TVET CERTIFICATE IV in Animal Health

COMMON INFECTIOUS DISEASES TREATMENT

ANHID401

Treat common infectious diseases

Competence

Credits: 90

Learning hours: 90

Sector: Agriculture and food processing

Sub-sector: Animal Health

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Purpose statement

This core module describes the skills, knowledge and attitude required to treat common infectious diseases. Any veterinary has to treat common infectious diseases during his professional work. So, this competence is very important for the Veterinary nurse training.

Upon completion of this module, the trainee will be able to:

- Describe common infectious diseases
- Perform diagnosis for infectious diseases
- Apply treatment for infectious diseases

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Learning Unit 1 – Describe common infectious diseases

Introduction

Infectious diseases constitute a serious threat of animal health and production in many countries especially in developing countries; where veterinary drugs, required infrastructures and skilled workers are not sufficient .Frequently, morbidity and mortality rates are very high, resulting in economic losses thus reducing the production. As the majority of the population of Rwanda is depending on agriculture, the government, in its priorities, decided to promote agriculture sector and the human resource strengthening is one of important strategies.

L O 1.1 – Identify common infectious diseases

Common infectious diseases are classified into bacterial, viral and fungal diseases.

- Topic 1: Common bacterial diseases

The following are the common bacterial diseases:

- Anthrax (Charbon bacteridien)
- Black quarter (black leg)
- Brucellosis
- Tuberculosis
- Tetanus
- Contagious bovine pleuropneumonia
- Pneumonia
- Leptospirosis
- Pasteurellosis

- Salmonellosis(Purollosis, thyphose)
- Mastitis
- Abces
- Colibacillosis
- Omphalitis
- Streptococcosis
- Kerato- conjunctivitis
- Stomatitis
- Otitis
- Metritis
- Gastro-enteritis
- Erysipelas
- Actinomycosis
- Joint ill (Arthrite des veaux)
- Wooden tongue
- Foot rot
- Gastro-intestinal infection

- Topic 2: Common viral diseases

The following are the common viral diseases:

- Lumpy skin diseases
- Newcastle diseases
- Canine distemper
- Rabbies
- Rinder Peste
- Small ruminant peste
- Gumbolo disease
- Sheep pox
- Blue tongue
- Contagious ecthyma
- Classical swine fever
- African swine fever

- Foot and mouth disease (FMD)
- Chicken pox

- Topic 3: Common fungal diseases

The following are the common fungal diseases:

- Aspergillosis
- Ringworm

L O 1.2 – Characterize common bacterial diseases

- Topic 1: Common bacterial diseases

Bacteria may be found almost everywhere, in different conditions according to their life requirements. Somme bacteria cause great loss to animals by causing diseases; fighting against bacteria diseases must be based on the characteristics of each one.

A. Anthrax

A.1. Definition

Anthax also known as splenic fever ,(Charbon bacteridien) is an acute infectious disease chiefly affecting cattle, sheep, swine, horses and other animal species including man caused by *Bacillus anthracis* and characterized by septicemia, enlargement of the spleen and hemorrhagic infiltration throughout the body.Probably this is the most septicemic disease.

A.2. Etiology

Bacillus anthracis is aerobic, non-motile, encapsulated, gram-positive, spore forming, facultatively anaerobic, roughly rectangular rod with squared ends (about µm by 3to5µm); chain of rods are common. Ability of spores to survive long periods in soil. Spores have been known to have infected animals over 70 years after burial sites of anthrax-infected animals were disturbed. Spores are relatively resistant to extremes of temperature, chemical disinfection, and desiccation; lye solution 5% useful in disinfecting.

A.3. Epidemiology

Distribution

Anthrax is cosmopolitan; the disease is enzootic in west Texas and Minessota; sporadic in South Texas, Nevada, eastern and South Dakota; and only seen elsewhere.

Dry soils are likely to perpetuate survival of spores also neutral to alkaline soils favour survival.

Transmission

Most often related to ingestion; herbivorous are often infected while grazing or browsing, especially when eating rough irritant or spiky vegetation; the vegetation has been hypothesized to cause wounds within the gastrointestinal tract allowing entry of bacteria spores. Indirect transmission by:

- Fomites – feed sacks;
- Bone meals;
- Biting flies - blood sucking insects;
- Birds and wildlife spreading straw, bedding and carcasses.

Susceptible hosts

Cattle, sheep, goats, mules, swine, mink, mice, guinea pigs, deer. Dogs, cats, rats, and most birds are relatively insusceptible; man is susceptible.

A.4. Pathogenesis

After wound inoculation, ingestion, or inhalation, spores germinate and proliferate. In cutaneous and GI infection, proliferation can occur at the site of infection and the lymph nodes draining the site of infection. Lethal toxin and edema toxin are produced by *B. anthracis* and respectively cause local necrosis and extensive edema, which is a frequent characteristic of the disease.

As the bacteria multiply in the lymph nodes, toxemia progresses and bacteremia may ensue. With the increase in toxin production, the potential for disseminated tissue destruction and organ failure increases. After vegetative bacilli are discharged from an animal following death (by carcass bloating, scavengers, or postmortem examination), the oxygen content of air induces sporulation. Necropsy is discouraged because of the potential for vegetative cells to be exposed to air, resulting in large numbers of spores being produced. Because of the rapid pH change following death and decomposition, vegetative cells in an unopened carcass quickly die without sporulating

A.5. Clinical signs and lesions

Picture 1: **Blood discharge as sign of Anthrax**

Symptoms

Typically the incubation period is 3-7 days (range 1-14 days).The clinical course ranges from peracute to chronic.

The peracute form: common in cattle and sheep is characterised by:

- Sudden and rapidly fatal course;
- Staggering, trembling and collapse;
- A few convulsive movements and death may occur in cattle, sheep or goats with only a brief evidence of illness.

In acute anthrax of cattle and sheep:

- An abrupt fever and a period of excitement followed by depression, stupor;
- Respiratory or cardiac distress, staggering, grinding the teeth, convulsions and death.
- Often, the course of disease is so rapid that illness is not observed and animals are found dead.
- The body temperature may reach 107^0F(41.5^0C);
- Rumination ceases, milk production is reduced, pregnant animal may abort;
- They may be blood discharges from natural body openings;
- Some infections are characterised by localized, subcutaneous swelling that can be quite extensive. Area most frequently involved are the ventral neck, thorax and shoulders.

Lesions

Rigor mortis is frequently absent or incomplete; lesions include the following:

- Dark blood may ooze from the mouth nostrils and anus with marked bloating and rapid body decomposition
- The blood is dark and thickened; and fails to clot readily;
- Hemorrhage of different sizes are common on the seronasal surface of the abdomen and thorax as well as on the epicardium and endocardium, haemorrhages frequently occur along GI tract mucosa and ulcers.
- An enlarged dark red or black, soft, semifluid spleen is common;
- The liver, kidney, and lymph nodes are congested.

A.5. Diagnosis

a) Clinical diagnosis

Combination of assessment of history, symptoms and lesions:

- The diagnosis is made from the examination of aspirated carcass blood.
- Exudation of tarry blood from the body orifices of the cadaver;
- Failure of the blood to clot; absence of rigor mortis and the presence of splenomegally.
- Incision of spleen shows a black mud like content. Confirmatory laboratory examination should be attempted.

Note that the Carcass is not opened if anthrax is suspected.

Laboratory diagnosis

- Optimal sample for laboratory is a cotton swab dipped in the blood and allowed to dry;
- In pigs with localised disease, a small piece of affected lymphatic tissue that has been collected aseptically should be submitted. Before submission the receiving reference laboratory should be contacted regarding appropriate cipecimen labelling, handling and shipping procedures.
- Specific diagnostic tests include bacterial culture, fluorescent antibody stains to demonstrate the agent in blood films or tissues.

Differential Diagnosis

- Blackleg
- Bacillary hemoglobinuria
- Piroplasmosis
- Anaplasmosis

A.8. Treatment

Several antibiotics may be used but the treatment should begin early as possible.

- Domestic livestock respond well to penicillin if treated in the early stages of the disease;
- Oxytetracyclines given daily in divided doses also is effective;
- Other antibacterial including amoxicillin, chloramphenicol, ciprofloxacin, doxycycline, erythromycin, gentamycin, streptomycin and sulphonamides also can be used.

A.9. Prevention

The prevention of the disease is based on different methods.

- Use of bacterins;
- Avoiding overgrazing;
- Early isolation and quarantine;
- Insect control;
- Developing sound immunization program(every year);
- Avoiding feeding carcasses of animals dying of this disease
- Proper burying and disposed of carcasses
- Burning of bedding;
- Disinfecting premises;
- Keeping birds, animals and insects away;
- Burning or burial of suspect and confirmed cases;
- Annual vaccination of all grazing animals endemic areas;
- Animals at risk should be immediately treated with along acting antibiotic to stop all potential incubating infections;
- Avoid the site where animal died with anthrax have been buried and avoid disturbing this area.

B. Blackquarter

B.1. Definition

Blackquarter is an acute febrile disease of ruminants caused by *Clostridium chauvoei*. The disease is characterized by crepitated emphysematous swelling, usually in heavy skeletal muscles of the body. Black leg, quarter ill, evil, Pseudoanthrax, gangrene are the synonyms of black quarter.

B.2. Etiology

Blackquarter is caused by *Clostridium chauvoei*. gram-positive, motile, obligatory anaerobic rod, spore-forming, bacteria - vegetative form, non-capsulated rod.

Spores develop from the vegetative form when the vegetative forms are exposed to air. Spores are highly resistant to environmental changes and disinfectants and persist in soil for many years, 5% formalin will rapidly kill organism in short time

B.3. Epidemiology

Distribution

The disease occurs throughout the world and is a considerable source of economic loss in cattle and has a seasonal incidence with most cases occurring in the warm months of the year. The highest incidence in spring or fall, depending probably on when calves reach the susceptible age group.

Transmission

Cattle: soil-born infection but portal of entry of organism is still in dispute. However, it is presumed that the portal of entry is through GI mucosa after ingestion of contaminated feed.

- Bacteria may be found in the spleen, liver and GI tract of normal animals.
- Contamination of soil and pasture - entry from alveoli.
- Sheep and Goat: Almost always a wound infection;
- Pigs: Not really known - Oral or wounds.

Susceptible hosts

- Cattle, sheep, goats and wild ruminants (deer**)** are naturally susceptible.
- Pigs are infrequently infected
- Dogs, cats, human, horses appear to be immune
- Mink fed infected beef liver showed disease -mice, rat, guinea pigs, hamster, rabbit experimentally infected.
- In cattle disease is largely confined to young ages (6-24 months).
- Age is not a factor in sheep, goats, and pigs - mortality reaches 100% in cattle.

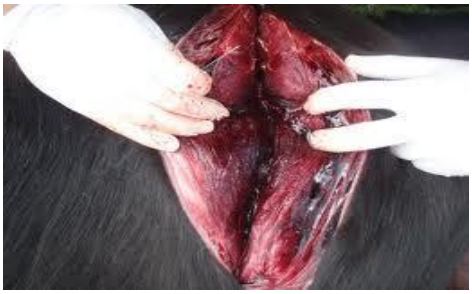
B.3. Pathogenesis

The causal agent of blackleg are probably ingested, pass through the wall of the GI tract and after gaining access to the bloodstream are deposited in muscles and other tissues (spleen, liver, and alimentary tract) and may remain dormant. There is no consensus on the pathogenesis of blackleg infection that occurs in ruminants, but toxins and neuraminidase produced by *Clostridium chauvoei* are believed to play a significant role in pathogenesis of the disease. Probably, spores reach skeletal muscles via intestine.

Conditions favouring spore germination, bacterial growth, and toxin production cause formation of local lesions marked by edema, hemorrhage and myofibrillar necrosis. The centers of lesions become dry, dark, and emphysematous due to bacterial fermentation while the periphery is edematous and hemorrhagic. A rancid-butter odour is typical.

B.5. Symptoms and lesions

Picture 2: Hindleg stuck out stiffly in cattle affected with blackquater



Picture 3: Dark almost black colour lesion in cattle affected with blackquater

Symptoms and lesions of Blackquater are:

- When infection begins, the animal may develop a fever, and the affected limb can feel hot to the touch.
- The limb usually swells significantly, and the animal can develop lameness on the affected leg.
- Crepitation (the sensation of air under the skin) can be noticed in many infections, as the area seems to crackle under pressure. These characteristic edematous and crepitant swellings develop in hip, shoulder, chest, back, neck or elsewhere.

At the first, the swelling is small, hot and painful. As the disease progresses, the swelling enlarges, there is crepitation on palpation and the skin becomes cold and insensitive with decreased blood supply to affected area.

A cut section of infected quarter shows a dark almost black, dry sponge-like central area due to the presence of small bubbles .There is a pervading rancid smell(rancid butter or cheese) which may be marked by the smell of putrefaction of carcass if it is not examined soon after death.

Cattle succumb to the disease without showing any symptoms, and only a necropsy reveals the cause. During a necropsy, a diagnosis is usually made very quickly, as the affected muscle is usually mottled with black patches, which are dead tissue, killed by the toxins the bacteria release when they infect live tissue. At the first, the swelling .Cattle found dead of Blackleg are often in a characteristic position, lying on the side with the affected hindleg stuck out stiffly. Bloating and putrification occur quickly.

- Blood stained froth exudate from nostrils and anus
- Rapid clotting of the blood
- Incision of the affected muscle mass reveals presence of dark, swollen tissue with rancid odor.
- Myocardial tissue and diaphragm may be the only tissues affected in some cases

Sheep

- When lesions in the limb muscle - stiff gait
- Edema and crepitation is not common before death - high fever, anorexia, depression, and quick death. Similar necropsy findings but muscle lesions are more localized and deeper.
- In all cases suspected of Blackleg smear of tissue should be made for bacterial examination.

Pigs

- Few cases reported.
- Very acute. Pigs die 48 hrs. after appear to be sick;
- Swelling of the throat region, even involving the face and the ear;
- Edema of pharynx is most prevalent.

Frequently the disease happens very quickly but sometimes it may happens more slowly; the animal is tired and weak ,usually ,the back legs are lame and they become swollen and hot; by palpation(pressure) you can feel bubbles under the skin over the back legs.

B.6. Diagnosis

Clinical symptoms and post-mortem are sufficient to warrant a preliminary diagnosis.

A positive diagnosis depends on lab work:

Differential diagnosis

- Anthrax
- Bacillary hemoglobinuria
- Malignant edema
- Emphysema due to injury

B.7. Treatment

Separate healthy and sick animal immediately;

- Treat sick animals with penicillin should be given in IV followed by IM injections.
- Blackleg antisera;
- Note that the treatment is often disappointing.

B.8. Prevention and Control

The prevention and control are based on vaccination, hygine and sanitation

- Bacterin - single or multiple antigens. It is advised to use a combined bacterin containing *Cl. chauvoei*, *septicum*, and *novyi*.
- In sheep - good immunity does not develop if vaccinated less than a year old.
- To prevent infection of ewe in enzootic area - vaccinate ewes three weeks before lambing on one occasion only.

Proper disposal of died animal:

- Burn the carcass or bury it deeply with lime;
- Burn any contaminated areas
- Disinfect any contaminated areas;
- Do not conduct a necropsy or any biopsy on the animal;
- Do not feed the carcass to any other animal(s).

C. Contagious Bovine Pleuropneumonia (CBPP)

C.1. Definition

Contagious bovine pleuropneumonia (called lung sickness or lung plague) is a highly contagious pneumonia accompanied by pleurisy.The disease progresses rapidly, animals lose condition, and breathing becomes very (rapid) labored, with a grunt at expiration. Contagious bovine pleuropneumonia (CBPP) is an acute, subacute or chronic respiratory disease of cattle caused by a Mycoplasma called *Mycoplasma mycoides mycoides* SC (small colony).

C.2. Etiology

The causal organism is Mycoplasma mycoides mycoides small colony type.The organisms are sensitive to dessication,and chemical disinfectants.They do not survive in direct sunshine for more than 24 hours.*M.mycoides mycoides* survive for up to 96hours in urine.They are rod-shaped,about 0.5µm wide.No spore,flagella and capsule.

C.3. Epidemiology

Distribution

It is present in Africa, with minor outbreaks occurring in the Middle East. The USA has been free of the disease since 1892, the UK since 1898, and Australia since 1973. The last outbreak of CBPP in Europe was seen in Portugal in 1999. Little is known about the disease in Asia, but China claims that its last outbreak was in 1995.

Transmission

Under natural conditions cattle become infected by inhaling droplets expelled by infected animals by coughing. Transmission is complicated by existence of carrier animal which appear clinically healthy but has a localized focus of infection in its lungs. The focus is surrounded by fibrous tissues in the form of sequestrate and vary in size.

The carrier animal does not disseminate infection when the capsule is intact, but in times of stress the capsule may be broken down allowing the viable organisms to escape through the bronchi and also infect susceptible animal. Septicemia produces lesions in the kidneys and very occasionally in the placenta, which can be sources of infection. Transplacental infection of carriers may not be detectable clinically or serologically, they constitute a serious problem in control programs. Breed susceptibility, management systems, and general health of the animal are important factors that influence the infection.

Susceptibility

Most affected animals include cattle, buffalo, camels and goat.

C.4. Pathogenesis

The mode of infection is most frequently by inhalation. Somme mycoplasma have a predilection for infecting mesenchymal cells lining serous cavities and joints; others parasitize tissues of the respiratory tract, including the lungs. Their attachment to cells of the respiratory tract may result in destruction of cilia predisposing to secondary bacterial infection. The fibrinous exudate frequently present in infection protects them from antibody and antimicrobial drugs and contributes to chronicity.

