phagocytosed by macrophages of the intestinal mucosa. Disease is produced via the elaboration of enterotoxins, cytotoxins (verotoxins) and endotoxins. The organisms may from this point get to the regional lymph nodes by the macrophages, and also to the liver through portal circulation.

The organisms may also colonize the small intestine, colon, mesenteric lymph nodes and gall bladder.

Young animals	Generally succumb to septicemia
Horses.....	Acute fatal colitis
Cow.....	A lingering febrile diarrhea and passage of pseudo-membranes.
Calves.....	Acute diarrhea
Dogs.....	Sudden bouts of acute diarrhea

Cats.....	Febrile enterocolitis
Pigs.....	Septicemia or enterocolitis (often followed by rectal stricture syndrome)

The disease patterns in domestic animals either take on the form of:

Septicemic Salmonellosis:

A disease of calves, foals and pigs. Younger animals are generally at greater risk than older animals. *S. choleraesuis* is the most incriminated species. Gross lesions may not be seen other than fibrinoid necrosis of blood vessels and widespread petechiations in organs. Death is attributable to disseminated intravascular coagulopathy.

Acute enteric salmonellosis:

Caused most frequently by *Salmonella typhimurium*. Occurs in cattle, pigs and Horses. Characterized by a diffuse catarrhal enteritis with fibrino-necrotic ileo-typhlo-colitis. Intestinal content is malodorous and mixed with mucus, fibrin and occasionally blood. Multiple foci of hepatocellular necrosis and Kupffer cell hyperplasia (paratyphoid nodules) may be seen. Lymphadenopathy is usually present. Fibrinous cholecystitis at necropsy is pathognomonic for acute enteric salmonellosis in calves.

Chronic Enteric Salmonellosis:

A disease of pigs, cattle and Horses.

Features of the chronic form of salmonellosis are:

- Presence of button ulcers (Discrete foci of necrosis and ulcerations especially in colon and cecum).
- Rectal strictures with resultant abdominal distention secondary to fecal retention.

LEPTOSPIROSIS

All mammals appear to be susceptible to at least one species of *Leptospira*. Disease is rare in cats, and less common in sheep than cattle.

- Serovars associated with disease in cattle include *hardjo*, *pomona*, *grippityphosa*, *canicola* and *icterohaemorrhagiae*.
- Serovars associated with disease in sheep and goats include *hardjo*, *pomona*, *grippityphosa* and *ballum*.
- Serovars associated with disease in pigs include *pomona*, *grippityphosa*, *bratislava*, *canicola*, *icterohaemorrhagiae*, *tarassovi* and *muenchen*.
- Serovars associated with disease in horses include *hardjo*, *pomona*, *canicola*, *icterohaemorrhagiae* and *sejroe*.
- Serovars associated with disease in dogs include *pomona*, *grippityphosa*, *canicola*, *icterohaemorrhagiae*, *pyrogenes*, *paidjan*, *tarassovi*, *ballum* and *bratislava*.

The primary reservoir hosts for most *Leptospira* serovars are wild mammals, particularly rodents. Reservoir hosts among domestic animals include cattle, pigs, sheep and dogs.

The specific reservoir host(s) vary with the serovar and the geographic region. Disease in reservoir hosts is more likely to be asymptomatic, mild or chronic.

Reservoir hosts include:

- Rats: serogroups *icterohaemorrhagiae* and *ballum*
- Mice: serogroup *ballum*
- Cattle: serovars *hardjo*, *grippityphosa* and *pomona*
- Sheep: serovars *hardjo* and *pomona*
- Pigs: serovars *pomona*, *tarassovi* and *bratislava*
- Dogs: serovars *canicola* and *bataviae*

Incubation Period

The incubation period is 4 to 12 days in dogs. Abortions usually occur 3 to 10 weeks after infection in cattle, and 15 to 30 days after infection in pigs.

Clinical Signs

Leptospira infections may be asymptomatic, mild or severe, and acute or chronic. The clinical signs are often related to kidney disease, liver disease or reproductive dysfunction. Chronically infected animals are often asymptomatic

Cattle

Acute leptospirosis occurs mainly in calves. The symptoms may include fever, anorexia, conjunctivitis and diarrhea. Severely affected animals may also develop jaundice, hemoglobinuria, anemia, pneumonia, or signs of meningitis such as incoordination, salivation and muscle rigidity. Some calves may die within 3 to 5 days, and the survivors can be unthrifty after recovery. The clinical signs vary with the serovar: infections with serovar *hardjo*, for example, are not usually associated with hemolytic anemia.

