Animal Health ii

Government of Nepal
Ministry of Education, Science and Technology
Curriculum Development Centre
Sanothimi, Bhaktapur
Phone : 5639122/6634373/6635046/6630088
Website : www.moecdc.gov.np

10

Feedback Copy

Technical and Vocational Stream

Resource Learning Materials

Animal Health-II

(Grade 10)

Secondary Level

Animal Science

Government of Nepal
Ministry of Education, Science and Technology
Curriculum Development Centre
Sanothimi, Bhaktapur

Publisher : Government of Nepal

Ministry of Education, Science and Technology

Curriculum Development Centre

Sanothimi, Bhaktapur

© Publisher

Layout by Khados Sunuwar

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted, in any other form or by any means for commercial purpose without the prior permission in writing of Curriculum Development Centre.

Preface

The curriculum and curricular materials have been developed and revised on a regular basis with the aim of making education objective-oriented, practical, relevant and job oriented. It is necessary to instill the feelings of nationalism, national integrity and democratic spirit in students and equip them with morality, discipline and self-reliance, creativity and thoughtfulness. It is essential to develop in them the linguistic and mathematical skills, knowledge of science, information and communication technology, environment, health and population and life skills. it is also necessary to bring in them the feeling of preserving and promoting arts and aesthetics, humanistic norms, values and ideals. It has become the need of the present time to make them aware of respect for ethnicity, gender, disabilities, languages, religions, cultures, regional diversity, human rights and social values so as to make them capable of playing the role of responsible citizens with applied technical and vocational knowledge and skills. This Learning Resource Material for Animal Science has been developed in line with the Secondary Level Animal Science Curriculum with an aim to facilitate the students in their study and learning on the subject by incorporating the recommendations and feedback obtained from various schools, workshops and seminars, interaction programs attended by teachers, students and parents.

In bringing out the learning resource material in this form, the contribution of the Director General of CDC Dr. Lekhnath Poudel, Prof. Dr. D.K. Singh, Dr. Shambhu Sah, Dr. Yam Bahadur Gurung, Dr. Ganesh Gautam, Dr. Kiran Pokhrel, Dr. Anita Subedi, Dr. Bhumika Paudel, Dr. Laba Kumar Jha, is highly acknowledged. The book is written by Dr. Hari Prasad Panta and the subject matter of the book was edited by Badrinath Timsina and Khilanath Dhamala. CDC extends sincere thanks to all those who have contributed in developing this book in this form.

This book is a supplimentary learning resource material for students and teachrs. In addition they have to make use of other relevnt materials to ensure all the learning outcomes set in the curriculum. The teachers, students and all other stakeholders are expected to make constructive comments and suggestions to make it a more useful learning resource material

2076 BS

Ministry of Education, Science and Technology

Curriculum Development Centre

Content

Unit : One

Diseases caused by helminthes and protozoa

Learning Outcomes

After the completion of this unit, student will be able to

- Explain the general concept of parasitology.
- Define common terminologies that is frequently used in parasitology.
- Identify the helminth and protozoa with life cycle pattern.
- Recognized different diseases causes by helminth and protozoa.

Prasitology

The word is derived from the word parasite "para" means besides and "Sito" means food. **Parasite** is an organism that lives inside or on another organism and drives substances from it without rendering any benefit to it. A parasite is a small organism that lives on or in, at the expense of a larger organism called host, host generally provides food and shelter for parasite and the study of the phenomenon of the parasitism is called parasitology.

Parasitology is multidisciplinary subject which is related to the field of biochemistry, Physiology, Cellbiology, Immunology, pathology and Pharmacology.

Animal association may be divided into

1. **Phoresis-** to carry.
   Commensalism- host is neither benefited nor harm.
2. **Symbiosis-** Interdependent upon each other that one can't live without the help of the other.
3. **Mutualism-** similar to symbiosis but association between host and parasite is not essential.
4. **Parasitism-** parasite is always benefited and host is almost always harmed.

Types of Parasites

1. **Internal parasite (Endoparasite**)- lives inside the body of the host in the

blood, tissue, body cavities, digestive tract and other organs eg trematodes, cestode and nematodes

2. **Exrernal parasite (Ectoparasites**)- live outside on the surface of the body of the host e.g lice ,ticks, mites, fleas, leech etc
3. **Temporary parasite**- visits it host for a short period e.g mosquito, sand fly, fleas, bedbug etc
4. **Permanent parasite**- live a parasitic life in or on the host body throughout the whole period of its life.
5. **Facultative parasite**- lie a parasite life when opportunity arises or a facultative parasite is able to live either free or as parasite eg. strongyloids spp. Noegleria fowleri etc
6. **Obligatory parasite**- can't exist without a parasite life e.g trichinella spiralis , malarial parasite
7. **Incidental parasite (accidental parasite**)- attacks an unusual host in which they do not occur normally eg. fasciola hepatica sometime occur in men.
8. **Wandering parasite-** happens to reach a place or organ where it can't live eg.stephanurus dentatus(sometimes found in the liver of pig but the usual site is the pelvis of the kidney.
9. **Periodic parasite**- visits their host at the time to obtain some metabolic requirement or some other benefits eg. mosquito, flies tabanus etc

Different types of Host

The organism that harbors the parasites is called the host eg. man, cattle etc.

1. **Definitive host**- Generally it is vertebrate animal and it either harbors the adult stage of the parasite or were the parasite utilize the sexual method of reproduction eg. sheep, goat, dog, poultry
2. **Intermediate host**- These are the host mainly vertebrates or invertebrate, which harbors the larval stages of the parasite. In some cases larval development are completed in two different intermediate hosts which has been referred to as first and second intermediate host respectively eg. snail, earthworm, dragonfly, grasshopper, sheep, goat, cattle
3. **Carrier host**- It is the host in which the parasite remains viable without

further development, later on the parasite host is ingested by the definitive host and parasite infection occurs.eg. Earthworms, slugs, flies, snail, dragon flies crab etc.

4. **Principal host-** It is that animal in which a particular parasite is most commonly found eg. haemonchus contortus-in sheep, Heterakis gallinarum-In fowl
5. **Reservoir host**- This is the host in which the parasite are found in side th host but which is not important. But parasite can affect other domestic animals or human being that host act as a resivior.eg. Echinococcus spp.in fox, Hydatid cyst in sheep, goat, cattle, dog, human.

Internal parasite (Endoparasite)

Endo parasite lives inside animal body and feed on host tissue, body, fluid and animal's food, helminthes (Trematodes, cestodes, Nematodes) and protozoa are the important endo parasites of animals and man.

Helminthes

The name helminth has been derived from a Greek word helmins or helminthos which means a worm and is usually applied to parasite and non parasitic species.

1. Platyhelminthes- a) Trematode eg. liverfluke, paramphistomum
   b) Cestode eg. tapeworms
2. Nematheminthes- Nematode eg. Stryangyloides spp., Haemonchus spp, Toxocara spp.

Protozoology is that branch of biology science, which deals with unicellular and microscopic organism/animal life known as protozoa.

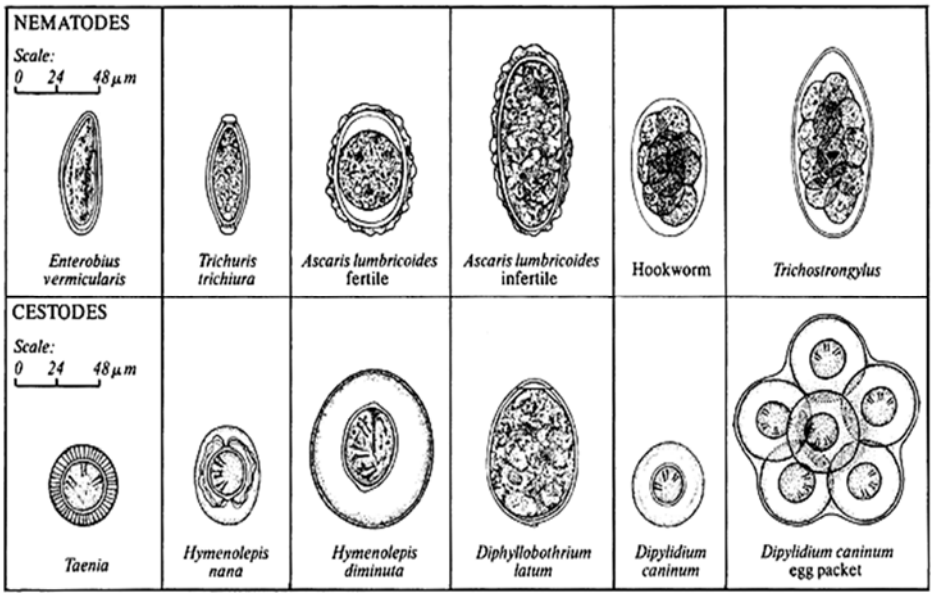
Morphological identification of Treamatodes

- They are unsegemented worm.
- Body dorsoventrally flattened leaf like called fluke.
- Three types of parasite i.e fleshy fluke (*Paramphistomum cervi* and *paramphistomum explanatum*), small flukes (*Dicrocolium eurytrema, D.lancanatem, D.dendriticum*) and large fluke(leaf like) (*Fasciola hepatica* and *Fasciola gigantica*).

- It has no body cavity
- Have suckers, hooks or clump for attachment to the host.
- Have indirect life cycle.
- Digestive system includes mouth and alimentary tract but no anus.
- Excretory system has flame (reddish orange-gold fiery color) cells.
- The respiratory system and circulatory system is absent.
- Reproductive system hermaphrodite (unisexual male/female) except family schistosomatidae.
- Adult flukes are found in intestine,liver and lungs.

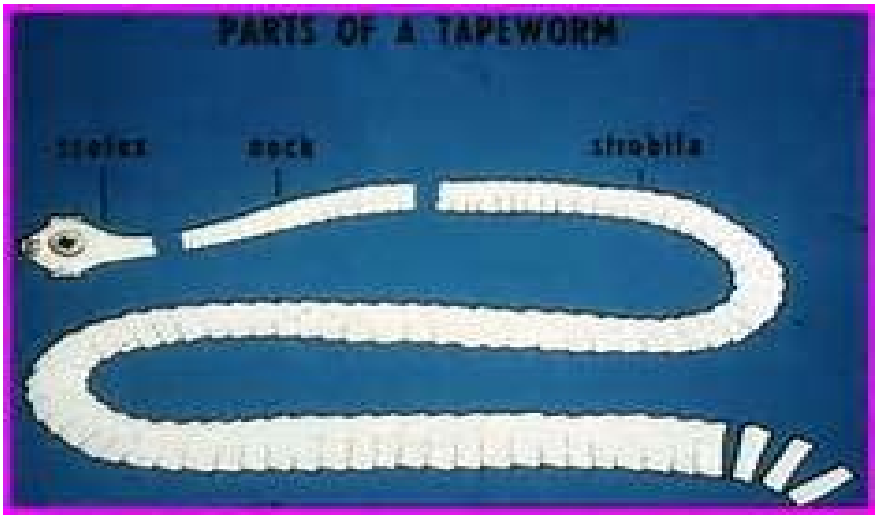
Morhological identification of Nematodes

- They are round and unsegmented worms.
- They usually cylindrical and elongated in shape.
- Extremities (ends) are tapering.
- Body contains an alimentary canal.
- Sexes are separate.
- Excretory system contains un-branched vessels.
- Life cycle generally consist of egg.
- Life cycle is direct or indirect.
- Generally affecting in intestine and lungs.
- Male is smaller than females.
- Some are oviparous and other ovo-viviparous.
- Eg.*Stryangyloides westeri, S. papillosa, S.avium, Haemonchus contortus,Toxocara canis*.



Morphological identification of Cestodes

- They are long, segmented and tape like hence called tape worms.
- They are dorsoventrally flattened.
- Sizes vary from a few millimeters to several meters long.
- Adult worms are mostly found in the intestine canal of animal and man.
- Have head and scolex.
- Sexes are unite i.e each individual worm is hermaphrodite.
- Body cavity or coelom (a fluid filled) is absent.
- Alimentary canal is entirely absent.
- Excretory and nervous system is present.
- Reproductive system is higily development and complete in each segments.
- Life cycle is usually complete in more than one host.
- Eg. *Monieza expansa, M.benedeni, Raillientina tetragona,Taenia solium,T.saginata,T.multiceps,Echinococcus granulosus*.



Diseases caused by helminthes and protozoa

The tiny animal or small insects which live and take food from the body of other animal is called parasites. This types of parasites causes a lot of diseases to the animals. The cattle diseases caused by this types of parasites are known as parasitic diseases. Parasites which affects the animals are of two types. They are external parasites and internal parasites. The most dangerous parasites of animals are listed below.

- Round worm
- Tape worm
- Fluke worm
- Ticks
- Lice
- Fly
- Mites

The parasites described above do a lot of harm to the domestic animal**.**

1.2. Liver fluke

Fascioliasis: Important parasitic disease.

Caused by Two species:

- Fasciolahepatica

- Fasciolagigantica

Morphology: Specie Size Shape *F. hepatica* 5cm length/1.5cm wide Leaf shape with prominent shoulders

F. gigantica; It is7.5cm length/1.2cm wide and leaf shaped with scarcely perceptible shoulders.*Fasciola hepatica*, also known as the common liver fluke or sheep liver fluke. It is a parasitic flatworm of the class Trematoda, phylum Platyhelminthes that infects liver of various mammals, including human. The disease caused by the fluke is called fascioliasis (also known as fasciolosis). *F.hepatica* is world-wide distributed and causes great economic losses in sheep and cattle.

Life cycle outside the animal

Eggs hatch in spring (>10 °C) to release miracidia which must penetrate a mud snail (Lymnaea [syn. Galba] truncatula) within 3 hours to develop inside snail. Then cercariae emerge from snail and encyst on grass (metacercariae). Infection of a snail with one miracidium can produce over 600 metacercariae

Life cycle inside the animal

- Once ingested metacercariae excyst in SI and immature fluke migrate through the gut wall to penetrate the liver.
- Tunnel through liver for 6-8 weeks before entering bile ducts where they reach maturity
- Time from infection of cattle/sheep to adult egg-laying fluke is 10-12 weeks
- Little or no development of immunity

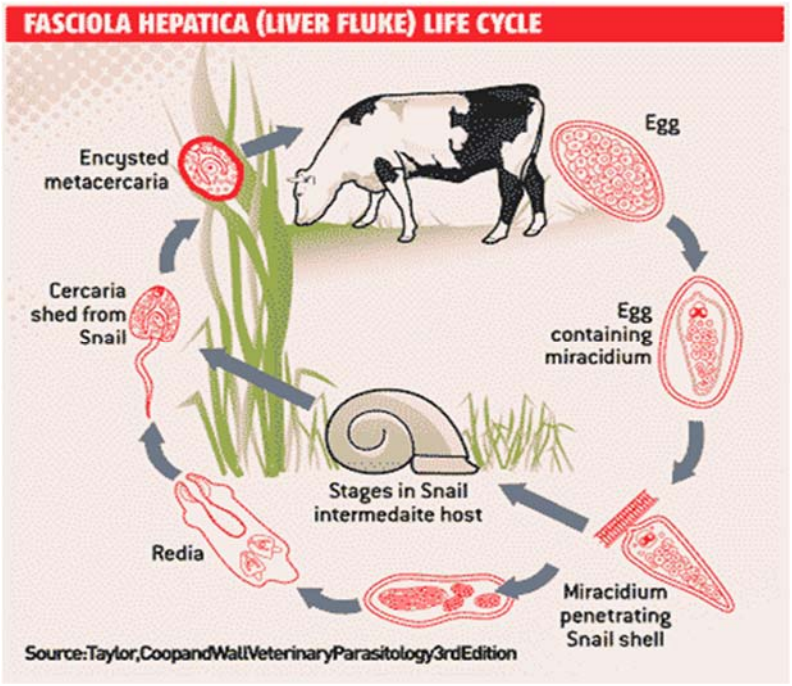
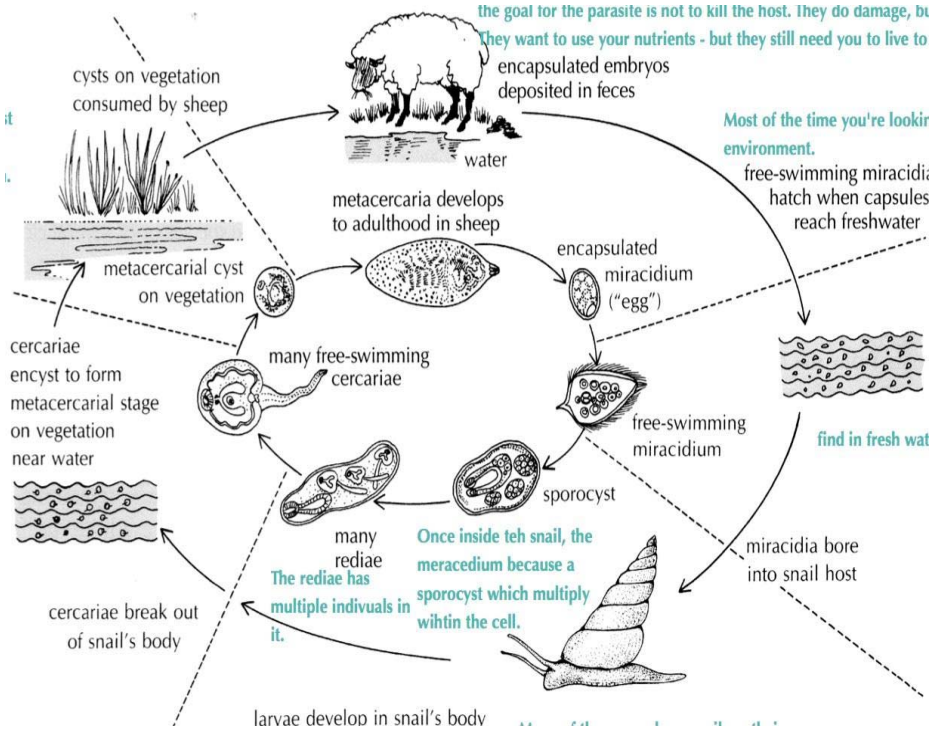
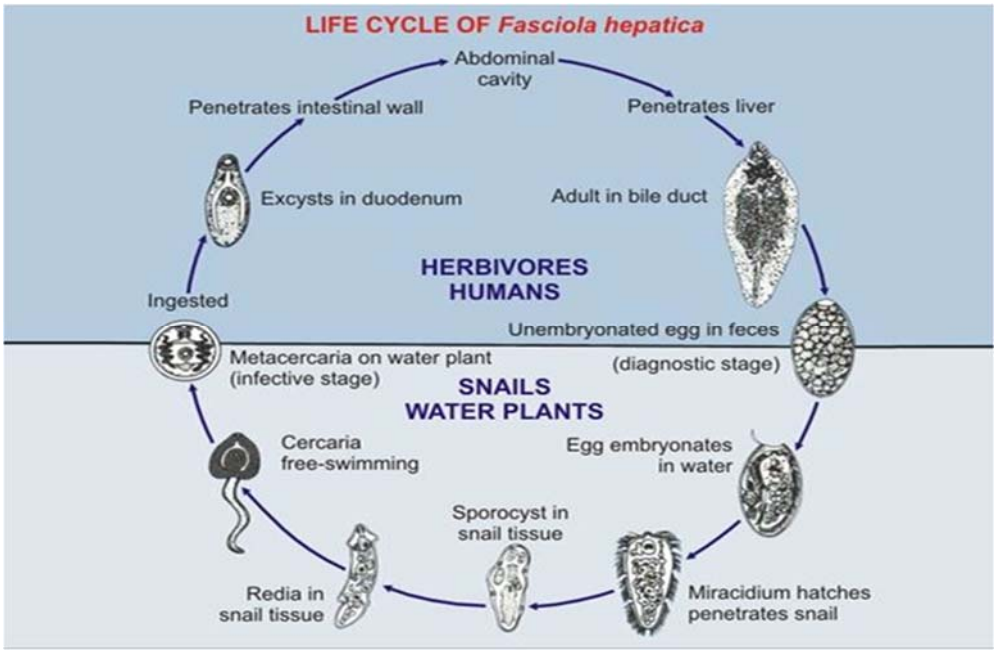


Fig-**Life Cycle of *Fasciola hepatica***





Clinical signs

- Fever
- Right-upper-quadrant pain
- Nausea
- Anorexia
- Jaundice
- Biliary obstruction
- Anemia Condemned Liver
- Adult-Biliary hyperplasia, dilatation, fibrosis, calcification
- Migration-Diffuse fibrosis, shrunken ventral lobe

Cattle: Partial immunity builds, self-cure at 5-8 months after major infection, most gone within 1 year. Sheep and goats: Little immunity, flukes live up to 10 years Rabbit, swine, other 'incidental' hosts are potential reservoirs. Zoonotic human infection via watercress, vegetables is a major problem in the altiplano of the Andes, Egypt, Iran, sporadic elsewhere

Rationale of treatment recommendations

- Treat in each region two months after transmission season ends to allow

flukes to become adult and susceptible to available drugs, but while still in maximum pathogenic phase.

- The end of season can be determined by producers when habitats dry up for >2 weeks in summer-fall or when sustained periods of winter cold
- Fluke worm looks like leaves of tree. This parasites has two sucker. With this they stay with the organ of animal. Generally domestic animals get infected by three types of fluke worm. They are listed below.
- Liver Fluke
- Stomach Fluke
- Blood Fluke

Symptoms

- Digestive system of fluke worm infected animal get damaged.
- They dedecate odor liquid.
- Suffers by inflammation in liver, bile duct and different organ if the fluke worm attack the liver.
- Infected animal become very week.
- They lose energy and productivity.
- Blood in their body get reduced and they suffer by anemia.
- Swelling place can be seen under the chin.
- Saliva may flows from the nose of fluke worm infected animal.
- Animal faces breathing problem.
- Infected animal stops eating food and die due to anemia.
- If fluke worm attacks the calf much then it may die.

Triclabendazole is effective at killing all stages of triclabendazole-susceptible flukes from two weeks old. Cattle may be slaughtered for human consumption only after 56 days from last treatment. Do not administer to cows producing milk for human consumption. Intensive use or misuse of preparations such as triclabendazole can give rise to drug resistance with reduced efficacy of the preparation.

Nitroxynil is licensed for the treatment of fascioliasis (infestation of mature and

immature Fasciola hepatica more than 8 weeks after infection). The interval between nitroxynil treatments must not be less than 60 days. Cattle may be slaughtered for human consumption only after 60 days from last treatment. Do not use in cattle producing milk for human consumption.

Clorsulon is only effective against adult flukes. Cattle may be slaughtered for human consumption only after 60 days from last treatment. Do not administer to cows producing milk for human consumption nor dairy cattle including heifers within 60 days of calving.