C.5. Symptoms and lesions

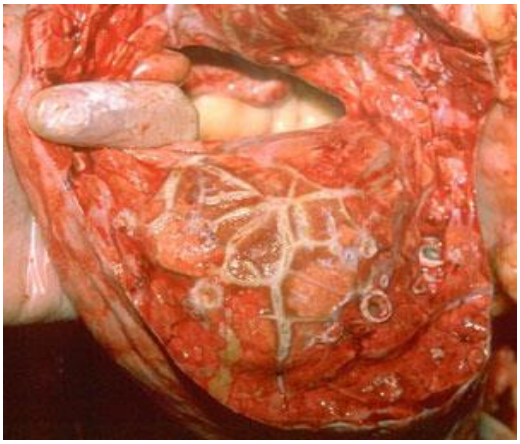
The incubation period varies, but most cases occur 3–8 week after exposure.

In acute cases

- Fever up to 107°F (41.5°C); anorexia; and painful, difficult breathing.
- Loss of appetite, and depression ;
- Mucoid nasal discharge;
- Sometimes abortions and stilbirths
- In hot climates, the animal often stands by itself in the shade, its head lowered and extended, its back slightly arched, and its elbows turned out.
- Percussion of the chest is painful; respiration is rapid, shallow, and abdominal. If the animal is forced to move quickly, the breathing becomes more distressed and a soft, moist cough may result.
- The animal becomes recumbent and dies after 1–3 week.

In chronical cases

Picture 4: Chronic pleuropneumonia in cattle



Picture 5: Section of affected lung: Enlarged and solid. Pleuro-pneumonic portion of lung

Affected cattle usually exhibit signs of varying intensity for 3–4 week, after which the lesions gradually resolve and the animals appear to recover. Subclinical cases occur and may be important as carriers.

The thoracic cavity may contain up to 10 L of clear yellow or turbid fluid mixed with fibrin flakes, and the organs in the thorax are often covered by thick deposits of fibrin.

The disease is largely unilateral with over 80–90% of cases affecting only one lung. The affected portion being enlarged and solid.

On section of the lung, the typical marbled appearance of pleuro-pneumonia is evident due to the widened interlobular septa and subpleural tissue that encloses gray, yellow, or red consolidated lung lobules.

In chronic cases, the lesion has a necrotic center sequestered in a thick, fibrous capsule, and there may be fibrous pleural adhesions. Organisms may survive only within the inner capsule of these sequestra, and the animals may become carriers.

C.6. Diagnosis

Diagnosis is based on clinical signs; as soon as an outbreak is suspected, slaughter and necropsy of presumptively infected cattle is advisable; confirmation is by isolation of the mycoplasma followed by growth inhibition or immunofluorescence test using hyper-immune rabbit sera against the mycoplasma. Confirmation of serologic reactions can be made by immune blotting test.

C.7. Treatment

Note that the treatment is recommended only in endemic areas because the organisms may not be eliminated, and carriers may develop. Tylosin (10 mg/kg, IM, bid for 6 injections) and danofloxacin 2.5% (2.5 mg/kg, sid for 3 consecutive days) have been reported to be effective.

C.8. Prevention

The disease is reportable by law in many countries from which it has been eradicated by slaughter of all infected and exposed animals.

- In countries where cattle movement can readily be restricted, the disease can be eradicated by quarantine, blood testing, and slaughter.
- Where cattle cannot be confined, the spread of infection can be limited by immunization with attenuated vaccine (eg, T1/44 strain). However, the vaccine is effective only if herd coverage within a country is high.
- Tracing the source of infected cattle detected at abattoirs, blood testing, and imposition of strict rules for cattle movement also can aid in control of the disease in such areas. .
- Carrier animals constitute a serious problem in control program because they are not detected clinically or serologically.

D. Brucellosis

D.1. Definition

Brucellosis is a bacterial infection that spreads from animals to people most often via unpasteurized milk, cheese and other daily products. More rarely, the bactia that cause brucellosis can spread through the air or through direct contact with infected animals; the disease is caused by bacteria of genus brucella.

D.2. Etiology

Brucella species are small, gram-negative, non-mobile, non-spore-forming, rode-shaped (coccobacilli) bacteria. At 60^0C brucella are killed in 10-15 minutes. Exposure to direct sunlight kills the organisms within a few hours. Brucellae have been recovered from fetuses and from manure that has remained in a cool environment for >2 monts.

The disease in cattle, water buffalo, and bison is caused almost exclusively by *Brucella abortus*; however, *B suis* occasionally is isolated from seropositive cows but does not appear to cause clinical signs and is not contagious from cow to cow. In some countries, the disease in cattle is caused by *B melitensis*.B.melitensis is found in sheep and goat; *B.suis* in pigs; *B.canis* indogs; *B.ovis* in sheep.

D.3. Epidemiology

Distribution

Brucellosis is found worldwide in cattle-raising regions, except in Japan, Canada, some European countries, Australian, New Zeland and Israel where it has been eradicated.

Transmission

Brucellosis is transmitted by ingestion of unsterilized milk or meat from infected animals. Infection can occur also when there is ingestion of contaminated pasture, drinking water, fodder, and bedding or from eating infected substances after birth or discharges or licking the skin of new-borne calf. Natural transmission occurs by ingestion of organisms, which are present in large numbers in aborted fetuses, fetal membranes, and uterine discharges. Venereal transmission by infected bulls to susceptible cows appears to be rare.

Transmission may occur by artificial insemination when *Brucella*-contaminated semen is deposited in the uterus but, reportedly, not when deposited in the midcervix. Brucellae may enter the body through mucous membranes, conjunctivae, wounds, or intact skin in both people and animals. Infection spreads rapidly and causes many abortions in unvaccinated cattle. In a herd in which disease is endemic, an infected cow typically aborts only once after exposure; subsequent gestations and lactations appear normal. After exposure, cattle become bacteremic for a short period and develop agglutinins and other antibodies; some cattle resist infection, and a small percentage of infected cows spontaneously recover.

Susceptibility

Suscptible animals may include cattle, water buffalo camels, sheep gaot, pig, dog and other species.

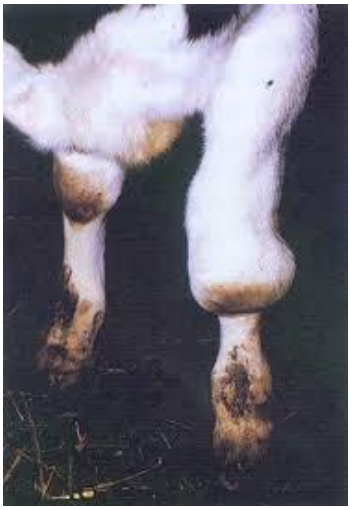
D.4. Pathogenesis

Following exposure, Brucella penetrate intact mucosal surfaces. In the alimentary tract, the epithelium cover the ileal Peyer's patches are preferred site for entry.

After penetrating mucosal barriers, organisms may be engulfed by phagocytic cells. Brucella are internalized in macrophages by generalized membrane ruffling and macropinocytosis. Various mechanisms are employed by Brucella to allow for survival inside phagocytic cells.

Brucella are capable of surviving and multiplying inside macrophage by modifying the phagosome maturation process and creating an intracellular environment suitable for multiplication. Following into the host, Brucella organisms, either free in the extracellular environment or in phagocytic cells, localize to regional lymph nodes. There they proliferate and infect other cells or are killed and the infection is terminated. There is preferential localization to the reproductive tract of pregnant animals.

D.5. Symptoms and lesions



Picture 6: Arthritic joints of a cattle affected by Brucellosis

The incubation period in cattle 2weeks to 5monts

Brucellosis symptoms may include:

- Fever, joint pain and fatigue; symptoms may disappear for weeks or months and then return.
- Clinical signs of the cattle infected with Brucella abortus include the abortion which usually occurs during the second half of gestation; it is accompanied by retention of placenta and brownish vaginal discharge.
- Infections may also cause stillborn or weak calves, retained placentas, and reduced milk yield.
- Arthritic joints;
- In case of sow many piglets are borne dead;
- Usually, general health is not impaired in uncomplicated abortions.
- Unilateral or bilateral testicular enlargement;
- Seminal vesicles, ampullae, testicles, and epididymis may be infected in bulls; therefore, organisms are present in the semen. Agglutinins may be demonstrated in seminal plasma from infected bulls. Testicular abscesses may occur. Longstanding infections may result in arthritic joints in some cattle resulting in hygroma.
- Very severe case may result in complete sterility;

D.6. Diagnosis

Diagnosis is based on bacteriology or serology. *B abortus* can be recovered from the placenta but more conveniently in pure culture from the stomach and lungs of an aborted fetus.

Foci of infection remain in some parts of the reticuloendothelial system, especially supramammary lymph nodes, and in the udder. Udder secretions are the preferred specimens for culture from a live cow.

- Serum agglutination tests have been the standard diagnostic method; agglutination tests may also detect antibodies in milk, whey, and semen.
- Screening tests: non-dairy and dairy herds in an area may also be screened for brucellosis by testing serum samples collected from cattle destined for slaughter or replacements through intermediate and terminal markets, or at abattoirs. Reactors are traced to the herd of origin, and the herd is tested. The cost of identifying reactors by this method is minimal compared with that of testing cattle in all herds.

D.7. Prevention

Efforts are directed at detection and prevention, because no practical treatment is available.

- Eventual eradication depends on testing and eliminating reactors. The disease has been eradicated from many individual herds and areas by this method. Herds must be tested at regular intervals until two or three successive tests are negative.
- Non-infected herds must be protected. The greatest danger is from replacement animals. Additions should be vaccinated calves or non-pregnant heifers. If pregnant or fresh cows are added, they should originate from brucellosis-free areas or herds and be sero-negative. Replacements should be isolated for at least 30 days and retested before being added to the herd.
- Vaccination of calves with *B abortus* Strain 19 or RB51 increases resistance to infection. Resistance may not be complete, and some vaccinated calves may become infected, depending on severity of exposure.
- A small percentage of vaccinated calves develop antibodies to Strain 19 that may persist for years and can confuse diagnostic test results. To minimize this problem, calves in the USA are mostly vaccinated with a vaccine of Strain RB51. It is a rough attenuated strain and does not cause production of antibodies, which are detected by most serologic tests. Whole-herd adult cattle vaccination using Strain 19 or RB51 has been practiced in certain high-incidence areas and selected herds in the USA with much success.
- Vaccination as the sole means of disease control has been effective. Reduction in the number of reactors in a herd is directly related to the percentage of vaccinated animals. However, when proceeding from a control to an eradication program, a test and slaughter program becomes necessary.

E. Tuberculosis

E.1. Definition

Tuberculosis is an infectious disease caused by certain pathogenic organisms of the genus mycobacterium and is usually characterized by the formation of nodular granulomas known as "Tubercles". Tuberculosis is commonly defined as a chronic, debilitating disease.

E.3. Etiology

The family Mycobacteriaceae contains one genus, Mycobacterium.

They are aerobic, slightly curved or straight rod, acid-fast. It is hard to stain by Gram's method, but are considered to be Gram-positive, non-mobile, non-spore forming, can resist for several weeks in dark areas, resistance in the milk if not boiled at 58^0C in 1hour; resistance to congelation and salting.

Three major types of tubercle bacilli are recognized in warn-blooded animals.

- Human type - *Mycobacterium tuberculosis*. It can also cause disease in pigs and occasionally in cattle.
- Bovine type - *Mycobacterium bovis*. It is closely related to the human type and causes TB in cattle, pigs, man, horses, and occasionally in sheep.
- Avian type - *Mycobacterium avium*. Produces diseases primarily in birds and occasionally in pigs, cattle, sheep, and man.

E.3. Epidemiology

Distribution

Bovine tuberculosis is known to exist in all parts of the world. It is of major importance in dairy cattle. The disease can occur in all species including man and is of importance for public health reasons as well as for its detrimental effect on animal production. In pigs the incidence is much lower. In horses and sheep the disease occurs rarely.

Transmission

Although the organism does not form spores, it is moderately resistant to heat, desiccation and many disinfectants. It is readily destroyed by direct sunlight unless it is in a moist environment.

E.4. Pathogenesis

Inhalation of infected droplets expelled from the lungs are the usual route of infection; although ingestion, particularly via contaminated milk, also occurs. Inhaled bacilli are phagocytized by alveolar macrophages that may either clear the infection or allow the mycobacteria to proliferate. In later instance, a primary focus may form, mediated by cytokinesis associated with a hypersensitivity reaction that consist of dead and degenerate macrophages surrounded by epithelioid cells, granulocytes, lymphocytes and later multinuclear giant cells. The purulent to gaseous, necrotic center may calcify and the lesions may become surrounded by granulation tissue and a fibrous capsule to form a classic tubercle.

E.5. Symptoms and lesions

Picture 7: **Miliary abscesses in lung of cattle affected by Tuberculosis**

The clinical signs reflect the extent and location of the lesions.

Generalised signs include:

- Progressive emaciation, lethargy, weakness, anorexia, fluctuating fever;
- The bronchopneumonia in the respiratory form of the disease causes a chronic, intermittent, moist cough with later signs of dyspnea and tachypnea;
- The destructive lesions of the granulomatous bronchopneumonia may be detected on auscultation and percussion.
- Superficial lymph node enlargement may be a useful diagnosis diagnostic sign when present.
- Tuberculosis granulomas may be found in any of the lymph nodes, particularly in bronchial and mediastinal lymph nodes.
- Tuberculosis granulomas usually have a yellowish appearance and are caseous. The caseous center is firm and covered by a fibrous thick capsule.

- In the lung, miliary abscesses may extend to cause a suppurative bronchopneumonia. The pus has a characteristic cream to orange color and varies in consistency from thick cream to thick cheese. Small nodules may appear on the pleura and peritoneum.

E.6. Diagnosis

The diagnosis of TB in animals after death is seldom difficult because the lesion has a characteristic appearance. In cases of doubt the causative organism can be demonstrated in stain films or cultures or by inoculation into experimental animals.

Because of the chronic nature of the disease and the multiplicity of signs caused by the variable localization of the infection, TB is difficult to diagnose on clinical examination and tuberculin test is usually necessary to diagnose the disease.

Differential diagnosis

- Lung abscesses
- Pleurisy and pericarditis following traumatic reticulitis and chronic contagious bovine pleuro-pneumonia.

Treatment of tuberculosis is contraindicated because it rarely successful.

E.7. Prevention

Test for tuberculosis with intradermal tuberculin and slaughter presumed infected animals is the only assured approach to eradicate the disease.

- Eradication of bovine TB is a major objective that has nearly been achieved in many countries of Europe, USA, Japan, New Zealand and Australia. The basis of these eradication programs has been the systematic application of the TB test and the slaughter of reactors.
- Hygiene measures to reduce contamination of facilities are also useful;
- Spread of TB from animals to man makes this disease an important zoonoses. Infection in man occurs largely through consumption of infected milk, but spread can also occur by inhalation.
- Transmission to man can be almost completely eliminated by pasteurization of milk, but only complete eradication of TB can protect the public. Because of the risk that TB will spread to man, the affected animal is usually destroyed rather than treated.

F. Wooden tongue

F.1. Definition

Wooden tongue or Actinobacillosis is a specific infectious disease caused by a gram-negative coccibacilli belonging to the genus Actinobacillus. Infection with *A. lignieresii* is responsible for the wooden tongue disease characterised by the presence of granulomas with pus containing small, hard yellow to white granules.

F.2. Etiology

Wooden tongue is caused by bacteria of genus *Actinobacillus* spp. (e.g. *A. lignieresii, A. suis, A.pleuropneumoniae* and *A. equuli*). Gram- negative coccobacilli, rod-shaped bacteria, pathogens affecting soft tissue.

F.3. Epidemiology

Distribution

The disease occurs worldwide. It usually occurs sporadically, preferentially in areas with copper deficiency or pasture with abrasive weeds. High prevalence is recorded in some areas in New Zealand.

Transmission

The organism enters tissues of the mouth through epithelial damage associated with rough fodder e.g. containing sharp stems or thorns, where it multiplies locally. *A. lignieresii* is considered to be a normal rumen inhabitant of sheep and cattle. It survives 4 to 5 days in forage or hay and consequently actinobacillosis is not considered to be a highly contagious disease.

Susceptible species

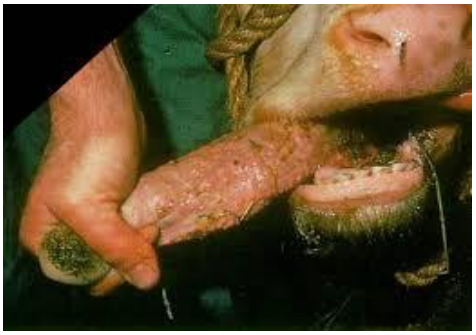
Actinobacillus lignieresii affects mostly cattle and sheep but the disease is also known to occur in horses, pigs, dogs and chickens.

F.4. Pathogenesis

Actinobacillus in the mouth, invade tissues through breaks in the lining of the mouth and multiply in soft tissues with the tongue and lymph nodes of the head. The disease starts suddenly with the tongue becoming hard, swollen and painful; affected animals drool saliva and appear to be shewing gently.

F.5. Clinical signs and lesions

In cattle, actinobacillosis mainly affects the tongue (‘wooden tongue’), the lymph nodes of the head and neck. The characteristic lesion is a granuloma of the tongue, with discharge of pus to the exterior.