In adult cattle, the early symptoms such as fever and depression are often transient and milder, and may go unnoticed. The most prominent signs of infection are abortions, decreased fertility or decreased milk yield. Some serovars cause late term abortions, stillbirths and increased neonatal mortality. The placenta is retained in up to 20% of the cows that abort, and infertility may be a sequela. Some serovars can cause sudden agalactia or decreased milk production. The milk may be thick, yellow, and blood-tinged but there is typically little evidence of mammary inflammation. The appearance of the milk usually improves in 4 to 5 days, and milk production returns to normal after 10 to 21 days. Jaundice may be seen in severely affected animals.

Sheep and goats

Leptospirosis in sheep and goats is similar to the disease in cattle. It is characterized by fever and anorexia and, in some animals, jaundice, hemoglobinuria or anemia. Abortions, stillbirths, weak lambs or kids and infertility can also be seen, either with or without other clinical signs. Clinical disease is relatively uncommon in sheep.

Swine

In swine, clinical leptospirosis is most often characterized by reproductive signs including late term abortions, infertility, stillbirths, mummified or macerated fetuses, and increased neonatal mortality. Fever, decreased milk production and jaundice may also be seen. In some infected herds, the only sign of infection may be a transient fever. Subclinical infections are common. In piglets, there may be fever, anorexia, depression, diarrhea, jaundice, hemoglobinuria and gastrointestinal disorders, as well as signs of meningitis. Affected piglets may grow more slowly than normal and high mortality rates can be seen in young or weak piglets.

Horses

Many infections in horses are subclinical. Ocular disease is the most common syndrome. During the acute phase, ocular signs may include fever, photophobia, conjunctivitis, miosis and iritis. Corneal opacity and periodic ophthalmia may be sequelae of acute infections. In the chronic phase, there may be anterior and posterior adhesions of the eye, a turbid vitreous body, cataracts, uveitis and other ocular abnormalities.

Although systemic disease is uncommon, severe cases of leptospirosis accompanied by liver, kidney or cardiovascular disease have been described. Recently, leptospirosis has also been associated with a number of abortions.

Dogs

The clinical signs and severity of disease are highly variable in dogs. Some infections are asymptomatic or mild, while others are severe or fatal. The initial signs are usually non-specific and may include fever, depression, anorexia, stiffness, myalgia, shivering and weakness. The mucus membranes are often injected. These symptoms may be followed by signs of kidney disease including anuria, hematuria or increased frequency of urination, vomiting, dehydration and oral ulceration. Abortions, diarrhea, gray stools, coughing, dyspnea, conjunctivitis, weight loss and jaundice may also be seen. Hemorrhagic syndromes occur in some dogs: the mucus membranes may have widespread petechial and ecchymotic hemorrhages and, in later stages of the disease, there may be hemorrhagic gastroenteritis and epistaxis. Some dogs die peracutely without clinical signs. Chronic kidney disease can be a sequela.

Chronic infections may be asymptomatic, or associated with fever of unknown origin and conjunctivitis. Some serovars are more likely to cause certain syndromes. Fever, hemorrhage,

anemia and jaundice are typically associated with the serovar *icterohaemorrhagiae*. Serovar *grippotyphosa* tends to cause severe acute kidney failure and/or chronic active hepatitis. Dogs infected with serovar *pomona* are often asymptomatic and chronic carriers. Serovar *canicola* often causes chronic interstitial nephritis.

Wild animals

Infections are often asymptomatic in wild animals, including rodents.

Seals

Symptoms reported in seals and sea lions have included depression, polydipsia, fever, abortions and neonatal deaths.

Transmissions: *Leptospira* spp. are shed in the urine of acutely infected animals. Chronic carriers may excrete them for months or years. In addition, organisms can be found in aborted or stillborn fetuses, as well as in normal fetuses or vaginal discharges after calving. Rarely, *Leptospira* is transmitted through rodent bites.

Post-Mortem Lesions

Cattle: In acute cases, there may be signs of anemia, as well as icterus, hemoglobinuria, and submucosal and subserosal hemorrhages. The kidneys are typically swollen and contain petechiae and ecchymoses, which become pale over time. The liver is sometimes swollen, with minute foci of necrosis. Ulcers and hemorrhages may be found in the mucosa of the abomasum. Petechiae can also be seen in other organs in some fulminating infections. Pulmonary edema and emphysema are rare but have been reported.

Dogs: In acute infections, the kidneys and/or liver can be swollen, and hemorrhages may be found in any organ. Lesions associated with acute uremia may also be seen. In chronically infected dogs, there may be gray or white foci and/or streaks in the kidney and liver.

Seals: Severe interstitial nephritis and gastroenteritis have been reported in infected seals. Hepatitis may not be grossly apparent but the gall bladder may contain inspissated black bile.