The recovery of chronically infected cattle is slow following treatment with a flukicide. Improved nutrition of affected cattle is essential to restore body condition and production. Treated cattle should be moved to clean pastures wherever possible.

Treatment

- According to the advice of veterinarian feed the animal wormicide medicine
- Don't let the animal to graze in the dampy field
- Apply wormicide medicine like round worm and tape worm

1.3 Round Worm

Round worms are of different types. For example, pinworm, hookworm, necator, ancylostoma, whipworm, trichuris trichiura, ascaris lumbricoides, strongyloides stercoralis, trichinella spiralis, strongyloidiasis, trichinosis etc. Round worm has similarity to earthworm. This types of round worm lives in the stomach, intestine duct and breathing system of cow, buffalo, goat, sheep etc. and lays eggs there. When the animal defecate, then the eggs fall in the ground. In favorable environment this eggs produce larva and awn. This types of larva lives in ground, leaves, grass etc. When other animal eat those, the larva go directly to the stomach and intestine duct of that animal. They live in the stomach and intestine duct and become an adult parasites. And they become very harmful for the animal. The symptoms, treatment and prevention method of round worm infected animal are described beneath.

Symptoms

- Round worm infected animal loss their appetite.

- Body weight get reduced slowly.
- Hair of their body seems rough.
- They defecate liquid closet.
- They suck the blood of animal. So, the animal suffers by anemia.
- Round worms consume all the nutrients so, the animal suffers by malnutrition.

- Big sized round worms gather in the intestine duct of animal. And they stop the path of intestine duct. As a result the infected animal die.

Treatment & prevention methods

- Clean the animal shade regularly.
- Keep the garbage of house in a pit hole.
- Don't let your animal grazing in muddy and dampy field.
- Always keep the animal house dry.
- Feed wormicide the calf regularly since they reach 3 months of age.

1.4 Babesiosis

Domestic animal gets infected by babesiosis disease due to a protozoa named babesia. The germ of this disease spreads by ticks from the wound place of infected animal. This disease is also known as (Piroplasmosis, Tick fever, Texas cattle fever,Red fever (Cf. Red water is Theleriosis), Spleenic fever,Biliary fever in horse)

Etiology

Various organism of *Babesia spp*:-

Species	Host	Intermediate host	Distribution
Babesiabigemina	Cattle, Buffalo	Boophilus microplus.	America, Africa,

		Boophilus annulatus. Rhipicephalus spp.	Australia, Asia.
Babesia bovis.	Cattle, Buffalo.	Boophilus calcaratus. Boophilus microplus. Rhipicephalus surss. Ixodes ricinus.	America, Africa, Australia, Asia and South Europe.
Babesia divergens	Cattle, Buffalo	Boophilus calcaratus. Boophilus microplus. Ixodes ricinus.	Europe (Britain)
Babesia motasi	Sheep/Goat	Hemaphysalis punctata. Rhipicephalus bursa. Dermacentor silvarum.	Asia, Africa.
Babaesia ovis.	Sheep/ Goat.	Rhipicephalus bursa. Ixodes persulcatus.	Europe, Asia.
Babesia equi.	Horse	Rhipicephalus sanguineus. Rhipicephalus bursa. Dermacentor spp.	Asia, Africa.
Babesia coballi	Horse/ Donkey	Rhipicephalus sanguineus. Rhipicephalus bursa. Dermacentor spp.	Asia, Africa.
Babesia canis.	Dog	Rhipicephalus sanguineus. Haemaphysalis leachi. Dermacentor spp.	Asia, Africa.
Babesia gibsoni.	Dog	Rhipicephalus sanguineus. Haemaphysalis bispinosa.	America.
Babesia felis.	Cat	Hyalomana scupense.	Indian subcontinent, South Africa.

Symptoms

- The red blood cells of animal breaks.
- For this reason the color of their urine become red.
- Temperature of the body of infected animal get increased highly.
- The pulse of artery get increased.
- Babesiasis disease infected animal suffers by anemia.
- They may suffers by dyspnoea.
- The animal caught by fever suddenly.
- Animal stops ruminating.
- The infected animal die due to lack of proper treatment.
- Life cycle
- Babesia spp. do not parasitize any vertebrate host cell other than erythrocytes.
- Each sporozoite (merozoite) penetrates the cell membrane of an erythrocyte with the aid of a specialized apical complex.
- Once inside, it transforms into atrophozoite from which two merozoites develop by a process of merogony (binary fission).
- In the passage of host blood to the mid gut of the tick vector, the development of two populations of ray bodies from the gamonts (gametocytes) occurs.
- The ray bodies undergo further multiplication within the erythrocytes, which continues after they have emerged.
- Large aggregations of multinucleated ray bodies form, but once division is complete, single-nucleated ray bodies that are now haploid and assumed to be gametes emerge from the aggregates and then fuse together in pairs (syngamy) to form a spherical cell (zygote) .
- The zygote selectively infects the digestive cell of the tick gut where they multiply and then the basophilic cells where further multiplication occurs with development tokinetes that escape into the tickhemolymph.
- In the gut cells, schizogony occurs with the formation of polyploidkinetes (large merozoites) .
- These motile club-shaped kinetes then escape .into the hemolymph and infect a variety of cell types and tissues, including the oocytes where successive

cycles of secondary schizogony occurs.

- Thus, transovarial transmission occurs with further development occurring in the larval stage.
- Kinetes enter the salivary glands and are transformed into multinucleated stages (sporogony) and these then break up to formsporozoites.

Incubation period

- Cattle: 2-3 weeks
- Horse: 8-10 days

Treatment & Prevention Methods

- Keep the living place of animal clean and dry always.
- Spray neocidol 40 wp 4-5 gram in the body of animal mixing with 2.5 liters water.
- When the animal start showing the symptoms of babesiasis disease then feed them boric acid and potassium aluminum sulphate by mixing with water.
- Imidocarb has been reported to protect animals from disease but immunity can develop. There are also concerns with regard to residues in milk and meat.

1.5 Coccidiosis

A protozoa named Eimeria causes coccidiosis disease in cattle. This disease affects much to animal aged between 6-12 months. Germ of coccidiosis disease enter into the intestine duct of animal through water and food. This germs causes sore in the alimentary tract of animal. Cattle housing in dampy and lower places is very favorable for spreading the germ of coccidiosis disease in cattle.

COCCIDIOSIS IN POULTRY

Coccidia are almost universally present in poultry-raising operations, but clinical disease occurs only after ingestion of relatively large numbers of sporulated oocysts by susceptible birds. Both clinically infected and recovered birds shed oocysts in their droppings, which contaminate feed, dust, water, litter, and soil. Oocysts may be transmitted by mechanical carriers (eg, equipment, clothing, insects, farm workers, and other animals). Fresh oocysts are not infective until they sporulate;

under optimal conditions (70°–90°F [21°–32°C] with adequate moisture and oxygen), this requires 1–2 days.

ETIOLOGY

Coccidia are host-specific, and there is no cross-immunity between species of coccidia. The prepatent period is 4–7 days. Sporulated oocysts may survive for long periods, depending on environmental factors. Oocysts are resistant to some disinfectants commonly used around livestock but are killed by freezing or high environmental temperatures.

There are seven different Eimeria species that infect chickens;

1. *Eimeria acervulina*
2. *Eimeria maxima*
3. *Eimeria tenella*
4. *Eimeria necatrix*
5. *Eimeria mitis*
6. *Eimeria brunetti*
7. *Eimeria praecox*

Clinical Signs

- Signs of coccidiosis range from decreased growth rate to a high percentage of visibly sick birds, Severe diarrhea,
- High mortality
- Feed and water consumption are depressed
- Weight loss
- Decreased egg production
- Increased mortality may accompany outbreaks.
- Mild infections of intestinal species, which would otherwise be classed as subclinical, may cause depigmentation and potentially lead to secondary infection, particularly Clostridium spp infection.
- Survivors of severe infections recover in 10–14 days but may never recover lost performance.

Control

Poultry that are maintained at all times on wire floors to separate birds from droppings have fewer infections; clinical coccidiosis is seen only rarely under such circumstances.

Other methods of control are vaccination or prevention with anticoccidial drugs.

Treatment

Sulfonamides are widely used: sulfadimethoxine, sulfaquinoxaline, sulfamethazine,

The supplementation of vitamins A and K promotes the recovery.10g/10 lit for 3 days Plain water for next 2 days10g/ 20 lit water for next 3 days supercox

CODRINAL- 4 gm of codrinal is to be added to 1 litre of drinking water and administered to the ill birds for 2-4 days. Each gm contains: Sodium salt of (P) - toluenesulphonyl 0.55 gm betamethoxyethylurethane Tetracycline hydrochloride 0.05 gm Crystalline lactose 0.375 gm Dried sodium bisulphite 0.025 gm-30 gm in 25 litres of water for 5-7 days.

Alternative Medicines

Oregano, green tea and cinnamon all help fight coccidiosis

Coccidiosis in Calves

Introduction

It is a disease of calves, lambs, kids & foals Characterized clinically by:- hemorrhagic enteritis, diarrhoea, dysentery, depression, weakness & loss of weight The chronic form characterized by retardation of growth rate & production the infection occur after ingestion of food & water contaminated with sporulated oocyst

Two most important spp are *E.bovis and E.zuernii*. Coccidiosis more frequently occurs in calves from 1-6 month of age. Young calves are usually infected when they are placed in pastures or lots contaminated by other infected cattle or calves.

Life Cycle

The oocysts are present in manure. Before they become infected, they undergo a developmental process called sporulation. Sporulation occurs outside the animal's

body. Sporulation requires 2-3 days which results in the formation of 8 infective sporozoites within each oocyst.

When a susceptible calf swallows infective oocyst, the sporozoites are released penetrate the epithelial Cells of intestine. The sporozoites then begin to divide into many intermediate stages, merozoites. The merozoites reinvade the intestinal epi.cells and can undergo an additional round of merogony or develop into either micro or macrogametes. These gametes unite to produce oocyst which passed out of the animal's body in manure.

Clinical Signs

- Severe diarrhea with foul smelling fluid feces contain mucous & blood
- Severe straining, rectal prolapse in severe case
- Pale M.M.
- anorexia, weakness & dehydration

Diagnosis

- May be made from a combination of herd history,
- Clinical signs,
- Gross lesions at necropsy,
- Microscopic examination of scraping of the intestinal mucosa and manure.

Treatment

- Amprolium → 10 mg/kg b.wt/orally/daily/for 5 days
- Sulfadimidine (sulfamethazine) → 140 mg / kg b.wt / orally / daily for 3 days
- Ringer solution → 1-2 liter / I.V/ daily

Control

Prevent manure contaminated fodder and water. Keep pens dry and supply with ample dry bedding. Use well drained pastures that are well drained. Raise watering troughs above the ground. Keep grazing to a minimum on grasses along the edges of ponds and streams. Prevent overgrazing. Animals forced to graze down to the roots of plants may eat large numbers of parasites. Heavily parasitized animals should be isolated from the rest of the herd and treated

Pasture should be well drained. Avoid grazing on lush grass along the edges of

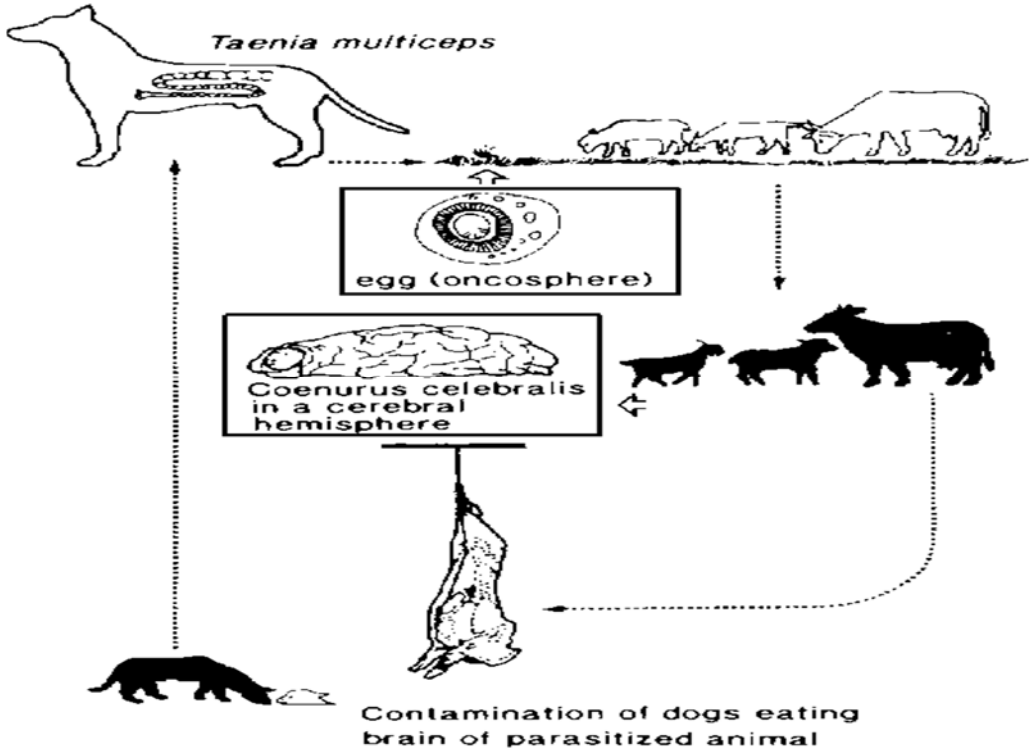
ponds and streams. Overgrazing should be avoided. Separate the infected animal and treat them. Reduce moisture on pasture

1.6 GID (*Coenurus cerebralis* infection, Sturdy)

Gid also known as Coenurosis is a disease of the central nervous system in goats and sheep. Coenurosis is infection by the metacestode larval stage (coenurus) of *Taenis multiceps* which infests the small intestine of carnivores. The definitive hosts for *Taenis multiceps* are members of the family Canidae. In 80–90% of cases, the cyst is located in one cerebral hemisphere, whilst in 5–10% of cases, it is localised in the cerebellum; rarely it involves two sites in the brain of the affected animal.

Coenurus cerebralis, the larval form of *Taenis multiceps* which is seen in the small intestines of carnivores. Infection occurs as a result of the oral intake of eggs spreading vis faeces of those animals by intermediste hosts. The disease is known as gid or sturdy which primarily localises in the central nervous system of sheep and goats mostly, but can also seen in camels, deer, pigs, horses, however, rarely in cattle and humans. Most of the cysts are located in the cerebral hemispheres and spinal cord, while rarely invading the subcutaneous and intramuscular tissues along with other organs Symptoms vary depending on the cyst's location, size and compression. *Coenurus cerebralis* initially causes purulent meningoencephalitis, later as the cyst grows, it leads to central nervous system symptoms resulting in. Most of the characteristic clinical findings are observed 2-8 months after the intake of pathogen. Infected animals manifest circling, head tilt towards the side of the cyst location, in coordinated and uncontrolled movements, ataxia, failure to hold the head straight, blindness, teeth grinding, salivation, paresis, convulsions.

Life cycle: *Eggs* expelled with dog faeces are ingested by the intermediate host (sheep). The *larvae* hatch in the intestine and pass with the blood stream towards different organs. The larvae which reach the brain and spinal cord grow to the *coenurid* stage. *Coenurus cerebralis* will further mature in the brain and spinal cord.



Life cycle of Taenia multiceps

Clinical findings

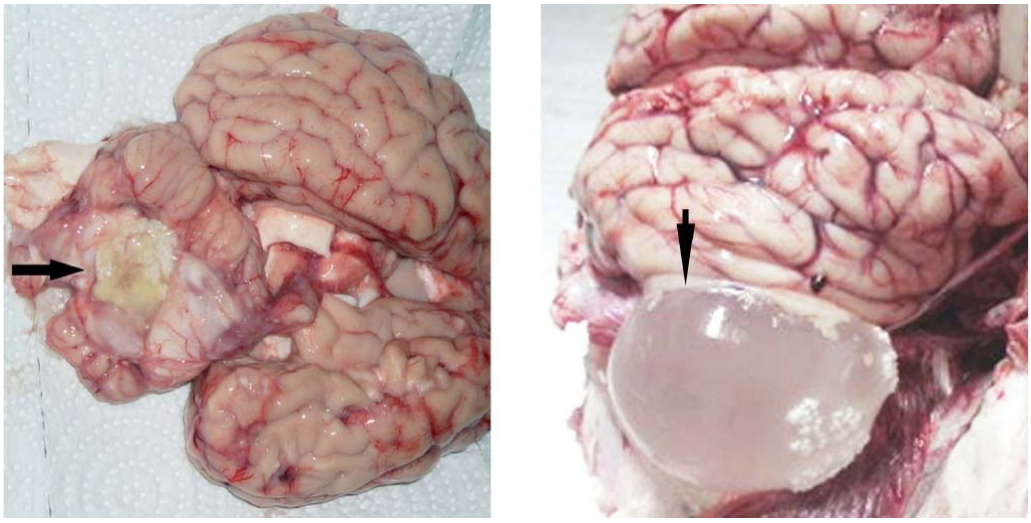
During migration of larval stage

1. Blindness
2. Muscular tremor and incoordination
3. Excitability and collapse
4. Infection with the fully developed larval stage
5. Salivation
6. Wild expressions
7. Frenzied running and convulsion
8. Deviation of eye and head
9. Loss of function
10. Dullness
11. Incomplete mastication

12. Head pressing
13. Incomplete paralysis and, in spinal cord involvement, inability to rise

Postmortem findings:

1. Thin walled cyst in the brain.
2. Lesion in the lumbar region and rarely, in the cervical area of the spine



Coenurus cerebralis. Thin walled cyst in the brain

Diagnostic information

Infected dogs pass *taenis*-type eggs in their faeces. Sheep at the chronic stage of the infection may show circular movements, jerky movements or staggering gait.

Treatment:

For adult tapeworm a number of effective drugs are available including praziquantel, mebendazole, fenbendazole, nitroscanate, and dichlorophen.

Surgical removal is possible if the cyst is situated on the brain surface.

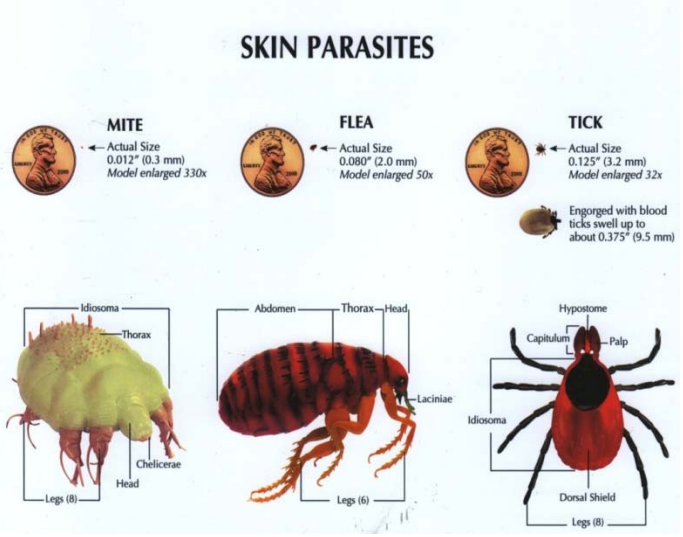
Unit: Two

Diseases caused by external parasites (Ectoparasites)

Learning Outcomes

After the completion of this unit, student will able to

- Explain the general concept of ectoparasites.
- Identify the different ectoparasites.
- Classify the different animal disease causes by ectoparasites.



2.1 Lice

1) Suckling lice (anaplura)

- The mouth parts are adopted for sucking the tissue fluids and the blood of the host.
- Head is more or less pointed interiorly; two antennas are visible at he side of the head and are composed of 5 segments.
- Thorax is small and it's 3 segments are composed of 5 segments
- Abdomen is large with 9 segments
- The first pair of the leg is smaller while the 3rd pair of leg is larger.

2) Biting lice (mallophaga)

- Biting lice feed on the epithelial debris on the skin of the host or on the

feathers of the birds and have mouth parts adopted for chewing of these materials.

- The mesothorax and metathorax are fused to form one piece in front of which the prothorax and distinct and separate segments.
- Thoracic spiracles are on the ventral side of the mesothorax.

Ticks

- Ticks are giant mites.
- All stages of the tick attacks to animal sucks blood and transmit diseases.
- A 6 legged larva hatches from the eggs sucks the blood and molts to an eight legged nymph which also suck blood. The nymph mouths to an 8 legged adult.
- Hosts are depending upon the how many times they drop off and seek a new host on moulting.
- It acts as the vector for *BABESIOSIS* diseases

Mites-

- It attacks fowl, pigeon, and also many wild birds. It may also feed on man.
- It is often called red mite of poultry. It becomes only red when it has recently fed on its host blood; otherwise it is whitish, grayish or black.
- The body is divided into two portions i.e. anterior minute gnathosoma which bear mouth parts and posterior idiosoma.
- Larval stages of mites normally have 3 pairs of legs; nymphs and adult have 4 pair's legs.

Three types mites

1. **Sarcoptes mites** - are roughly circular in outline. All the legs of the both sexes are short and 3rd and 4th pair's .Do not project beyond the margin of the body female bear interiorly 3 short spines and posterior 6 longer spines.
2. **Psoroptes mites**- are oval in shape and tarsal suckers have jointed pedicles.
3. **Chorioptes mites**- are similar to Psoroptes mites but tarsal suckers have un jointed pedicles.

Control measures of parasites

The following control measures may be taken:

1. Treatment of the animal by using appropriste anthelmantics.
2. The animal should not be allowed to graze in the field of low land areas where water reservoir like river, pond, lake are present.
3. Water reservoir should be fenced.
4. Faeces should be disposed properly.
5. The grasses in the form of hay or silage should be provided
6. Underground water should be provided for drinking water.
7. Control of intermediste host (mollusca) is done by use of molluscicidal agent like copper sulphate solution and N-Tritylmorpholine in spring and mid summer.
8. Vaccination is a recent trend for control.

Unit: Three

Bacterial diseases of livestock

Learning Outcomes

After the completion of this unit, student will be able to

- Identify the different bacterial diseases of livestock.
- Recognize causative agent, pathogenesis and clinical sign and symptoms of livestock diseases.

Anthrax

Synonyms

- Splenic fever
- Milzbrand (German term)
- Charbon (Malignant charcoal)
- Wool sorter's disease
- Patkhe (Nepal**)**

Distribution

- It is one of the ancient diseases that is worldwide in distribution**.**

Susceptible hosts

- Most of the animals food
- The most susceptible animals are cattle and sheep.
- Horses and pigs are also affected.
- Not uncommon in camels.
- Anthrax is a zoonotic disease.

Etiology

- *Bacillusanthracis.*

Mode of transmission

Soil borne

- Animals while grazing in the infected pasture pick up the infection (spore) through ingestion or through breach in the oral mucosa or skin.