Picture 8: Ulcers on the tongue and Hardening and ulcer on the tongue of cattle

Infection usually begins as an acute inflammation with sudden onset of:

- inability to eat or drink for several days;
- drooling saliva ;
- rapid loss of condition;
- painful and swollen tongue ;
- nodules and ulcers on the tongue ;
- Animals may occasionally die from starvation and thirst in the acute stages of the disease. As the infection becomes chronic, fibrous tissue is deposited and the tongue becomes shrunken and immobile and eating is difficult.
- Local lymph nodes may be enlarged and abscesses may form and discharge creamy pus, which may contain granules.
- Less commonly the jaw, lungs, oesophageal groove, or udder may be affected. Rarely granulomas may occur anywhere on the skin or internal organs.
- In sheep, the tongue is not commonly affected. Multiple purulent granules occur in the skin if the face, lips, nose, jaw and neck with regional lymph nodes usually being involved. Lesions develop into abscesses that rupture and discharge yellow-green pus containing granules. Affected sheep have difficulty in eating and many die of starvation.
- At post-mortem, animals will often be in poor condition. Granulomatous lesions containing pus may be seen in and around the mouth. There are often ulcers up to 1 cm in diameter. Encapsulated abscesses may be found in local lymph nodes. In chronic cases there is fibrous connective tissue proliferation and hardening and distortion of the tongue (‘wooden tongue’) is common.

F.5. Diagnosis

The disease can be strongly suspected on clinical grounds. Diagnosis can be confirmed by microscopic examination of smears made from pus, or by culturing the organism.

The pus contains small brownish-white granules, which upon microscopic examination are seen to consist of colonies of Gram- negative, rod-shaped bacteria surrounded by club-shaped structures.

Differential diagnosis

Similar granulomatous lesions may be seen with infections caused by:

- *Actinomyces bovis*,
- *Staphylococci* spp,
- *Streptococci* spp,
- *Corynebacterium pyogenes.*

F.6. Treatment and prevention

Actinobacillosis is readily treated.

- Treatment can involve surgical debridement and flushing with iodine.
- Administration of potassium iodide orally (6 to 10 g a day for 10 days) or intravenous injection of sodium iodide at 10 % (8 g for 100kg) is effective to stop the acute signs of the disease within two days.
- Antibiotics can be used; streptomycin is considered the treatment of choice, tetracylcines and tilmicosin are also effective.
- Control of actinobacillosis is best achieved by early recognition and prompt treatment of cases; isolation or disposal of animals with disease is recommended.
- The risk of actinobacillosis can be reduced by not grazing animals on poor quality, coarse fodder, but this is often not feasible
- No vaccines are available.

G. Actinomycosis

G.1. Definition

Actinomycosis is an infectious disease caused by bacteria of genus Actynomyces which are the normal flora of the oral and nasal pharyngeal membranes; several species are associated with the diseases in animals.
It causes disease when it gains access to adjacent soft tissues via penetrating wounds. It causes localized infection and can spread via lymphatics to other tissues

G.2. Etiology

Actinomyces are gram-positive, non-motile, non-spore-forming, which require enriched media for growth anaerobic, non-acid-fast rods, many of which are filamentous or branching. Branches are <1 µm in diameter, as opposed to fungal filaments, which are >1 µm in diameter.

G.3. Epidemiology

Actinomyces bovis is found in the oropharynx of cattle and other domestic animals, and *Actinomyces viscosus* is a commensal in the oral cavity of dogs and humans. *Arcanobacterium pyogenes* is commonly present on the nasopharyngeal mucosa of cattle, sheep and pigs.

G.4. Pathogenesis

Arcanobacterium pyogenes produces a haemolytic exotoxin, pyolysin, which is cytolytic for several cell types including neutrophils and macrophages, and is dermonecrotic and lethal for laboratory animals. It is a major virulence factor of *A. pyogenes* as mutants lacking the gene for pyolysin production are cleared rapidly from experimentally infected mice.

Neuraminidases produced by *A. pyogenes* are thought to assist in adherence of the organism to host tissues, probably by exposing hidden host cell receptors for otheradhesins. *Arcanobacterium pyogenes* produces a number of other adhesins also including extracellular matrix-binding proteins and fimbriae. This bacterium also produces proteases whoserole as potential virulence factors are not yet determined.

Virulence attributes of *Actinomyces* species important inveterinary medicine have not been established. Purulent reactions are typical of infections with *A. pyogenes*, whereas *A. bovis* and *A. viscosus* provoke pyogranulomatous reactions.

G.5. Diagnosis

Species of animal affected, clinical presentation and type and location of lesions may suggest the species involved.

- Specimens suitable for laboratory procedures include exudates, aspirates and tissue samples for culture and histopathology.
- Gram-stained smears may reveal morphological forms typical of the aetiological agent.
- Histo-pathological examination of specimens from lesions caused by *A. bovis* reveals aggregates of filamentous organisms surrounded by eosinophilic club-shaped structures.
- Blood and MacConkey agars are inoculated with the specimen and incubated at 37°C for up to 5 days

G.6. Symptoms and lesions

Canine actinomycosis

Actinomyces viscosus is the aetiological agent of canine actinomycosis. Infection can result in subcutaneous pyogranulomatous lesions and extensive fibrovascular proliferation on the peritoneal or pleural surfaces with sanguinopurulent exudate in the affected cavity. The thoracic lesions closely resemble those of canine nocardiosis. The main clinical finding is respiratory distress. *Actinomyces viscosus* has also been isolated from cutaneous lesions in a horse and from a heifer which had aborted.

The lesions associated with *A. hordeovulneris* infection in the dog include cutaneous and visceral pyogranulomas, pleuritis, peritonitis and arthritis. In uncomplicated infections, *A. viscosus* is usually responsive to treatment with penicillin. A new species, *Actinomyces canis*, which was isolated from a number of different clinical conditions in dogs, has been described.

Bovine actinomycosis (lumpy jaw

Picture 9: Swelling of the jaw in cattle

Invasion of the mandible and, less commonly, the maxilla of cattle by *A. bovis* causes a chronic rarefying osteomyelitis. The organism is presumed to invade the tissues following trauma to the mucosa from rough feed or through dental alveoli during tooth eruption. A painless swelling of the affected bone enlarges over a period of several weeks. The swelling becomes painful, and fistulous tracts discharging purulent exudate develop. Spread to contiguous soft tissues may occur but there is minimal involvement of regional lymph nodes.

G.7. Diagnosis

- Clinical signs are often distinctive in advanced cases.
- Radiography can be used to determine the degree of bone destruction.
- Lumpy jaw should be distinguished from other conditions which result in swelling of the bones of the jaw and from actinobacillosis which may involve the soft tissues of the head.

G.7. Treatment

- When lesions are small and circumscribed, surgery is the therapy of choice. In advanced cases, surgical treatment is frequently unrewarding.
- Prolonged therapy with penicillin, given parentally to animals early in the disease, may be of value. Isoniazid *per os* for 30 days has also been recommended.

H. Footrot

H.1. Definition

Foot rot, or infectious pododermatitis, is a hoof infection commonly found in sheep, goats, and cattle. As the name suggests, it rots away the foot of the animal, more specifically the area between the two toes of the affected animal. It is extremely painful and contagious disease.

H.2. Etiology

The cause of the infection in cattle is two species of anaerobic bacteria (bacteria that can grow without oxygen), *Fusobacterium necrophorum* and *Bacteroides melaninogenicus*. Both bacteria are common to the environment in which cattle live, and *Fusobacterium* is present in the rumen and fecal matter of the cattle. This is one of the reasons foot rot is such a major problem in the summer. Footrot is easily identifiable by its appearance and foul odor. *Fusobacterium necrophorum* and *Dichelobacter nodosus*, are the cause of the disease in sheep.

H.3. Epidemiology

The infected animals can serve as the source of infection for the whole herd because they will spread the bacteria throughout the environment. The bacteria can live without a host for up to seven days. Once another animal gets a cut or crack in the soft tissue between its toes, the bacteria can infect the animal.

H.4. Pathogenesis

In sheep, the *Fusobacterium necrophorum* first invades the interdigital skin following damage to the skin, and causes interdigital lesions and slight inflammation. The second stage of the disease is marked by the invasion of the foot by the foot rot bacterium *Dichelobacter nodosus*, a gram-negative anaerobe. Usually, there is an injury to the skin between the hooves that allows the bacteria to infect the animal. Another cause of foot rot may be high temperatures or humidity, causing the skin between the hooves to crack and let the bacteria infect the foot.

H.5. Symptoms and lesions

Picture 10: Lesions of hoof infection of cattle affected by Footrot

Regular monitoring of herd may help to discover clinical cases:

- The first sign of a foot rot infection is when the skin between the claws of the hoof begins to swell.
- Swelling usually appears 24 hours after infection. The skin between the toes may be very red and tender and the toes may separate because of all the swelling. This is very painful to the animal and can cause lameness.
- The animal may also have a raised body temperature.
- A crack can develop along the infected part and is yellow in color.
- The foot will have a foul odor.
- Tendons and joints in the area can become infected, which is much harder to treat.
- Grazing on knees and reluctant to walk;
- Holding limbs above the ground;

In chronic form:

- Loss of body condition;
- Swollen painful hoof, making animal go lame;
- Parts of hoof may contain pus and smell rotten;

H.6. Diagnosis

The diagnosis can be based on clinical signs.

H.7. Treatment

The best way to treat foot rot is to catch it as early as possible.

- The infected animals should be separated away from the herd as soon as possible to prevent the infection from spreading and allow the animal a better environment for healing.
- The first treatment is to clean the foot thoroughly and examine the foot to determine it is definitely foot rot causing the infection.
- Keeping the wound clean and using an antibiotic ointment may help reduce the spread of infection.
- Foot rot is usually treated with an antimicrobial product. Penicillin, tetracycline, and other antibacterial medicines are often used to treat normal cases of foot rot.
- It is critical to closely monitor the animals to make sure they are responding to treatment.

- The infected animals should be kept dry until the healing has occurred. If the animal is showing no signs of recovery after three to four days, the bacteria could have infected the other tissues of the foot. Infusing antibiotic into the veins of the foot may be an effective way to treat those cases.
- Claw amputation and in very severe cases, euthanasia, may also have to be considered.

H.8. Prevention

Infected animals must be kept away from the rest.

- A good way to prevent foot rot is to keep any foreign objects that may cut or damage the foot out of the environment. This should be a practice regardless of whether a herd has foot rot or not. The cuts are what allow the bacteria to enter the foot tissue and cause the infection.
- Some cattle feeders add zinc to the feed mixes and may vaccinate the animals for foot rot. Zinc is important to maintaining the skin and hooves of cattle. Cattle deficient in zinc will become infected more easily than cattle with adequate zinc in their diets.
- Regular footbathing may help reduce the incidence of foot rot in a herd. The bath usually contains copper sulphate or formalin.
- Vaccines have been developed but their efficacy is questionable and the immunity they provide is of short duration.

I. Omphalitis

I.1. Definition

Omphalitis or navel ill is an inflammation of the ombilicus and surrounding parts. Omphalitis is a disease of young calves, usually less than one week of age. It occurs as a result of infection entering via the umbilical cord at, or soon after, birth. This infection can result in a range of signs depending on where the bacteria spread to.

I.2. Etiology

Bacteria that have been associated with omphalitis are mainly gram-positive and include staphylococci, streptococci, *Corynebacterium* spp, *Actinomyces* spp, and *Erysipelothrix rhusiopathiae*, as well as gram-negative coliforms.

I.3. Clinical Signs

Picture 11: Swollen navel of a cattle affected by Omphalitis

If infection stays mostly confined to the navel, the primary sign is a swollen, painful navel that does not dry up. An abscess may develop from which pus (often like a thick custard) may burst; The calf may have a high temperature and reduced appetite.

I.4. Diagnosis

The diagnosis of omphalitis is usually based on the clinical signs.

- If a swollen navel is the main sign, ensure that it is not a hernia before treating
- All calves that die suddenly should be have a post-mortem examination.

I.5. Treatment

Early prompt treatment is important as early treatment is much more effective. Separate the infected animals and isolate them.

- Antibiotics and painkillers are effective in most mild cases. Antibiotic treatment should continue until after the signs have disappeared (which can take over a week even in mild cases)
- Severe cases may not recover even with prolonged antibiotic treatment
- For large navel abscesses, veterinary intervention to drain and remove the infected tissue is often necessary.

I.6. Prevention

Prevention is the key to this disease. Ensuring that the cow calves in a clean environment will significantly reduce the risk of omphalitis (and many other diseases such as toxic mastitis and metritis).

- Proper planning and preparation can prevent the build-up of disease that occurs in too many calving areas.
- Applying a disinfectant (such as iodine) to the navel can reduce the risk of bacteria entering via the navel, but it is no substitute for good hygiene. No amount of disinfectant can overcome being born in a dirty wet yard. Because of the anatomy, bulls navels tend to dry slower than heifers and they

are thus at more risk of navel ill. Applying disinfectant two or three times to bulls can reduce the risk.

- It is also important to ensure that if cattle are born in a nice clean environment that they aren't moved to other pens or contaminated pastures until the navel has dried completely.
- Finally, like all diseases of young calves getting sufficient colostrum is essential. Ensure that all calves get a good suck in the first 6 hours of birth. If this doesn't happen ensure that they get at least 2 litres of colostrum as soon as possible. Colostrum works best if calves take it from birth
- Environmental bacteria gain entry to the neonate via the umbilical cord. Predisposing factors include lack of routine asepsis of the umbilical cord; poor sanitation in the living pens.

J. Joint ill

J.1. Definition

Joint ill is a disease of young calves (but kids and lambs are also susceptible), usually less than one week of age. It occurs as a result of infection entering via the umbilical cord at, or soon after, birth. This infection can result in a range of signs depending on where the bacteria spread to.

J.2. Etiology

Bacteria that have been associated with joint ill are mainly gram-positive and include staphylococci, streptococci, *Corynebacterium* spp, *Actinomyces* spp, and *Erysipelothrix rhusiopathiae*, as well as gram-negative coliforms; most of them reach articulations following untreated omphalitis.

J.3. Clinical Signs

Picture 12: Inflammation of joints

Joint ill is characterized by the following signs:

- With joint-ill, more than one joint is hot, swollen, stiff and painful.

- Often, the affected limb(s) cannot bear weight, and kids with more than one leg affected may be unable to stand. The more commonly affected joints include the carpus, shoulder, hock, and stifle.
- If infection spreads from the navel, or navel ill is not treated, further signs will develop as bacteria spread via the bloodstream and settle in other parts of the body.
- Temperature will be raised while the bacteria spread but by the time the disease is noted it may be normal
- Loss of appetite and depression
- Usually only a few calves in a batch are infected though outbreaks can occur where hygiene is very poor.
- Other sites where bacteria can settle include the eyes, around the heart and the brain.
- Death is common in the latter cases.
- In some calves infection spreads from the navel to the liver causing a liver abscess. In this case problems may not be noted until the calves are older (1 -3 months).

J.4. Diagnosis

The diagnosis of joint ill is usually based on the clinical signs.

Swollen stiff painful (often hot) joints in the presence of navel ill suggests joint ill.

J.5. Treatment

Early prompt treatment is important as early treatment is much more effective.

- Separate the infected animals and isolate them.
- Antibiotics and painkillers are effective in most mild cases. Antibiotic treatment should continue until after the signs have disappeared (which can take over a week even in mild cases)
- Severe cases may not recover even with prolonged antibiotic treatment

J.6. Prevention

The prevention relies mostly on hygiene and sanitation;

- Ensuring that the cow and calves are in a clean environment will significantly reduce the risk of joint ill (and many other diseases such as toxic mastitis and metritis).
- Proper planning and preparation can prevent the build-up of disease that occurs in too many calving areas.

- Applying a disinfectant (such as iodine) to the navel can reduce the risk of bacteria entering via the navel, but it is no substitute for good hygiene.
- No amount of disinfectant can overcome being born in a dirty wet yard. Because of the anatomy, bulls navels tend to dry slower than heifers and they are thus at more risk of navel ill. Applying disinfectant two or three times to bulls can reduce the risk.
- It is also important to ensure that if cattle are born in a nice clean environment that they aren't moved to other pens or contaminated pastures until the navel has dried completely.

K. Erysipelas

K.1. Definition

Erysipelas is an infectious disease of swine, turkeys and sheep. It is caused by *Erysipelothrix rhusiopathae*, it is charactrised by localized skin infections.

K.2. Etiology

Erysipelothrix rhusiopathiae is a non-motile, gram-positive, facultative anaerobe, resistant to high salt concentrations and grows in the temperature range 5°C to 42°C and in the pH range of 6.7 to 9.2.

Erysipelothrix rhusiopathiae causes erysipelas in pigs and turkeys worldwide. Sheep and other domestic animals are occasionally infected. The bacterium also causes erysipeloid, a localized cellulitis, in humans.

K.3. Epidemiology

Susceptibility

Infections with *E. rhusiopathiae* are encountered in pigs, turkeys and sheep. In addition, several major outbreaks of disease have been reported in poultry. The bacterium has also been isolated from sheep, cattle, horses, dogs, cats and poultry and from 50 species of wild mammals and over 30 species of wild birds.

Transmission

It is claimed that up to 50% of healthy pigs harbour *E. rhusiopathiae* in tonsillar tissues. Carrier pigs excrete the organism in faeces and in oronasal secretions. Although soil and surface water can become contaminated with *E.rhusiopathiae*, survival time in soil probably does not exceed 35 days under optimal conditions. The bacterium is often present in the slime layer of fish, a potential source of human infection.

K.4. Pathogenesis

Infection is usually acquired by ingestion of material contaminated by pig faeces. Entry may occur through the tonsils, skin or mucous membranes.

Virulence factors include a capsule which protects the organism against phagocytosis by macrophages and neutrophils and appears to protect the organism within macrophages, allowing intracellular replication.

The ability to produce neuraminidase is correlated to virulence; this enzyme plays a major role in adherence of the organism to endothelial cells and subsequent invasion of these cells.

Other factors that contribute to the pathogenicity of *E. rhusiopathiae* include surface proteins which aid adherence to tissues and biofilm formation. A hyaluronidase enzyme is important in dissemination of the bacteria within tissues.