Diagnosis

Leptospirosis can be diagnosed by culture, detection of antigens or nucleic acids, or serology. The location of the organisms varies with the form of the disease.

In acute infections, *Leptospira* may be found in the blood, milk, and cerebrospinal, thoracic or peritoneal fluids. During chronic infections, they are sometimes found in the urine. The liver, lung, brain and kidney are collected at necropsy from acute cases, and the kidney and genital tract are tested in chronic cases. Organisms can also be found in the body fluids or tissues of aborted fetuses. Identification to the species, serogroup and serovar level is done by reference laboratories, using genetic and immunologic techniques. *Leptospira* can also be identified in clinical samples by immunofluorescence and immuno histochemical staining, as well as DNA probes and polymerase chain reaction (PCR) techniques. Darkfield microscopy can be used but is non-specific. Silver staining is sometimes useful as an adjunct technique. The commonly used serological tests are the microscopic agglutination test (MAT) and enzyme-linked immunosorbent assays (ELISAs).

Serovar-specific ELISAs are available for animals and cross-reactions are less common in animals than in humans.

JOHNE'S DISEASE (PARATUBERCULOSIS)

Aetiology: *Mycobacterium paratuberculosis* (*Johnii*) closely related to *M. avium*.

Disease is characterized in cattle by intractable diarrhea, emaciation, and hypoproteinemia in animals older than 19mths. In small ruminants, diarrhea is uncommon.

Infection is acquired by ingestion of organisms. The bacilli penetrate the gastrointestinal mucosa from where they are taken up by macrophages. Gross lesions include the presence of non-caseating granulomas in the intestines and regional lymph nodes in cattle. (The granulomas in small ruminants are tuberculoid i.e caseating, and sometimes there is central mineralization).

Histologically, lesions in the lamina propria of the intestines include accumulation of large macrophages with foamy cytoplasm which contain large numbers of acid-fast bacilli in cattle. The cells are well-differentiated epithelioid cells in a whorled pattern and a variable number of Langhan's type giant cells.

MYCOPLASMOSIS (CBPP and CAPP)

Contagious bovine pleuropneumonia (CBPP) is an infectious and highly contagious disease of cattle and water buffaloes considered to be amongst the most important infectious diseases.

The disease is caused by a bacterium called *Mycoplasma mycoides sub species mycoides var bovis*.

Clinical signs:

Hyper-acute Form: In this form, death is sudden without any premonitory signs. Clinical diagnosis is difficult and there may not be any gross lesions at postmortem

Acute form: Sudden onset of fever ~ 40C Drop in milk yield and sudden anorexia. Sick cattle tend to isolate themselves from the herd and they stop eating. Breathing is labored and obviously painful. Abdominal breathing may be seen with respiratory grunts. Some animals may develop a shallow, dry and painful cough particularly noticeable on exercise. Acutely affected cattle stand with head and neck extended and fore legs spread apart. Nostrils are dilated with mouth open and panting for air. There may be nasal discharge, sometimes streaked with blood and frothy saliva accumulates around the mouth. Some animals develop swellings of the throat and dewlap.

Pregnant cows and heifers may abort.

Chronic Form: Clinical signs regress but cattle still have intermittent fever with loss of appetite and weight.

Pathology:

Lesions are generally confined to the thorax except in the young calves where inflammation of the carpal and tarsal joints with increased synovial fluid has been observed.

The most striking feature of the acute disease is the large volume of yellow fibrinous fluid (up to 30litres) found in the thoracic cavity. Usually the lesion is unilateral and in most cases the caudal lobe is affected (contrast with cranio-ventral lobe in pasteurellosis).

The pleura is markedly thickened and opaque sometimes with adhesions to the rib cage. The cut surface of the lungs show typical marbling with intermingling dark red, red and pale pink areas separated by a network of pale bands (interlobular septae). This appearance is typical of CBPP.

In the chronic form, fluid is rarely seen in the pleural cavity but we have fibrous adhesions between lung lobes, between lungs and rib cage. There is sequestration of dead lung tissue which becomes surrounded by a capsule of fibrous connective tissue. The sequestrum is typical of chronic CBPP The content of the sequestrum may shrink and later become dry or cheesy.

Regional lymph nodes may become enlarged and edematous.

Renal cortical necrosis and infarcts can sometimes be seen.

CAPP is the counterpart of CBPP in goats and not sheep. *M.mycoides sp mycoides large clony*, *M.mycoides sp Capri* and *Mycoplasma strain F38* have been incriminated. The condition is clinically similar to CBPP. Presents with fibrinous bronchopneumonia and pleuritis. Sequestra is uncommon in this condition. In addition, there is fibrinous polyarthritis septicemia, meningitis, mastitis, and abortion.