- The new area may be infected due to contaminated animal products such as bone meal, fertilizers, hide, hair, wool, grain or forage.

Clinical findings

Per-acute form

- Sudden death of animal

Acute form

- Elevation of body temperature (104 to 1080F).
- Animal refuses to eat and there is development of bloat.
- Ruminal stasis is evident.
- Animal is extremely depressed.
- Pulse and respiratory rates are accelerated to a great extent.
- With the advancement of the disease process there is development of muscular tremor.
- Some animals may show extreme aggressive behaviour
- At the end, animal show distressed breathing and mouth breathing.
- Death usually takes place within 48 hours.
- Death is followed with bleeding from all the natural orifices.

Sub-acute form

- It is characterized by oedema.
- Edema is predominantly noticed under the neck, brisket region, thorax, abdomen and flank.
- Pregnant cattle may abort.
- Some may survive for 2 to 3 months.

Diagnosis

- Diagnosis is based on clinical history of abrasion of skin, history of occurrence near about area.
- Post mortem examination
- Exudation of tarry blood from body orifices.
- Failure of the blood clotting
- Absence of rigormortis.

- Splenomegaly.
- If anthrax is suspected post mortem examination should be avoided to reduce environmental contamination and health hazard.

Confirmation

- To confirm the diagnosis blood from ear vein and local edema fluid should be collected and stained with 1% polychrome methylene blue (Mc Faydean reaction).
- Mice inoculation test
- Ascoli's Ppt. test

Differential Diagnosis

- ***Lighting strike:*** Singeing of hair & history of electrical storms
- ***Per acute BQ:*** Lightly restricted to young animals & crepitating swellings
- ***Bacillary hemoglobinuria*****:** hemoglobinuria & presence of characteristic infarcts in liver
- ***Acute leptospirosis:*** occurs only sporadically & presence of hemoglobinuria
- *Per acute lead poisoning, hypomagnesemia tetany:* obvious nervous signs & a complete different necropsy findings.

Treatment

- Penicillin 10,000 units per kg body weight twice daily through parenteral route.
- Oxytetracycline, erythromycin, or sulphonamide has been used.
- Anti anthrax serum @ 100 – 200 cc through intravenous route along with a course of penicillin may be given.

Prevention & Control

- Control of meat & milk from animals in infected herds–
- Avoid any risk to the human population
- Avoid unnecessary waste & imposition of unnecessarily harsh prohibitions on farmer
- When an outbreak occurs, followings are part of animal disease control program & indirectly reduce human exposure–

- Placing of farm on quarantine
- Destruction of discharges & cadavers
- Vaccination of survivors
- Prohibition of movement of meat & milk from farm during quarantine period should prevent entry of infection into the human food chain
- Hygiene is the biggest single factor in the prevention of spread of disease–
- Careful disposal of infected material
- Infected carcasses should not be opened but immediately burned /buried, together with bedding & soil contaminated by discharges
- Burial should be at least 2 m deep with an ample supply of quicklime added
- All suspected case & in-contact animals must be segregated until cases cease & for 2 wks thereafter the affected farm placed in quarantine to prevent movement of animals

Black leg

Synonyms

- Black quarter
- Quarter ill
- Quarter evil
- Emphysematous gangrene
- Charchare (Nepal)

Definition

This is an acute infectious disease of cattle, goat and sheep. The disease is characterized by development of focal gangreenous and emphysematous myositis.

Etiology

Black quarter is caused by *Clostridium chauvoei,* a gram positive rod shaped, spore forming toxin producing anaerobe.

Epidemiology

Occurrence

When the disease occurs it is usual for a number of animals to be affected within the space of a few days.

- Disease is enzootic in particular areas, especially when they are subject to flooding.
- Case-fatality rate in blackleg approaches 100%.

Source of infection

- Blackleg is soil-borne infection.
- Bacteria may be found in the spleen, liver, and alimentary tract of normal animals, and contamination of the soil and pasture may occur from infected feces or decomposition of carcasses of animals dying of the disease.

Transmission

- Disease spreads from contaminated soils.
- Contamination of the soil is due to infected feces and carcasses which cause pollution of the land.
- Organisms gain entry through ingestion of infected feeds or possibly through contamination of wounds.

Risk factors

Environmental risk factors

- Typical blackleg of cattle has a seasonal incidence with most cases occurring in the warm months of the year.
- The highest incidence may vary from spring to autumn, depending probably on when calves reach the susceptible age group.
- In some areas there is an increased prevalence in years of high rainfall.
- Outbreaks of blackleg in cattle have occurred following excavation of soil which suggests that disturbances in soil may expose and activate latent spores.

Animal risk factors

- True blackleg is usually thought of as a disease of cattle and occasionally sheep but outbreak of the disease has been recorded in deer and and it is reported in a horse.
- In cattle the disease is largely confined to young stock between the ages of 6 months and 2 years although disease occurs occasionally in younger animals and cattle up to 3 years.

Pathogenesis

- Ingestion of the spores
- The spores from the alimentary tract penetrate tissues from the places of breach of alimentary mucosa due to trauma.
- The ingested organisms also carried from the intestine via circulation to the skeletal muscles.
- Some of the spores in the muscles are destroyed by phagocytosis and others remain latent for at least several weeks.
- Very often, welformed heavy muscles like muscles of gluteal region, loin, shoulders are affected.
- There is necrosis of the muscles and blood capillaries.
- Gases accumulate within the muscles fibres due to fermentation.
- Exotoxin is produced from the organisms Which causes systemic reaction characterized by toxaemia and local reaction characterized by necrotising myositis.

Clinical findings

Cattle

- The first sign is rise in body temperature which may be as high as 1060 F - 1080 F.
- The appetite is lost and rumination is suspended and there is stiffness or lameness in one of the limb.
- Very soon characteristic swelling develops in one of the thick layers of muscles. Most commonly the lesions are located on the thigh, buttock, shoulder, neck and lumbar region. Swelling are hot and painful in the early stage and become cold and painless later.
- On pressure swellings emit crackling or crepitating sound due to emphysema.
- Skin over the swelling is discolored and dry.
- There is labored breathing and accelerated pulse rate (100-120/minute).
- Occasionally there is distinct abdominal pain.
- Finally, the temperature drops and the patient dies within 12 to 48 hours.

Diagnosis

- History of outbreak.
- Clinical observation and postmortem findings.
- Microscopic examination of smear
  - By examination of smear made from the affected tissues or fluid of the swellings, gram positive rods with sub-terminal spores will be seen.
- FAT
- Differential diagnosis
- Black quarter must be differentiated from anthrax, malignant edema and bacillary hemoglobinuria.

Treatment

- Satisfactory response has been reported from the use of penicillin, aureomycin and oxytetracycline.
- The antibiotic may be injected into the affected muscles.
- Penicillin is extensively used and considered as drug of choice.
- Penicillin @ 20,000 to 40,000 units per kg body weight per day may be used.
- Crystaline penicillin may be given through intravenous route followed by procaine penicillin through i/m route.

Control

- Since the disease is associated with infection from the soil, the cultivation in that soil may be avoided.
- The young animals should be kept out of such area.
- The dead body should be burnt or buried.
- The dead body should not be allowed to skinned.
- The calf should not be allowed to graze in endemic pasture.
- All the animals of the endemic zones should be vaccinated with suitable vaccine.

Brucellosis

Synonyms

- Bang's disease

- Contagious abortion
- Infectious abortion
- Enzootic abortion
- Undulant fever
- Malta fever
- Mediterranean fever

Definition

Brucellosis is an acute or chronic contagious disease of domestic animals that mainly causes placentitis and abortion in females; and orchitis and epididymitis in males.

Distribution

- **World wide**

Etiology:

Brucella spp

Brucella abortus (cattle), *Brucella melitensis* (goat), *Brucella suis* (swine), *Brucella ovis* (sheep)e.t.c.

Clinical findings

- Abortion takes place from 6 months onwards (last trimester of pregnancy).
- Greyish white mucoid or muco-purulent discharges from vagina.
- Prior to parturition cow may show the clinical pattern of normal parturition like swelling of vulva, relaxation of pelvic ligament, enlargement of udder and discharges from vulva.
- Retention of foetal membranes.
- There may be chronic endometritis.
- In bull epididymitis, orchitis and inflamation of accessory genital organ.

Diagnosis

- Byclinical findings.
- Laboratory diagnosis
- Isolation and identification of organisms.

- The materials used for such examinations include stomach content, spleen, muconium, lung of the aborted foetus, chorion of placenta, uterine exudate, vaginal discharge of dam, milk, abscess of testis and epididymis.
- Modified acid fast and Grams stain of the impression smear will reveal Gram negative cocco-bacilli.
- Serological test: RBPAT, Standard tube agglutination test, CFT, MRT, ELISA.

Treatment

- The organisms live inside the cells of the infected animal and that is why the success rate of the treatment is not impressive.
- Long acting Oxytetracycline 20 mg/kg b wt i/m 5 injections at 3-4 days intervals and streptomycin 25 mg/kg b wt i/m or i/v daily for 7 days.
- In case of infected horse, Chloramphenicol 1 gm/ 100 kg b wt i/m or i/v daily for 12-20 days.

Hemorrhagic Septicemia

Synonyms

- Pasteurellosis
- Shipping fever

Definition

- It is an acute septicemic disease occurring most often in cattle, buffalo, camel, sheep, goat and pig usually following some form of stress, such as transportation, exertion etc.

Distribution

- Tropical and subtropical countries.
- most commonly during or following monsoon.
- The outbreaks are seen during the period of high humidity.

Etiology

- The disease is caused by *Pasteurella multocida type -1* organism.
- The organism may be present in the respiratory tract as commensal and may not be able to produce the disease alone.

- Predisposing factors like fatigue, transportation, over exertion, starvation, close confinement to damp and humid atmosphere may help the organism to assume virulent role and set up the disease process.

Susceptible hosts

- Cattle and buffaloes are the most susceptible species.
- Young growing cattle within the age group of 6 months to 2 years are most often affected.
- Over fed cattle may suffer when they are put under stress.
- Sheep, goats and pigs

Clinical findings

- The incubation period is 2-5 days.
- There is high rise of temperature (104-1070F) with concurrent shivering.
- Profuse salivation, lacrimation and nasal discharge.
- There is sign of conjunctivitis and the visible mucous membrane assume deep red color.
- There is sharp drop in milk yield in lactating cows.
- Abdominal pain, severe diarrhea or dysentery.
- Respiration rate is rapid in nature.
- Auscultation of lung reveals increased vesicular rales and moist rales.

Due to the progression of the disease lung sound may become inaudible as a result of consolidation.

- In less acute cases localization of edema in the subcutaneous pocket of the head, neck, dewlap and brisket region.
- The edematous swelling are hot and painful.
- The edema produces severe dyspnea due to obstruction of respiratory passage.
- Death usually occurs within 20-24 hours.

Diagnosis

- History of predisposing factors and clinical findings.
- Nasal swab or washing and the exudates may be used for culture from living

animals.

- Smears from heart blood, liver, lungs, spleen and intestinal content from dead animals should be stained with leishman stain. Bipolar short ovoid rods may be located.

Treatment

- In the initial phase of the disease intravenous administration of sulphonamides i.e. sulphamethazine @ 150 mg / kg body weight for 3 days or sodium sulphadimidine in the similar dose may give effective response.
- Oxytetracycline @ 5-10 mg/kg body weight for 3-5 days.
- Besides, treatment may be extended with chloramphenicol @ 10 mg/kg body weight or ampicillin @ 10 mg/kg body weight.
- Long acting oxytetracycline may be given to reduce the treatment cost.
- Symptomatic treatment with anti-inflammatory drugs eg. Betamethazone or Dexamethazone @ 1 mg/5kg body weight may be required.

Mastitis

Synonyms

- Mammitis
- Mammite
- Inflamation de la ubres.

Definition

Mastitis is the term which denotes inflammatory condition of the udder irrespective of causes. It is characterized by physical, chemical and microbiological changes in the milk and pathological changes in the glandular tissues of the udder. The changes in the milk include change of color, change of consistency (clot) and presence of abnormally large number of leukocytes.

Etiology

Cattle

Bacteria: Staphylococcus aureus; Str. agalactiae; Str. zooepidemicus; Str. faecalis; Str. pyogenes; Corynebacterium pyogenes; Klebsiella Spp.; Mycobacterium bovis; Escherichia coli; Brucella abortus; Pseudomonas pyocyaneus; Leptospira pomona;

Pasteurella multocida.

Mycoplasma: Mycoplasma bovis; Mycoplasma bovigenetelium.

Fungus: Trichosporon spp.; Aspergillus fumigatus; Aspergillus midulus; Candida Spp.; Cryptococcus neoformans.

Virus: Some viruses (Vesicular stomatitis, Infectious rhinotracheitis) have been implicated as cause of mastitis in cattle.

Buffalo

Bacteria: Staphylococcus aureus; Streptococcus agalactiae; Streptococcus dysagalactiae; Streptococcus uberis; Streptococcus bovis; Escherichia coli; Pseudomonas spp.

Mycoplasma: Mycoplasma bovis; Mycoplasma bovigenetelium.

Fungus: Trichosporon spp.; Aspergillus fumigatus; Aspergillus midulus; Candida Spp.;Cryptococcus neoformans.

Horse

Bacteria: Corynebacterium pseudotuberculosis; Streptococcus zooepidemicus; Streptococcus equipment.

Epidemiology

Occurrence and prevalence of infection

- Prevalence of infection is about 50% of cows and 25% of quarters. Quarter infection may be as low as 10%. Incidence of clinical mastitis ranges from 10-12% per 100 cows at risk per year. Case fatality rate depends on cause of mastitis.
- Prevalence of infection with contagious pathogens ranges from 7-40% of cows and 6-35% of quarters.
- Prevalence of infection with environmental pathogens:coliforms 1-2% of quarters; streptococci less than 5%.
- Environmental pathogens are most common cause of clinical mastitis in herds which have controlled contagious pathogens.
- Contagious pathogens transmitted at time of milking; environmental

pathogens between milking from environment.

Mode of transmission

- The avenue of infection is the teat canal. Through teat canal the infection reaches the mammary gland.
- There are two sources of infective agents. They are the *udder*-where many bacteria like *Streptococcus agalactiae* and *Staphylococcus aureus* may continue to persist as normal inhabitant and the *environment*-where bacteria like *Escherichia coli* and *Pseudomonas pyocyaneus* continue to persist.
- These organisms when multiply and invade the tissues produce much damaging effect and they are not amenable to common hygienic practices.
- Cutaneous surface of the cow may have many organisms as resident population and from where the organisms may have the chance to invade through contamination by handlers.
- Contamination of milkers' hands, clothes and machine cup by milk from the affected quarter may lead to the spread of the disease to other non-infected teats of cows.
- Fly and other insects may also spread the infection from one place to the other.
- Spread of infection is also possible through bedding ground by discharges of affected gland.

Risk factors

- **Age and parity:** The disease is more prevalent in high age group. More the number of lactation more the possibility of the disease.
- **Breed:** Incidence is more in exotic and crossbred cows than the zebu ones.
- **Stage of lactation:** Infection rate is more at the initial and end stage of lactation.
- **Complete milking:** Incomplete removal of milk from the udder is a conductive factor.
- **Size of the herd:** Small herd seldom suffer. The disease is more frequent in large herd.
- **Feed offered:** Heavy protein feeding may act as predisposing factor.
- **Seasons:** In western countries more prevalent in summer but in our country

it occurs irrespective of season.

- **Genetic factors:** Some breeds are more susceptible to this infection due to obvious reasons of low slung udder and long teats which render more mechanical damage and pave the way for infection.
- **Trauma:** Any mechanical injury to teat or udder helps the micro organisms to enter the organ. Even trauma during faulty milking may assist in bacterial invasion.
- **Hygiene:** Bad hygiene and sanitation help bacterial multiplication. Maintenance of adequate hygiene at pre and post milking is important in relation to setting up of infection.
- **Resistance:** Some animals may have more resistance obviously due to teat shape and morphology of teat duct.
- **Skin lesions:** The mastitis organisms may be present in the skin and teat canal orifice thus infection may be more prevalent in cows where the resident bacterial population in the skin is abundant in proportion.
- **Infected quarter:** The udder which is previously infected is less likely to be infected again. There is less possibility of establishment of new infection.
- **Retained placenta:** Incidence of mastitis has been observed to be frequent in nature amongst cows with retained placenta.

Clinical findings

Mastitis may clinically be classified as (i) Per-acute mastitis (ii) Acute mastitis (iii) Sub acute mastitis and (iv) chronic mastitis.

Per-acute mastitis

- Affected animal shows a very high temperature (106-1070 F).
- Offfed and show respiratory distress.
- Udder is swollen and extremely painful.
- Cessation of milk secretion and exudates are often blood stained.

Acute mastitis

- There is no systemic reaction.
- Udder becomes swollen and there is changes in milk.

- Milk may be replaced by custard material yellow or brown fluid with flakes or clots.
- Infection may be localized in one quarter or the entire udder may be involved.

Subacute mastitis

- Variable changes in the milk but practically no
- Changes in the udder tissues.
- Culture of milk will show presence of pathogenic bacteria.

Chronic mastitis

- Chronic mastitis is the terminal stage of the disease.
- Udder becomes hard due to fibrosis.
- Supra-mammary lymphnodes become palpable.
- Quarters may become thickened, firm, nodular and atrophic.
- Parenchymatous cells are replaced by connective tissues.
- Milk changes vary considerably. The milk may appear as yellowish fluid or white with clots and flakes. Sometime it may look as green or yellow-green and foul smelling.
- Teats may reveal injuries or sores near the orifices with involvement of supra-mammary lymph nodes.

Diagnosis

- History
- Clinical findings
- *Physical examination of the udder:* Palpation and inspection of the udder are directed for the detection of fibrosis, inflammation, swelling and atrophy.
- Pathological tests to detect bacteria, cells, clots and chemical changes.
- Direct microscopic examination for detection of bacteria and WBC.
- ***Differential diagnosis:*** udder edema, blood in milk, cyst & abscess, cancerous growth, actinomycosis in udder.

Tests for milk abnormalities

- Strip cup test

  Before initiating the proper test, milk should be collected properly

- Udder should be cleaned with warm water.
- The udder should be wiped with clean cloth soaked in disinfectant.
- The udder should be allowed to dry.
- The teat orifices should be painted with Tr. Iodine or 70% alcohol.
- Milk should be collected directly in the tube.
- At least 5 ml of milk should be collected.

The strip cup consists of a flat enamel plate, partitioned into 4 areas. The plate is black in color so that the clot is clearly visible. The milk is to be stripped directly into the cup. The stripping should be made from left hind, then right fore and lastly right hind. Presence of clot or flakes will indicate abnormality of milk. Such cow, should be put for further testing and kept in isolation.

- Bromothymol blue test
- Bromocresol purple test
- Chloride test
- California mastitis test (CMT)
- White side test.

Direct test

- Isolation and identification of the organisms.
- Cultural examination.
- Bio-chemical test.
- Animal inoculation test.
- Serological test
    - Slide agglutination test.
    - Precipitation test.
    - Plasma coagulase test.

Treatment

Treatment strategies vary with the clinical severity of the disease.

- The sub-acute case requires only intramammary therapy for 3 days after each milking.
- The acutely affected cow should be treated with systemic and intramammary

antibiotics for a minimum of 3 days.

- The per-acute cow needs to be treated with systemic and intra-mammary antibiotics, oral or intravenous fluids and anti-inflammatory drugs.

■ **Per-acute treatment**

- Although the rate of diffusion of antibiotic is greater but still the selection of parenteral antibacterial drugs should be made on the basis of diffusion from the blood stream into the milk.
- Erythromycin, tylosin, penethemate, chloramphenicol and trimethoprim are diffused well.
- Penicillin and tetracyclines are medium performers and neomycin and streptomycin are poor diffusers.
- Treatment of mastitis should be made on the basis of resistant of the antibacterial drugs.

Udder infusions

- Disposable tube containing suitable drugs in a water soluble ointment base are best suited for dispensing and the treatment of individual cow.
- Complete emptying of the quarter before infusion by the parenteral injection of oxytocin is advisable in cases of acute mastitis. Strict hygienic measures is necessary during treatment to avoid the introduction of bacteria, yeasts and fungi into the treated quarters.
- The drugs which have the best record of diffusion through the udder after intra-mammary infusion are penethemate, ampicillin, amoxycillin, chloramphenicol, novobiocin, erythromycin and tylosin.
- Those of medium performance are penicillin G, cloxacillin and tetracyclines.

Tetanus

Synonyms

- Lock Jaw

Definition

It is a non-contagious, non-febrile, infectious disease of mammals caused by toxin characterized by spasmodic contraction of skeletal muscles.

Etiology

- The disease is caused by *Clostridium tetani.* The organism is long, slender, rod shaped anaerobe with rounded ends. The organism develops terminal spores which give it a drum stick appearance.

Epidemiology

Occurrence

- Tetanus occurs in all parts of the world and is most common in closely settled areas under intensive cultivation.
- It occurs in all farm animals mainly as individual, sporadic cases, although outbreaks are occasionally observed in young cattle, young pigs and lambs following wounding management procedures.

Case-fatality rate

- In young ruminants the case-fatality rate is over 80%, but the recovery rate is high in adult cattle.
- In horses it varies widely between areas. In some areas almost all animals die acutely, in others the mortality rate is consistently about 50%.

Source of infection

Soil-borne

- Feces of infected animals.

Transmission

- The organisms gain entrance through deep punctured wound contaminated with bacterial spores.
- Organisms may gain access during parturition and manual handling of the genitalia with contaminants.
- Retention of placenta and prolapse may augment the transmission if not attended properly.
- Castration by open methods is an important avenue of transmission in bull calf, kid, lamb and piglet.
- Shearing, docking and vaccination may also help in setting up the disease process.

- Neo-natal animals may get the infection through contaminated umbilicus.
- Deep wound in the feet during grazing, ploughing or transport may contribute in establishment of the disease.
- Wound of oral mucosa, dental caries etc. may also influence the disease transmission.
- Wound due to surgical interference may also help in localization of infection.
- Wounds by a penetrating object e.g. (nail etc.) contaminated by dirt may act as a significant factor in establishment of the disease.
- Trauma and damage of the tissues caused by injection, vaccination or chemical agents such as calcium salt, lactic acid, or by infection with other bacteria may help in the initiation of the disease processes.

Animal risk factors

- Horse is more susceptible than cattle.

Importance

- Tetanus is important because of its high case fatality and the very long convalescence in the survivors.