In the septicaemic form of the disease, vascular damage is characterized by swelling of endothelial cells, adherence of monocytes to vascular walls and widespread hyaline microthrombus formation.

Localization of the bacteria in joint synovia and on heart valves during haematogenous spread accounts for the development of chronic lesions at these sites.

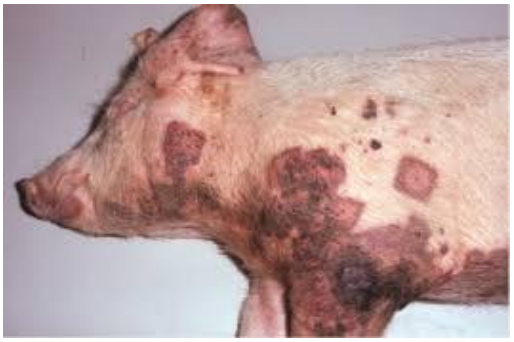
Long-term articular damagemay result from an immune response to persistent bacterial antigens. Viable *E. rhusiopathiae* are rarely isolated from chronically affected joints.

Swine Erysipelas

Subclinically infected carrier pigs are the main reservoir of infection. Pigs with acute disease excrete large numbers of organisms in faeces. Organisms are also excreted in urine, saliva and nasal discharges. Infection is usually acquired through ingestion of contaminated food or water and less commonly through minor skin abrasions. In pigs kept outdoors, repeated faecal contamination of the soil occurs and may represent a source of infection.

The susceptibility of individual pigs and the virulence of the *E. rhusiopathiae* strain, both of which are highly variable, determine the course and outcome of infection. Pigs under 3 months of age are normally protected by maternally-derived antibodies while animals over 3 years of age usually have acquired a protective active immunity through exposure to strains of low virulence. Stress factors appear to be important in the occurrence of disease, with acute cases frequently observed following sudden changes in weather, transport or weaning.

K.5. Clinical signs and lesions



Picture 13: Cutaneous diamond lesions on the skin of pig affected by Erysipelas

Swine erysipelas can occur in four forms. The septicaemic and cutaneous ('diamond') forms are acute while arthritis and vegetative endocarditis are chronic forms of the disease. Chronic arthritis has the most significant negative impact on productivity.

Septicaemia occurs after an incubation period of 2 to 3 days.

- During an outbreak of acute disease, some pigs may be found dead and others are febrile, depressed and walk with a stiff, stilted gait or remain recumbent.
- Mortality may be high in some outbreaks.
- Pregnant sows with the septicaemic form of disease may abort.
- In the diamond-skin form, systemic signs are less severe and mortality rates are much lower than in animals with septicaemia. Pigs are febrile, and cutaneous lesions progress from small, light pink or purple, raised areas to more extensive and characteristic diamond-shaped erythematous plaques. Some of these lesions resolve within a week; others become necrotic and may slough.
- Arthritis, which is commonly encountered in older pigs, can present as stiffness, lameness or reluctance to bear weight on affected limbs. Joint lesions, which may be initially mild, can progress to erosion of articular cartilage with eventual fibrosis and ankylosis. *Erysipelothrix rhusiopathiae* is one of the most frequently detected causes of non-suppurative arthritis in pigs at slaughter.
- In vegetative endocarditis, the least common form, wart-like thrombotic masses are present, usually on the mitral valves. Many affected animals are asymptomatic but some may develop congestive heart failure or die suddenly if stressed by physical exertion or by pregnancy.

K.6. Diagnosis

- Diamond-shaped skin lesions are pathognomonic;
- Specimens for laboratory examination include blood for haemoculture and post-mortem specimens of liver, spleen, heart valves or synovial tissues. Organisms are rarely recovered from skin lesions or chronically affected joints.
- Microscopic examination of specimens from acutely affected animals may reveal slender gram-positive rods.
- Filamentous forms may be demonstrable in smears from chronic valvular lesions.
- Blood agar and MacConkey agar plates inoculated with specimen material are incubated aerobically at 37°C for 24 to 48 hours.

K.7. Treatment

Both penicillin and tetracyclines are effective for treatment although resistance to tetracyclines has been reported in some countries. When chronic lesions have developed, antibiotic therapy is ineffective.

K.8. Prevention

Hygiene and management practices should be evaluated and, where necessary, brought to a satisfactory standard.

- Chronically affected animals should be culled.
- Affected pigs should be isolated.
- Both live attenuated and inactivated vaccines are available. Attenuated vaccines can be given orally, systemically or by aerosol. They should not be administered to animals receiving antibiotic therapy. However, use of attenuated vaccines may be associated with an increase in the chronic arthritic form of erysipelas.

L. Pasteurellosis

L.1. Definition

Synonyms: Fowl Cholera, Avian Cholera, avian pasteurellosis, avian hemorrhagic septicemia.
Acute septicemic disease of somestic fowls and wild birds caused by *Pasteurella multocida* characterized by high morbidity and mortality.

L.2. Etiology

Pasteurella multocida is small, non-motile, gram-negative, ovoid or elongated rod, encapsulated. The organism survives several months in soil, litter as decayed matters, but is killed by disinfectants.

L.3. Epidemilogy

Chicken, turkey and ducks are most commonly affected. However, domestic fowls, game birds, cage birds and wild birds are susceptible. Fowl cholera occurs sporadically or enzootically in most countries of the world. It is a serious problem in turkey and chicken.

- The mode of transmission among water fowl is unknown.
- In domestic Fowls, "healthy" carriers provide a source of infection.
- The natural spread of the disease among domestic fowl is believed to be by ingestion, inhalation and transmitted by vectors mechanically.

L.4. Pathogenesis

After ingestion or inhalation, *P. multocida* in the hosts are probably protected by the capsule (a highly polysaccharide located externally and adherent to the bacterial cell wall) against phagostosis thus multiplication and production of toxin is possible.Signs and outcome of the disease are attributed to endotoxemia.

L.5. Symptoms and lesions

Picture 14: Cyanotic combs and wattles on chicken affected by Pasteurellosis

Peracute form: Fowl died suddenly

Acute form:

- Birds may live for a day or longer;
- Greenish and yellowish diarrhea. Thirst, increased water consumption, anorexia.
- Fever, 100-112°F.

- Mucous in mouth and nostrils.
- Cyanotic combs and wattles. A sticky mucous in the mouth and nasal passage may be found.
- Reddish discoloration of the skin and breast muscles is a common but not constant finding. In general the abdominal organs appear congested and darken.
- Hemorrhage of various sizes is found on the heart muscles, particularly around the coronary groove, abdominal subcutaneous tissue and gizzard fat.
- The pericardial sac often contains an excess of yellowish fluid.
- Liver, either very dark or lighter than usual, its surface may be spotted with many white necrotic foci.
- Severe inflammation and hemorrhage in the lining of the duodenum are lesions frequently present. In lower section a yellow sticky material is found.
- Lung, consolidation and congestion with numerous small hemorrhages.
- Cheesy, yellowish deposits are often observed in various parts of the body, especially on the membrane of the air sacs and the intestine.

Chronic form:

Longer for weeks or months and act as carriers.

- Swollen wattles and eyes.
- Inflammation of joints, tendon sheath of the legs or wings.
- Torticollis
- Dried cheesy, yellow yolk material is found free in the abdominal cavity or adherent to some organ, ruptured yolk.
- Hemorrhages of the ovary, soft, flabby, irregular in outline and pedunculated ova, greenish colored ovum is observed occasionally.
- Salpingitis.
- Caseous, swollen wattles and joints.
- Supportive meningitis as a result of the organism localized at the base of the skull and ear.

L.6. Diagnosis

Clinical diagnosis

- Fever 110-112 ^{0}F,
- Cyanotic comb,
- Swollen and enlarged wattles and joints,
- Greenish diarrhea, these symptoms can lead to suspicion of the disease.

Laboratory diagnosis

- Finding bipolar characteristic organisms in Giemsa stained smear of circulating blood.
- Isolation and characterization of bacteria from circulating blood liver or other viscera.
- Whole blood strained antigen agglutination test.
- Laboratory animal inoculation, chicks and mice.

L.7. Treatment

Sulfonamides especially sulfaquinoxalene (SQ); sulfamenthazine, in the feed at 0.33% level for 14 days.Teramycin in water or feed is also effective.

L.8. Prevention

- Water sanitation;

- Good management would help;
- Control flies and rodents;
- Vaccination at 12-16 weeks for placement flocks in problems area and repeat 4-8 weeks later, subcutaneous injection of autogenous bacteria in the neck or thighs. Action of immunity develops within 4-5 days, length of immunity is about 6 months.

M. Salmonellosis

M.1. Pullorum Disease

M.1.1. Definition

Synonym: Bacillary White Diarrhea (BWD) Pullorum disease is primarily an acute disease of chicks during first month of lives, characterized by high mortality. It is often found in mature fowl as a chronic infection.

M.1.2. Etiology

Salmonella pullorum, isolated by Rettger in 1900, he described this disease as a "Fatal septicemia of young chicks".This bacteria is a long slender gram-negative rod. Resistant to cold, sunlight, drying and disinfectants, nonmotile, non-sporogenic and faculatively anaerobic.

M.1.3. Epidemiology

Distribution

Worldwide distribution. Pullorum is virtually eradicated in poultry in the U.S. since National Poultry Improvement Plan (NPIP) was organized in 1940 for the purpose of controlling pullorum disease**.**

Transmission

The most important source of infection is the infective egg laid by carrier hen. Mode of dissemination of pullorum disease is described below:

- Infected hen;
- Infected egg laid by infected hen;
- Incubator containing infected egg;
- Chick box in which infected chicks may be present;

- Brooder;
- Surviving infected pullet which may be a carrier.

Susceptibility

Chickens and turkeys are most susceptible. However, natural infections have been reported in pheasants, ducks, sparrows, guinea fowl, quail, canaries and pigeons. Among mammals, rabbits appear to be readily susceptible, and infection has been reported as occurring in hogs, foxes, mink and man.

M.1.4. Symptoms and lesions

Symptoms

Incubation period of 5 - 7 days; usually chicks under 3 weeks of age are more affected;

- Peracute type, no detectable symptoms, affected animals die suddenly;
- It takes acute form in baby chicks, and chronic form in mature fowls.
- Mortality: If chicks are hatched from infective eggs, dead and sick chicks may be observed in the hatchery.
- Those infected after hatching, mortality reach a peak at 7 - 10 days. Mortality as high as 30 - 40% has been recorded.
- Affected birds huddle near a heat source, are anorectic, appear weak and have whitish fecal pasting around the vent diarrhea; some of eggs laid by such hens hatch and produced progeny.
- Droopness, unthriftness and stunted growth, sleepy eyes are present;
- Difficult breathing (pneumonia).
- Swelling of joints - arthritis, more common in hens (chronic).

Lesions

Peracute form - no lesions.

Subacute form:

- Enlarged and congested liver. The normal yellow color may be streaked with hemorrhage.
- Necrotic foci may be present in the cardiac muscle, liver, lungs, ceca, large intestine and muscles of the gizzard.
- Unabsorbed yolk sacs ;

- Firm, cheesy material in ceca;
- Pericarditis and epicarditis
- Pneumonia - firm grayish nodules.
- Classic gray nodules in the liver, lungs, heart, gizzard and ceca.

Chronic Form - Adult fowl

- Adult carriers usually have no gross lesions but may have nodulae pericarditis, fibrous peritonitis, atrophic regressing ovarian follicles with caseous contents
- Oval and shrunken misshaped, greenish or leaden-colored egg yolk.
- Salpingitis and arthritis.

M.1.5. Diagnosis

By history, age, symptom.

Laboratory diagnosis:

- Definitive diagnosis: Isolation and identification of Salmonella pullorum bacteria.
- Tissue samples from liver, spleen, heart, and yolk sac are good source for bacterial culture in septicemic cases.

In the case of chronic infection or carrier, serological tests are required.

M.1.6. Treatment

No drug or combination of drugs has been found capable to eliminate infection, thus it is no substitute for eradication program. If chicks showed symptoms of pullorum increase brooder temperature to 100-105 F.

- Furazolidone is the drug of choice, it is administered in feed at the level of 0.04 percent for 10 days for treatment and 0.01-0.015 per cent continuously for prevention, or 200 grams/ton feed.
- Sulfonamides: Sulfadiazine and sulfamerazine are very effective in reducing mortality.

M.1.6. Control and Prevention

Any plan for controlling pullorum disease must its principle objective the elimination of the most prolific and constant source of infection, the carrier hen or turkey.

Procure eggs and chicks only from known pullorum free flocks.

- The hatchery should be dust free, cleaned after each hatch.
- Fumigate between hatches with KMN04 method.
- Fumigate at 18 days of incubation (chicken)
- 25 days of incubation (turkey)
- Length - 1-3 hours
- Also pre-incubation fumigation may be applied.

M.2. Fowl typhoid

M.2.1. Definition

Synonyms – Typhoid. It is a septiemic disease of domesticated birds. Characterized by an acute and chronic phase, enlarged spleen, colored liver, and diarrhea with pasting around the vent.

M.2.2. Etiology

Salmonella gallinarum is closely related to Salmonella.

M.2.3. Epidemiology

Distribution

The disease is worldwide spread in the poultry producing area of the country, but out breaks is sporadic, depending on the season, environment, and control methods. Incidence is low in USA, Canada, and some Eurpean countries; but is much higher in other countries.

Tranmission

Similar to pullorum disease. Transmission through eggs, carrier hens, flies also play an important role. Discharge from nose, mouth and bowl of infected birds are infectious.

Susceptibility

Young adult chickens are very susceptible 2 weeks guineas and a few other species are also susceptible.

M.2.4. Symptoms and lesions

It takes acute to chronic forms and signs and lesions depend on the form.

- Incubation period -four to five days, varies considerably with the virulence of the organism.
- Course of disease about five days;
- High fever of about 110-112°F;
- In acute cases there is sudden death.
- Loss of appetite, restless, ruffled feathers, increased thirst, unthriftiness, greenish diarrhea.
- Respiratory signs may include:difficult breathing,couthing accompanied by nasal discharge;
- Swolling of joints which can lead to lamness;
- Mortality depends on the resistance of birds may run as high as 75% in untreated flocks.
- Swollen and cyanotic comb and Wattles

Post-mortem lesions

- Peracute type - no lesions;
- Liver: enlarged 2-3 times, bronze color, necrotic foci is evident in subacute cases.
- Spleen: enlarged (2-3 times of normal size), mottle, and sometimes hemorrhages.
- Kidney: enlarged, hard, swollen and congested.
- Oviduct: appeared cook, due to high fever,
- Ova: hemorrhagic.

Misshapen and discoloration

- Heart - necrotic foci in myocardium and pericardium.
- Intestine -catarrhal inflammation.
- In young chicks, grayish-white foci may sometimes be observed in the lungs, heart, and gizzard.

M.2.5. Diagnosis

There are similarities with pullorum, fowl cholera, fowl plague.

Differential diagnosis:

- The marked congested and enlarged organs are not found in pullorum disease, fowl cholera or fowl plague (Avian influenza).
- The bronze colored liver is very diagnostic.

Laboratory diagnosis:

- Cultivation and identification of the organism
- Serological identification.
- *Salmonella pullorum* and gallinarum are serologically undifferentiated. However*, S. gallinarum* ferments maltose, most strains of *S. pullorum* does not attract this sugar. *S. gallinarum* acidifies dulsitol*. S. gallinarum* is anaerogenic, *but S. pullorum* is aerogenic.

M.2.6. Treatment

Among the drugs used to treat fowl typhoid disease are furazolidone, gentamycin sulfate, and sulfa drugs (sulfadimethoxine, sulfamethazine, and sulfamerazine.

M.2.7. Prevention

Start with Salmonella clean flocks, blood test breeders. Sanitation and immunization with killed fowl typhoid bacteria, not effective.

N. Leptospirosis

N.1. Definition

Leptospirosis is a disease caused by bacteria of genus leptospira that affect humans as well as other animals including mammals, amphibians and reptiles.

N.2. Etiology

Leptospirae are coiled spiral organisms and typical cells have a hook at each end making them S-or C-shaped; they are gram-negative.

N.3. Epidemiology

Rodents are the most frequent leptospiral carriers with wild carnivores ranking second. They have been isolated from birds, reptiles, amphibians and invertebrates the epidemiology of such associations is not established. No mammal can be excluded as possible host.

Exposure is through contact of mucous membranes or skin with urine-contaminated water, fomites, or feed. Other sources are milk from infected cow and genital secretion from cattle and swine of either sex.

Leptospirosis is perpetuated by the many tolerant hosts and the protracted shedder state. Indirect exposure depends on mild and wet conditions, which favour environmental survival of leptospirae. More direct transfer occurs by urine aerosols in milking barns, cattle sheds, or canine courting habits, which may explain the male bias of canine leptospirosis.

Contaminated bodies of water are important sources of infection to livestock, aquatic mammals, and humans. Animal handlers, sewer workers, field hands, miners, and veterinarians are at increased risk of exposure.

N.4. Pathogenesis

The leptospira enter the bloodstream subsequent to mucous membrane or reproductive inoculation colonizing particularly liver and kidney where they produce degenerative changes. Other affected organs may be muscles, eyes, and meninges, where non suppurative meningitis may develop. Hemorrhages results from damaged vascular endothelium. Leptospira serovar Pomona in causes intravascular hemolysis due to hemolytic exotoxin. Secondary changes include icterus due to liver damage and blood destruction, and acute, subacute or chronic nephritis due to renal tubular injury.

In surviving animals, leptospirae are removed from circulation with the appearance of antibody but persists in the kidney for many weeks.

N.5. Symptoms and lesions

Canine

The serovars involved are icterohemorrhagiae and canicola, grippotyphosa, Pomona and blatislava.