PASTEURELLOSIS

Bovine Pasteurellosis is a conglomerate of four distinct conditions namely:

Enzootic Pneumonia (Calf/ viral pneumonia) of multifactorial aetiology

Bovine pneumonic pastuerellosis caused by *Pasteurella (Mannheima) hemolytica*

Shipping Fever caused by *Pasteurella hemolytica* and *Pasteurella(Mannheima) multocida*

Haemorrhagic septicemia caused by *Pasteurella multocida* serotypes B and E

Enzootic Pneumonia of calves

A respiratory disease of calves caused by a variety of etiological agents. Condition is sometimes called viral pneumonia because it often begins with infections with viruses such as PI-3 virus and BRSV (Bovine Respiratory syncytial virus). Sometimes Chlamydia and mycoplasmas may also precipitate the infection. *Pasteurella multocida* is part of the normal flora of bovine respiratory tract. Following infection with any of the above listed pathogens, *Pasteurella multocida* could cause a secondary suppurative bronchopneumonia as an opportunistic infection. The establishment of the infection depends on the compromise of the respiratory defense mechanism either by poor ventilation, relative humidity and overcrowding.

Pathology: The acute phase of infection is purely a broncho-interstitial pneumonia (because of the viral involvement). Microscopic lesions are those of necrotizing bronchiolitis, necrosis of Type 1 pneumocytes and mild interstitial and alveolar edema. Viral inclusions and multinucleated giant cells may be seen depending on the precipitating viral infection.

At the later stages, when *Pasteurella* complication is evident, the pneumonia becomes a suppurative bronchopneumonia. The lungs at this stage contain a creamy mucoid exudate in the airways. Pulmonary abscesses and broncho-ectasis may also follow.

Bovine pneumonic pasteurellosis (Shipping Fever):

Condition is caused by *Pasteurella hemolytica* biotype A, serotype 1

Clinical Signs: Clinically, the condition presents as a severe toxemia. Cattle usually become depressed, febrile, and anorexic. They also have a productive cough, with encrusted nose, shallow respiration and mucopurulent nasal exudates.

Once established in the lungs, *P. hemolytica* causes lesions by means of different virulence factors including endotoxins and outer membrane proteins but the most important is probably the production of a leukotoxin (exotoxin) which binds and kills bovine macrophages and neutrophils. The disease is characterized by a severe toxemia.

Pathology: Grossly, there is an extensive lobar fibrinous bronchopneumonia with prominent fibrinous pleurisy and pleural effusions. Lesions are always cranio-ventral and usually ventral to a horizontal line through the tracheal bifurcation.

A fibrinous, lobar, bronchopneumonia with prominent fibrinous pleuritis and pleural effusions. Lesions are always cranioventral. Distended interlobular septae with yellow gelatinous edema and fibrin. Marbling of lobules occurs as a result of intermingling areas of coagulative necrosis with normal areas.

Histologically: The necrotic areas are typically bordered by a rim of elongated cells often referred to as 'oat-cells' or 'swirling macrophages'. These cells are now known to be degenerating neutrophils mixed with a few alveolar macrophages.

Haemorrhagic Septicemia:

Caused by serotypes B and E of *P. multocida*. Usually spread by hematogenous route to many organs.

Clinical Signs: Severe and acute septicemia, high fever, and rapid death.

Lesions: Petechiations on serosal surfaces of heart, lungs, skeletal muscles swollen and haemorrhagic lymph nodes. There may also be a fibrino-haemorrhagic pneumonia and blood-tinged fluid in the thorax.

The disease in sheep bears some resemblance to what is obtained in bovine. The following are well-recognized syndromes in sheep:

Ovine Septicemic Pasteurellosis:

A common ovine disease caused by *P. hemolytica* biotype T in animals 5 months and older, biotype A in lambs younger than 2 months of age. These are part of the normal flora of the tonsils and oropharynx in sheep. Under stress and immunosuppression, the agents invade adjacent tissues and enter blood stream causing septicemia.

Clinical Signs: Affected animals may die without premonitory signs. In some cases, signs of dullness, recumbency, and dyspnea may be observed.

Pathology: A distinctive necrotizing pharyngitis and tonsillitis, severe congestion and edema of the lungs, focal hepatic necrosis. Infarcts and petechiae in tongue, esophagus and intestines. The hallmark microscopic lesion is a disseminated intravascular thrombosis often associated with bacterial colonies in affected tissue

Ovine Pneumonic Pasteurellosis (Acute Ovine enzootic pneumonia):

Aetiology: *P. multocida* biotype A

Pathogenesis is similar to pneumonic pasteurellosis of cattle.