Clinical findings

Horse

- Stiff gait and apathy to feed is the initial sign of the disease.
- Prolapse of the third eye lid.
- Erection of the ears. Immobility of the ears.
- With the advancement of the disease horse turn progressively more and more stiff, stands with its feet widely apart and its head and neck in an extension position.
- Rigidity of the facial muscles gives an anxious expression.
- Spasms of the masseter muscle leads to trisumus (Lock Jaw).
- Mouth is held tightly closed and separation of jaw is hardly possible.
- Restriction of mastication and dribbling of saliva from the mouth.
- Animal remains hypersensitive.
- Reflex irritability is noted right from the start of symptom.
- Light noise, air current, any disturbance aggravate the spasms of the muscles.

- Sometimes, this way lead to violent trembling resulting profuse sweating all over the body.
- In acute case, temperature and pulse rate may be elevated but sub-acute case will show normal to level.
- Appetite may persist to some extent but chewing and swallowing become difficult rather impossible.
- Finally, the horse is unable to stand and dies due to asphyxia within 3-10 days.

Cattle and buffalo

- Clinical manifestations in cattle are more or less similar to horse.
- Mild cases may recover. The initial signs consist of restricted movement, muscular stiffness and difficulty in walk.
- Fall in milk yield, lock jaw and hypersensitivity on little stimuli.
- Prolapse of the third eyelid, pump handle position of the tail are common feature.
- Lateral recumbency supervenes along with extensor rigidity and opisthotonus.
- Suppression of rumination and bloat are the important attributes in cattle.
- Rumen may appear like drum.
- Cow dies due to asphyxia.

Diagnosis

- Clinical signs are so suggestive that it is hardly difficult to arrive at a diagnosis.
- *C. tetani* can be recognized in stained smears of exudates.
- In the laboratories, the organisms can be cultivated by anaerobic method.
- Biological methods may be employed using rabbit or guinea pig.
- Bio-chemical changes that do occur is elevation of CPK values in acute cases of tetanus.

Differential diagnosis

- The disease may be confused with the followings
- *Strychnine poisoning:* History of access to drug or malacious act; duration of

illness; absence of wound; chemical analysis of bowel content will help in confirmatory diagnosis.

- *Grass tetany:* History of grazing pasture; characteristic signs of tetany; low magnesium level; absence of wound; response to magnesium therapy.
- *Milk fever:* History of parturition; sternal or lateral recumbency; low calcium level; absence of wound; response to calcium therapy.
- *Rabies:* History of dog bite; change of behaviour; salivation; biting tendency; absence of wound; ascending paralysis and death.

Treatment

- The principle treatment lies with (a) destruction of tetanus organisms (b) neutralization of circulating toxin and (c) relaxation of muscles to prevent asphyxation.
- Penicillin is the drug of choice and it should be given in massive doses. Penicillin may be infiltrated locally to minimize further multiplication of the bacteria. 1% hydrogen peroxide may be used to clean the wound.
- To neutralize toxin, antitoxin has to be injected. 3,000 to 7,000 i. u. of antitoxin may be used depending on the size of the animal at 12 hours interval. Antitoxin can also be given in the wound locally.
- Muscle relaxants like chlorpromazine @ 500 mg for an adult horse or 0.2 mg per kg body wt may be given to other animals.
- Sterile 10% solution of magnesium sulphate (30-60 ml) may be given through s/c route.
- Intra-gastric or parenteral administration of nutrients and fluids should be given as supportive treatment.

PNEUMONIA

Pneumonia is the inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleurisy. It is manifested clinically by an increase in respiratory rate, cough, abnormal breath sound on auscultation and in most bacterial pneumonia, by evidence of toxemia.

Causes

Cattle

1. Pneumonic pasteurellosis (Shipping fever). Pasteurella hemolytica, P.multocida with or without parainfluenza –3 virus (PI3).
2. Enzootic pneumonia of calve: PI3, adenoviruse 1,2, and 3 Rhinovirus, Bovine Syncytial Virsus (BSV), reoviruses, Bovine herpes virus l (BSV virus) plus chlamydia spp. Mycoplasma spp. Pasteurella spp. Corynebacterium pyogenes, Strept spp. and bedsomia spp. (Actinobacillus actinoides).
3. Viral Pneumonia of yearling and adult cattle is caused by either PI3 or adenoviruses.
4. Contagious bovine pleuropneumonia caused by Mycoplasma mycoides.
5. Atypical interstitial pneumonia.
6. Massive infestation with ascarid larvae.
7. Lungworm pneumonia (Dictyocaulus viviparous)
8. Klebsiella pneumonia infection in calves and nursing cows suffering from mastitis caused by this organism.
9. Sporadically in T.B. (Mycobacterium bovis).
10. Sporadically in calf Diphtheria (Spherous necropherous).
11. Hemophilus somnus, possibly in young cattle affected with the more common septicemic form of the disease. Its role as primary cause is uncertain.

Horses

1. **Newborn foals:** Any of the septicemia which occur at those time, Strep. Sp., E. Coli, Actinobacillus equi.
2. In immunodeficient foals, pneumonia is caused by adenoviruses or pneumocystis carini.
3. **Older foals:** Corynebacterium, Rhodococcus equi and equine herpesvirus.
4. Dictycaulus arnifeldi and Parascarsis equorum rarely cause significant pneumonia.
5. As a sequel to Strangles.
6. Rarely as sequele to equine viral arteritis or equine viral rhinopnumonitis in adult animals.
7. Glanders and epizootic lymphangitis (Histomonus farcinicus) usually include

pneumonic lesions.

Sheep

1. Pneumonic pasteurellosis (Pasteurella sp.) as acute primary pneumonia in feedlot lambs, or secondary to PI3 or Chlamedia spp.
2. Newborn lambs: Uncommonly Strept. Zooepidemicus, Salmonella abortus ovis.
3. Mycoplasma spp. (Severe pneumonia).
4. Symptomless pneumonia without secondary infection adenovirus, RSV, reovirus Mycoplasma sp.
5. Corynebacterium pseudotuberculosis (sporadic).
6. Melioidosis (*Pseudomonus pseudomallei*).
7. Lungworm pneumonia (*Dictycolus filarial*).
8. Progressive interstitial pneumonia

Goats

1. Pleuropneumonia caused by mycoplasma strain F38 or *M.Capri* is a devastating disease.
2. Chronic interstitial pneumonia with pulmonale as common sequel by a number of mycoplasma spp. *M. mycoides var. mycoids* appears to be the most commonly recorded.
3. Rotavirus infection.

All species

1. Toxoplasmosis (sporadic cases).
2. Systemic mycosis lesion are focal only.
3. Aspiration pneumonia.
4. Secondary pneumonia caused by Strept. Spp. Corynbact. spp. dermatophilus sp.
5. Interstitial pneumonia, pulmonary consolidation and fibrosis by toxins in plants (*Eupatorium glandulosum* in horses), *Zieria arborescens* (stinkwood) in cattle, *Astragalus sp.* in all species.

Clinical findings:

1. Rapid, shallow respiration, is the cardinal signs of early pneumonia, dyspnea occurring in the later stages.
2. Polypnoea may be quite maker with only minor pneumonic lesions and the rapidity of the respiration is an inaccurate guide to the degree of pulmonary involvement.
3. Cough is other important sign (the type of the cough varies with the type of the lesion). Bronchopneumonia is usually accompanied by a moist painful cough, interstitial pneumonia by frequent, dry, backing cough, often in paroxysms.
4. Cyanosis is not a common signs and occurs only when large of the lung are affected.
5. A nasal discharge may or may not be present in the bronchioles whether or not there is accompanying inflammation of the upper respiratory tract.
6. The odour of the breath may be informative. It may have an odour of the decay when there is a large accumulation of inspissated pus present in the air passages, or putrid, especially in horses, when pulmonary gangrene is present.
7. Auscultation of the thorax before and after coughing may detect exudate in the air passages.
8. By auscultation in the early congestive stage of bronchopneumonia and interstitial pneumonia the vascular murmur is increased. Moist rales develop in broncho-pneumonia as bronchiolar exudation increases but in uncomplicated interstitial pneumonia, clear, harsh bronchial tones are audible. When complete consolidation occurs in either forms, bronchial tones are the only sounds audible over the affected lung but moist or crepitant rales can be heard at the periphery of the affected area in bronchopneumonia.
9. Consolidation also causes increased audibility of the heart sounds. When pleurisy is also present it causes a pleuritic friction rub in the early stages and muffling of the pulmonary sounds in the late exudative stages. Consolidation can be detected also by percussion of the thorax or by tracheal percussion.
10. There may be an observable difference in the amount of movement in the two side of the chest if the degree of consolidation is much greater in one lung.

Additional signs evident in pneumonia include fever of variable severity, anorexia, depression, an increase in pulse rate.

a. Swab or tracheal sputum and determination of the sensitivity of the isolated bacteria to antibacterial agents.
b. Transtracheal aspiration has been described earlier and is valuable tool for an intensive investigation of a respiratory tract infection.
c. Radiographic examinations are undertaken only in animals of suitable size.
d. Hematological examination usually reveals a leukocytosis with shift to the lift in bacterial pneumonia.
e. A leukopenia and lymphopenia occurs in some cases of acute viral pneumonia. In viral pneumonia, the serological testing of acute and convalescent sera, in addition to the isolation of the virus, are useful tools supports in evidence of the presence of an active infection.

Diagnosis

There are two kinds of errors in the clinical diagnosis of pneumonia.

- One of that is the pneumonia is not detected clinically, because the abnormal lung sound are apparently not obvious.
- The other is to make a diagnosis of pneumonia, because of the presence of dyspnoea which was due to disease in some other body systems.
- The major clinical findings of pneumonia are polypnoea in the early stage and dyspnoea later, abnormal lung sounds, and fever and toxemia in bacterial pneumonia. Polypnoea and dyspnoea may result from involvement of other body systems as congestive heart failure, terminal stage of anemia, poisoning by histotoxic agents such as hydrocyanic acid, hyperthermia and acidosis are accompanied by respiratory embarrassment, but not by the abnormal sounds typical of pulmonary involvement. Pulmonary edema and congestion, embolism of the pulmonary artery and emphysema are often mistaken for pneumonia, but can be usually differentiated by the absence of fever and toxemia, on the basis of the history and on auscultation findings.
- All of the practical laboratory aids described should be used when necessary.

They are of particular importance when outbreaks of pneumonia are encountered. In a single routine case of pneumonia, the cause is usually not determined.

- However the age and class of the animal, the history and epidemiological findings and the clinical findings can usually be correlated and a presumptive etiological diagnosis done.

Differential diagnosis

- Differential diagnosis must be done from pleurisy, that characterized by shallow, abdominal type of respiration, pleuritic friction sounds when effusion is minimal and a muffling of lung sound and a fluid line detectable by auscultation and percussion when fluid is plentiful.
- Differential diagnosis must be done from pneumothorax, where the later have inspiratory dyspnea and on the affected side the abnormalities include; an absence of the vesicular sounds, but bronchial tones are still audible over the base of the lungs, an increase in absolute intensity of the heart sound and increased resonance on percussion. Finally differential diagnosis must be done from diseases of the upper respiratory tract such as laryngitis, and tracheitis which are accompanied by varying degrees of inspiratory dyspnea which is often loud enough to be audible without stethoscope.
- In less severe cases, auscultation of the mid- cervical trachea will reveal moist wheezing sounds on inspiration. These sounds are transmitted down into the lungs and are audible on thorax auscultation.
- In some cases of severe laryngitis and tracheitis, the respiratory sound are audible over the trachea and over the lungs are markedly reduced because of almost total obliteration of these organs.
- In laryngitis and tarcheitis there is usually a more frequent cough than in pneumonia an the cough can be readily stimulated by squeezing of the larynx or trachea.
- In pnumonia the abnormal lung sounds are audible on both inspiration and expiration. Examination of the larynx through oral cavity in cattle and with the aid of rhinolaryngeoscope in the horse will usually reveal the lesions.

Treatment

1. In specific infection isolation of affected animals and careful surveillance of the remainder of the group to detect cases on the early stages should accompany the administration of the specific antibacterial drugs or biological preparations to affected animals. The choice of antibacterial agents will depend on the tentative diagnosis, the experience with drugs in previous cases and the results of the drug sensitivity tests.
2. The common bacterial pneumonia of all species will usually recover quickly (24 hr.) if treated with an adequate dose of the drug of choice early in the course of the disease.
3. Animals with severe pneumonia will require daily treatment for the several days until recovery occurs.
4. Those with bacterial pneumonia and toxemia must be treated early on an individual basis. Each case should be identified and carefully monitored for failure to recover.
5. Antimicrobial agents in a long acting base may be used to provide therapy over a 4-6 day period instead of the daily administration of the shorter-acting preparations. However, the blood level from the long-acting preparations are not as high as the shorter-acting preparations and may not be as effective in severely affected animals.
6. The common causes for failure to respond favourly to treatment for bacterial pneumonia include: (A) Advanced disease when treatment was undertaken, (B) The development of pleurisy and pulmonary abscesses, (C) Drug resistant bacteria.
7. There is no specific treatment for the viral pneumonia because viral and mycoplasmal pneumonia are commonly complicated by secondary bacterial infections. It is common practice to treat acute viral and mycoplasmal pneumonia with antibacterial until recovery is apparent.
8. In outbreaks of pneumonia where many animals are affected and new cases occur each day for several days, the use of mass medication of the feed and / or water supplies should be considered.

9. Mass medication may assist in the early treatment of subclinical pneumonia and is a labor-saving method of providing convalescent therapy to animals which have been treated individual.
10. When outbreaks of pneumonia occur and new cases are being recognized at the rate of 5-10 % per day of the total in the group, all the remaining in contact animals may help to treat subclinical cases before they become clinical and thus "abort" the outbreak.
11. Corticosteroids have been used for their anti-inflammatory effect in the treatment of acute pneumonia. However, there is no clinical evidence that they are beneficial. Affected animals should be housed in warm, well ventilated, draft-free accommodation, provided with ample, fresh water, and light nourishing food.
12. During convalescence condition, the return to work or exposure to bad or cold weather should be avoided. If the animal does not eat, oral or parenteral force-feeding should be initiated. If fluids are given intravenously care should be exercised in the speed with which they are administered. Injection at too rapid rate may cause overload on the heart ventricle and death may occur due to acute heart failure.
13. Supportive treatment may include the provision of O2 supply to be available especially in the critical stages when hypoxia is evident. In foals, the oxygen can be administered through an intranasal tube passed back to the nasopharynx and delivered at the rate of about 3 liters / min. for several hours. Expectorants may be of value in chronic cases and during convalescence.

Enterotoxaemia

Enterotoxaemia is an infectious disease of ruminants that result due to absorption of certain bacterial toxin which is being formed in the intestine.

Etiology

Enterotoxaemia is caused by *Clostridium perfringens*that produce toxions starting from A to F of which type A, B, C, D, E are important.

Clinical signs

- Dysentery
- Abdominal pain
- Spasms and aimless wandering
- Weakness
- Depression
- Haemorrhagic diarrhea

Treatment

Oral antibiotics (tetracycline, chlortetracycline, penicillin) may be used.

Unit: Four
Viral diseases of livestock

Learning Outcomes

After the completion of this unit, student will be able to

- Explain the general concept of viral diseases.
- Identify the virus, pathogenesis, pathology and prevention from diseases.

Rabies

Rabies is a fatal encephalitis of all warm-blooded mammals caused by a lyssavirus and manifested mainly in either a furious or dumb (paralytic) form. The infection usually originates in a bite wound and ascends a nerve trunk to the cord and brain. The incubation period is variable and, on occasions, has been longer than six months.

Etiology- A labile, single-stranded RNA virus in the Rhabdoviridae family.

Distribution- The disease occurs worldwide except for Australia (a rabies variant virus recovered from bats), New Zealand, the British Isles, Hawaii, the Scandinavian countries, Cyprus and Japan. All domestic animals are susceptible. It is frequently endemic in wild animals including the skunk, fox, raccoon, wolf, bobcat and coyote. There are periodic epidemics among wild animals. Asymptomatic salivary gland infections occur in vampire bats resulting in prolonged viremia. Insectivorous bats may also be infected. In recent years in the US there have been more feline cases than canine, probably because cats are less frequently vaccinated.

Host- Domestic animals including the dog and cat are moderately susceptible, as are humans, racoons, bats and skunks.

Mode of Infection and Transmission- The virus is shed in the saliva from infected salivary glands. The disease almost always results from the bites or scratches of infected or rabid animals. Several cases in humans have resulted from aerosol exposure. There have been two known recoveries in humans. Although susceptible to common disinfectants and ultraviolet light, the virus retains its viability in tissues for several weeks at room or refrigerator temperatures.

Clinical Signs- The incubation period is usually 2 - 8 weeks but can be several months. The course is 3 - 10 days. The disease is seen in the forms described below; however, these forms are not always clearly demarcated.

- ***Prodromal form-*** Animals show apprehension, anxiety and changes in temperament and behavior. Severe pruritis may develop at the site of exposure. This stage lasts from 1 - 3 days. Paralysis ensues rapidly and death occurs within 10 days after the first signs are seen.
- ***Furious form-*** Aimless wandering; bumps into objects; excitement; irritability; bites or attempts to bite animals, people and inanimate objects (mad-dog syndrome); depraved appetite; voice altered; muscle paralysis, salivation, convulsions, ataxia, paralysis and death.
- ***Paralytic form-*** This form is most common. The animal is lethargic and hides; doesn't usually bite; muscular tremors; perceived difficulty in swallowing; terminal paralysis.
- ***Inapparent form-*** This form has been observed in dogs, cats, skunks and bats. These animals may seroconvert, survive and serve as a source of the virus for extended periods. Bats may be asymptomatic or have protracted clinical signs with transmission of the virus for months.

Diagnosis

The different forms of the disease make a clinical diagnosis difficult. History of exposure to potentially infected wild animals should be considered. Canine distemper, pseudorabies, canine hepatitis, feline infectious peritonitis, listeriosis, cryptococcosis, toxoplasmosis, other infections of the CNS and poisonings due to lead, strychnine and various pesticides which produce neurologic signs should be considered.

Rabies can only be definitively diagnosed by laboratory means.

After human exposure to a dog or cat suspected of having rabies, the dog or cat may be killed and submitted to the laboratory immediately, or be confined for 10 days. If suspicious signs develop, the animal should be killed and submitted to the laboratory for tests.

The fluorescent antibody (FA) procedure is widely used and is the preferred method.

Treatment - None.

Control - A variety of live attenuated and inactivated vaccines are available.

Avian flury strain: A modified live virus (MLV) vaccine. The high egg passage can be used in dogs, cats and cattle. The low egg passage can only be used in dogs.

Cell culture MLV vaccines- Causes fewer allergic reactions than the avian strain. They are given intramuscularly. There have been postvaccinal reactions in cats (see discussion of Vaccinosarcoma).

Cell culture produced, inactivated vaccines have largely supplanted the MLV vaccines because of safety considerations. Only inactivated vaccines are used in the US.

Vaccination Schedule- Maternal antibodies from vaccinated females will protect most neonates until three months of age. There are several types of vaccines but the first injection should be given at three months (dogs and cats) and the second injection at one year of age. Animals should be revaccinated every 2 - 3 years.

Human Exposure- Decisions frequently have to be made by veterinarians regarding human exposure. It is important to remember that the virus can be in the saliva of infected animals from 1 to 13 days before clinical signs occur. The location and severity of the bite are quite important. Persons bitten in the head region by a stray animal should begin treatment immediately if the animal cannot be found. When the animal is available and has not been vaccinated, it should be sacrificed and the brain examined by the FA test. Attempts, still largely in the experimental stage, are being made to immunize certain wild animals in endemic areas with vaccine-laced baits in order to prevent rabies and thus halt its spread.

Public Health Signicance - All individuals at high risk of exposure, e.g., veterinary practitioners, staff of veterinary clinics, diagnostic laboratory workers, animal control personnel, park rangers, etc..., should be regularly vaccinated. Vaccination may be advisable for those travelling to countries where rabies is endemic. In the

event that a vaccinated individual is exposed they are given the same post exposure regimen that an unvaccinated person would receive. It is most important that all bite or scratch wounds be thoroughly cleaned and washed with plenty of water and soap or detergent. The risk of rabies transmission to humans needs to be carefully evaluated. To be considered are the prevalence of the disease in the area, the nature and extent of the exposure and the behavioral status of the suspected animal. Dogs, cats, wild carnivores and bats are the most frequent sources of human infections. If the animal responsible for the possible exposure, regardless of its behavior, can be sacrificed it can be subjected to immediate diagnostic testing. As was mentioned above, suspected animals should be confined for ten days. If suspicious signs develop, the animal should be killed and submitted to the laboratory for tests. Regimens employing inactivated human or animal cell culture vaccines are use for prevention in high-risk personnel and those exposed. Rabies immune globulin, or antirabies serum (equine) are widely used for prophylaxis in exposed humans.

Foot and Mouth Disease

Foot and mouth disease is a very fatal viral diseases. The cloven footed animals get infected by this disease. This disease spreads through the food, used water, saliva, contiguity and using other equipment of affected animal. The disease may spread through air too. High productive cows get infected by this disease very much.

Causes

Foot and mouth disease is a very fatal contagious viral disease. This disease affect cow, buffalo, goat, sheep etc. But cow get infected much. A virus named picorna virus causes this disease.

Symptoms

- In the initial state the body temperature of the cow become about 104-106 Fahrenheit.
- Blister can be seen inside the mouth, tongue and middle of the hoofs.
- This blister get fractured and turned to sore.
- Saliva exude from nose and mouth continuously and for this thy can consume grass or other food properly.
- Due to sore in the hoof the cow can’t walk well.

- Constipation can be seen in sick animal.
- Infected cattle lick the sore of their hoof with their tongue.
- Sometimes they raise their legs high.
- The cow loses their appetite.
- The hoof may drop off day by day.
- Abortion may happen to the pregnant cow.
- The cow infected by foot and mouth disease become very sick.

Prevention Method

- If foot and mouth disease affect any cow, separate it from the healthy one.
- Keep the infected animal to a clean and dry place.
- Wash the wounded place with 3% iosan solution 3 times a day. Do this for 3-5 days.
- After washing with iosan, apply antibiotic powder immediately in the wounded place.
- Feed the animal soft feed.
- Apply medicine in the mouth and hoof sore of infected animal.
- Vaccinate the cattle timely to defense foot and mouth disease.
- Timely applying disease preventive vaccine is the only method to prevent this disease.

Treatment

Apply foot and mouth disease preventive vaccine at the age of 2-3 month for the first time. After one month apply second vaccine. The third vaccine should apply after six-month latter of first vaccination. Do this after every six months. Apply the vaccine through injection under the skin of dewlap.

Peste Des Petits Ruminants (PPR)

PPR is an acute highly contagious viral disease of goats, less commonly sheep and closely related wild bovidae, and is clinically mimics cattle plague, characterized by fever, errosive stomatitis, enteritis, pneumonia, and death.

Etiology

- PPR virus is a Morbillivirus of the Family Paramyxoviridae.

Hosts

The natural hosts are goats, sheep, and closely related wild bovidae, sheep.