The most acute form affects pups producing fever, hemorrhages often apparent antemortem on mucous membranes and skin or manifested by epistaxis or blood stained feces and vomitus.

Icteric type: it may be acute and rapidly fatal with signs of gastrointestinal upsets, uremic breath, and ulceration in the anterior alimentary tract or it may run a slow course with delayed onset.

Cattle

Abortion usually late term but it may occurs at any time following infection, abortion is due to foetal death. The infection may be subclinical and it is marked by milk–drop syndrome, reproductive failure and infertility;

Acute leptospirosis affect mostly calves and sometimes adult cattle and it is marked by:

- Fever;
- Haemoglobunuria;
- Icterus;
- Anaemia;
- Fatality rate of 5% to 10%

Swine

- Icterus and haemorrhage occur especially in piglets
- Abortion and infertility are the manifestations in sow.

N.6. Diagnosis

Diagnosis of leptospirosis depends on a good clinical and vaccination history and laboratory testing.

- Routine dark field microscopy should be limited to urine;
- Fluorescent antibody used on fluids, tissue section, organ impression on aborted foetuses.
- Isolation and identification; cultures (sample may be blood, serum or fresh kidney biopsy) are examined microscopically.
- The gold standard in diagnosis of leptospirosis is MAT (Microscopic agglutination test).

N.7. Treatment and prevention

Leptospirae are susceptible to penicillin, tetracycline, chloramphenicol, streptomycin, and erythromycin. Streptomycin or dihydrostreptomycin is routinely used to eliminate the carrier state in animals. However, sustained leptospiral infection in the kidneys and reproductive tracts of cattle subsequent to antibiotic treatment is not uncommon, again suggesting the presence of leptospiral biofilm formation.
Vaccination generally prevents disease. It prevents neither infection nor shedding.

O. Mastitis

Mastitis in dairy cows

O.1. Definition

Mastitis is the inflammation of the mammary gland and udder tissue, and is a major endemic disease of dairy cattle. It usually occurs as an immune response to bacterial invasion of the teat canal by variety of bacterial sources present on the farm, and can also occur as a result of chemical, mechanical, or thermal injury to the cow's udder.

Milk-secreting tissues and various ducts throughout the udder can be damaged by bacterial toxins, and sometimes permanent damage to the udder occurs. Severe acute cases can be fatal, but even in cows that recover there may be consequences for the rest of the lactation and subsequent lactations.

The illness is in most respects a very complex disease, affected by a variety of factors: it can be present in a herd **subclinically**, where few, if any, symptoms are present in most cows.

O.2. Etiology

Bacterial cells of *Staphylococcus aureus*, one of the causal agents of mastitis in dairy cows. Its large capsule protects the organism from attack by the cow's immunological defense.

Bacteria that are known to cause mastitis include:

- Members of **enterobacteriaceae** family: *Enterobacter aerogenes*, *Escherichia coli, (E. coli)*, *Klebsiella pneumonia*, *Klebsiella oxytoca, Proteus spp.*
- Members of **pasteurellaceae**: *Pasteurella species*
- Members of **staphylococcus** genus: *Staphylococcus aureus*, *Staphylococcus epidermidis*
- Members of **streptococcus** genus: *Streptococcus agalactiae*, *Streptococcus uberis*
- Members of **Brucella** genus: *Brucella melitensis*
- Members of **achlorophillic** algae: *Prototheca zopfii*, *Prototheca wickerhamii*
- Members of **pseudomonas** genus: *Pseudomonas aeruginosa*
- Members of ***Arcanobacterium*** *genus*: *Trueperella pyogenes* (previously *Arcanobacterium pyogenes*)
- Members of **Corynebacterium** genus: *Corynebacterium bovis*
- Members of **Mycoplasma** (various species)

Types of mastitis and predisposing factors

Mastitis may be classified according two different criteria: either according to the clinical symptoms or depending on the mode of transmission.

Clinical symptoms

- Clinical mastitis
- Sub-Clinical mastitis

Mode of transmission

- Contagious mastitis
- environmental mastitis

Predisposing factors includes:

- Climatic conditions and seasonal variation, bedding and hygiene of environment;
- Bad hygiene of milker's hands and cloths.
- Constant running of water over the floor of a milking shed;
- Lack of footbath with disinfectant where machine milking is practiced
- Lack of normal milk room hygiene including washing of containers and equipment.
- Insufficient hygiene on the body of animals or wounds on external surface of teats;
- Incomplete milking process.

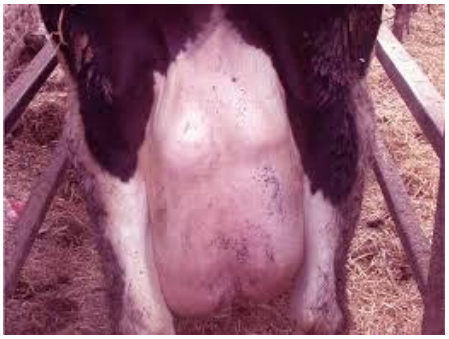
O.3. Transmission

Mastitis is most often transmitted by contact with the milking machine, and through contaminated hands or materials in housing, bedding and other equipment.

O.4. Pathogenesis

Mastitis occurs when white blood cells (leukocytes), are released into the mammary gland, usually in response to an invasion of bacteria of the teat canal. Milk-secreting tissue, and various ducts throughout the mammary gland are damaged due to toxins by the bacteria. Mastitis can also occur as a result of chemical, mechanical, or thermal injury. The udder sac is hard, tight, and firm.

O.4. Clinical signs




Picture 15: Inflamed mammary gland and milk with pus flakes in cattle affected with Mastitis

This disease can be identified by abnormalities in the udder such as swelling, heat, redness, hardness or pain if it is clinical. Other indications of mastitis may be abnormalities in milk such as a watery appearance, flakes, or clots, milk production decreases. When infected with subclinical mastitis, a cow does not show any visible signs of infection.

O.5. Diagnosis

The diagnosis may be based on clinical symptoms in clinical case; using CMT test.

O.6. Treatment

Treatment is possible with long-acting antibiotics, but milk from such cows is not marketable until drug residues have left the cow's system.

Antibiotics may be systemic (injected into the body), or they may be forced upwards into the teat through the teat canal (intramammary infusion). Cows being treated may be marked with tape to alert dairy workers, and their milk is syphoned off and discarded. Vaccinations for mastitis do exist, but as they only reduce the severity of the condition, and do not prevent new infection they should be used in conjunction with a mastitis prevention program.

O.7. Prevention

A good milking routine is vital. This usually consists of applying a pre-milking teat dip or spray, such as an iodine spray, and wiping teats dry prior to milking. The milking machine is then applied.

- After milking, the teats can be cleaned again to remove any growth medium for bacteria. A post milking product such as iodine-propylene glycol dip is used as a disinfectant and a barrier between the open teat and the bacteria in the air.

- Mastitis can occur after milking because the teat holes close after 15 minutes if the animal sits in a dirty place with dung and urine.
- Practices such as good nutrition, proper milking hygiene, and the culling of chronically infected cows can help. Ensuring that cows have clean, dry bedding decreases the risk of infection and transmission.
- Dairy workers should wear gloves while milking, and machines should be cleaned regularly to decrease the incidence of transmission.
- Practices such as close attention to milking hygiene, the culling of chronically-infected cows, good housing management and effective dairy cattle nutrition to promote good cow health are essential in helping to control herd mastitis levels.

P. Metritis

P.1. Definition

Metritis is inflammation of the wall of the uterus, while endometritis is inflammation of the functional lining of the uterus, called the endometrium . Amongst domestic animals, metritis and endometritis are most common in cattle after parturition, and the diseases are often called postpartum metritis or postpartum endometritis.

P.2. Etiology

The most common bacteria that cause postpartum metritis and endometritis in cattle are *Escherichia coli*, *Trueperella* (previously *Arcanobacterium*) *pyogenes* and anaerobic bacteria such as *Prevotella* species and *Fusobacterium necrophorum*. "Several specific diseases are associated with metritis or, cendometritis. These include brucellosis, leptospirosisampylobacteriosis, and trichomoniasis".Susceptible animals may include: cows, mares, ewes, does, or sows and others.

P.3. Clinical signs

Metritis occurs within 21 days and is most common within 10 days of parturition. Metritis is characterized by an enlarged uterus and a watery red-brown fluid to viscous off-white purulent uterine discharge, which often has a fetid odour. The severity of disease is categorized by the signs of health:

- Grade 1 metritis: An abnormally enlarged uterus and a purulent uterine discharge without any systemic signs of ill health.

- Grade 2 metritis: Animals with additional signs of systemic illness such as decreased milk yield, dullness, and fever >39.5°C.
- Grade 3 metritis: Animals with signs of toxemia such as inappetence, cold extremities, depression, and/or collapse.

Clinical endometritis is defined in cattle as the presence of a purulent uterine discharge detectable in the vagina 21 days or more postpartum.

Subclinical endometritis is characterized by inflammation of the endometrium and the presence of neutrophils in cytology or biopsy histology, in the absence of signs of clinical endometritis.

Metritis in large animals

In all species, acute puerperal metritis occurs within the first 10–14 days postpartum. It results from contamination of the reproductive tract at parturition and often, but not invariably, follows complicated parturition.

Acute puerperal metritis responds well to systemic antimicrobial therapy combined, if necessary, with NSAID and other supportive measures such as fluid therapy. Cephalosporin antibiotics or penicillin are considered most appropriate for systemic treatment of cows with metritis because they are active against most common pathogens, reach therapeutic levels in endometrial tissues, and may help prevent some of the potential sequelae of metritis and endometritis, such as endocarditis or renal disease. Oxytetracycline requires administration at high levels (11 mg/kg, twice a day) to maintain uterine tissue concentrations of required for many strains of pathogenic pyogenes.

Drainage of the uterine content may be advantageous but should be attempted only after initiation of antimicrobial therapy; it should be done very carefully because the inflamed uterus may be friable, and manipulation of the uterus may result in bacteremia.

Cows are more resistant to uterine infection during estrus, and as cows undergo more estrous cycles after parturition, the prevalence of endometritis diminishes. This has led to increased use of prostaglandin (PG) $F_{2\alpha}$ or its analogs, at usual luteolytic doses, for the management of endometritis. Another potential advantage of the use of $PGF_{2\alpha}$ or its analogs is stimulation of uterine contraction and expulsion of uterine exudate

Metritis in small animals

Metritis is post-partem infection of the uterus. Predisposing causes include prolonged delivery, dystocia, and retained foetuses or placentas. *Escherichia coli* is the most common bacterium isolated from the infected uterus; streptococci, staphylococci, *Proteus* sp, and others are isolated less frequently.

The primary clinical sign is purulent vulvar discharge. Bitches or queens with metritis are usually depressed, with signs of fever, lethargy, and inappetence, and may neglect their offspring. Pups may become restless and cry incessantly. Metritis should be considered in any postpartum animal with signs of systemic illness or an abnormal vaginal discharge. A large, flaccid uterus may be palpable. Radiographs should be taken to determine whether foetuses or placentas are retained.

Treatment includes stabilization with IV fluids, supportive care, and antibiotic therapy based on culture and sensitivity testing of the vulvar discharge. Prostaglandin $F_{2\alpha}$ (0.1–0.25 mg/kg, SC, for 2–3 days) or oxytocin (in bitches and queens, IM) may help evacuate the uterine contents. Ovario-hysterectomy is recommended after initial stabilization if the animal is extremely ill or if future reproduction is unimportant. Otherwise, it should be considered an elective procedure to be performed when lactation has ceased.

Q. Abscess

Picture 16: Abscess on the mandibular area of the Rabbit

Q.1. Definition

An abscess is a collection of pus that has built up within the tissue of the body. Abscesses are usually caused by an infection. Often many different types of bacteria are involved in a single infection. Localized infections are common in farm animals and many are bacterial infections secondary to traumatic injuries.

Q.2. Etiology

The most common pus-forming abscess are bacteria including:

Arcanobacterium pyogenes, Fusobacterium necrophorum, streptococci and staphylococci. Clostridial infections are common but occur sporadically. They are described under malignant edema.

Q.3. Pathogenesis

The local infection may take the form of a circumscribed aggregation of bacterial debris and necrotic tissue, known as an abscess. This may be firmly walled off by a dense fibrotic capsule or be contiguous with normal tissue.

The species of bacteria in the abscess determines the type of pus present and its odor. Staphylococci produce large quantities of thick yellow pus, streptococci produce less pus and more serous-like exudates. The pus of *Fusobacterium necrophorum* is very foul-smelling and usually accompanied by the presence of gas.

Q.4. Clinical signs

Classical local signs of abscess include:

- redness: the area of redness often extends beyond the swelling
- heat
- Swelling that when pressed feels like it is fluid filled.
- Pain and loss of function.

General clinical signs may include:

- Fever, depression, lack of appetite;
- Weight loss, which can be dramatic in degree and rapidity;
- Obstruction of lymphatic and venous drainage, which can cause local swelling and oedema.
- The presence and location of the local infection can be demonstrated at necropsy.

Q.5. Treatment

Surgical treatment must be combined with antibiotheurapy.

- Surgical drainage of readily accessible intact abscesses is the treatment of choice and in most cases the only effective method of therapy. During the procedure, the surgeon will make a cut (incision) in the abscess, to allow the pus to drain out.
- The site is prepared surgically and the abscess is drained, flushed and topically medicated. If the abscess has not yet pointed with a soft spot, hot fomentations and hydrotherapy may aid in the maturation of a superficial abscess.
- An analgesic may be required during this stage of therapy. Tooth root abscesses require extraction of the affected tooth to effect a cure.
- Antimicrobial agents given parentally can be used for the treatment of deep abscesses not readily accessible to surgical drainage.

- However, antimicrobial agents alone may be ineffective, even if the organism appears sensitive to the drug in vitro in cases where the abscess is surrounded by a dense capsule - presumably the capsule prevents diffusion of the drug into the abscess cavity.
- Lipophilic antibiotics, such as rifampin, florfenicol or macrolides, are theoretically advantageous in penetrating into abscesses. Rifampin should be administered with another antimicrobial agent in order to delay the development of antibiotic resistance.
- Ideally, a sample of the contents of the abscess should be cultured and antimicrobial susceptibility determined.

R. Pneumonia

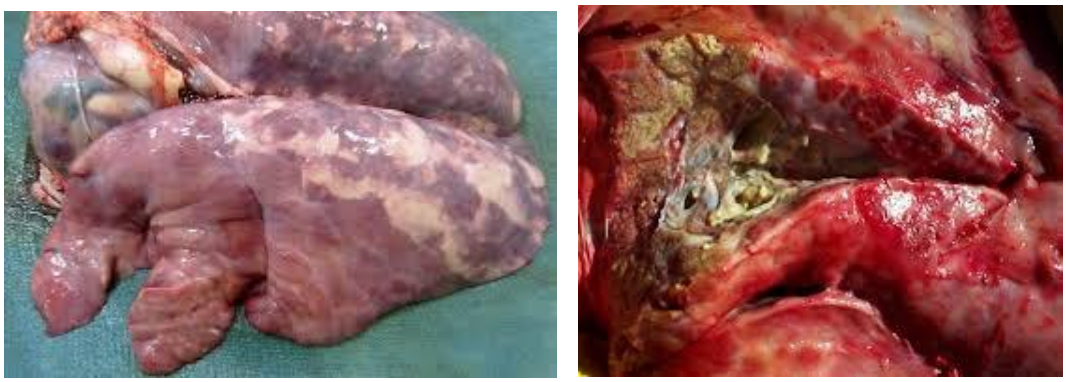
R.1. Definition

Pneumonia is an acute or chronic inflammation of the lungs and bronchi characterized by disturbance in respiration and hypoxemia and complicated by the systemic effects of associated toxins.

R.2. Etiology

The usual cause is primary viral, bacterial or fungal infection of the lower respiratory tract. Contagious bovine pleuropneumonia, tuberculosis, pasteurellosis, Canine distemper virus, parainfluenza virus, and others cause lesions in the distal airways and and thus result in pneumonia by their invasion of the lungs. Parasitic invasion of the bronchi like *Filaroides*, *Aelurostrongylus*, or *Paragonimus* spp may result in pneumonia. Injury to the bronchial mucosa and inhalation or aspiration of irritants may cause pneumonia directly and predispose to secondary bacterial invasion. Aspiration pneumonia may result from persistent vomiting, abnormal esophageal motility, or improperly administered medications (eg, oil or barium) or food (forced feeding); it may also follow suckling in a neonate with a cleft palate.

R.3. Clinical symptoms and lesions



Picture 17: Inflamed lung and Lesion of lung section in case of pneumonia in cattle

The initial signs are usually those of the primary disease**.**

- Lethargy and anorexia are common.
- A deep cough is noted.
- Progressive dyspnea, “blowing” of the lips and cyanosis may be evident, especially on exercise.
- Body temperature is increased moderately, and there may be leukocytosis.
- Auscultation usually reveals consolidation, which may be patchy but more commonly is diffuse.
- In the later stages of pneumonia, the increased lung density and peribronchial consolidation caused by the inflammatory process can be visualized radiographically.
- Complications such as pleuritis, mediastinitis, or invasion by opportunistic organisms may occur.
- Clinical signs of bacterial pneumonia are often proceeded by signs of viral infection of the respiratory tract.

With the onset of bacterial pneumonia clinical signs increase in severity and are characterized by:

- Depression and toxemia;
- Fever
- Rapid shallow respiration rate;
- Auscultation may reveal increased bronchial sound ,crackles and wheezers;
- Pneumonia may become chronic and associated with the formation of pulmonary abcesses.

R.4. Diagnosis

Generally, diagnosis relies on gross necropsy findings and bacteria culture because the bacteria involved are inhabitant in upper respiratory tract, if possible the specimens should be collected from animal that have not been treated with antibiotics.