Lesions: A severe fibrinous bronchopneumonia, lobar and cranio-ventral with pleuritis. Subacute to chronic cases appear as a fibrinopurulent bronchopneumonia and sequelae include: Abscesses and fibrous pleural adhesions.

Chronic Enzootic pneumonia:

Pasteurella participates in the combination of infectious, environmental and managerial factors responsible for this condition. PI-3, Adenoviruses, Reovirus, RSV, Chlamydiae, and mycoplasmas (*M. ovipneumoniae*).

In the early stages of the disease, a cranio-ventral broncho-interstitial pneumonia is observed characterized by moderate thickening of alveolar walls due to hyperplasia of Type 2 pneumocytes. In the late stage, Hyperplastic bronchitis, atelectasis, alveolar and bronchiolar fibrosis and a severe peribronchiolar lymphoid hyperplasia (cuffing pneumonia) becomes evident.

BRUCELLOSIS:

A bacterial disease caused by members of the genus *Brucella*, is an important zoonotic disease and a significant cause of reproductive losses in animals. Brucellosis is usually caused by *Brucella abortus* in cattle, *B. melitensis* or *B. ovis* in small ruminants, *B. suis* in pigs, and *B. canis* in dogs. Abortions, placentitis, epididymitis and orchitis are the most common consequences. The main impact of the disease is economic as deaths are rare except in fetuses and neonates. Most species of *brucella* can infect animals other than their preferred hosts when they come in close contact.

Transmission: Infection is usually transmitted between animals by contact with the placenta, fetus, fetal fluids and vaginal discharges from an infected animal. Animals are infectious either after an abortion or full-term parturition. Ruminants may be asymptomatic after their first abortion but may remain chronic carriers and continue to shed brucella in milk and uterine discharges during subsequent pregnancies. *Brucella* organisms are also found in semen. Venereal transmission is important in *B. ovis*, *B. suis*, and *B. canis*.

Brucella organisms can be spread on fomites including feed and water. The organisms can withstand drying and desiccation and may remain viable for many months.

Bovine Brucellosis (*B. abortus*)

In cattle, *B. abortus* causes abortion, stillbirths and weak calves; abortions usually occur during the second half of gestation. The placenta may be retained and lactation may decrease. After the first abortion, subsequent pregnancies are generally normal; however, cows may shed organism in milk and uterine discharges. Epididymitis, seminal vesiculitis, orchitis, and testicular abscesses are sometimes seen in bulls. Infertility occur occasionally in both sexes due to metritis or orchitis / epididymitis. Hygromas, particularly on leg joints, are a common symptom. Arthritis can develop after long-term infections.

Similar lesions occur in other ruminants such as; camels, bisons, and water buffaloes.

Ovine/ Caprine Brucellosis:

(*B. melitensis*): Affects both sheep and goats. Infection is usually characterized by abortions, stillbirths and the birth of weak offspring. Animals that abort may retain placenta. Sheep and goats usually abort only once, but re-invasion of the uterus and shedding of organisms can occur during subsequent pregnancies. Milk yield is significantly reduced in animals that abort. Clinical signs of mastitis are uncommon in infected small ruminants. Acute orchitis and epididymitis can occur in males and may result in infertility. Arthritis is seen occasionally in both sexes.

B. Ovis: Affects only sheep and **not** goats. This organism can cause epididymitis, orchitis, and impaired infertility in rams. Initially, only poor quality semen may be seen; later lesions become palpable in the epididymis and scrotum. The testis may atrophy. Abortions, placentitis and perinatal losses are observed in *B. ovis* infections.

Porcine Brucellosis (*B. suis*)

In pigs, the most common symptom is abortion, which can occur at any time during gestation. There may also be weak or stillborn piglets. Vaginal discharge is often minimal. Occasionally some sows develop metritis. Temporary or permanent orchitis may be seen in boars. Swollen joints and tendon sheaths accompanied by lameness and incoordination can occur in both sexes. Less common signs include; posterior paralysis, spondylitis and abscesses in various organs.

Canine Brucellosis (*B. canis*)

Abortion and stillbirth in pregnant dogs. Most abortions occur late (7-9th week of gestation) Abortion is usually followed by a mucoid, serosanguinous, vaginal discharge that persist for up to 6weeks.

Early embryonic deaths and fetal resorption do occur and sometimes pups are born weak or with congenital defects and may die soon after birth. Epididymitis, scrotal edema, orchitis and poor semen quality may be seen in males. Scrotal dermatitis can occur due to self-trauma. Unilateral or bilateral testicular atrophy can be seen in chronic infections. And some males may become infertile. Lymphadenitis is common in infected dogs.