Goats are clearly more susceptible than sheep and the disease often occurs in goats without affecting sheep in close proximity. The highest incidences are found in young stock less than two years old.

Cattle and pigs also infected.

Transmission

- The virus is present in all body excretions and secretions such as tears, nasal discharge, sputum, and diarrheic feces.
- PPR virus spreads by direct contact or close indirect contact and infection is mainly by inhalation of infective aerosols but could also occur through the conjunctiva and oral mucosa.

Clinical Sign

- Firstly, body temperature of goat increases and may suffers by fever.
- Saliva flows from their nose and eyes.
- Diarrhea pneumonia can be seen.
- Nose and eyes get closed always**.**
- Sore can be seen in the mouth of goat.
- Mouth of infected goat spreads bad smell.
- PPR disease infected goat suffers by dyspnoea.
- The goat stops eating food.
- They do liquid defecate containing blood.
- Infected goat loses its disease preventive power
- PPR has acute and subacute forms.

The acute form

- The acute form occurs frequently in goats with a clinical course mimics that of cattle plague but crusty scabs and pneumonia development is more prominent in PPR.
- After an IP 2-6 days the first clinical sign is short fever 40-41°C accompanied by dullness, serous oculonasal discharge that rapidly becomes profuse and

purulent. The nasal discharge may block the nares and encrust the muzzle, causing the animal to snort and sneeze, whereas the ocular discharge may mat the eye lids together.

- Congestion and necrosis affects the gums, the lower lip, and may extend over the entire oral mucosa. The tongue becomes coated with fetid diphtheric plaques, the lips swollen, and the animal unable to eat.
- Profuse diarrhea begins 2-4 days after the onset of fever and feces may be mucoid and blood tinged.
- Pulmonary involvement usually occurs during the later stage of the disease and the death usually occurs within a week of the onset of illness. The mortality rate in goats is generally high and ranges from 77-90%, but goats of the endemic Sahel area have a lower rate.
- In the absence of complications recovery may occur within 8-10 days from illness.

Subacute form

- Subacute reactions are more common in sheep but they also occur in goats and are manifested by low-grade signs and lesions.
- Most affected animals recover wthin 2 weeks and a few day. Sheep fatality rate is less than 10%.

Diagnosis

A presumptive diagnosis of the acute forms of PPR can be made from the epidemiology, clinical signs, and lesions but subacute form may require laboratory diagnosis.

Samples and specimens

For virus isolation or viral antigen detection, from live animals during early stages of fever until the beginning of the erosions, ocular or nasal swabs, scrapings of early oral lesions, blood with heparin or EDTA "buffy coat". From deceased animals portions of the spleen, lymph nodes, and lungs collected within 2 hours of death. The specimens should be chilled on ice, but not frozen, and examined as soon as possible.

For serology collect serum from acutely ill and recovered animals.

For histopathology slices of lymph node, spleen, tonsils, and mucosal lesions are collected in 10% formalin saline for.

Differential diagnosis

PPR should be differentiated clinically from RP, bluetongue, contagious caprine pleuropneumonia "CCPP", contagious ecthyma, foot and mouth disease, sheep and goat pox, bacterial and parasitic diarrhea, and viral and parasitic pneumonia.

Treatment

There is no specific treatment and the disease is notifiable. In valuable individuals, good nursing, fluid replacment to compensate electrolyte losses from diarrhea, and antibiotics to suppress bacterial infections that complicate viral pneumonia should be considered.

Prevention and control measures

- Kill the seriously infected animal as soon as possible, so that the germ can't spread everywhere.
- Vaccinate the healthy animal timely to prevent this disease.
- Keep the house clean and germ free always.
- Separate the infected goat form healthy one and keep them in dry place.
- Keep communication with the nearest veterinarian always.
- The used equipments of goat should keep under soil or burn it with fire.
- Don't transport or sell the infected goat.

Canine Distemper

Canine distemper is a highly contagious, systemic, viral disease of dogs seen worldwide. Clinically, it is characterized by a diphasic fever, leukopenia, GI and respiratory catarrh, and frequently pneumonic and neurologic complications.

Etiology and Pathogenesis

Canine distemper is caused by paramyxovirus closely related to the viruses of measles and rinderpest.

Clinical Findings

- Diphasic fever
- Oculonasal discharge
- Rhinitis
- Conjunctivitis
- Gastroenteritis
- Loss of appetite
- Vomition
- Abdominal pain
- Restlessness
- Excitement
- Convulsion
- Thickened foot pads (Hard pad)

Diagnosis

- On the basis of clinical signs
- Pathological lesions
- Detection of inclusion bodies
- Serological tests

Treatment

Treatments are symptomatic and supportive, aimed at limiting secondary bacterial invasion, supporting fluid balance, and controlling neurologic manifestations. Broad-spectrum antibiotics, balanced electrolyte solutions, parenteral nutrition, antipyretics, analgesics, and anticonvulsants are used, and good nursing care is essential. No single treatment is specific or uniformly successful.

Prevention

- Vaccination

Swine Fever

Classical swine fever (CSF) is a highly contagious and economically significant viral disease of pigs. The severity of the illness varies with the strain of the virus, the age of the pig, and the immune status of the herd.

Etiology

Pesti virus

Transmission

Pigs are mainly thought to become infected by the oral or oronasal routes. CSFV may also enter the body via other mucus membranes (including genital transmission in semen), and skin abrasions. It can be shed in oronasal and ocular secretions, urine, feces and semen; one study reported that pigs infected with strains of low virulence excreted the virus mainly in oronasal secretions

Clinical Signs

The clinical signs vary with the strain of CSFV, and the age and susceptibility of the pigs. While highly virulent strains were prevalent in the past, most outbreaks are now caused by moderately virulent strains, and the clinical signs are often less severe and distinctive. Highly virulent strains of CSFV tend to cause acute, severe illness in naive herds. Common clinical signs in the acute form include a high fever, huddling, weakness, drowsiness, anorexia and conjunctivitis, which can cause severe crusting of the eyelids. Constipation, with the passage of hard fecal pellets, is typically followed by, or intermittent with, watery diarrhea. Pigs may be incoordinated or exhibit an unsteady, weaving or staggering

gait, which often progresses to posterior paresis. Some pigs may vomit yellow, bile-containing fluid, or develop respiratory signs. The skin can become hyperemic, and may develop hemorrhages (especially on the abdomen, inner thighs, ears) or a purple cyanotic discoloration, which tends to be seen on the snout, ears and tail. Severe leukopenia is a common laboratory abnormality. Pigs with acute classical swine fever often die within 1-3 weeks, and convulsions may occur in the terminal stages. The subacute form is similar; however, the signs are less severe, the course is prolonged, and the mortality rate lower.

Treatment

There is no treatment for classical swine fever, other than supportive care. Control Disease reporting a quick response is vital for containing outbreaks in CSFV-free regions. Veterinarians who encounter or suspect classical swine fever should follow their national and/or local guidelines for disease reporting. In the U.S., state or

federal veterinary authorities should be informed immediately.

Prevention In countries where classical swine fever is endemic, this disease may be excluded from a herd by buying animals from CSFV-free herds, quarantining the new stock for 4 months and testing the animals before allowing them to contact the rest of the herd. Vaccines can be used to protect animals from clinical signs, and may also be employed to reduce the prevalence of infections during an eradication program. Both modified live and subunit (marker) vaccines are manufactured.

Unit: Five

Bacterial diseases of poultry

Learning Outcomes

After the completion of this unit, student will be able to

- Identify the different bacterial diseases of poultry.
- Differential diagnosis of bacterial diseases.

Avian Salmonellosis

- Two main important diseases
- Pullorum disease.
- Fowl typhoid.

Pullorum disease

Synonyms

- White diarrhoea.
- Bacillary White Diarrhoea.
- BWD.

Definition

Pullorum disease is an infectious, egg-transmitted disease of poultry, especially chick and turkey poults, often characterized by white diarrhoea and high mortality in young birds and by asymptomatic adult carriers.

Etiology

Salmonella pullorum.

Distribution and Incidence

- Acute systemic infectious disease of chicks.
- Chronic form in adults.
- May remain as carrier.
- World wide.
- Birds of 3 weeks are mostly affected.

Host

Chicken, Duck, goose, Parrot, Cockatoos. Sparrows, Finches, Canaries, Ostrich, Emu, Kiwi, Crane, Rail, Pheasants, Moynah, Peacocks.

Transmission

- Vertically through egg.
- Horizontal
- Contaminated utensils, feed water.
- Dead embryos and chicks.
- Rodents, flies.
- Visitors.

Clinical signs

- Excessive number of dead-in-shell chicks.
- Death shortly after hatching.
- Acutely affected birds
- Depressed.
- Anorexic.
- Respiratory distress.
- White viscous dropping (diarrhoea) adhere to feathers around vent (Bacillary White Diarrhoea).
- Dehydration.
- Finally death.
- Occasional blindness.
- Occasional brown colored droppings

Sub-acute form

- Lameness.
- Swollen hock joint.
- Poor growth rate.

Chronic form

- Inappetance, depression.
- Ruffled feathers.

- Greenish brown diarrhoea.
- Drooping of wings and head.
- Death, if any---severe dehydration.

Diagnosis

- History,Clinical signs,Gross lesions.
- Isolation and identification of organisms.
- Gram negative,Aerobic,Non spore forming rods.

Serology

- TAT (Tube Agglutination Test).
- RST (Rapid Serum Test).
- Stained Ag whole blood test.

Treatment

- Sulphonamides: 0.5% in feed for 5 days.
- Trimethoprim-Sulfadiazine preparations 1-2 gm/L of DW b.i.d. for 5-7 days.
- Trimethoprim-Sulfamethoxazole preparations 1 gm/4 L of DW for 5 – 7 days.
- Furazolidone preparations 250 – 500 gm / ton of feed.

Antibiotics

1. Chloramphenicol 0.5% in feed for 10 days.
2. Chlortetracycline 200 mg / kg in ration for 10 days.
3. *CS test.

Fowl Typhoid

Definition

Fowl typhoid is an infectious disease, primarily of chickens and turkeys, with many of the clinical and epizootiologic features and lesions that occur with pullorum disease.

Etiology

Salmonella gallinerum.

Distribution and Incidence

Worldwide.

Birds > 3 months – most affected.

Host: Same as PD.

Transmission

- Vertical.
- Horizontal.
- Recovered bird as carriers for long periods.
- Feces- at least one month.
- Clinical signs

Acute form

- Infected eggs→Hatched →Die shortly after hatch.
- Before death →Appear depressed and weak with white chalky material adhere to vent.
- Mortality → 93%.
- If birds don't die within 2-3 days: Progressive loss of condition and anaemia → Shrunken and pale comb and wattles.
- Watery to mucoid yellowish diarrhoea.
- Decreased egg production in case of layers

Sub-acute form

- Increased dead-in-shell embryos.
- Small, weak, moribund / dead chicks on hatching trays.

Chronic form

- Dyspnea.
- Depression.
- Ruffled feathers.
- Closed eyes → Blindness.

Diagnosis and treatment and control. Same as PD

Chronic respiratory disease (Mycoplasm gallisepticum)

This is a chronic slow spreading contagious disease in birds characterized by obstinate hacking cough, sneezing and tracheal rales. Large numbers of birds in a

flock used to suffer causing heavy economic loss especially in broilers.

Etiology: Mycoplasma gallisepticum

Clinical findings

- Tracheal rales
- Sneezing
- Respiratory distress
- Open beak breathing
- coughing and nasal discharge
- Young chicks show signs of conjunctivitis with lacrimation
- 40% reduction in egg production
- Hatchability may be affected.
- Sign of ataxia and lameness accompanied with enlargement of hock joint may appear in some birds.

Diagnosis

- History of slow spread of the disease.
- Clinical signs manifested as reduced food intake, hacking type of cough, reduced egg production.
- Serological test: rapid serum agglutination test, whole blood agglutination test
- ELISA (enzyme linked immunosorbent serologic assy test)

PM lesion

Treatments

1. Tylosin tartrate@1 gm/ lit of water for 24-72 hrs.
2. Timulin@ 0.025% in water for 3 consecutive/Regular days
3. Lincomycin powder@1 gm/litre of drinking water

Unit: Six
Viral Diseases of Poultry

Learning Outcomes

After the completion of this unit, student will be able to

- Identify the different viral diseases of poultry.
- Differential diagnosis of viral diseases of poultry.

Avian Influenza

- Definition
- Avian influenza is an infectious disease caused by A type influenza virus of the family Orthomyxoviridae.

Occurrence

Most species of birds.

A few outbreaks in chickens.

Most outbreaks in duck and turkeys.

All age group of birds, if previously unexposed, are presumed susceptible.

Zoonotic importance.

Etiology

- Influenza virus in the group of orthomyxovirus.
- There are several serotypes.

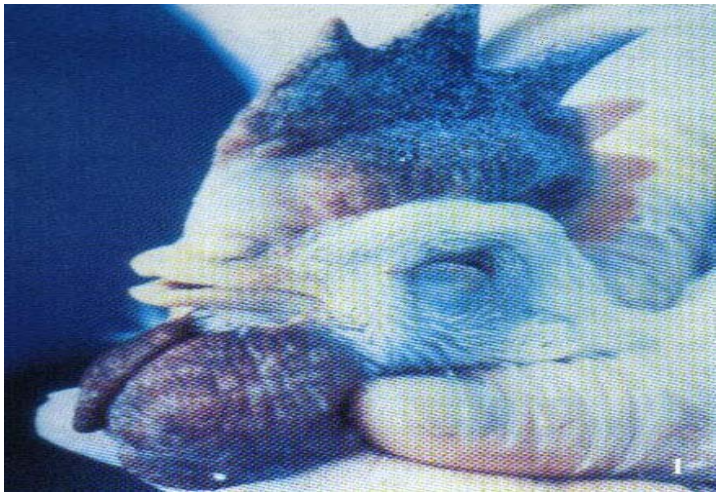
Transmission

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

Clinical signs

- Symptoms are diverse according to species, age, sex, virus strain, mixed infections, and environmental factors.
    - Loss of activity.
    - Loss of appetite for food and water.

- Egg production drops.
- Weakness.
- Coughing, sneezing, rales.
- Lacrymation
- Ruffling of feathers.
- Edema and cianosis in the head, comb, and wattle.
- Nervous symptoms and diarrhoea.



Gross lesions

- Air sac hypertrophy.
- Catarrhal or fibrinous peritonotis or enteritis.
- In layers, exudate may be observed in the oviduct.

Diagnosis

- As influenza of fowl has varied symptoms, it cannot be diagnosed by clinical signs, except in the epidemic period.
- Isolation and identification of the virus gives definitive diagnosis.
- Serological tests
  - Agar gel precipitation test (AGPT).
  - HI test and ELISA are performed to determine the subtypes.

Avian influenza should be differentiated from ND, fowl pox.

Prevention/treatment/measures

- There is no treatment for fowl influenza.
- Although H5 and H7 virus inactivated vaccines are used for prevention in U.S.A. and Australia, it is not practicable in our country.
- It is important to prevent contact with wild birds, turkeys and ducks.
- Apply all-in, all-out systems.
- Disinfection and hygienic control of humans, foodstuff, vehicles, and other equipment should be effectively managed.

Infectious Bursal Disease

Definition

- IBD is an acute, contagious viral disease of young chickens characterized by diarrhoea, vent picking, trembling, incoordination, inflamation followed by atrophy of the bursa of Fabricius and by a variable degree of immunosuppression.

Synonyms

IBD.

Gumboro Disease.

Etiology

- Family: Birnaviridae.
- Genus: Birnavirus.
- 2 serotypes-
  - Serotype 1: Pathogenic.
  - Serotype 2 : Non- pathogenic.

Host

- Chickens
- 3 to 6 weeks old.
- Ducks, geese, swan.
- Pheasant, grouse.
- Peacocks.

Transmission

- Excreted through faeces.
- Vectors / Reservoirs
  - Meal worms.
  - Litter mites.
  - Wild birds.
  - Vermin.
  - Rats.
  - Visitors.

Route

- Oral.

Clinical signs

Depends on

- Age.
- Breed.
- Maternal antibody level.

Morbidity

- High- upto 100%.

Mortality

- Usually 20-30%.
- Occasionally 50%.
- In case of vvIBDV- 90-100%.

Acute form

- Depression.
- White watery diarrhoea.
- Soiled vents.
- Anorexia.
- Ruffled feathers.
- A reluctance to move.

- Closed eyes.
- Finally death.

Mild form

- Little / no signs.
- Suboptimal growth

Gross lesions (BF)

From 2-3 days

- Develop transudate.
- White color to cream color.
- Mucosal surface – Hemorrhagic.
- Purulent exudates.
- Necrotic foci.
    - On 3rd day- Increase in size and weight.
    - On 4th day – Double in size and begins to reduce. Transudate starts to disappear.
    - On 5th day – Returns to its normal size and then atrophied.
    - After 5th day – grey in color.
    - On 8th day – One third to its normal size.

Gross lesions

Hemorrhages

- Thigh muscle.
- Pectoral muscle.
- Proventriculus**.**
    - Splenomegali.
    - **Liver –** Necrotic foci, infarcts.
    - **Intestine –** Increased mucus.
    - **Kidney –** Hemorrhagic, urates deposition.

Diagnosis

- Clinical signs.

- Gross lesions.
- Isolation and identification of IBDV.
- Serology
  - ELISA

Treatment and control

- No practical therapeutic measures.
- Management procedures
  - All in all out system.
  - Sanitation program to reduce the level of IBDV.
- Vaccination of parent breeders and/or young chicks.
  - Live and killed vaccines.
- Maternal antibody will normally protect chicks for 1 to 3 weeks but booster vaccination in breeder flocks in which the passive immunity may be extended to 4 to 5 weeks.

Newcastle Disease

Synonyms

- Ranikhet Disease.
- Paramyxovirus 1 infection.
- Avian pneumoencephalitis.

Definition

- It is an infectious several virus disease of birds, caused by Paramyxovirus 1 (PMV-1) and characterized by characteristic respiratory, alimentary and nervous signs.

Occurrence

- Usually in chickens or (less often) in turkeys.
- Most poultry and cage birds are susceptible.
- All age groups are susceptible.
- All poultry raising countries.
- Zoonotic importance.

Etiology

- Newcastle disease is caused by a paramyxovirus.
- The many known strains vary greatly in pathogenecity. They often are classified or referred to as
    - Lentogenic- these are mildly pathogenic (eg. B-1, F, LaSota).
    - Mesogenic- these are moderately pathogenic.
    - Velogenic- these are markedly pathogenic.

Transmission

- Through infected droppings and respiratory discharge between birds.
- Spread between farms is by
    - Infected equipment
    - Trucks
    - Personnel
    - Wild birds or air.
- The incubation period is variable but usually about 3 to 6 days.

Clinical signs

- **The severity of the ND depend on the**
    - infecting virus (amount and pathotypes).
    - The host species and its age and immune status.
    - Coinfection with other organisms.
    - Environmental conditions (stress).
    - Route of exposure and dose.
- **Infection with the highly virulent viruses**
    - It may produce peracute infections of fully susceptible chickens where the first indication of disease is sudden death.
    - Typically, disease signs such as
        - Depression
        - Prostration
        - Diarrhoea
        - Odema of the head and

- Nervous signs may occur with mortality reaching 100%.

- **Infection with moderately virulent virus**
  - Severe respiratory disease, followed by nervous signs.
  - Laboured breathing with sneezing and gurgling, accompanied by the nervous signs like paralysis or twisted necks (torticollis) are the main signs.
  - The appearance of shell-less or soft-shelled eggs, followed by complete cessation of egg laying may be an early signs in adult fowl.
- **Infection with low virulence virus**
  - These viruses cause no disease, or mild respiratory distress for a short time in chickens and turkeys.
- **Internal lesions**
  - Inflamed tracheas.
  - Pneumonia and/or froth in the airsacs are the main lesions.
  - Haemorrhagic lesions are observed in the proventriculus and the intestines.
- **Diagnosis**
  - Although the history of outbreak, clinical signs and pathological changes are suggestive for ND but none of the clinical signs or lesions of Nd may be regarded as pathognomonic.
  - Diagnosis is made by virus isolation from tracheal or cloacal swabs together with blood testing to demonstrate high antibody levels.
  - Infectious bronchitis or infectious laryngotracheitis can give similar clinical signs, but lesions, blood tests and virus isolation tests are decisive.
- **Treatment**
  - There is no satisfactory treatment due to viral etiology.
  - Some poultry raisers use PPM 0.01% in drinking water.
  - Some farmers use sulphadiazin or sulphamethazine 0.5% in feed to check secondary infections.

- **Prevention and control**
  - Chickens and turkeys can be immunized against Newcastle by proper vaccination.
  - Live and/or inactivated (killed) adjuvant vaccines is the only reliable control method.
  - Furthermore, in order to prevent virus spread to other flocks, general hygiene and prevention of invasion of infected wild birds are required.

Marek's Disease

Synonyms

- MD.
- Neurolymphomatosis.

Definition

- Marek's disease is a herpes virus induced neoplastic disease of chickens characterized by infiltration of various nerve trunks and/or organs with pleomorphic lymphoid cells.

Occurrence

- Important only in chickens.
- However, turkeys, quail and other species have limited susceptibility.
- Most commonly in youngs, sexually immature chickens 2 to 7 months old.
- It can occur at virtually any age beyond 3 weeks.
- Worldwide distribution.

Etiology

- Herpes virus.

Transmission

- The MD virus is present in desquamated feather follicle epithelial cells and in oral, nasal and tracheal secretions of infected birds.
- Main transmission is by infected premises, where day-old chicks will become infected by oral and respiratory routes.
- Dander from feather follicles of MD-infected chickens can remain infectious for more than one year.

- Young chicks are particularly susceptible to horizontal transmission.
- Susceptibility decreases rapidly after the first few days of age.

Clinical signs

- Classical MD (typical)
  - The signs depend on the peripheral nerves affected and involvement of the brachial and schiatic nerves is common, leading to progressive spastic paralysis of the wings and legs, respectively.
  - Leg nerve involvement causes a bird to lie on its side with one leg stretched forward and the other backward.
  - When the cervical nerves are involved, there may be torticollis and if the vagus and intercostal nerves are affected, respiratory signs may develop.
  - Classical type MD occurs mainly among chickens of 3-5 months old.
  - Mortality rate is less than 10%.

Acute MD

- Many birds die suddenly without preceding symptoms.
- Some appears depressed before death and some show paralytic signs.
- Mortality reaches 10-30% and occasionally 50%.
- Mostly seen in 2-4 months of age, but is sometimes also seen in chicks less than one-month old.
- In case of broiler it is called visceral type MD.

Skin type MD

- Tumors form mainly in the feather follicles.

Eye type MD

- Contraction of pupils.
- Deformation and decoloration of the iris

Gross lesions

- Tumors formation is observed in the liver, which occasionally can be enlarged even ten times or more.
- Diffuse nodular lesions with a white appearance are observed.