- Analysis of bronchoalveolar lavage fluid is valuable for the diagnosis of bacterial infections.
- Cytologic examination can demonstrate the animal's immune response and indicate the intracellular or extracellular location of bacteria.
- Bacterial culture and sensitivity testing is required and may include anaerobe and mycoplasma culture, especially in refractory cases.

- A viral etiology generally results in an initial body temperature of 104°–106°F (40°–41°C). Leukopenia, often expected, may not be seen in many viral respiratory infections (eg, canine infectious tracheobronchitis, feline infectious peritonitis pneumonia).
- A history of recent anesthesia or severe vomiting indicates the possibility of aspiration pneumonia.
- Acutely affected animals may die within 24–48 hr of onset. Mycotic pneumonias are usually chronic in nature ;

Miliary nodules seen at necropsy may suggest protozoal pneumonia.

R.5. Treatment

Early recognition by trained personal skilled at detecting the early symptoms of disease and treatment with antibiotics are essential for successful therapy.

- Long-acting antimicrobial such as florfenicol tulathromycin, tilmicosin enrofloxacin are indicated in large animals.
- The animal should be placed in a warm, dry environment**.**
- Anemia, if present, should be corrected.
- If cyanosis is severe, oxygen therapy may be used, administered by means of an oxygen cage, with a concentration of 30%–50%.
- Empirical antimicrobial chemotherapy should be initiated and changed if needed based on results of culture of bronchoalveolar lavage fluid.
- Supportive therapy should be instituted as needed and may include oxygen supplementation, pulmonary physiotherapy (nebulization and coupage), and bronchodilators. If no response is seen after 48–72 hour of therapy, the treatment plan should be reassessed. Antimicrobial chemotherapy should be continued 1 week after clinical and radiographic signs resolve.
- Animals should be reexamined frequently.
- Chest radiographs should be repeated at regular intervals to monitor recurrence or note a primary underlying disease process and to detect complications such as lung consolidation, atelectasis, or abcessation.

S. Infectious Kerato-Conjunctivitis

S.1. Definition

Infectious kerato-conjunctivitis of cattle, sheep, and goats (also called pink-eye or infectious ophtalmia) is the disease characterized by blepharospasm, conjunctivitis, lacrimation, and varying degrees of corneal opacity and ulceration. Infectious bovine kerato-conjunctivitis (IBK) is the most common ocular disease of cattle and is seen worldwide.

S.2. Etiology

The gram-negative rod *Moraxella bovis* is the only organism demonstrated to cause IBK in cattle. Seven different serogroups of *Moraxella bovis* are currently recognized. Most other ocular infections of cattle are characterized by conjunctivitis with minimal or no keratitis.

In sheep and goats, naturally occurring conjunctivitis or kerato-conjunctivitis can be associated with *Chlamydophila pecorum*, *Mycoplasma* spp (notably *M conjunctivae*), *Moraxella ovis*, *Colesiota conjunctivae, Listeria monocytogenes, Acholeplasma oculi,* and *Thelazia* spp.

S.3. Pathogenesis

M. bovis invades the lacrimal and tarsal glands of the eye, causing keratitis, opacity, uveitis, aqueous flare and corneal ulcers.

The bacterium adheres to the cells via its fimbriae and pili proteins, and produces β-haemolysin toxins which lyse the corneal epithelial cells *Moraxella bovis* also secretes cytotoxic toxin and pathogenic fibrinolysin, phosphatase, hyaluronidase and aminopeptidases.

S.4. Clinical symptoms

Picture 18: Mucopurulent ocular discharge in cattle affected by kerato-conjuctivitis

The disease usually is acute and tends to spread rapidly. In all species, young animals are affected most frequently, but animals of any age are susceptible. One or both eyes may be affected.

- The earliest clinical signs are photophobia, blepharospasm, and epiphora;
- Later, the ocular discharge may become mucopurulent;
- Conjunctivitis, with or without varying degrees of keratitis, is usually present. In sheep and goats, concurrent polyarthritis may be present in association with *Chlamydophila pecorum* infections.
- Appetite may be depressed because of ocular discomfort or visual disturbance that results in inability to locate food. The usual clinical course varies from a few days to several weeks.
- Most corneal ulcers in cattle with IBK heal without loss of vision; however, corneal rupture and severe or untreated cases can lead to panophthalmitis and permanent blindness**.**

S.5. Lesions

Lesions vary in severity.

- In cattle, one or more small ulcers typically develop near the center of the cornea. Initially, the cornea around the ulcer is clear, but within a few hours a faint haze appears that subsequently increases in opacity.
- Lesions may regress in the early stages or may continue to progress. After 48–72 hr in severe cases, the entire cornea may become opaque, blinding the animal in that eye.
- Blood vessels may invade the cornea from the limbus and move toward the ulcer at least1 mm/day.
- Corneal opacity may result from edema (hazy white to blue corneas), which is a part of the inflammatory process, or leukocyte infiltration (milky white to yellow corneas), which indicates severe infection.

- Continued active ulceration may cause corneal rupture. Relapse may occur at any stage of recovery.

S.6. Diagnosis

In all species, presumptive diagnosis is based on ocular signs and concurrent systemic disease. It is important to distinguish that the lesions are not due to foreign bodies or parasites. In IBR, upper respiratory signs and conjunctivitis predominate, while keratitis accompanied by ulceration is rare. In bovine malignant catarrhal fever, respiratory signs are prominent with primary uveitis and associated keratitis. Microbial culture may be beneficial in confirming the causative organisms. *Chlamydophila* and *Mycoplasma* spp require special media; the diagnostic laboratory should be consulted before sample collection. Cytologic evaluation of stained slides prepared from conjunctival scrapings of sheep and goats may reveal *Chlamydophila* organisms; however, intracytoplasmic inclusion bodies can be difficult to recognize. PCR analysis can be used to detect *Chlamydophila* and *Mycoplasma* spp.

S.7. Treatment

Because antibiotic susceptibility may vary in different geographic locations, bacterial culture and susceptibility testing is advised.

- *M bovis* is susceptible to many antibiotics. One common treatment is bulbar conjunctival injection with penicillin.
- Currently (in USA) long-acting oxytetracycline (two injections of 20 mg/kg, IM or SC, at a 48- to 72-hr interval) and tulathromycin (2.5 mg/kg, SC, given once) are approved antibiotics to treat IBK in cattle.
- Florfenicol (20 mg/kg,
- IM, two doses at a 2-day interval). A single injection of long-acting oxytetracycline (20 mg/kg, IM) along with oral oxytetracycline (2 g/calf/day for 10 days) fed in alfalfa pellets has also been shown to be effective at reducing severity of IBK during a herd outbreak.
- Topical applications of ophthalmic preparations should be applied at least three times a day to be effective, and thus are often not cost-effective or practical in herd settings. Effective antibiotics for topical ophthalmic use include triple antibiotic, gentamicin, and a combination oxytetracycline/polymyxin B ointment. A third-eyelid flap or partial tarsorrhaphy, which will shade the cornea from sunlight, together with subconjunctival injection, may reduce morbidity in severely affected animals.

A temporary eye patch glued to the hair surrounding the eye is an inexpensive and easily applied treatment. The eye patch provides shade, prevents exposure to flies, and may help to decrease spread of organisms.

- For sheep and goats in which chlamydophilal and mycoplasmal infections are suspected, respectively, topical tetracycline, oxytetracycline/polymyxin B, or erythromycin ointments are treatments of choice. These preparations are all effective against *Chlamydophila* or *Mycoplasma* and should be applied 3–4 times daily. If topical therapy is not practical, long-acting oxytetracycline (20 mg/kg, IM) or the addition of oxytetracycline to the feed (80 mg/animal/day) may be beneficial.
- Animals with substantial uveitis secondary to kerato-conjunctivitis that is particularly painful may benefit from topical ophthalmic application of 1% atropine ointment 1–3 times daily. This will prevent painful ciliary body spasms and reduce the likelihood of posterior synechia formation that occurs with miosis.
- Because of mydriasis caused by atropine, treated animals should be provided with shade. Systemic NSAID treatment may be used to provide relief from secondary uveitis.

S.8. Prevention

Good management practices are of paramount importance to reduce or prevent spread of infection in cattle, sheep, and goats.

- Separation of infected animals is beneficial when possible. Gloves and protective clothing should be worn and then disinfected between animals when affected individuals are being handled.
- Temporary isolation and preventive treatment of animals newly introduced to the herd may be helpful, because some of these animals may be asymptomatic carriers.
- Ultraviolet radiation from sunlight may enhance disease (particularly in cattle); therefore, affected animals should be provided with shade.
- Dust bags or insecticide-impregnated ear tags can be used to reduce the number of face flies (*Musca autumnalis*), an important vector for *M bovis*.
- *M bovis* bacterins are available and can be administered before the beginning of fly season. Cattle should be started on *M bovis* vaccine series 6–8 weeks before the anticipated first cases of IBK to allow time for adequate immune responses to develop.
- The efficacy of current commercially available *M bovis* bacterins is controversial and likely varies because of vaccinal versus outbreak strains of *M bovis* and varying degrees of cross-protection afforded by vaccination. Vaccination may reduce the severity and duration of infection in affected

animals. IBR may predispose cattle to infection with *M bovis*; thus, vaccination of herds against IBR may reduce outbreaks of *M bovis*.

- The use of modified live IBR vaccines has been associated with outbreaks of IBK in cattle; IBR vaccination must be appropriately timed with cattle shipments so that these events do not coincide. Vaccination of cattle with a modified live IBR vaccine could likely exacerbate an outbreak of IBK associated with *M bovis* and/or *Moraxella bovoculi* because of increased ocular and nasal secretions spreading bacteria between herdmates as well as corneal epithelial damage.
- In recent studies, the efficacy of autogenous *Moraxella* spp bacterins to prevent IBK has not been demonstrated in randomized controlled field trials. Nevertheless, anecdotal evidence has suggested that, for some herds, *M bovis* and/or *Moraxella bovoculi* autogenous bacterins have provided benefit in reducing IBK problems. It is unlikely that any *Moraxella* spp vaccine will ever completely control IBK in the face of overwhelming challenge from and exposure to other risk factors such as flies, dust, other infectious agents, and trace mineral deficiencies. As such, planning and implementing a successful IBK control program should address multiple issues that may potentially reduce susceptibility of cattle to IBK beyond just vaccines against *Moraxella* spp.

T. Colibacillosis

T.1. Definition

Colibacillosis is acute fatal septicemic or cubacute pericarditis and airsacculitis caused by infection with *Escherichia coli.*It is a common systemic disease of economic importance in poultry and seen world-wide. The disease is also called Colisepticemia or *Escherichia coli* infection. It affects many species of animals.

T.2. Etiology

Bacteria belonging to the family *Enterobacteriaceae mainly Escherichia coli*.

- Gram-negative rods up to 3 µm in length ;
- non-spore-forming ;
- facultative anaerobes ;
- Motile enterobacteria with peritrichous flagella.

T.3. Epidemiology

Bacteria belonging to the *Enterobacteriaceae* have a worldwide distribution, inhabit the intestinal tract of animals and humans and contaminate vegetation, soil and water. Some members of the family constitute part of the normal flora of the intestinal tract whereas some of the pathogenic organisms are disseminated by clinical and subclinical execrators and by survival in the environment especially in area inhabited by animal; transmission is through the fecal-oral route.

T.4. Pathogenesis

Enterotoxigenic diarrhea (in neonatal pigs, calves, lambs and weaning pigs)is caused by strains of E.coli that produce adhesions that promote attachment to glycol-proteins on the surface of epithelial cells of the jejunum and ileum, and an enterotoxin that affects the epithelial cell(to which the entorotoxigenic strain of E.coli is adherd), resulting in fluid secretion and diarrhea. Following ingestion by the host, enterotoxinec strains of E.coli adhere to target cells, multiply, and secrete entorotoxin. Fluid and electrolytes accumulate in the lumen of the intestine resulting in diarrhea, dehydration, and electrolyte imbalances.

T.5. Symptoms

Clinical infections in young animals may be limited to the intestines (enteric colibacillosis, neonatal diarrhoea), or may manifest as septicaemia (colisepticaemia, systemic colibacillosis) or toxaemia (colibacillary toxaemia). In older pigs, post-weaning enteritis and oedema disease are manifestations of toxaemia. Extraintestinal localized infections in adult animals, many due to opportunistic invasion, can involve the urinary tract, mammary glands and uterus.

Enteric colibacillosis

Enteric colibacillosis primarily affects newborn calves, lambs and piglets.

Enteric colibacillosis in calves:

Picture 19: Depression in calves suffering Enteric colibacillosis:

In enteric colibacillosis symptoms may include:

- Diarrhoea develops within the first few days of life. Faecal consistency issomewhat variable. In some cases faeces are profuse and watery; in others they are pasty; white or yellowish and rancid. This rancid faecal material may accumulate on the tail and hind limbs.
- Depression becomes marked as dehydration and acidosis develop.
- Mildly affected animals may recover spontaneously.
- Without treatment, severely affected calves die within a few days.

Enteric colibacillosis in Piglets

- May succumb to enteric colibacillosis within 24hours of birth.

- Often, an entire litter is affected and, as the disease progresses, piglets refuse to suck.
- A profuse watery diarrhoea rapidly leads to dehydration;
- Weakness and death.

Colisepticaemia

Systemic infections with *E. coli* are relatively frequent in calves, lambs and poultry. Septicaemic strains of *E. coli* have special attributes for resisting host defence mechanisms. They invade the bloodstream following infection of the intestines, lungs or umbilical tissues (navel ill).

Colisepticaemia often presents as an acute fatal disease with many of the clinical signs attributable to the action ofendotoxin.

- Pyrexia, depression, weakness and tachycardia, with or without diarrhoea, are early signs of the disease.
- Hypothermia and prostration precede death which may occur within 24 hours.
- Meningitis and pneumonia are commonly encountered in affected calves and lambs.
- Postsepticaemic localization in the joints of calves and lambs results in arthritis with swelling, pain, lameness and stiff gait.
- Watery mouth occurs in lambs up to 3 days of age and has been associated with systemic invasion by *E. coli* It is characterized by severe depression, loss of appetite, profuse salivation and abdominal distension. The condition is encountered in lambs born in confined lambing areas.
- Morbidity rates may exceed 20% and mortality in affected lambs is high, many dying within 24 hours of clinical onset.
- Death is attributed to endotoxic shock.
- In poultry, airsacculitis and pericarditis may develop following septicaemia.
- Coligranuloma is characterized by chronic inflammatory changes resembling tuberculous lesions which are encountered at post-mortem examination in laying hens.

Oedema disease of pigs

- Oedema disease is a toxaemia which usually occurs 1 to 2 weeks after weaning in rapidly growing pigs.
- The onset of oedema disease is sudden, with some animals found dead without showing clinical signs.
- Characteristic signs include:
    - Posterior paresis, muscular tremors and oedema of the eyelids and the front of the face.
    - The squeal may be hoarse due to laryngeal oedema. The faeces are usually firm.

- Flaccid paralysis precedes death which typically occurs within 36 hours of the onset of clinical signs.
- Animals that recover frequently have residual neurological dysfunction.

The characteristic post-mortem lesions are:

- Oedema of the greater curvature of the stomach and the mesentery of the colon. Perivascular oedema in the central nervous system, detectable on histological examination, accounts for the neurological dysfunction.
- Cerebrospinal angiopathy, in which there is marked fibrinoid necrosis in vessel walls, may develop in animals surviving acute oedema disease.

Post-weaning diarrhoea of pigs

This condition occurs within a week or two after weaning, often following changes in feeding regimens or in management and with possible involvement of rotaviruses.

T.6. Clinical signs

- Vary from an afebrile disease with inappetence to watery diarrhoea in severe cases.
- Diarrhoea and purplish discoloration of areas of the skin are often observed. Some animals may die suddenly.

Coliform mastitis

Infection of the mammary glands of cows and sows by members of the *Enterobacteriaceae*, including *E. coli*, occurs opportunistically. In dairy cows, the source of infection is faecal contamination of the skin of the mammary gland, and relaxation of the teat sphincterfollowing milking increases vulnerability to infection.

- The acute form of the disease is characterized by endotoxaemia and can be life-threatening.
- Peracute disease may be fatal within 24 to 48 hours.
- Affected animals are severely depressed with drooping ears and sunken eyes.
- Mammary secretions are watery and contain white flecks.

Urogenital tract infections

Opportunistic ascending infections of the urinary tract by certain uropathogenic strains of *E. coli* result in cystitis, especially in bitches. These strains possess virulence factors such as fimbriae which facilitate mucosal colonization. Invasion of hyperplastic endometrium by opportunistic strains of *E. coli* is a critical factor in the pathogenesis of canine pyometra. Prostatitis in dogs is also associated with invasion by opportunistic *E. coli* strains.

T.7. Diagnosis

The diagnosis is based on:

- The age and species of the affected animal, the clinical signs and the duration of illness which suggest the type of infection and the category of disease.
- The selection of specimens, the laboratory procedures for diagnosis and appropriate treatment and control measures are influenced by the history, the progress of the disease and the system or organ affected.

T.8. Treatment

The nature and duration of therapeutic measures are determined by the severity and duration of the disease process.