PATHOLOGY: Some aborted fetuses appear normal; others are autolysed or have variable amounts of subcutaneous fluid in body cavities. In ruminant fetuses, the spleen and /or liver may be enlarged and there may be pneumonia and fibrous pleuritis.

Abortions caused by *Brucella* species are usually accompanied by placentitis. The cotyledons may be red, yellow, normal or necrotic. In cattle and small ruminants, the inter-cotyledonary region is typically leathery, with a wet appearance and focal thickening. There may be exudates on the surface. In adults, granulomatous and purulent lesions may be found in the male and female reproductive tracts, mammary glands, supra-mammary lymph-nodes, other lymphoid tissues, bones and joints. Mild to severe endo-metritis may be seen after an abortion, and males can have unilateral/bilateral epididymitis and / or orchitis. In *B.abortus*-infected cattle, hygroma may be found on the knees, stifles, and other joints.

In rams infected with *B. ovis*, lesions are usually limited to epididymitis and orchitis. Epididymal enlargement can be unilateral or bilateral and the tail is affected more often than the head or body. Fibrous atrophy can occur in the testis. The tunica vaginalis is often thickened and fibrous with extensive adhesions. Placentitis is occasionally seen in infected ewes.

In *Brucella canis* infections, aborted puppies are often partially autolysed and have evidence of generalized bacterial septicemia. Fetal lesions include; subcutaneous edema, congestion and haemorrhages in the abdominal region. Peritoneal fluid is serosanguinous. Degenerative and necrotic lesions are also seen in the liver, spleen, kidneys and intestine. There is lymph node enlargement in infected adult animals. . The spleen is frequently enlarged, firm and nodular. There is also hepatomegaly. Scrotal edema, scrotal dermatitis, epididymitis, orchitis, prostatitis, testicular atrophy, and fibrosis occur in some infected males. Metritis and vaginal discharge may be seen in females. Less commonly reported lesions include: spondylitis, meningitis, focal non-suppurative encephalitis, osteomyelitis, uveitis and abscesses in various organs.

LISTERIOSIS

Disease caused by *Listeria monocytogenes*.

Listeriosis presents in three disease forms:

- **Meningoencephalitis**
- **Abortion and stillbirth**
- **Septicemia**

Listeria organisms amongst others have also been incriminated in blood borne diseases causing fetal pneumonias (necrotizing interstitial) in domestic animals.

Septicemia is commonly seen in young animals possibly from an in-utero infection.

In the encephalitic form, gross lesions may be in apparent except for leptomeningeal opacity. Foci of necrosis in the terminal brain stem and cloudy CSF have been noticed. Microscopically, a meningo-encephalitis is seen in the pons and medulla. Occasionally, the anterior part of the cervical spinal cord may participate in the lesions. There is a diffuse suppurative leptomeningitis, focal malacia and neuronal necrosis with characteristic accumulation of neutrophils and gitters cells (micro-abscesses) in the brain stem.

Listeriosis is also a confirmed cause of sporadic abortions in cattle, sheep and goats. *Listeria* abortions occur in the last trimester of pregnancy. There may be septicemia and placentitis in the aborting dam. The placental lesions include; a severe diffuse necrotizing and suppurative inflammation of both the cotyledons and intercotyledonary areas.

Fetal lesions include an enlarged liver with numerous tiny necrotic foci from where the organisms may be readily demonstrated.

COLIBACILLOSIS

The group of diseases caused by the bacterium *Escherichia coli* . These diseases include:

Enterotoxic Colibacillosis:

Common in animals in the age group 2days - 3weeks. Faeces of affected animals are profuse, yellow to white, watery and pasty. Affected animals are dehydrated with tucked up abdomen and sunken eyeballs. Gross lesions are those of dilated, flaccid small intestine filled with translucent fluid.

Microscopically, the intestine appears normal but on careful examination, bacteria may be seen lining the luminal surface of the enterocytes.

Similar lesions have also been observed in adults infected with the 'attaching and effacing *E.coli* strain'. This syndrome in adults often occurs in association with other enteropathogens such as: rotavirus, cryptosporidium sp et.c. In contrast to enterotoxigenic *E.coli* infection, the brush border of the enterocytes in this 'attaching and effacing *E.coli*' is usually disrupted (effaced).

Septicemic colibacillosis:

Also a disease of newborn calves, lambs and occasionally foals that were denied colostrums. Lesions seen are generally those of septicemia, but in addition, there may be localization in the intestines, lungs, oral cavity or umbilicus.