- The spleen is markedly swollen and uniformly white or develops a lot of tumors.
- In the bursa of Fabricius, tumor lesions white in color uniformly form.
- As to skin lesions, various sizes of tumors are formed in the skin.
- Other organs and tissues like proventriculus, ovary, kidney, heart and muscle tumors also form.
- There are swelling, edema and disappearence of the normal striation of peripheral nerve fibres, and the peripheral nerve in a serious case enlarges more than ten times.
- In MD of broilers, however, invisible nerve lesions do occur.

Diagnosis

- A tentative diagnosis can be made on history, clinical signs and pathological changes.
- A definitive diagnosis can be made on isolation and identification of the causative virus.

Treatment and control

- There is no effective treatment.
- Prevention and control of MD is based on
  - Management and hygiene.
  - Rearing of genetically resistant stock.
  - Vaccination.

Infectious Bronchitis

Synonyms

- IB

Definition

- Infectious bronchitis is an acute, highly contagious, virus-caused disease of chickens characterized by respiratory signs (gasping, sneezing, coughing, and nasal discharge), severe renal disease associated with nephrotropic strains, and a marked decrease in egg production.

Occurrence

- Only chickens are susceptible to IB virus.
- All ages are susceptible, assuming they have not had prior exposure to the virus.
- The disease is more severe in baby chicks.
- Worldwide in distribution.

Etiology

- The disease is caused by the several different serotypes of IB virus (Corona-virus).

Transmission

- The nasal discharge and faeces are the main sources of organism and transmission by aerosol (droplet) and contact to the susceptible birds.
- The virus can also be transmitted via the air between chicken houses and even from farm to farm.

Clinical signs

- In young chicks, it causes a cheesy exudate in the bifurcation of the bronchi, thereby causing asphyxia, preceded by severe respiratory distress("pump handle breathing") like
  - Gasping
  - Coughing
  - Tracheal rales and
  - Nasal discharge.
- In older birds IB does not cause mortality.
  - Respiratory signs include
    - Wet rales
    - Gurgling and
    - Wheezing.
  - Egg production will decrease dramatically.
  - Deformed eggs with wrinkled shells will often be laid, and shell irregularities with bumps of calcium deposit.

Internal lesions

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

Diagnosis

- There are three main factors to be considered in order to arrive at a diagnosis.
  - Isolation of the virus in the laboratory.
  - A rising antibody titre when the serum is tested against a known strain of IB virus.

Treatment

- There is no treatment for IB but it is helpful to increase the temperature of the room as well as the brooder in young flocks.
- Secondary bacterial infections may be prevented by or treated with broad-spectrum antibiotics

Prevention and control

- Recovered chickens are immune for at least a year and local immunity in the respiratory tract is important.
- Prevention by vaccination is the best method to control infectious bronchitis

Fowl Pox

Synonyms

- Avian pox.
- Pox.
- Avian diphtheria.

Definition

- Fowl pox is a slow spreading viral disease of chickens, turkeys and other birds characterized by cutaneous lesions on unfeathered skin of the head, neck, legs and feet and / or by diphtheric lesions in the upper digestive and respiratory tract.

Etiology

- Family: Poxviridae.
- Pox virus.
- Distribution
- Chickens.
- More than 200 species of wild birds.
- Parrot, cockatoos, myna, canary.
- Crane, rail, emu, ostrich, raptors.
- Canary pox (Kikuths's disease).

Transmission

- Mechanical
  - Injured / lacerated skin.
- Insects
  - Mechanical vectors.
- Humans
  - Hands
  - Cloths.
- Routes
  - Aerosol.

Clinical signs

- Cutaneous form
- Nodules
  - Comb.
  - Wattles.
  - Eyelids.
  - Non feathered parts.

Diphtheric form

- Cankers / Diphtheric yellowish lesions.
  - Mucous membrane of mouth.
  - Esophagus.

- Trachea.

- Tracheal involvement
  - Mild respiratory signs (coryza like).
- Blindness
  - Cutaneous eye lesions.
- Starvation
  - Most losses.
- Mortality
- 50%.

Gross lesions

- Cutaneous form
  - Local epithelial hyperplasia.
  - Nodules as white-yellow foci.
  - Papules by 5^{th} day.
  - Vescicular stage with formation of extensive thick lesions which may coalesce become scars.

Diphtheric form

- Slightly elevated, white opaque nodules develop on the MM.
- Nodules coalesce becoming a yellow, cheesy necrotic, pseudo-diphtheric / diphtheric membranes.

Diagnosis

- Clinical sins.
- Gross lesions.
- Sample collection.
- Isolation and identification of virus**.**

Treatment and control

- Topical application-
  - Antiseptics.
  - Tinctures.
  - Fat solvents.

Antibiotic therapy (CS test)

- To prevent secondary bacterial infections.

Antifungal therapy

- Nystatin orally.

Vitamin A

- Small birds- water and food.
- 10,000-25,000 iu/im- Psittacine bird weighing 300-500 gm.
- Proper husbandry should be practiced to alleviate environmental stress.
- Immunization
- Fowl pox vaccine.

Unit: Seven

Fungal diseases of livestock and poultry

Learning Outcomes

After the completion of this unit, student will able to

- Identify the different fungal diseases of poultry and livestock.

Mycotoxicosis

Definition

- Mycotoxicosis is a disease caused by a toxic fungal metabolite. Mycotoxicoses may affect both man and animals. Poultry mycotoxicoses are usually caused by fungi that colonize and invade grains and feeds, but other environmental aspects may be involved.

Occurrence

- Grains and forages (feed stuffs).
- Fungi produce metabolites that are toxic to birds.
- Although specific mycotoxins from more frequently in certain geographic locations, interstate and international shipment of grains may result in widespread distribution of a mycotoxin problem.

Transmission

Ingestion or cutaneous exposure

Ergotism

Etiology

- The ergot alkaloids.
- Claviceps spp. that colonize wheat, rye, and triticale are the most common causes of ergotism.
- Clinical signs
- Retarded growth.
- Low egg production.
- Nervous incoordination**.**

Gross lesions

- Abnormal feather development.
- Necrosis
  - Beak.
  - Comb.
  - Toes.
  - Enteritis

Trichothecene mycotoxicosis

Synonyms

- Fusariotoxicosis

Etiology

- Fungus Fusarium sp. produce trichothecene.
- More than 40 trichothecene mycotoxins are known to exist.
- Only two of the most toxic found naturally are
  - T-2 toxin and
  - Diacetoxyscirpenol.

Clinical signs

- Reduced growth.
- Severe depression.
- Bloody diarrhoea.

Gross lesions

- Necrosis of the oral mucosa.
- Reddening of the mucosa of the gastrointestinal tract.
- Mottling of the liver.
- Distention of the gall bladder.
- Atrophy of the spleen.
- Visceral hemorrhages.

Aflatoxicosis

- Etiology

- Aflatoxin
- Group B1, B2, G1, G2.

- Aflatoxin produced by *Aspergillus flavus*.

Clinical signs

- Reduces growth.
- Reduces pigmentation of carcass.
- Reduces egg production.
- Reduces immune function.
- Increases nutrient requirements for protien, trace elements (Selenium), and vitamins.

Gross lesions

- Jaundice.
- Generalized edema and hemorrhages.
- Tan or yellow discoloration of the liver.
- Swelling of the kidneys**.**

N.B. Aflatoxin B1 is a potent, naturally occuring carcinogen and thus has special public health considerations.

Ochratoxicosis

- Etiology
  - Ochratoxin A, B and C.
  - Produced by *Penicillium viridicatum* and *Aspergillus ochraceus.*
  - Ochratoxin A is the most toxic and is the greatest threat to poultry production.

Clinical signs

- Reduction in feed intake.
- Increases mortality.
- Reduces growth.
- Reduces immune function.
- Reduces egg production.

Gross lesions

- Visceral gout.
- Lesions in the liver and kidney.

Treatment

- Remove the toxic feed and replace it wih unadulterated feed.
- Treat concurrent diseases (parasitic, bacterial) identified in the diagnostic evaluation.
- Substandard management practices should be immediately corrected.
- Vitamins, trace minerals (selenium) and protein requirements are increased by some mycotoxins and can be compensated by feed formulation and water-based treatment.

Prevention and control

- Detection and control of mycotoxin contamination.
- Proper storage of fedd ingredients.
- Good processing, shipping and handling procedures of feed.
- Storage of ground feeds more than 14 days should be avoided.
- Store feeds under cool, dry conditions.
- Feeds and grains can now be screened for several mycotoxins (aflatoxin, T-2 toxin, ochratoxin, zearalenone) using monoclonal antibody detection kits.
- Detection of toxin by other techniques.
- Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills and storage bins and these should be controlled.
- Antifungal agents should be added to feeds.
- Toxin binder may be added to the feed.

Dermatophytosis (Ringworm)

Dermatophytosis is a contagious disease caused by fungi of the genera Microsporum and Trichophyton which infect the keratin-containing tissues (skin, nails and hair) of domestic animals, humans and other animals.

Etiology

- The principal dermatophytic fungi affecting dogs and cats are as follows.
- Microsporum canis (Hosts: dog and cat)
- M. gypseum (Source: soil. Host: dog)
- Trichophyton mentagrophytes (Source: rodents. Host: dog)
- Microsporum canis causes more than 95 % of ringworm in the cat and about 70 % in dogs
- Other species of dermatophytes rarely cause ringworm in dogs and cats.

Distribution

- Worldwide. Microsporum gypseum occurs naturally in soil.

Mode of Infection

- Direct and indirect contact. Infected animals and fomites are the usual source of the dermatophytes. Inapparent infections are common in kittens.

Clinical Signs and Diagnosis

The circular or roughly circular lesions of ringworm are variable in size and develop from raised plaques on the skin. The hair becomes thin, broken and finally there is bareness that may become scaly and scabby. Most lesions occur on the head and neck. Bald patches and hair loss are characteristic of feline ringworm. Lesions of M. gypseum in the dog are mainly on the muzzle. Some heavily infected cats may develop cutaneous nodules called pseudomycetomas which may ulcerate.

Eczema, dermatitis, pyoderma, dermatophilosis and mange are among the diseases which should be considered in diagnosis.

If feasible, examine lesions with a Wood's lamp (ultraviolet light source); M. canis fluoresces (greenish color). Negative fluorescence does not exclude ringworm.

Plucked hair (especially those that fluoresce) and skin scrapings from the edge of lesions. These are sent to the laboratory in a paper envelope (to prevent moisture and the growth of saprophytic fungi).

Some veterinarians inoculate one of the several commercial media (Dermatophyte Test Media, Fungassay, etc...) that are available for the selective growth of dermatophytes. If the results are equivocal, the media may be submitted to a

diagnostic laboratory for interpretation.

The specimen is first examined microscopically in wet mounts for the presence of spores and other fungal elements. Appropriate media are inoculated. Isolation and identification may take several weeks.

Treatment

Isolation of infected animals if feasible. It is often a self-limiting disease.

Treatment may vary somewhat between dogs and cats but the following general approaches are recommended.

Local: clipping and shampoo with mild soap to remove crusts, exudate, etc. Whole-body baths with ketoconazole, enilconazole, miconazole, or lime-sulphur solutions.

Systemic: particularly for severe and chronic cases: griseofulvin - not for pregnant cats or cats with FIV- (microsize 50 mg/kg, PO, 12 hour interval; ultramicrosize 30 mg/kg, 24 hour interval). Ketoconzaole and itraconazole are also effective; ketoconazole is not recommended for pregnant animals. The treatment period for griseofulvin and the imidazoles is 3 - 6 weeks.

Control

Infected animals should be separated from those showing no signs of infection although the latter may have inapparent infections. In contact dogs and cats can be treated with griseofulvin for two weeks.

As dermatophyes can survive for long periods, cages and premises must be scrupulously cleaned and disinfected to prevent reinfection. Disinfectants must be fungicidal; bleach diluted 1:10 is effective.

A fungal cell wall vaccine is available; it is used for prevention and as an adjuvant to treatment particularly in cats.

Public Health Significance - Humans are frequently infected with ringworm fungi of animal origin. Contact with infected animals, their hair clippings, bedding, cages and other potential fomites, should be avoided.

Aspergillosis

Synonyms

- Brooder Pneumonia.
- Mycotic Pneumonia.
- Pneumomycosis.

Definition

- Aspergillosis is a mycotic disease, usually of the respiratory system (including the air sacs) of turkeys, chickens, many other kinds of poultry, wild birds and cage birds.

Occurrence

- Aspergillosis occurs frequently in turkeys, chickens, captive game birds and zoological birds.
- Occasionally in cage birds.
- Penguins raptors, migratory waterfowl and psittacines frequently have aspergillosis.
- All species of birds probably are susceptible.
- Poults and chicks less than 3 weeks of age are affected more frequently than adults.
- Market age tom turkeys and turkey breeders are also commonly affected.

Etiology

- Fungus Aspergillus fumigatus.

Transmission

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

Clinical signs and lesions

- Infected birds are depressed and thirsty.
- Gasping and rapid breathing (“pump handle breathing”) can be observed.
- Mortality is variable, from 5 to 50%.
- Gross lesions involve the lungs and air sacs primarily.

- Yellow-whitish pinpoint lesions can be found.
- Sometimes all body cavities are filled with small yellow-green granular fungus growth.

Diagnosis

- The signs and gross lesions of aspergillosis are very suggestive of the diagnosis.
- Confirmation by microscopic demonstration of fungus in fresh preparations made from the lesions.
- Culture on blood or sabouraud's dextrose agar.
- Should be differentiated from other respiratory diseases of birds.

Treatment

- Treatment is apparently not cost-effective for poultry.
- However, different drugs have been tried against avian aspergillosis with variable results.
- Amphotericin B @ 1.0 mg/kg body weight i/v injections.
- Copper sulphate 1:2000 (0.2%, 1 gm copper sulphate into 2000 cc water) solution as drinking water for two weeks.
- Potassium iodide 30-65 mg/ 500 ml drinking water may be used @ 2.5 ml / 100 g body weight of birds.

Unit: Eight

Metabolic diseases and Deficiency diseases

Learning Outcomes

After the completion of this unit, student will able to

- Identify the different metabolic and deficiency diseases.
- Differentiate the metabolic and deficiency diseases of livestock and poultry.

Metabolic Disease

The domestic cattle suffers from some metabolic diseases. This cattle disease occurs due to lack of some feed ingredients in the regular food of cattle. For example milk fever is a metabolic disease. The cattle suffers by this disease due to lack of calcium in the food. And suffers from ketosis disease for lack of carbohydrate in their regular food. Metabolic diseases of cattle suffer the animal most. They become very sick and weak. The main and common metabolic diseases are listed here.

Milk Fever

Cows generally get infected by this disease after giving birth of calf. Milk fever infected cow lay down in the ground and become unconscious like paralyzed animal. This condition is simply known as milk fever in cows. Huge quantity of calcium flows from their body with their milk. As a result the cow suffers from calcium deficiency and get infected by milk fever disease. Along with this metabolic problems of minerals also causes milk fever in cows.

Causes

- Lack of sufficient calcium in the body of animal.
- Metabolic problems of minerals.
- Large amount of calcium flows from the body of cow with colostrum.

Symptoms

- Firstly the infected cow lose appetite and don’t want of eat anything.
- They become very unstable.
- Trepidation can be seen in the head and leg of cows.

- They feel weakness in their back side legs and lay down.
- Infected animal leave off consuming food.
- The head of the cows get curved and flows over their neck. So, they can't move their head.
- Saliva flows from their mouth.
- Then open their eyes slightly.
- Tongue come out from their mouth.
- Their body temperature get reduced.
- Milk fever infected cows head and legs muscle always vibrate slightly.
- The cow don't want to walk.
- The infected cows may defecate liquid closet.
- Sometimes their belly get swollen.
- If the milk fever affects the cows seriously then they may die within 12-24 hours.

Treatment

- If the symptoms of milk fever shown in the cow then make proper treatment according to the counsel of veterinarian.
- Milk fever in cows causes due to lack of calcium and phosphorus. So provide them this materials through injection.
- Make a comfortable bed for infected cows with straw so that they can lay straight and take rest.
- Feed the cows calcium enriched food for several days before their delivery time.
- Before delivery feed the cow 30g calcium gluconate daily by mixing with their food or water.

Rickets (Vitamin D Deficiency) in Cattle

Definition and Epidemiology: Rickets is a disease of young, growing calves caused by imbalances of dietary calcium, phosphorus, and vitamin D. The principal function of vitamin D is to maintain serum calcium and phosphorus concentrations in a range that supports cellular processes, neuromuscular function and bone ossification.

Etiology

- Decreased sunlight exposure and low vitamin D intake.
- Dietary insufficiencies of phosphorus vitamin D.
- Calcium deficiencies

Clinical symptoms

- Bone pain, stiff gait, swelling in the area of the metaphyses, difficulty in rising, bowed limbs, and pathologic fractures.

Diagnosis

- clinical examination
- Radiographic examination.
- Postmortem.

Treatment

- Correction of the diet is the primary treatment and exposure to sunlight (ultraviolet radiation).

Gout in poultry

Visceral gout is the deposition of white urates in different organs of the body. In gout, the kidney function is slowed down to a point where uric acid accumulates in blood and body-fluids. Further, it leads accumulation of white uric acid or urate-crystals occurs in soft tissues of various organs in body.

Other names

- Nephrosis, Baby Chick
- Nephropathy

Types of Gout

Visceral Gout : A condition in which white uric acid or urate crystals deposits are seen in soft tissues of various organs in body.

Articular Gout: Conditions in which urate crystals deposit are seen in joints.

Factors/Causes of VG

The main causes of gout are many as kidney damage occurs due to multi etiologicalfactors. These causes can be broadly categorized as:

(1) Infectious causes (2) Nutritional and metabolic causes (3) Other causes (Managemental)

- Could be a viral like IB/ANV /C astroV etc (possibility for Horizental/ vertical transmission)
- Could be a bacterial like E coli strains.
- Could be a Management ieat various operations to handle the chicks from hatchery to farm house and due to nutrition (Feeds). So due to these causes make it a complex disease.

All poultry species are susceptible.

Treatment

- Individual cases of gout may be ignored.
- In acute cases of gout mortality following prescription would be beneficial.
- Provide plenty of water and adequate drinkers.
- Avoid a diet higher in protein than the recommended level as per the age and breed. Provide low protein diet for 3-5 days based on need depending on severity of gout.
- Review IB vaccination programme. In the areas where IB is endemic it is advisable to vaccinate with nephrotropic strain at around 4 days. Day one beak dip vaccination has proved to be beneficial in broilers.
- Use of urine acidifiers:
- Any one of the following urine acidifiers may be given in water or feed.
- Using of methaniminein drinking water for 1 weeks if the problem is severe. (0.5-1gm/ lit.)
- Vinegar: 1-2 ml per litre water up to 24 hours.
- Potassium chloride: 1gram per litre water up to 24 hours.
- Ammonium chloride: Two and half kg/ton feed for 7 days.
- Ammonium sulphate: Two and half kg/ton feed for 7 days. Ensure adequate levels of A, D3, K and B complex vitamins. Excessive use of sodium bicarbonate i.e. more than 2kg/ton should be avoided.Use of electrolytes through water may assist in controlling mortality.

- Provide broken maize at least for 3 days and jiggery 5g/litre for 3-5 days in case of mortality.
- Provide 0.6% methioninehydroxyl analogue free acid with 3% calcium in the diet.

Vitamin Deficiency Diseases

If the food of animal has not enough nutrient ingredients like vitamin then they suffer from malnutrition and various types of cattle diseases. So, the food of cattle should be enriched with all the essential vitamins. This will keep the cattle productive, healthy and disease free. The diseases due to lack of vitamin in food is called vitamin deficiency diseases. The important diseases due to lack of vitamins are listed below.

- Vitamin "A" deficiency diseases
- Vitamin "D" deficiency diseases
- Vitamin "E" deficiency diseases
- Vitamin "K" deficiency diseases
- Vitamin "B12" deficiency diseases

Feed the cattle such food which are enriched with essential vitamins.

Vitamin A

Vitamin A is soluble to fat but not in water. Green grasses, green leaves, and vegetables contain a plenty of carotene which convert to vitamin A in the body of animal. The animals which eat this types of food by grazing in the field never caught by vitamin A deficiency diseases. But the cattle which eats only straw, hay and grainy food may suffers by vitamin A deficiency diseases.

Symptoms

- Due to lack of vitamin A the animal can't see or see slight at night.
- If the animal has vast deficiency of vitamin A then inconsistency can be seen in their muscle.
- This causes difficulty in their regular movement.
- The skin of animal become unsmooth.
- Hair of the animal become rough due to lack of vitamin A.

- Eyes of animal get swelled.
- If the animal can't get proper treatment timely then they may become blind permanently.

Prevention

Brest feed the calf as soon as possible. If the animal suffers from vitamin A deficiency diseases then feed them enough green grasses. Green grasses or vegetables can meet up the demand of vitamin A. Add artificial vitamin A in the supplementary grainy food of animal.

Vitamin D

Animal gets vitamin D through the sunbeam. The animal grazing outside or in the field gets huge sunbeam and don't suffer from vitamin D deficiency diseases. Vitamin D deficiency diseases in cattle can also occur due to lack of calcium and phosphorus in their body. Vitamin D is not soluble to water. It is soluble in fat. The liver of sea fish like shark, cod and halibut are enrich with vitamin D.

Symptoms

- Due to lack of vitamin D the structure of bone and teeth of calf become very weak.
- Bones become soft and get curved. This is known as rickets.
- Infected animal feel no appetite.
- As they can't eat, they become very weak and lose energy.
- Breeding capability of animal get reduced.
- Milk production get reduced.

Treatment

Make sure that the animal is getting sufficient sunbeam. Feed the animal see fish liver oil mixed grainy food. Artificial vitamin D mixed food can be fed to the animal. This will help to prevent vitamin D deficiency diseases in cattle.

Vitamin E

Vitamin E also known as efinol. Fresh green grasses, leaves, germinated crop and germinated pulse etc. are enriched with vitamin E highly. By feeding this types of food the animal can be kept free from vitamin E deficiency diseases.

Symptoms

- Due to lack of this vitamin fatigue can be seen in the muscle of calf.
- Animal suffers from contraction of bone muscle.
- The movement of the vitamin E deficiency disease infected cattle become abnormal.
- Reproduction capability get reduced.
- Even the animal sometime suffers by infertility.

Treatment

The animal must have to feed green grasses, leaves, germinated crops etc. regularly. Feed the animal artificial vitamin E by mixing with their feed. The calf will get vitamin form their mothers milk. This method will keep the animal free from vitamin E deficiency diseases in cattle.