- In calves with neonatal diarrhoea, milk should be supplemented with fluids containing electrolytes.
- Severely dehydrated calves require parenteral fluid replacement therapy.
- Calves with hypogammaglobulinaemia can be given bovine gammaglobulin intravenously.
- In most domestic species, enteric diseases may be treated, if required, by oral administration of antimicrobial compounds which are active in the gastrointestinal tract. Systemic and localized infections require parenteral administration of therapeutic agents.
- Treatment should be based on susceptibility testing of isolates.
- Antimicrobial resistance is a major problem with respect to *E. coli* organisms, particularly those isolated from intensively-reared farm animals such as pigs and poultry. Multiple resistance to three or more classes of antimicrobial agents is common in isolates of *E. coli* from pigs and poultry at time of slaughter and in clinical isolates from all animals, both farm animals and pets. Increasing resistance is of importance because resistant *E. coli* may be transferred from animals to humans, either through food or by direct contact. In addition, multiple resistance in *E. coli* infections in animals is significant because it limits therapeutic options and may lead to treatment failure in some cases. Antimicrobial resistance to newer agents is a particular concern in animal isolates of *E. coli*, and isolates producing extended spectrum β-lactamases have been reported in many countries and in most domestic animal species. Resistance to fluoroquinolones is an emerging problem in animals, particularly in samples from clinical cases
- Because of the extensive local tissue damage, intramammary treatment of coliform mastitis is often of limited value. Therapy is aimed at counteracting shock and eliminating toxic material from the mammary gland by frequent stripping of affected quarters.

T.9. Prevention

Newborn animals should receive ample amounts of colostrum shortly after birth. Colostral antibodies can prevent colonization of the intestine by pathogenic *E. coli*. Absorption of gammaglobulin from the intestine declines progressively after birth and is negligible by 36 hours.

- A clean, warm environment should be provided for newborn animals.
- Dietary regimes may contribute to the development of oedema disease and other post-weaning conditions. To avoid factors that may contribute to the occurrence of disease, new feed should be introduced gradually.
- Vaccination is of value for a limited number of the diseases caused by *E. coli*. Vaccination methods used for prevention of enteric disease in piglets and calves include:
    - Vaccination of pregnant cows with purified *E. coli* K99 fimbrial or whole-cell preparations, often combined with rotavirus antigen, can be used to enhance colostral protection.
    - Commercially available killed vaccines containing prevalent pathogenic *E. coli* serotypes can be given to pregnant sows. Research is ongoing into the development of oral vaccines for post weaning diarrhoea in pigs which contain live attenuated *E. coli* strains carrying the particular fimbrial adhesins involved in disease production.
    - A commercial vaccine is available for protection against *E. coli* mastitis in cows.
- Breeding for disease resistance can be employed and has been used commercially to reduce the number of susceptible pigs. In Switzerland, breeding policies have been implemented which greatly reduced the number of pigs which were susceptible to *E. coli* F18-associated diarrhoea

- Control of *E. coli* infections in animals is important not only for the prevention of disease in animals but also for public health reasons.

U. Streptococcosis

U.1. Definition

Streptococcosis is infectious disease caused by bacteria of streptococcus genus; frequently associated localized suppirative conditions and inflammatory processes in different parts of the body.

U.2. Etiology

Strictly anaerobic or facultative aerobes, gram-positive cocci.

They are approximately 1.0 µm in diameter and form chains of different lengths, non-motile.

U.3. Epidemiology

The streptococci are distributed worldwide. Most species live as commensals on the mucosae of the upper respiratory tract and lower urogenital tract. These fragile bacteria are susceptible to desiccation and survive for only short periods off the host.

U.4. Pathogenesis

Most of the streptococci that are pathogenic for animals are pyogenic and are associated with abscess formation, other suppurative conditions and septicaemia. *Streptococcus suis*, which is non-pyogenic, is a major pathogen of pigs, causing septicaemia, meningitis and pneumonia among other conditions.

Virulence factors include enzymes and exotoxins such as streptolysins (haemolysins), hyaluronidase, streptokinase and proteases. Polysaccharide capsules, which are major virulence factors of *S. pyogenes, S. pneumoniae* and most strains of *S. equi*, are antiphagocytic. The cell-wall proteins of *S.pyogenes, S. equi* and *S. porcinus* are also antiphagocytic. In the absence of antiphagocytic factors, these bacteria are rapidly killed by phagocytes.

Streptococcosis of pig

Streptococcus suis is recognized worldwide as a cause of significant losses in the pig industry.

It is associated with meningitis, arthritis, septicaemia and bronchop-neumonia in pigs of all ages, and with sporadic cases of endocarditis, neonatal deaths and abortion.

Clinical signs and epidemiology

Asymptomatic carrier pigs harbour *S. suis* in tonsillar tissue. Disease outbreaks are most common in intensively reared pigs when they are subjected to overcrowding, poor ventilation and other stress factors. Sows carrying the organisms can infect their litters, leading either to neonatal deaths or to carrier animals in which characteristic signs develop later in life. Meningitis, which is often fatal, is characterized by fever, tremors, incoordination, opisthotonos and convulsions.

In North America, *S. suis* is often isolated from cases of respiratory disease in conjunction with *Mycoplasma* and *Pasteurella* species. Serious infections occur periodically in humans directly involved in pig husbandry or processing. Infections with *S. suis* have also been recorded in cattle, small ruminants, horses and cats.

Treatment

Most strains of *S. suis* are susceptible to penicillin or ampicillin.

Control

- These bacteria tend to become endemic in a herd and eradication is not feasible.
- Improved husbandry may decrease the prevalence of clinical disease.

- Research is ongoing on the development of subunit vaccines containing capsular antigens.
- Prophylactic long- acting penicillin, given by injection to sows 1 week prior to farrowing and to piglets during the first 2 weeks of life, has proved worthwhile in herds experiencing neonatal deaths or meningitis at weaning.

Bovine streptococcal mastitis

Streptococcus agalactiae, S. dysgalactiae and *S. uberis* are the principal pathogens involved in streptococcal mastitis.

Enterococcus faecalis, S. pyogenes and *S. zooepidemicus* are less commonly isolated from cases of mastitis:

- *Streptococcus agalactiae* colonizes the milk ducts and produces persistent infection with intermittent bouts of acute mastitis.
- *Streptococcus dysgalactiae*, which is found in the buccal cavity and genitalia and on the skin of the mammary gland, causes acute mastitis.
- *Streptococcus uberis*, a normal inhabitant of skin, tonsils and vaginal mucosa, is a major cause of clinical mastitis, usually without systemic signs.

Diagnosis

- Clinical signs include inflammation of mammary tissue and clots in the milk.
- Milk samples should be collected carefully to avoid contamination.
- Samples should be cultured on blood agar, Edwards medium and MacConkey agar and incubated aerobically at 37°C for 24 to 48 hours.
- Sugar fermentation tests.

Treatment and control

- Localized suppurative conditions are drained of pus.
- For systemic treatment, penicillin G and ampicillin are effective;
- Cephalosporin and chloramphenicol are alternative;
- Streptococcal endocarditis is treated with combined penicillin and gentamicin.
- Penicillin (intra-mammary) are effective for treating mastitis due to *S. agalactiae* and most other streptococci.

V. Otitis

Definition

The otitis is inflammation of the external ear canal (otitis externa), middle ear infection (otitis media), and inner ear infection (otitis interna).

V.1. Otitis externa

Otitis externa an acute or chronic inflammation of the epithelium of the external ear canal. It may develop anywhere from the tympanic membrane to the pinna. It is variably characterized by erythema, edema, increased sebum or exudate and desquamation of the epithelium. The ear canal may be painful and or pruritic depending on the cause or duration of the condition.

V.1.1. Etiology

The causes of otitis externa have been grouped into 4 areas:

- Primary factors: that directly cause the otitis they include parasite (otodectes, psoroptes, sarcoptes, demodex); foreign bodies (gras awn, concreted wax, medication); tumor (cerumin gland adenoma, inflammatory polyps); hypersensitivity (atopic dermatitis, food sensitivity, contact dermatitis); disorders of keratinization, hypothyloidism.
- Secondary factors: they intensify and complicate primary and perpetuating conditions; these are yeasts and bacteria.
- Predisposing factors: are conditions that place an individual at risk of developing otitis. They are often congenital or environmental and include conformation(pinnal carriage, narrow ear canal ,excessive hair or ceruminous gland);
- Perpetuating factors: that tend to prevent the resolution of the otitis once it develops. They include otitis media and progressive pathogenic changes.

V.1.2. Symptoms

- The history will determine if the otitis is acute, chronic or recurrent.
- Acute conditions tend to be parasitic or foreign body.
- Chronic condions point toward a hormonal, allergic, or neoplastic process.
- The outside of the ear should be examined, and any erythema, edema, crusts, scale, ulcers, lichenification, hyperpigmentation or exdates should be noted.

V.1.3. Diagnosis

- An otoscopic examination will allow identification of deep foreign bodies, tumors, impacted debris, ruptured or abnormal tympanin membrane.
- Otoscopic examination conducted using a handheld otoscope or video otoscope.
- During an otoscopic examination the ear canal should be inspected for changes in diameter, pathologic changes in the skin, quality and type of exudate, parasite, foreign bodies, neoplasms and change in tympanic membrane.
- A stained smear can quickly determine if microbial overgrowth is present.

V.1.4. Treatment

All primary and secondary causes and predisposing factors need to be identified, managed, and treated.

- Management of pain or pruritus must be included in the initial treatment protocol. Tramadol for the first 5–7 days at 5 mg/kg, PO, tid, may be especially beneficial.
- It is important to remember that topical medications are inactivated by exudates, and excessive cerumen may prevent medications from reaching the epithelium. The ears should be gently cleaned with an ear cleaner that will remove the debris in the canal. Thick, dry, or waxy material requires a ceruminolytic solution such as carbamide peroxide or dioctyl sodium sulfosuccinate (DSS). If rods are seen, the ear cleaner should contain squaleae, because one possible cause is *Pseudomonas*, which can produce a biofilm that protects bacteria from antibiotics. The ears should be thoroughly rinsed with warm water to remove residual ear cleaner.
- Effective treatment may require both topical and systemic antimicrobial therapy, along with pain medications and glucocorticoids. The duration of treatment may vary from 7–10 days to >30 days, depending on the diagnosis. In treatment of acute bacterial otitis externa, antibacterial agents in combination with corticosteroids reduce exudation, pain, swelling, and glandular secretions. The least potent corticosteroid that will reduce the inflammation should be used.
- Irritating medications (eg, home remedies and vinegar dilutions) should be avoided. They cause swelling of the lining of the ear canal and an increase in glandular secretions, which predispose to opportunistic infections. Substances that are usually not irritating in normal ear canals may cause irritation in an ear that is already inflamed. This is particularly true of propylene glycol. Powders, such as those used after plucking hair from the canal, can form irritating concretions within the ear canal and should not be used.
- Yeast infections in dogs can be treated with oral ketoconazole 5 mg/kg/day, PO, for 15–30 days. Ketoconazole should not be used in cats; itraconazole 2–3 m/kg/day for 15–30 days or one week on/one week off is recommended.
- Duration of treatment will vary depending on the individual case but should continue until the infection is resolved based on reexamination and repeat cytology and culture.
- Animals with bacterial and yeast infections should be physically examined, with cytologies evaluated weekly to every other week until there is no evidence of infection. For most acute cases, this takes 2–4 weeks. Chronic cases may take months to resolve.

- Methicillin-resistant *Staphylococcus intermedius* and *Pseudomonas* otitis (caused by *Pseudomonas aeruginosa*) have emerged as frustrating and difficult perpetuating causes of otitis because of the development of resistance to most common antibiotics. These infections are often chronic in course (>2 months) and associated with marked suppurative exudation, severe epithelial ulceration, pain, and edema of the canal.
- Successful treatment is multifaceted and should include the following steps:
  - identify the primary cause of the otitis and manage it;
  - remove the exudate via irrigation of the ear canal;
  - identify and treat concurrent otitis media;
  - select an appropriate antibiotic ;
  - Treat topically and systemically until the infection resolves (weeks to months).
- Polymyxin B and fluoroquinolone antibiotics have shown the best success in controlling *Pseudomonas* infections in cases in which resistance has been identified through culture. However, resistance is developing to fluoroquinolones.

V.1.5 Prevention

The prevention must be carried out by applying different actions:

- Owners should be shown how to properly clean the ears.
- The frequency of cleaning usually decreases over time from daily to once or twice weekly as a preventive maintenance procedure.
- The ear canals should be kept dry and well ventilated. Using topical astringents in dogs that swim frequently and preventing water from entering the ear canals during bathing should minimize maceration of the ear canal. Chronic maceration impairs the barrier function of the skin, which predisposes to opportunistic infection.
- Preventive otic astringents may decrease the frequency of bacterial or fungal infections in moist ear canals.
- Clipping hair from the inside of the pinna and around the external auditory meatus, and plucking it from hirsute ear canals, improves ventilation and decreases humidity in the ears. However, hair should not routinely be removed from the ear canal if it is not causing a problem, because doing so can induce an acute inflammatory reaction.

- Ear hygiene is important; in particular, the hair from the pre- and periauricular area should be clipped, as well as hair from the surface of the inner pinnae and ends of the ears. Plucking of hair from the ear canal is controversial but may be needed to adequately resolve the ear infection. Hair plucking is painful and should be done under anesthesia.

V.2. Otitis media and interna

Inflammation of the middle ear structures, is seen in small and large domestic animals, including dogs, cats, rabbits, ruminants, horses, pigs, and camelids. It can be unilateral or bilateral and can affect animals of all ages. Although typically sporadic, outbreaks are possible in herds.

Otitis media usually results from extension of infection from the external ear canal through the tympanic membrane or from migration of pharyngeal microorganisms through the auditory tube. Occasionally, infection extends from the inner ear to the middle ear, or reaches the middle ear by the hematogenous route. Primary otitis media has been reported in certain breeds of dogs, particularly Cavalier King Charles Spaniels. Untreated otitis media can lead to otitis interna (inflammation of the inner ear structures) or to rupture of an intact tympanic membrane with subsequent otorrhea or otitis externa

V.2.1. Clinical symptoms

Clinical signs of otitis media include the following:

- Head shaking, rubbing or scratching the affected ear, and tilting or rotating the head toward the affected side; self-trauma can lead to aural hematoma.
- The most common cause of recurrent otitis externa is undiagnosed otitis media.
- When otitis externa accompanies otitis media, the external ear canal may look inflamed and contain an abnormal discharge.
- The pinna or ear canal may be painful and malodorous, and the hair surrounding the base of the ear may be wet or matted.
- Because the facial (cranial nerve VII) and sympathetic nerves course through the middle ear, animals with otitis media may exhibit signs of facial nerve paralysis (eg, ear droop, lip droop, ptosis, and collapse of the nostril) and/or Horner syndrome (eg, miosis, ptosis, enophthalmos, and protrusion of the nictitating membrane) on the same side as the affected ear.
- Exposure keratitis and corneal ulceration may develop. With facial paralysis, the nasal philtrum or lip may deviate away from the affected side. These signs help to distinguish otitis media from simple otitis externa.

- With otitis interna, inflammation impairs function of the vestibulocochlear nerve (cranial nerve VIII), resulting in hearing loss and signs of peripheral vestibular disease such as head tilt, circling, leaning or falling toward the affected side, general incoordination, or spontaneous horizontal nystagmus with the fast phase away from the affected side.
- Extension of infection from the inner ear to the brain leads to meningitis, meningoencephalitis, or abscesses, with signs referable to those conditions.
- Whereas animals with otitis media/interna are usually alert, nonfebrile, and have a good appetite, those with meningitis or meningoencephalitis are usually depressed, febrile, and inappetent. A major differential diagnosis for otitis media/interna in ruminants is listeriosis. However, in listeriosis, there areother signs such as dysphagia or loss of facial sensation and depression.

V.2.2. Diagnosis

- In large animals, otitis media and interna are presumptively diagnosed based on history and clinical signs. A history of bottle feeding or feeding of contaminated milk to neonates, concurrent or previous respiratory disease, chronic ear infection, or aural foreign body, in conjunction with typical signs of otitis media/interna, should prompt examination of the ear canal.
- Otitis media is confirmed by visualizing a bulging, discolored, or ruptured tympanic membrane. Although the tympanic membrane may be visualized using a simple otoscope in many cases, the anatomy of the ear canal hinders visualization in some species, such as horses and llamas; endoscopy, or video otoscopy, is an alternative approach. Imaging methods assist in diagnosis and assessment of lesion severity. Radiography can detect osseous changes in the tympanic bulla and fluid in the tympanic cavity if appropriate positioning and techniques are used.
- In some cases, diagnosis is made only at necropsy, using special techniques to expose the tympanic region. Diagnosis of clinical otitis media/interna in one ear should always prompt examination of the other ear to determine whether subclinical otitis is present.
- Diagnosis can be challenging, because the tympanic membrane can be intact. Palplation of the tympanic membrane with a blunt instrument is not an accurate method of determining the patency of the tympanic membrane. Advanced imaging techniques are more sensitive than routine radiographs but are not 100% sensitive and specific. In some cases, exploratory (and therapeutic) bulla osteotomy may be necessary.