Lesions are those of fibrinous arthritis, ophthalmitis, serositis and meningitis. Cortical abscesses may also be observed in the kidneys.

Edema Disease (Enterotoxaemic colibacillosis):

A disease of pigs. Best pigs in the group are affected. Associated with dietary changes at weaning. Condition is characterized by incoordination of hind legs, sagging, and swaying, difficulty in rising, irritability, muscle tremors, aimless wandering, and clonic convulsions.

Pathogenesis: The hemolytic *E.coli* proliferates in the small intestines subsequent to dietary changes and elaborates an exotoxin called the '*edema disease principle*'. This is an angiotoxin capable of generalized vascular endothelial injury of arterioles and arteries resulting in fluid loss and edema.

Pathology: Edema of gastric submucosa, eyelids, gall bladder, and mesentery of spiral colon. Arterial damage in the brain results in malacia of medulla, thalamus, and basal ganglia (focal symmetric encephalomalacia or swine cerebral angiopathy)

Entero-invasive Colibacillosis;

This is principally a human condition. Occasionally observed in cattle and pigs. Pathogenesis is similar to that of invasive bacteria such as salmonella sp.

A verotoxin elaborated by this strain of *E. coli* results in haemorrhagic colitis. In humans, it's often a food-borne illness.

VIRAL DISEASES OF POULTRY

Newcastle disease

Shell-less, soft shallow egg or Inn-shallow egg are characteristic white misshapen/right eggs are not a feature.

NEWCASTLE DISEASE

Aetiology

Infection caused by a virus of the family paramyxoviridae.

Pathogenicity: virus with the host chicken vetogenic highly susceptible, but duck and geese may be infected and show signs. In chicken, the pathogenically is neurotically determined chiefly by the of the virus, close age and environmental condition. In general the younger, the chicken, the more acute the disease.

Transmission:-

Vertical transmission (virus from parent to progeny) via the embryo is controversial

Clinical Signs

With strain – resp., digestive, ocular neurological. Sudden death. In layer – egg production affected – quantity and quality soft shelled misshapened-eggs, retention in the oviduct – Egg binding.

Pathology

Gross lesion

Haemorrhagic lesion is prominent in the proventriculus , ceaca and small intestine.

Histopath

Nervous system – Non-suppurative encephalomyelitis with neuronal degeneration. Foci of gliosis, lymphocytic perivascular cuffing and proliferation of endothelial cells.

Lesions marking seen in cerebellum, medulla, brain stem and spinal cord and rarely cerebrum.

Vascular system

Hyperaemia, oedema and haemorrhage are found in the blood vessels of many organ. Also may be seen hydropic degeneration of the media, hyalinization of capillaries and arterioles and nervous of endothelial cells of the vessels.

Lymphoid System

1. Lymphoid depletion
2. Loss of germinal center.
3. Lesion for the spleen lymphocytes in the depicted destruction of cortical areas and germinal centers of spleens thymus.
4. Marking degeneration of the medullary region is seen in the bursa.

Intestinal tract

Haemorrhagic – necrotic lesions – seen in intestines with infection of virulent forms. Other lesions are related to vascular changes.

Respiration

Nasal– loss of cilia, upper respiratory tract- congestion, oedema and dense cellular infiltration of lymphocytes and MQs in the lungs. Haemorrhage and erythrophagocytosis in the alveolar areas of the lymph. Para bronchi. Eodema, cellular infiltration and increased thickens of air sack may be observed. Intramuscular hycytoplasm inclusion in the oesoph lung and brain.

EGG DROP SYNDROME

Egg drop syndrome 1976 (EDS' 76) has become a major cause of egg production loss throughout the world. It is probably in produced into chickens through a contaminated vaccine. Since its initial recognition, it has become apparent that sporadic outbreaks of EDS 76 occur as a result of fowl becoming be infected through direct or indirect contant with infected wild or domestic waterfowl. The viral affects only avian species and, therefore has no public health significance.

Aetiology

Caused by Egg drop syndrome 76 virus classified as an adenovirus on the basis of morphopogy, replication and chemical composition.

Transmission

Horizontal transmission mainly, and there is evidence that spread can occur when birds are transported in inadequately cleaned form one site to another.

1. Hortal transmission also being documented.
 2. Spread from both domestic or wild duck geese and possibly other wild bird to hen through during water contaminated by droppings.
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1. Fall in egg production
 2. Loss of colour of pigmented eggs,
 3. Production of thin –shelled, soft - shelled or shell – less eggs.
 4. Inappetence and dullness – not consistant findings.