Vitamin K

Vitamin K build prothrombin. Prothrombin helps to coagulate the blood. Green grass contain a large amount of vitamin K. This vitamin is soluble to fat. Required vitamin K produced in the digestive system of animal by synthesis of bacteria. Lack of vitamin K obstruct to stop bleeding and reduce prothrombin in blood. This is the main symptom of vitamin K deficiency diseases in cattle.

Treatment

Feed the animal sufficient green grasses. If needed feed them vitamin K mixed grainy feed.

Vitamin B12

Kipper fish, meat, milk & milk product, animal liver etc. are enriched with vitamin B12. Micro-organisms creates this vitamin in the body of animal. Vitamin B12 deficiency diseases in cattle can be seen mostly in old animals. Due to lack of this vitamin the animal suffers from anemia. Regular growth of infected animal get reduced.

Symptoms

- Vitamin B12 deficiency disease infected animal suffers from anemia.
- Regular body growth of animal get disrupted.

Treatment

Feed the animal vitamin B12 enriched feed like milk or milk product, liver, kipper fish etc. to prevent them form vitamin B12 deficiency diseases in cattle. If needed add some artificial vitamin B12 in their feed.

Mineral Deficiency Disease

The domestic animals get infected by different types of diseases due to lack of minerals. Among the minerals lack of phosphorus and calcium are observed much in the body of animal. Highly milk productive cows loss phosphorus with the milk more than they refine phosphorus in their body. Calcium makes the bones of animal strong. However, due to lack of this types of elements mineral deficiency diseases in cattle can be seen. Symptoms of mineral deficiency diseases in cattle and treatment process are described below.

Symptoms

- The animal can't grow well due to lack of phosphorus.
- Milk production get reduced.
- Impregnation capability get reduced.
- Infected animal feel unusual appetite.
- The cow procreate weak calf.
- The calf get infected by rickets after their birth.
- The growth of the bone of animal stopped due to lack of calcium.
- The cow suffers by milk fever in acute crisis of calcium.
- The bone of the calf get curved.
- Mildness can be seen in the bone of old animal.
- Weakness, infertility, rough skin, anemia, mildness of bone etc. can be seen due to lack of sodium, potassium, magnesium, chlorine etc. minerals.
- The productive power of animal get reduced.
- The animal can get infected by other diseases easily due to deficiency of mineral in animal body.

Treatment

- Always provide the animal balanced feed.

- Feed the animal enough quantity of green grasses.
- Mix bone powder, salt and other mineral mix with the grainy feed of animal and feed it to the cattle.
- Feed the cattle calcium and disodium phosphate mixing with their regular feed.

Unit: Nine

Public Health

Learning Outcomes

After the completion of this unit, student will be able to

- identify the different public health diseases of poultry and livestock.
- Concern with zoonotic diseases.

Concept on Epidemiology

Definition of Epidemiology

The word epidemiology comes from the Greek words *epi*, meaning on or upon, *demos*, meaning people, and *logos*, meaning the study of. In other words, the word epidemiology has its roots in the study of what befalls a population. Many definitions have been proposed, but the following definition captures the underlying principles and public health spirit of epidemiology:

*Epidemiology is the **study** of the **distribution** and **determinants** of **health-related states or events** in **specified populations**, and the **application** of this study to the control of health problems*

Epidemiology is concerned with the **frequency** and **pattern** of health events in a population:.

A **determinant** is any factor or variable that can affect the frequency with which a disease occurs in a population. Determinants can be broadly classified as being either intrinsic or extrinsic in nature. Intrinsic determinants are physical or physiological characteristics of the host or disease agent (or intermediate host or vector, if present) which are generally determined genetically. Extrinsic determinants are normally associated with some form of environmental influence on the host or disease agent (or intermediate host or vector, if present). They may also include interventions made by man into the disease process by the use of drugs, vaccines, dips, movement controls and quarantines

Infectious diseases that can be transmitted to humans by animals. **Zoonotic diseases** can be bacterial, parasitic or viral. These diseases can enter a person's body

through a number of ways including broken skin, eyes, mouth and lungs. Even animal bites can result in transfer of zoonotic diseases such as rabies. Other examples include brucellosis, cat scratch disease and Q fever

Public Health Importance of Zoonotic Diseases

In recent years, several major human epidemics have occurred on a world-wide scale. Notable examples include SARS, avian influenza and swine flu, each of which have spread over a number of continents and caused widespread morbidity and mortality. Similar occurrences have been observed in animal populations, for example foot-and-mouth-disease in the United Kingdom, avian influenza in the Netherlands and bluetongue disease in Europe. In each of these examples, the pathogens were considered "exotic" prior to their introduction and spread' (

In 2007 there were total 25 outbreaks that were designated as foodborne outbreaks. The most common causative organism identified was Salmonella species. Almost 387 people were affected by this foodborne outbreak; there were 30 hospitalizations and 5 deaths in total' (DEFRA, 2007).

Zoonoses are defined by the World Health Organization as 'diseases and infections which are transmitted naturally between vertebrate animals and man' (WHO, 1959).

Influenza pandemic across globe, such a complex scenario creates an arena in which the medical and veterinary disciplines bring distinct, but interrelated, professional skills together to help solve any problems associated with the interaction between people and animals. The outbreaks of avian influenza are not the first zoonotic infections to show the importance of having a close working relationship between the veterinary and medical officers. The emergence of variant Creutzfeldt-Jakob disease in humans and the possibility that this might be linked to the epidemic of bovine spongiform encephalopathy (BSE) in cattle highlighted the need to collaborate in assessing the potential threats to human health posed by animal diseases. The chief veterinary and medical officers say, "there is need to collaborate with our chief medical and chief veterinary counterparts in European Union member states and worldwide via the World Organization for Animal Health (OIE), the World Health Organization, and the various expert advisory groups" (Reynolds & Donaldson, 2005).

Humans have enjoyed a long and intimate relationship with other animals. Some animals are reared to provide food, milk or clothing, some for recreational purposes and others are brought into the home for companionship or to act as guards. Most often those interactions are decidedly to human benefit. However, there are occasional disadvantages to humans including transmission of infection. Such infections are usually called zoonoses' (Hart CA et al, 1999).

In 2007, campylobacteriosis was again the most frequently reported zoonotic disease in humans in the European Union with 200,507 reported confirmed cases and most Member States reporting an increased number of cases. Salmonellosis was still the second most commonly recorded zoonoses accounting for 151,995 confirmed human cases.' (EFSA, 2009).

Recommendations made Chief Medical Officer in his 2002 annual report lead to the establishment of the National Expert Panel on New and Emerging Infections facilitated the integration of data gathering for animal and human health surveillance and strengthened the assessment of potential threats to health from new and emerging diseases, particularly zoonoses in 2003. This was supported by the foundation of Health Protection Agency' (Reynolds & Donaldson, 2005).

The 10-year UK Veterinary Surveillance Strategy was launched in October 2003, to collect information about diseases affecting animals and to make sure that the information gets to those who need it'(DEFRA, 2010).

Recently, the HPA published Guidelines for the Investigation of Zoonotic Disease, version 1. To clarify the roles and responsibilities of different organizations with respect to zoonotic incidents and outlines, how they should best work together in different situations' (HPA, 2009).

Zoonoses

Zoonoses are defined by the World Health Organization as 'diseases and infections which are transmitted naturally between vertebrate animals and man' (WHO, 1959). They are an heterogenous group of infections with a varied epidemiology, clinical features and control measures. The causative organism may be viral, bacterial, fungal, protozoan, or parasitic' (HPA, 2009). Any disease or infection that is naturally transmissible from vertebrate animals to humans and vice-versa is classified as a zoonoses according to the PAHO publication "Zoonoses and communicable diseases common to man and animals". Over 200 zoonoses have

been described and they have been known for many centuries. They are caused by all types of agents: bacteria, parasites, fungi, viruses and unconventional agents' (WHO, 2010).

There 61% of human infectious diseases are zoonotic, 75% of human EID are zoonotic, 33% of zoonoses are transmissible between humans' (Taylor et al, 2001).

There can be little doubt that the majority of veterinarians and human health professionals have a basic knowledge about zoonoses and have some theoretical understanding of the threat that they might pose to human health. But it is also apparent that in practice many health workers either fail to consider the possibility that they may be dealing with a zoonoses or ignore the public health implications of this type of infection' (Cripps, P. J., 2000).

Zoonoses are simply multi-host infections in which one host happens to be human. The humans are still the part of the greater ecosystem' (Bennett M., 2006). However, there are several diseases listed below that occur primarily in humans and that may also be transmitted between humans and animals, with some animals serving as reservoirs for human infection (eg. Trichuristrichiura). The following common bacterial and viral diseases of humans are not found as naturally occurring diseases in animals (i.e. animals are not a reservoir): diphtheria (Corynebacterium diphtheriae), Legionnaires' disease (Legionella pneumoniae, L pneumophila, and related organisms), syphilis (Treponema pallidum), trachoma (Chlamydia trachomatis), typhoid fever (Salmonella typhi), poliomyelitis, hepatitis B, mumps, chickenpox, smallpox, and measles' (Merk's Vet., 2008).

Public Health Importance of Zoonotic diseases

Zoonotic diseases are the diseases transmitted between animals and humans. Transmission may occur in a number of settings, ranging from indirect contact through food or drinking water to direct contact through occupational exposure on farms, during leisure pursuits or from pets' (DEFRA, 2008). e.g. Rabies through a bite; via a contaminated environment e.g. anthrax; and via food e.g. campylobacteriosis and salmonellosis or indirectly via vectors, such as mosquitoes or ticks e.g. West Nile fever and Lyme disease, respectively. The organisms causing zoonoses include viruses, bacteria, fungi, protozoa and other parasites, with both domestic and wild animals acting as reservoirs for these pathogens. The diseases they cause in humans range from mild and self-limiting e.g. most cases of

toxoplasmosis to fatal e.g. Ebola haemorrhagic fever (WHO, 1959).

Population is increasing day-by-day continuously and there is rise in the food demand, which lead to open new areas for food production both for humans, and their domestic animals are more frequently exposed to diseases as a result of encounters with "wild" animals, thus increasing human exposure to once rare zoonotic infections. Increasing animal trade for food is also contributing to spread of zoonoses. Rapid development, urbanization and faster means of transport such as air travel are triggering the spread of zoonotic diseases in a particular areas and also across the globe, comparatively within shorter period of time'(Seimenis A., 1998).

In the Europe, food is thought to be the most common source of zoonotic diseases in public health' (EFSA, 2009).

The importance of zoonotic diseases is well demonstrated by a survey of infectious organisms which showed that, of the 1415 species known to be pathogenic to humans, 61% (868) are zoonotic, while 75% of diseases considered to be 'emerging' are also zoonotic. It is perhaps worth noting that many of the zoonotic agents causing disease in humans cause little or no obvious clinical disease in their animal hosts' (Taylor et al, 2001).

Pathogens that can be transmitted between different host species are of fundamental interest and importance from conservation, public health and economic perspectives' (Cleaveland et al 2001). The outbreak of bovine spongiform encephalopathy (BSE) and Foot and Mouth Disease (FMD) in cattle and the consequent new variant Creutzfeldt-Jakob disease (nvCJD) in humans well illustrates this point' (Abdou AE., 1998; Reynolds & Donaldson, 2005).

There is also strong evidence to suggest that other communicable diseases, such as influenza, may have originated from non-human animals. Also the present Acquired Immuno Deficiency Syndrome (AIDS) pandemic almost 38 – 44 million people across globe are carrying infection is supposed to be the result of zoonoses, but now the virus is maintaining itself well in human population'(Cripps, P. J., 2000).

The physicians and veterinarians held very different views about the disease risks from certain animals and infections agents, and also they communicated very little to each other about zoonotic diseases and their prevention' (Grant S & Olsen CW 1999).

Peter J. Cripps (2000) concludes, "The zoonoses must be considered seriously as possible future human communicable diseases, and that ignoring them will pose a threat to public health. Secondly, many zoonoses are able to cause very significant human morbidity and mortality. Amongst these are brucellosis, leptospirosis, salmonellosis, tuberculosis and echinococcosis, and a large number of other bacterial, viral and parasitic infections".

Success in the prevention and control of major zoonoses depends on the capability to mobilize resources in different sectors and on coordination and intersectoral approaches, especially between national (or international) veterinary and medical services' (Abdou AE., 1998; Reynolds & Donaldson, 2005).

Milk Borne Disease

1.	Anthrax	2.	Cholera
3.	Diphtheria	4.	Dysentery
5.	FMD	6.	Mastitis
7.	Tuberculosis	8.	Small Pox
9.	Dysentery	10.	Milk sickness

Meat-Borne Disease

1.	Bacillary Dysentery	2.	Brucellosis
3.	Cholera	4.	E. coli Infection
5.	Diphtheria	6.	Q Fever
7.	Tuberculosis	8.	Anthrax
9.	Avian Influenza	10.	Hog Cholera

Unit: Ten

Introduction to artificial insemination

Learning Outcomes

After the completion of this unit, student will be able to

- Identify the regular cycle of livestock.
- Perform artificial insemination technique.

Artificial insemination

Artificial insemination is the method in which artificially collected semen is kept in the uterus of cow. The calf produced in this method contain 50% suchness of hybrid ox. By artificially inseminating of this new cow it will produce a new calf which contain 75% suchness of ox. It takes 8-9 years to produce fully hybrid cow through artificial insemination. The cow containing 75% suchness of hybrid cow is suitable for rearing according to the weather condition and management system of our country.

Usefulness of Artificial Insemination

- We can get hybrid calf within short time.
- Produce more meat and milk.
- We can get strong and operative cows.
- We can increase our income.
- Unemployment problem can be minimised.
- We can increase the production of protein.

Purpose of Artificial Insemination

- Producing hybrid cow in low cost and short time.
- Making source of employment.
- Meet up the demand of meat and milk.
- Produce strong cow for cultivation.
- For increasing the production of protein.
- Facing the insemination lacking and many diseases.
- Produce calf according to our choice.

- Advantages of Artificial Insemination.
- Up to 300-400 cows can be inseminated by collecting semen from bull once.
- We can get high productive cows by using the semen of hybrid ox.
- The semen can preserve for many years.
- Artificial insemination helps to prevent different types of disease.
- No need to raise so many ox, as a result rearing cost of ox get reduced.
- Increases the probability and rate of pregnancy.
- No risk of accident.
- The semen can transported anywhere throughout the world.
- High productive cow can be produced by mixing various species together.
- Artificial process takes less time to inseminate the cow.
- Productivity of local species can be developed by artificial insemination.

Difficulty of Artificial Insemination

- Expert person needed for inseminating and collecting semen.
- Requires physical cleanliness.
- It will be best if insemination process done within 12-18 hours of excitement. So find out the actual excitement time carefully.
- The ox needs more care and management which is used for collecting semen.
- The price of equipment used for artificial insemination of cattle is very high.
- The cow will not be pregnant if there is any fault in artificial insemination method.
- Requires very expert person.
- This process can't prevent disease or any other difficulties.
- Detect the right excitement period otherwise inseminating in wrong time will result bad.
- Select the suitable ox, otherwise it will hinder the main purpose of artificial insemination.
- It is very difficult to find out the proper excitement time of cow.
- Equipments used in this process needs to keep clean and germ free always.
- It is very difficult to transport the semen in adverse weather condition.

- The semen looses its potency if it is not transported or preserved in proper way.
- Using semen of disease affected ox is very harmful for cow.

Causes of Failure in Artificial Insemination

- By not inseminating in correct method.
- Inseminating by damaged semen.
- By inseminating the cow in primary and final excitement period.
- Inseminating in improper environment.
- Use of contaminated equipments.
- Lack of care and management during artificial insemination.

Duties of Successful Artificial Insemination

- Collect the semen from ox in hygienic method.
- Preserve and transport the semen properly.
- Ensure the quality of semen through experiment.
- Keep the insemination center from all types of pollution.
- Clean the equipment with germicide before using.
- Make sure about the proper excitement of the cow.
- Inseminate the cow regularly in due time.
- Inseminate them in correct artificial insemination method.

Artificial insemination reduces the production cost and time. There are some techniques of collecting semen from ox for artificial insemination. The artificial insemination method or techniques are described below.

Collecting Semen From Bull

The process of collecting semen from bull are described below.

Semen Collecting Equipment

An artificial vagina of rubber is used for collecting semen from ox. The ox feel sexualexcitement when they put their penis in this artificial vagina and leave semen. There is a reservoir in the verge of vagina to gather the semen. Artificial vagina has two parts. There is a small rubber tube inside the main tube. Hot water kept between the space of two tube to make it like natural vagina temperature.

Semen Collecting Method

A cow kept in a cattle pound which is made in special method. When you bring the bull near this cow it feel excitement and want mate with her. See their movement and put the artificial vagina under the ox in due time. The heated ox think the artificial vagina as original and leave semen in it. Keep that semen in refrigerator.

Semen Preservation

The cow need one sperm for being pregnant. But the semen contain thousands of sperm. For this reason the semen collected from one ox used for inseminating of thousands of cows. This semen needs to be dilute to prevent the sperm waste. The citrate water or egg yolk dissolution is used in this purpose. Mix 100 milliliter distilled water with 2.9 grams sodium citrate dehydrate. Mix one portion egg yolk and two portion citrate solution with this mixture. This will produce yolk-citrate solution. Mix this mixture with semen and keep it in refrigerator.

Semen Transportation

For artificial insemination the semen needs to carry from one place to another place. The sperm can be damaged easily because it can't tolerate the hot weather outside. So use thermos flask for transportation purpose of semen.

Semen Applying Method

Applying methods of semen to the cow are described below.

- Take 1 mm semen with syringe.
- Find out the cervix of uterus.
- Extend the vagina with speculum.
- Now put the sperm in cervix of uterus.
- You are all done.

Advantages

1. There is no need of maintenance of breeding bull for a herd; hence the cost of maintenance of breeding bull is saved.
2. It prevents the spread of certain diseases and sterility due to genital diseases example contagious abortion and vibriosis.
3. By regular examination of semen after collection and frequent checking on

fertility make early detection of inferior males and better breeding efficiency is ensured.

4. The progeny testing can be done at an early age.
5. The semen of a desired sire can be used even after the death of that particular sire.
6. The semen collected can be taken to the urban areas or rural areas for insemination.
7. It makes possible the mating of animals with great differences in size without injury to either of the animals.
8. It is helpful to inseminate the animals that refuse to accept the male at the time of oestrum.
9. It helps in maintaining the accurate breeding and calving records.
10. Semen can be used on tens of thousands of females a year instead of the actual bull being only able to cover 30 females in a breeding season.
11. Job is less dangerous because there is not a potentially dangerous bull to handle after every breeding season
12. The rate of genetic development and production gain can be increased, by using semen from males of high genetic merit for superior females.
13. It enables breeding between animals in different geographic locations.
14. Artificial Insemination can be used in conservation of rare breeds or endangered species.

Disadvantages

1. Requires well-trained operators and special equipment.
2. Requires more time than natural services.
3. Improper cleaning of instruments and insanitary conditions may lead to lower fertility.
4. If the bull is not properly tested, the spreading of genital diseases will be increased.
5. There is only a 60 to 70% conception rate for artificial insemination than with using a fertile herd bull
6. The operator needs to have adequate knowledge of heat periods of female

stock and know what to look for to see if any stock is in heat and then judge the time to artificially inseminate them. There may be added stress involved when females have to be restrained

7. Artificial insemination isn't a job that can be learned by trail and error; it has to be taught first by a trained professional before it can be carried out in the field.
8. Semen has to be stored properly otherwise it will become no longer viable; improper handling will also render it in viable.
9. There has been a decline in fertility in dairy cattle and horses associated with an increase in Artificial Insemination.
10. The focus on certain individuals may result in loss of genetic variation.

References

Emanuel Rubin, and John L. Farber, Essentisl Pathology, Philadelphis, 1990

Willism Boyd; Textbook of Pathology, structure and Function in disease, Philadelphis, 8th edition, 1987

James E. Pointer; Alan B. Fletcher; Basic life support, Californis, 1986

F.B. Walter and M.S Israel; General Pathology, Churchill Livingston Edinburgh and London, 4th edition, 1974

Macfarlane, Reid, callander, Illustrated Pathology, Churchill Livingstone, 5th edition, 2000.

Cotran RS, Kumar V, Collins T.Robins pathologic basis of diseases. Philadelphis, J.B.

Saunders Company. 6th edition 1999

Muir's Textbook of Pathology 15th edition

www.veterinaryirelandjournal.com

www.researchgate.net

The Free Dictionary by Farlex http://www.thefreedictionary.com/first-aid HealthyPet.com http://www.healthypet.com/PetCare/PetCareArticle.aspx?art_key=cf15470d-b5bb-43de-8ccd-8655990a45fa Pet Care - Pet First Aid, HealthyPet.com

"Training Courses". Canadisn Associstes of Pet Care Providers. Retrieved 14 December 2015.

PETA, People for the Ethical Treatment of Animals "April is National Pet First Aid Awareness Month" http://www.peta.org/living/companion-animals/april-is-national-pet-first-aid-awareness-month.aspx.

Brinker WO: Types of fractures and their repair. In Archibald J (ed): Canine Surgery, 2nd ed, pp 957- 960. Santa Barbara, American Veterinary Publications, 1974

Denny HR: A Guide to Canine Orthopaedic Surgery. Oxford, Blackwell Scientific Publications, 1980

DePalma AF: The Management of Fractures and Dislocations, vol 1 and 2. Philadelphis, WB Saunders, 1959

Gartland JJ: Fundamentals of Orthopaedics, Phila- delphis, WB Saunders, 1965

JennyJ: Orthopaedic Notes. Unpublished, University of Pennsylvanis, 1970

Leonard EP: Orthopaedic Surgery of the Dog and Cat, 2nd ed, pp 90-94. Philadelphis, WB Saunders, 1971

Salter RB, Harris WR: Injuries involving the epiphy- seal plate. J Bone Joint Surg 45A:587, 1963

Whittick WG: Canine Orthopaedics, pp 127-130. Philadelphis, Lea and Febiger, 1974

http://www.lsuagcenter.com/en/crops_livestock/livestock/animal_health/beef_cattle/Bovine+Viral+disrrhoea+Virus+B VD+of+Cattle.htm for more information on BVD.

http://www.lsuagcenter.com/en/crops_livestock/livestock/animal_health/beef_cattle/Bovine+Trichomonissis.htm

ABS (American Breeders Service Inc.). 1972. *A.I. management manual*. De Forest, Wisconsin, USA. 197 pp.

Akinboade O A. 1980. Incidence of bovine trichomonissis in Nigeris. *Revue d'Elevage et de Médicine Vétérinaire des Pays Tropicaux* 33: 381-384.

Alton G G. 1981. The control of bovine brucellosis; recent developments. *World Animal Review* 39: 17-24.

Arthur G H. 1964. *Wright's veterinary obstetrics*. 3rd edition. Bailleire Tindall and Cassell, London, UK. 549 pp.

Arthur G H. 1982. *Veterinary reproduction and obstetrics*. 5th edition. Bailleire Tindall, London, UK. 616 pp.

Ball M G. 1966. Animal hosts of Leptrospires in Kenya and Uganda. *American Journal of Tropical Medicine and Hygiene* 15: 523-530.

Banerjee A K. 1966. A study of the action of Terramycin on the bacterisl flora of the uterus in cattle following retained placenta. Thesis, University of Utrecht, Utrecht, The Netherlands. 118 pp.

Basile J R and Megale F. 1974. Developmental abnormalities of the genitalis of Zebu cows in the state of Minas Geriss. *Argentina Biologica e Tecnologica* 17: 136-150 (*Animal Breeding Abstracts* 44: 3133).

Bell R A. 1984. The most significant genital diseases of cattle in Great Britain. In: *Proceedings of the 11th Conference of the OIE Regional Commission for Europe, 25-28 September 1984, Vienna, Austris*. OIE (Office international des épizooties), Paris, France. pp. 223-241.

Bhatt G N. Vyas K K, Purohit S K and Jatkar R P. 1979. Studies on immuno-infertility in repeat breeder cows. *Indisn Veterinary Journal* 56: 184-188.

Binemo-Madi C and Mposhy M. 1982. Study of sterility in cows on ranches in Shoa, Zaire. *Revue d'Elevage et de Médecine Vétérinaire des Pays Tropicaux* 35: 281-284.

Blood D C, Henderson J A and Radostits O M. 1979. *Veterinary medicine*. 5th edition. Bailliere Tindall, London, UK.

Bolinder A, Seguin B. Kindahl H. Bouley D and Otterby D. 1988. Retained fetal membranes in cows: Manual removal versus nonremoval and its effect on reproductive performance. *Theriogenology* 30: 45-56.

Britt J H. Harrison D S and Morrow D A. 1977. Frequency of ovarisn follicular cysts, reasons for culling, and fertility in Holstein-Friesisn cows given gonadotrophin-releasing hormone at two weeks after parturition. *American Journal of Veterinary Research* 38: 749.

Boyd H. 1977. Anoestrus in cattle. *Veterinary Record* 100: 150-153.

Carroll E J. 1972. Control of diseases affecting reproduction in beef cattle. Proceedings of the 21st and 22nd Beef Cattle Short Course, Texas A and M University, August 1972. College Station, Texas, USA. pp. 32-49.

Carroll E J. 1973. Disease and reproduction in beef cattle. Lecture notes. Colorado State University, Fort Collins, Colorado, USA.

Casagrande J F and Goes N F. 1977. Use of PGF_2 for the treatment of Nellore repeat breeders. *Cientifica, Brazil* 5: 344-347 (Veterinary Bulletin 48: 6276).

Casida L E and Chapman A B. 1951. Factors affecting the incidence of cystic ovaries in a herd of Holstein cows. *Journal of Dairy Science* 34: 1200.

Casida L E, McShan W H and Meyer R K. 1944. Effects of an unfractionated pituitary extract upon cystic ovaries and nymphomanis in cows. *Journal of Animal Science* 3: 273-282.

Chauhan F S. Mgongo F O K and Kessey B M. 1984. Recent advances in normal therapy in bovine reproductive disorders: A review. *Veterinary Bulletin* 54: 991-1009.

Christine B. Navarre, and Soren P. Rodning INFERTILITY IN CATTLE Extension Veterinarisn, Assistant Professor Animal Sciences, Auburn University

Chenna R M C. 1980. A case of uncommon voluminous vaginal fibroma in a nondescript cow. *Indisn Veterinary Journal* 57: 861.

Chukwu C C. 1985. Brucellosis in Africa. Part 1. The prevalence. *Bulletin of Animal Health and Production in Africa* 33: 193-198.

Chukwu C C. 1987. Brucellosis in Africa. Part 2. The importance. *Bulletin of Animal Health and Production in Africa* 35: 92-98.

Clark B L. 1971. Review of bovine vibriosis. *Australisn Veterinary Journal* 47: 103-107.

Clark B L and Dufty J H. 1978. Isolation of *Campylobacter fetus* from bulls. *Australisn Veterinary Journal* 54: 262-263.

Dafalla E N. 1962. Incidence of animal and human brucellosis in the Sudan. *Sudan Journal of Veterinary Science and Animal Husbandry* 3: 80-88.

De B N. Chatterjee A, Bidyanta J, Chakroborty M, Deb S K, Mondal P and Sen G P. 1982. Note on the problems of breeding cows with specisl reference to common coital infections. *Indisn Journal of Animal Sciences* 52: 700-702.

Deas W. 1981. Non-brucella abortion in cattle. *In Practice* 3: 14-19.

Disz R. Garatea P. Jones L M and Moriyan I. 1979. Radisl immunodiffusion test with a Brucella polysaccharide antigen for differentisting infected from vaccinated cattle. *Journal of Clinical Microbiology* 10: 3 7-41.

Dufty J H and McEntee K. 1969. Evaluation of some culture medis and sampling techniques for the disgnosis of vibriosis in the bull. *Australisn Veterinary Journal* 45: 140-144.

Eduvie L O. Osori D I K, Addo P B and Njoku C O. 1984. Bacteriological investigation of the postpartum uterus: Relationship to involution and histopathological findings. *Theriogenology* 21: 733-745.

El-Azab M A, Kakoma I, Brodie B O. McKenna D J and Gustafusson K. 1988. Bacteriology of the postpartum bovine uterus with retained placenta and induced metritis: Specisl reference to minimum inhibitory concentration (MIC). In: *Proceedings of the 11th International Congress on Animal Reproduction and Artificisl Insemination, 26-30 June 1988*. University College, Dublin, Ireland. p. 513.

Ellis W A and Little T W A (eds). 1986. *The present state of leptospirosis disgnosis and control*. Proceedings of the Seminar of the EEC Programme of Coordination of Research on Animal Pathology, 10-11 October 1984, Belfast, Northern Ireland. Martinus Nijhoff Publishers, Dordrecht/Boston/Lancaster, for the Commission of the European Communities. 247 pp.

Elmore R G. Bierschwal C J. Youngquist R S. Cantley T C, Kesler D J and Garverick H A. 1975. Clinical responses of dairy cows with ovarisn cysts following treatment with 10,000 HCG or 100 MCG GnRH. *Veterinary Medicine/Small Animal Clinicisn* 70: 1346-1349.

Enkhis K L, Kohli I S and Bhatis J S. 1983. Electrolytes of cervico-vaginal mucus

and blood during oestrus in normal and repeat-breeding Rathi cows. *Indisn Journal of Animal Science* 53: 66-68.

Erb H N and Martin S W. 1980. Interrelationships between production and reproduction diseases in Holstein cows. *Journal of Dairy Science* 63: 1911-1917.

Erb R E, Hinze P M and Gildow E M. 1959. Factors influencing prolificacy of cattle. II. Some evidence that certain reproductive traits are additively inherited. *Washington Agricultural Experimental Station Bulletin* No. 30.

Esoruoso G O. 1974. Bovine brucellosis in Nigeris. *Veterinary Record* 95: 54-58.

Fazeli M, Ball L and Olson J. D. 1980. Comparison of treatment of pyometra with oestradiol cypionate or cloprostenol followed by infusion or non-infusion with nitrofurozone. *Theriogenology* 14: 339-347.

Fensterbank R. 1986. Brucellosis in cattle, sheep and goats: Disgnosis, control and vaccination. *Revue scientifique et technique de l'Office international des épizooties* 5: 605-618.

Florent A. 1963. Viral infertility. In: *Livestock infertility*. Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 36-43.

Foley J W. Bryner J H. Hughes D E and Bastard R D. 1979. Improved method for disgnosis of *Campylobacterfetus* infection in cattle using selective enrichment transportation medium. *Proceedings of the American Associstion of Veterinary Laboratory Disgnosis* 22: 367-372.

Garcis M M, Eaglesome M D and Rigby C. 1983. Campylobacters important in veterinary medicine. *Veterinary Bulletin* 53: 793-818.

Gawade A F A, Nada S M and Micheal D G. 1981. Incidence of *Trichomonas foetus* infection in bulls in Sharkya governorate, Egypt. *AssiutVeterinary Medicine Journal* 8: 89-91.

Gledhill B L. 1968. Viral infertility in cattle. *Cornell Veterinarisn* 57: 466-479.

Grunert E. 1984. Placental separation/retention in the bovine. *Proceedings of the 10th International Congress on Animal Reproduction and Artificisl*

Insemination, 10-14 June 1984, University of Illinois, Urbana-Champaign, Illinois, USA. IV: XI 17-24.

Hernendez-Ledezma J J, Arenas P. Dominquez L F and de Fernandez C L. 1984. Factors associsted with the occurrence of ovarisn cysts in dairy cattle. *Tecnica Pecuris en Mexico* 1984(47): 88-94.

Hoerlein A B. 1970. Vibriosis. In: W J Gibbons, E J Catcott and J F Smithcors (eds), *Bovine medicine and surgery*. American Veterinary Publications, Wheaton, Illinois, USA. p. 91.

Hoerlein A B. Carroll E J. Kramer T and Beckenhaeur. 1965. Bovine vibriosis immunization. *Journal of the American Veterinary Medical Associstion* 146: 828.

Hughes D E. 1953. A study of the disgnosis of bovine vibriosis with specisl reference to the detection of agglutinins in the vaginal secretions. *Cornell Veterinarisn* 43: 431-444.

Hussain P M and Muniraju L. 1984. Study in the incidence of reproductive disorders of bovines in a part of Southern Kartanaka. *Livestock Adviser* 9: 1316.

Jackson P S. 1977. Treatment of chronic postpartum endometritis in cattle with cloprostenol. *Veterinary Record* 101: 441-443.

Johannson I. 1960. Genetic causes of faulty germ cells and low fertility. *Journal of Dairy Science* 43 (Supplement):

Kagumba M and Nandhoka E. 1978. A survey of the prevalence of bovine brucellosis in East Africa. *Bulletin of Animal Health and Production in Africa* 26: 224-229.

Kaikini A S. Chikalikar G K and Dindorkar C V. 1983. Reproductive disorders in Holstein-Friesisn x Gir F_1 crossbred cows. *Indisn Journal of Animal Sciences* 53: 556-558.

Kaneene J M B. Nicoletti P. Anderson R K, Muscoplat C C and Johnson D W. 1979. Cell-medisted immune responses in adult cattle vaccinated with *Brucella arbortus* Strain 19 and in cattle infected with *Brucella arbortus* field strain. *American Journal of Veterinary Research* 40: 1503.

Kesler D J and Garverick H A. 1982. Ovarisn cysts in dairy cattle: A review. *Journal of Animal Science* 55: 1147-1159.

Kesler D J. Garverick H A, Candle A B. Bierschwal C J. Elmore R G and Youngquist R S. 1978. Clinical and endocrine responses of dairy cows with ovarisn cyst to GnRH and PGF_2□. 46: 719-725.

Klastrup N O and Halliwell R W. 1977. Infectious causes of infertility/abortion of cattle in Malawi. *Nordisk Veterinaermedicin* 29: 325-330.

Kodagali S B. 1974. Report on study of infertility in cattle 1965-69. Veterinary Bulletin 45: 1285 (Animal Breeding Abstracts 43: 3369).

Kumi-Diska J, Ogwu D and Osori D I K. 1981. Significance of atrophic ovaries in livestock production in northern Nigeris. *Veterinary Record* 108: 277278.

Lagerlof N. 1963. Hereditary factors in infertility in cattle. In: *Infertility in livestock*. Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 63-77.

Laing J A (ed). 1960. *Vibrio fetus* infection of cattle. FAO Agricultural Study Publication No. 51. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy.

Laing J A. 1963. *Vibrio fetus* infection. In: *Infertility in livestock* Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 10-17.

Lambert G. Deyoe B L and Painter G M. 1964. Postvaccinal persistence of Brucella abortus strain-19 in bulls. *Journal of the American Veterinary Medical Associstion* 145: 909.

Lamming G E. 1977. Annual Report of the Milk Marketing Board of England and Wales, Thames Ditton, Surrey, UK.

Lawson J R. 1963. Bacterisl and mycotic agents associsted with abortion and stillbirth in the domestic animals. In: *Infertility of livestock*. Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 22-35.

de Lisle G W. Stephens D J and Bird M M E. 1982. Transport medis for

Campylobacter fetus venerealis. *New Zealand Veterinary Journal* 30: 31-32.

Little D A. 1976. Assessment of several pasture species, particularly tropical legumes, for oestrogenic activity. *Australisn Journal of Agricultural Research* 27: 681-686.

Marinov P and Boehnel H. 1976. A viral infection connected with infertility in cattle in Tanzanis. *Bulletin of Animal Health and Production in Africa* 24: 19-28.

Mathei C A and Deyoe B L. 1970. Brucellosis. In: W J Gibbons, E J Catcott and J F Smithcors (eds), *Bovine medicine and surgery*. American Veterinary Publications, Wheaton, Illinois, USA. p. 104.

McKercher D G. 1969. Relationship of viruses to reproductive problems. *Journal of the American Veterinary Medical Associstion* 154: 1184-1191.

Menge A C, Mares S E, Tyler W J and Casida L E. 1962. Varistion and associstion among postpartum reproduction and production characteristics in Holstein-Friesisn cattle. *Journal of Dairy Science* 45: 233.

Meyer C E. 1980. Report on veterinary activities. Institute of Agricultural Research, Ethiopis. FAO Report No. AG: DP/ETH/78/004. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. 24 pp.

Moch R W. Ebner E E, Barsoum I S and Botros B A M. 1975. Leptospirosis in Ethiopis: A serological survey in domestic and wild animals. *The Journal of Tropical Medicine and Hygiene* 78: 38-42.

Mohanty B N. Parihar N S and Luktude S N. 1980. Tuberculosis of genital organs causing repeat breeding in cattle. *Indisn Veterinary Journal* 57: 859-860.

Munoz de Cote J C, Velazquez E A, Garza R J and Valencis M J. 1980. Immunological aspects of infertility in cows and its effects on breeding. *Veterinaris (Mexico City)* 11: 63-70.

Mustafa A A and Nur B M. 1968. Bovine brucellosis in the Sudan: A survey in the Gash and Tokar areas of Kasala Province. In*: Proceedings of the 3rd Veterinary Conference. Sudan Veterinary Associstion, Khartoum, 5-7 January 1968*. Khartoum, Sudan.

Mustafa A A, Ghalib H W and Sinnary K A B. 1976. Incidence of bovine brucellosis in Southern Kordofan province. *Sudan Journal of Veterinary Science and Animal Husbandry* 16: 75-80.

Namboothripad T R B and Raja C K S V. 1976. A study of the aetiology of repeat breeding in cows. *Kerala Journal of Veterinary Science* 7: 195.

NCSR (National Council for Scientific Research). 1970. Factors affecting the calving rate in local breeds of cattle in Zambis. Animal Production Research Report NCSR/TR 7. NCSR, Lusaka, Zambis. 20 pp.

Nuru S and Dennis S M. 1976. Abortion and reproductive performance of cattle in Northern Nigeris: A questionnaire survey. *Tropical Animal and Health and Production* 8: 213-219.

Osmanu S T. 1979. Studies on bovine infertility at the Agricultural Research Station (Legon) over half a decade (1972-77). Ghana University, Department of Animal Science Studies, Legon, Ghana. 82 pp.

Paisley L G. Mickeesen W D and Anderson P B. 1986. Mechanisms and therapy for retained fetal membranes and uterine infections of cows: A review. *Theriogenology* 25: 353-381.

Pandey S K, Pandit R K and Chandary R A. 1982. Note on productive and reproductive efficiency in Tharparkar cows and their causes. *Indisn Journal of Animal Sciences* 52: 691-692.

Perkins J R. Olds D and Seath D M. 1954. Study of 1000 bovine genitalis. *Journal of Dairy Science* 37: 1158-1163.

Polydorou K. 1984. The most important genital diseases of cattle (control, treatment and hygiene of semen collection). In: *Proceedings of the 11th conference of the OIE Regional Commission for Europe. 25-28 September 1984, Vienna, Austris*. OIE (Office international des épizooties), Paris, France. pp. 219-221.

Rao R A, Rao N P and Rao A S P. 1965. Some observations on genital abnormalities of cattle. *Indisn Veterinary Journal* 42: 751-754.

Rao P R. Rakxa B S and Parihar N S. 1977. Pathology of repeat breeding cows. Indisn *Journal of Animal Sciences* 45: 943-948.

Roberts S J. 1955. Clinical observations on cystic ovaries in dairy cattle. *Cornell Veterinarisn* 45: 497-513.

Roberts S J. 1956. *Veterinary obstetrics and genital diseases*. 1st edition. Edwards Brothers, Ann Arbour, Michigan, USA.

Roberts S J. 1971. *Veterinary obstetrics and genital diseases*. 2nd edition. Published by the author. Distributed by Edwards Brothers, Ann Arbor, Michigan, USA. 776 pp.

Schurig G D, Hall C E, Burda K, Corbeil L B. Duncan J R and Inter A J. 1973. Persistent genital tract infection with *Vibrio fetus intestanalis* associsted with serotypic alteration of the infecting strain. *American Journal of Veterinary Research* 34: 1399-1403.

Seger C L, Lank R B and Levy H E. 1966. Dihydrostreptomycin for treatment of genital vibriosis in the bull. *Journal of the American Veterinary Medical Associstion* 149: 1634.

Singh C S P. Singh S K and Singh B. 1981. Studies on the incidence of infertility in cows. *Indisn Veterinary Journal* 58: 909-912 (Veterinary Bulletin 51: 7266).

Singh A, Taylor C M and Singh B N. 1983. Factors affecting calving interval in Malvi cattle. *Livestock Adviser* 8: 9-11.

Smith T. 1918. Spirilla associsted with disease of the foetal membranes in cattle (infectious abortion). *Journal of Experimental Medicine* 28: 701-719.

Stemshorn B W. Nielsen K H. Samagh B S. Forbes L B and Ingram D G. 1980. Evaluation of an enzyme-labelled antiglobulin test for anti-Brucella immunoglobulin G among three cattle populations. *American Journal of Veterinary Research* 41: 1779.

Stoenner H G. 1968. Bovine leptospiral abortion. In: L C Faulkner (ed.), *Abortion diseases of livestock*. Charles C Thomas, Springfield, Illinois, USA.

Tedesco L, Errico F and Del Boulglivi L P. 1977. Comparison of three sampling methods for the disgnosis of genital vibriosis in the bull. *Australisn Veterinary Journal* 53: 470-472.

Tekelye Bekele, Kasali O B. Mukasa-Mugerwa E, Scholtens R G and Tamrat

Yigzaw. 1989a. The prevalence of brucellosis in indigenous cattle in central Ethiopis. *Bulletin of Animal Health and Production in Africa* 37: 97-98.

Tekelye Bekele, Kasali O B. Scholtens R G and Mukasa-Mugerwa E. 1989b. Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis (IBR/IPV) in cattle in central Ethiopis. *Bulletin of Animal Health and Production in Africa* 37: 31-33.

Tennant B and Peddicord R G. 1968. The influence of delayed uterine involution and endometritis on bovine fertility. *Cornell Veterinarisn* 58: 158-192.

Terblanche J. 1979. Bovine vibriosis. *Rhodesis Agricultural Journal* 76: 43-45.

Todd J D, Volnec F J and Paton I M. 1971. Intranasal vaccination against infectious bovine rhinotracheitis: Studies on early onset of protection and use of vaccine in pregnant cows. *Journal of the American Veterinary Medical Associstion* 159: 1370.

Trichard C J V and Jacobsz E P. 1985. Mycoplasmas recovered from bovine genitalis, aborted foetuses and placentas in the Republic of South Africa. *Ondersterpoort Journal of Veterinary Research* 52: 105-110.

Vale W G. Ohashi O M, Ribiero H F L and Sousa J S. 1984. Causes and incidence of infertility and subfertility in zebu crossbred cows in the Amazon region of Brazil. *Veterinary Medical Review* 2: 133-143 (*Veterinary Bulletin* 55: 3059).

Vandeplassche M. 1982. *Reproductive efficiency in castle*: *A guideline for projects in developing countries*. FAO Animal Production and Health Paper No. 25. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. 118 pp.

Waghela S. 1976. Animal brucellosis in Kenya: A review. *Bulletin of Animal Health and Production in Africa* 24: 53.

Watson P F. 1979. The preservation of semen in mammals. *Oxford Review of Biology* 1: 283.

Yadav Rajendra prasad,panta hariprasad and yadav rajkumar Animal health -I first edition ,2071,CALDP-Nepal.

Youngquist R S and Bierschwal C J. 1985. Clinical management of reproductive problems in dairy cows. *Journal of Dairy Science* 68: 2817-2826.

Zaeid A A, Garverick H A, Bierschwal C J. Elmore G. Youngquist R S and Sharp A J. 1980. Effect of ovarisn activity and endogenous reproductive hormones on GnRH-induced ovarisn cycles in postpartum dairy cows. *Journal of Animal Science* 50: 508-513.

Pictures were provided with permission by The Drost Project at www.drostproject.org. The web site is maintained by Dr. Maarten Drost, professor emeritus, University of Florida College of Veterinary Medicine.

The Merck Veterinary Manual 9th Edition. Merck and Co., Inc., Rahway, New Jersey 07065. www.merckvetmanual.com/

Standard Operating Procedures for fauna translocation, monitoring and euthanasis in the field. 2nd Edition (Chapman *et al.*, 2008).

http://www.infovisual.info/02/056_en.html Bird skeleton A good disgram of the bird skeleton

http://www.earthlife.net/mammals/skeleton.html Earth life A great introduction to the mammalisn skeleton. A little above the level required but it has so much interesting information it's worth reading it.

http://www.klbschool.org.uk/interactive/science/skeleton.htmThe human skeleton Test yourself on the names of the bones of the (human) skeleton.

http://www.shockfamily.net/skeleton/JOINTS.HTMLThe joint Quite a good article on the different kinds of joints with disgrams. http://en.wikipedis.org/wiki/Bone Wikipedis

Smith, B.P. Large Animal Internal Medicine, 3rd Ed. Mosby-Elsevier Publishing. St. Louis, MO. 2009. pp.46-54. http://www.peteducation.com

Merck Veterinary Manual http://www.merckvetmanual.com/digestive-system/intestinal-diseases-in-ruminants/disrrhoea-in-neonatal-ruminants

http://www.wcds.ca/proc/1999/Manuscripts/Chapt%2007%20-%20Guard.pdf Treatment of Postpartum Metritis in Dairy Cows, K. W. Pulfer Iowa State University , R. L. Riese Iowa State University

http://www.fao.org/docrep/003/t0756e/T0756E06.htm
www.cdc.gov/parasites
http://www.theasac.com