5. Transient diarrhoeas
6. Mortality is almost 0.

Pathology

Gross Lesions

1. In naturally occurring outbreaks, in active ovaries and atrophied oviducts are often observed but these are not consistent present.
2. Mild splenomegaly has been described.
3. Egg in various stages of formation may be seen in the abdominal cavity.

Histopath

The major pathological changes occur in the pouch of shell gland and epithelial cells.

- Cellular desquamation (slough off) into the lumen, severe inflammation involving macrophages, plasma cells, lymphocytes with few heterophils in the lamina propria. Intranuclear inclusion bodies are seen in the epithelial cells – These indicate where viral replication takes place.

Diagnosis

Clinical signs, PM lesions and viral isolation.

The most sensitive indicator system is either embryonated duck or goose eggs from a flock that is free of EDS 76 virus infection of duck or goose cell cultures.

FOWL POX (FP, POX, AVIAN POX)

Fowl Pox is a viral disease of chicken, turkeys and other birds distinguished by cutaneous lesions on the head, neck, legs and feet. It has a worldwide distribution and affects birds of all age groups, except the recently hatched.

Transmission: The virus is present in lesions and in desquamated scabs. It is resistant to environmental factors and persists in the environment for many months. It usually infects birds through minor abrasions. Mechanical transmission occurs by cannibalism. Some mosquitoes can transmit the virus from infected to uninfected birds. The virus can be also transmitted by injury to the skin.

Antemortem findings: Two forms of lesions are recognized, - the cutaneous (dry form) and the diphtheric (wet form).

Cutaneous form

1. Low mortality
2. Lack of flock vigour and weight loss
3. A mild to moderate loss in egg production.
4. Scabby lesions on the head, neck and unfeathered parts of the skin (Fig. 196)

Diphtheric form

5. Mortality low to moderate
6. Difficult breathing
7. Nasal and ocular discharge

Postmortem finding: The following stages of the pox lesions papules, vesicles and pustules may be observed.

Cutaneous lesions

1. Papules are light coloured nodules.

2. Vesicles and pustules are raised and commonly yellow.

Diphtheric lesions

3. Buff to yellow plaques on mucous membranes in the mouth (Fig. 197), Oesophagus and upper respiratory tract.
4. occlusion of trachea, and death due to asphyxiation.

Histopathology shows characteristic intracytoplasmic inclusion bodies (Bollinger bodies) in the infected epithelium.

Judgement: Carcass affected with fowl is condemned if progressive generalized lesions in a bird are accompanied with emaciation. Fowls with localized lesions and recovered birds are approved after the removal of scales.

Differential diagnosis: Pantothenic acid and biotin deficiency, vitamin E deficiency, infectious laryngotracheitis and other respiratory disease in poultry, injuries caused by external parasites and cannibalism.

Avian leucosis complex occurs in four separate disease entities:

1. Leucosis-sarcoma group (Lymphoid leucosis)
2. Marek's disease (MD)
3. Reticuloendotheliosis group (REV)
4. Lymphoproliferative agent of turkey.

Transmission: L/S virus is transmitted by egg in vertical transmission and by shedders in horizontal transmission (chicken). Tumor lesions may or may not develop. Developed tumours can be differentiated by the bird's age on necropsy, histology and impression smears. In horizontal transmission, chicken which contract the virus after hatching develop antibodies; some will remain shedders, some will develop tumours and die, and others will overcome the infection. Infection from flock is unlikely as the virus does not survive a long time in the environment. In many chickens the virus may be also in a latent state.

(A) Lymphoid leucosis (big liver disease, visceral lymphomatosis) this disease is being studied because of its economic significance, and also as an experimental model of cancer. Lymphoid leucosis is a B cell tumour which starts in the bursa and, before sexual maturity, may spread to other organs. Mature birds are more affected than young birds. Male birds are also affected in lesser numbers than female due to the earlier regression of bursa in male birds.

Antermortem finding:

1. The disease occurs in 14 – 30 weeks old birds.
2. Pale, shriveled comb and loss of appetite
3. Dehydration and emaciation.
4. Diarrhoea, green scant faeces.
5. Enlarged liver, bursa of Fabricius and kidneys.
6. Distended abdomen due to enlarged liver.
7. Reduced egg production.

Postmortem finding

1. Grey tumour lesion in the liver (Fig. 198), spleen and bursa
2. Other organs such as lung, heart, proventriculus, gonads, bone marrow and mesentery are sometimes affected.
3. Ecchymotic haemorrhages around the skin follicles of the wing.

Judgement: The carcass of a bird affected with lymphoid leucosis is condemned. The condemned material may be used for animal food.

Differential diagnosis: Between lymphoid leucosis and Marek's disease.