V.2.3. Treatment and Prognosis

- Treatment of otitis media/interna is most successful when started early in the course of the disease.
- Chronic cases are often refractory to treatment or recur after apparent remission.
- When otitis externa accompanies otitis media/interna, the ear should be examined closely for mites and foreign bodies, such as plant awns, and the discharge cultured for bacteria.
- Many aerobic and anaerobic bacteria have been cultured from the ears of animals with otitis media/interna, and mixed infections are common. Pathogens that warrant mentioning because of their frequency of isolation include *Malassezia* spp and *Pseudomonas* spp in small animals; *Streptococcus suis* in pigs; *Streptococcus* spp in horses; *Mycoplasma* spp in goats; and *Mannheimia haemolytica*, *Pasteurella multocida*, *Histophilus somni*, and *Mycoplasma bovis* in cattle. *M bovis* is particularly problematic in dairy calves fed unpasteurized waste milk from cows with intramammary infection. However, other pathogens, such as coliform bacteria, *Staphylococcus* spp, *Neisseria* spp, corynebacteria, and *Arcanobacterium pyogenes* are frequently isolated from the ears of affected animals. Isolation of a bacterial pathogen(s) or mites from the ear helps direct initial treatment but does not necessarily imply causation of otitis media/interna, because the same organisms can be isolated from the external ear canals of apparently healthy animals.
- Ear mites, when present, should be treated with an appropriate systemic antiparasitic agent.
- Topical acaricides can be instilled into the external ear canal once it is cleaned. Bacterial infection should be treated with appropriate systemic antimicrobial agents, based on culture and susceptibility test results.
- In addition to antimicrobial and/or anthelmintic therapy, the external ear canal should be cleaned and flushed if otorrhea or otitis externa is present; physiologic saline or dilute antiseptic solutions, such as iodine, chlorhexidine, or hydrogen peroxide, are commonly used for flushing. Steroids or NSAIDs can help reduce inflammation and pain associated with otitis media/interna.
- Corneal ulceration, aural hematomas, and concurrent infections should be treated appropriately, if present, and the animal protected from further self-injury.
- Early diagnosis and treatment of otitis media/interna can result in complete resolution of infection and clinical signs. However, with severe, chronic, or nonresponsive cases, clients should be advised that neurologic deficits and hearing loss may persist even if infection is resolved.

W. Gastro-enteritis

W.1. Definition

Gastroenteritis or infectious diarrhea is a medical condition from inflammation of the gastrointestinal tract that involves both the stomach and the small intestine. It causes some combination of diarrhea, vomiting, abdominal pain and cramping. Dehydration may occur as a result.

W.2. Etiology

Salmonella typhi and *paratyphi* (as well as *Shigella*) are strictly human pathogens and domestic animals play no role in the epidemiology of these infections (although non-human primates may be infected and serve as a source for their handlers). All of the other *"non-typhoid" Salmonella spp.* (*Salmonella enterica*) are ubiquitously present in the environment and reside in the GI tracts of animals. As such, animals are the ultimate source for virtually all *Salmonella*. The vast majority of human cases of salmonellosis are, however, acquired not through direct contact with animals, but rather by ingestion of contaminated foods. For example:

- *Salmonella enteritidis* from undercooked eggs
- *Salmonella typhimurium* from undercooked meats or fecal contamination of a variety of foods.

W.3. Clinical signs

Salmonellosis in dogs and cats

The severity of salmonellosis in dogs and cats varies: subclinical carrier state (most common) and acute enterocolitis:

- fever, anorexia, lethargy ;
- diarrhea, possibly with mucus or blood ;
- abdominal pain (infection is often associated with mesenteric lymphadenitis;
- Cats may present with prolonged periods of fever and anorexia without diarrhea.
- Septicemia/endotoxemia ;
- Salmonellosis in cats has also been caused "song bird fever," reflecting association with predation on infected migratory birds.

The role of dogs and cats in zoonotic transmission

Salmonella infections in dogs and cats deserve special comment for several reasons related to zoonotic transmission:

- S*almonella spp.* can be isolated from healthy dogs and cats at rates of up to 36% and 18%, respectively.
- Dogs and cats tend to shed *Salmonella* organisms for very prolonged periods of time after infection.
- Dogs and especially cats can shed *Salmonella* organisms in both their feces and saliva, meaning that transmission can occur via licking.
- Pig ear dog treats may be a source of *Salmonella* infection for both dogs and humans that handle the treats.
- Dogs and cats may suffer salmonellosis as a "reverse zoonosis," with infection transmitted from human-to-dog and subsequently back to other humans.

The role of other domestic animals

- The feces of virtually any animal may be a potential source of *Salmonella.* Hospitalized horses and cattle must be considered as potential sources of *Salmonella* for veterinary clinicians and students.
- Recognizing the potential for pigs to serve as a source of *Salmonella* is particularly important since human infection with *S. cholerae-suis* is associated with a 20% mortality rate!
- A recent report highlighted the potential for inadvertent contamination of household carpets with *Salmonella* when residents had occupational exposure to cattle on farms or cats in veterinary clinics.

The role of exotic pets and wild animals

- Salmonellosis is well recognized as a zoonosis associated with many species of reptiles. In 2002, there was even a report of a reptile owner who donated blood products and two transfusion recipients became infected with a strain of Salmonella matching that of the donor's snake.
- Salmonellosis has also been associated with ritual consumption of raw marine mammal blubber or meat.

W.4. Diagnosis of *Salmonella* infections

- Diagnosis is based on fecal culture

W.5. Treatment

- Simple gastroenteritis should be treated with supportive therapy, fluids.
- Treatment with antibiotics should probably be restricted to those animals that develop evidence of systemic spread of infection because salmonella is resistant to antibiotics.
- A recent report suggests that bleach is the most effective disinfectant in veterinary hospital settings.

X. Tetanus

X.1. Definition

Tetanus is an acute, potentially fatal intoxication which affects many species including humans. However, species susceptibility to toxin varies considerably. Horses and humans are highly susceptible, ruminants and pigs moderately sow and carnivores are comparatively resistant. Poultry are not susceptible to tetanus.

X.2. Etiology

Clostridium tetani, the aetiological agent, is a straight, slender, anaerobic, gram-positive rod.

The endospores are resistant to chemicals and boiling but are killed by autoclaving at 121°C for 15 minutes. *Clostridium tetani* has a swarming growth and is haemolytic on blood agar due to the production of tetanolysin. The neurotoxin, tetanospasmin is produced by *C. tetani*. Synaptic inhibition of neuromuscular transmission

X.2. Epidemiology

Almost all mammals are susceptible; dogs and cats are relatively more resistant than other domestic or laboratory mammals. Birds are quite resistance; horses are more sensitive than other species. The disease is found worldwide**.** Usually a history of a wound or other tissue trauma.

Infection occurs when endospores are introduced into traumatized tissue from soil or faeces. Common sites of infection include deep penetrating wounds in the horse, castration and docking wounds in sheep, abrasions associated with dystocia in cows and ewes, and the umbilical tissues in all young animals. The presence of necrotic tissue, foreign bodies and contaminating facultative anaerobes in wounds may create the anaerobic conditions in which *Clostridium tetani* spores can germinate.

X.4. Pathogenesis

The neurotoxin binds irreversibly to ganglioside receptors on motor neuron terminals and is transported to the nerve cell body and its dendritic processes in the central nervous system in toxin-containing vesicles, by retrograde intra-axonal flow.

Toxin is transferred trans-synaptically to its site of action in the terminals of inhibitory neurons and enters these cells by endocytosis. Because release of inhibitory neurotransmitters is prevented, spastic paralysis results. Toxin can also be blood-borne, especially when produced in large amounts, and can then bind to motor terminals throughout the body prior to transfer to the central nervous system. Bound toxin not neutralized by antitoxin.

X.5. Clinical signs

Picture 20: Opisthotonos and Orthotonos signs in puppy and lamb affected by Tetanus

The incubation period of tetanus is usually between 5 and 10 days but may extend to 3 weeks. When the development of clinical signs is delayed, the wound at the site of infection may have healed and the condition is then referred to as latent tetanus. The clinical effects of the neurotoxin are similar in all domestic animals. However, the nature and severity of the clinical signs are dependent on the anatomical site of the replicating bacteria, the amount of toxin produced and species susceptibility.

Wounds on or near the head are usually associated with a shorter incubation period and an increased tendency to generalized tetanus. Localized tetanus, which usually affects less susceptible species such as dogs, presents as stiffness and spasm of muscles close to the site of injury as a result of the effect of toxin on local nerve endings.

Clinical signs include:

- Stiffness, localized spasms, altered heart and respiratory rates,
- Dysphagia and altered facial expression.
- Spasm of masticatory muscles may lead to 'lockjaw'.
- Opisthotonos (head, neck and spinal column enter into a complete arching position) due to spasm of axial column.

- Orthotonos or orthotonus (titanic spasm that fixes the head, body and the limbs in a rigid straight line).
- Animals that recover from tetanus are not necessarily immune because the amount of toxin that can induce clinical disease is usually below the threshold required to stimulate the production of neutralizing antibodies.

X.6. Diagnostic

- The diagnosis of tetanus is usually presumptive and is based on characteristic clinical signs and a history of recent trauma (wound) in unvaccinated animals
- Differentiation from strychnine poisoning is necessary, particularly in dogs. Gram-stained smears of material from lesions may reveal the characteristic 'drumstick' forms of *C.tetani*
- Anaerobic culture of *Clostridium tetani* from necrotic wound tissue may be attempted but is often unsuccessful.
- Serum from affected animals may be used to demonstrate circulating neurotoxin, using mouse inoculation.

X.7. Treatment

The treatment must begin quickly and directed toxin or toxin production:

- To neutralize unbound toxin, antitoxin should be administered promptly, either intravenously or into the subarachnoid space on three consecutive days.
- Toxoid may be given subcutaneously to promote an active immune response even in those animals that have received antitoxin.
- Penicillin is administered intramuscularly or intravenously to kill toxin-producing vegetative cells of *Clostridium tetani* in the lesion.
- Surgical debridement of wounds and removal of foreign bodies, followed by flushing with hydrogen peroxide, produces aerobic conditions which help to inhibit bacterial replication at the site of injury.
- Affected animals should be housed in a quiet dark environment. Fluid replacement therapy, sedatives, muscle relaxants and good nursing can minimize clinical discomfort and maintain vital functions.

X.8. Prevention

- Farm animals should be vaccinated routinely with tetanus toxoid. A booster dose of vaccine may be advisable if a vaccinated animal sustains a deep wound.

- In horses, prompt surgical debridement of wounds is desirable.
- Unvaccinated animals that have sustained deep wounds or are presented for surgery should be given antitoxin. This passive protection usually lasts about 3 weeks.

Y. Stomatitis

Stomatitis is a clinical sign of many diseases in large animals. Oral trauma or contact with chemical irritants (e.g., horses that lick at their legs after having been blistered with caustic agents) may result in transient stomatitis. Traumatic injury from the ingestion of the awns of barley, foxtail, porcupine grass, and spear grass, as well as feeding on plants infested with hairy caterpillars, also will result in stomatitis in horses and cattle.

Clinical signs commonly associated with acute active stomatitis include ptyalism, dysphagia, or resistance to oral examination. Oral examination is facilitated by sedation, after which the mouth can be examined carefully with the aid of a mouth speculum and a light source. Ulcers should be visually and digitally evaluated to determine whether embedded foreign material (e.g., grass awns) is present. If the etiology is ingestion of foreign material, changing the quality and quantity of the hay or removing the animal from a pasture with grass awns may affect recovery.

Vesicular Stomatitis

Y.1. Definition

Vesicular stomatitis is a viral disease caused by two distinct serotypes of vesicular stomatitis virus: New Jersey and Indiana. Vesculation, ulceration, and erosion of the oral and nasal mucosa and epithelial surface of the tongue, coronary bands, and teats are typically seen in clinical cases, along with crusting lesions of the muzzle, ventral abdomen, and sheath. Clinical disease has been seen in cattle, horses, and pigs and very rarely in sheep, goats. Serologic evidence of exposure has been found in many species, including cervids, nonhuman primates, rodents, birds, dogs, antelope, and bats.

Y.2. Etiology

The viruses are members of the family Rhabdoviridae and genus *Vesculovirus*. Vesicular stomatitis viruses are the prototypes of the *Vesculovirus* genus.

They are bullet shaped and generally 180 nm long and 75 nm wide. Although there are many members of the *Vesiculovirus* genus, the New Jersey and Indiana serotypes are of particular interest in the Western hemisphere. These two viruses are similar in size and morphology but generate distinct neutralizing antibodies in infected animals. They have both been isolated in recent outbreaks in the USA.

Y.3. Epidemiology

Vesicular stomatitis viruses are endemic in South America, Central America, and parts of Mexico but have not been seen naturally outside the Western hemisphere. The virus can be transmitted through direct contact with infected animals with clinical disease (those with lesions) or by blood-feeding insects. In the south-western USA, black flies (Simulidae) are the most likely biologic insect vector. Other insects may act as mechanical vectors.

Exposure to insects that carry the virus is often associated with nearby moving water sources such as creeks or rivers or irrigation of pastures. Experimental studies have shown that feeding of infected insects on mucosal surfaces and nonhaired areas of the body were more often associated with development of lesions at those sites than if insects fed on haired areas of the body.

The prevalence of clinical cases in a herd is generally low (10%–20%), but seroprevalence within the herd may approach 100%. Viremia has not been detected in livestock species that exhibit clinical signs of vesicular stomatitis, although experimental studies have shown transmission of virus, presumably via lymphatic, between co-feeding black flies on cattle.

Virus is routinely isolated from active lesions in affected animals, and these lesions serve as a source of virus spread by direct contact and contamination of shared feed and water stations. Many vertebrate species have serologic evidence of exposure and may serve as reservoirs of infection. No definitive reservoir or amplifying host of vesicular stomatitis viruses in the USA has been identified.

Y.4. Clinical Findings

The incubation period is 2–8 days and is typically followed by a fever:

- By the time animals develop other signs and are examined, however, they are rarely febrile. Ptyalism is often the first sign of disease.

- Vesicles in the oral cavity are rarely seen in naturally occurring cases because of rupture soon after formation; therefore, ulcers are the most common lesion seen during initial examination. Ulcers and erosions of the oral mucosa, sloughing of the epithelium of the tongue, and lesions at the mucocutaneous junctions of the lips are commonly seen in both cattle and horses.
- Ulcers and erosions on the teats are not uncommon in cattle and may result in secondary cases of mastitis in dairy cows.
- Coronitis with erosions at the coronary band are seen in some cattle, horses, and pigs, with subsequent development of lameness.
- Crusting lesions of the muzzle, ventral abdomen, sheath, and udder of horses are typical during outbreaks in the western USA.
- Loss of appetite due to oral lesions, and lameness due to foot lesions, are normally of short duration, because the disease is generally self-limiting and resolves completely within 10–14 days.
- Virus-neutralizing antibodies to either serotype persist and have been documented in individual horses that had previous clinical disease for >8 yr. after an outbreak, but reinfection can occur after a second exposure.

Y.5. Diagnosis

Clinical diagnosis

Diagnosis is based on the presence of typical signs and either antibody detection through serologic tests, viral detection through isolation.

- Samples for viral isolation may include vesicular fluid, epithelial tags from lesions, or swabs of lesions.
- Of primary concern in diagnosis is differentiation of vesicular stomatitis from clinically indistinguishable but much more devastating viral diseases, including foot-and-mouth disease in ruminants and swine, swine vesicular disease, and vesicular exanthema of swine. Horses are not susceptible to foot-and-mouth disease. Both non-infectious and infectious causes of oral lesions must be considered.

Differential diagnosis

Differential diagnoses include actinobacillosis, foot-and-mouth disease, malignant catarrhal fever, and bovine viral diarrhea. Epidemic diseases such as bluetongue in ruminants, swine vesicular disease, and vesicular stomatitis in horses must be differentiated from other forms of acute noninfectious or contagious stomatitis.

Y.6. Treatment and Prevention

No specific treatment is available or warranted and prevention methods are:

- Cachexia can be avoided by providing softened feeds.
- Cleansing lesions with mild antiseptics may help avoid secondary bacterial infections.
- Management factors suggested to reduce risk of exposure to the virus include limiting time on pasture during insect season, providing shelters or barns during insect feeding times, and implementing other procedures that reduce animal contact with insects, such as application of insecticides.
- Affected animals should be isolated, and movement of other animals from the affected premises restricted.
- Vesicular stomatitis is a reportable disease in most areas, including the USA, so state and federal animal health officials must be notified when it is suspected.
- Commercially produced vaccines are not available in the USA, but vaccines for livestock are available in some Latin American countries.
- Veterinarians act as a part of the surveillance network as they examine animals involved in shows, exhibitions, races, and interstate or international movement in order to write a health certificate (i.e., certificate of veterinary inspection).
- When practitioners observe suspect cases of vesicular stomatitis, they should report to both their state and federal animal health officials. Reporting will prompt a regulatory investigation.
- Mucosal swab and serum samples from suspected animals are submitted for testing to veterinary diagnostic laboratories.

The vesicular stomatitis viruses are zoonotic and may cause self-limiting influenza-like disease (headache, fever, myalgia, and weakness) last 3–5 days in people working in close contact with the virus (e.g., laboratory exposure, direct contact with lesions in infected animals). Rarely, people can develop vesicles on the buccal and pharyngeal mucosa, lips, and nose.

- Topic 2: Gastro-Intestinal Infection

A. Definition

Gasto-intestinal infection is the pathologic condition involving different causal agents which are normally part of normal intestinal flora; the disease develops after a stressful event. The disease is mostly characterized by gastro-intestinal tract inflammation and diarrhea.

B. Etiology

Several microbes may be involved in gastrointestinal infection: bacteria, virus fungi and protozoa.

Definitive etiologic diagnosis of infectious disease of the GI tract depends on demonstrating the pathogen in the tract or in the feces of the affected animal.

C. Diagnosis

In herd epidemics, such as an outbreak of acute undifferentiated diarrhea in newborn calves or piglets, the best opportunity to establish a diagnosis is in the earliest stage of the disease by selecting untreated animals and submitting them for necropsy and detailed microbiologic examination of the intestinal flora. When selective necropsy is not an option, a series of carefully collected daily fecal samples should be submitted to a diagnostic laboratory with a request for special culture techniques, depending on the infectious disease suspected.

D. Treatment of infection

Antimicrobial agents are used for the treatment of bacterial diseases. There is no specific therapy for treatment of viral diseases. Antimicrobials are commonly given PO daily for several days until recovery is apparent, but there is little objective evidence of efficacy. There is evidence that overdosage or prolonged oral treatment may be detrimental (eg, bacterial overgrowth, villous atrophy).

Parenteral administration of antimicrobials is indicated when septicemia is apparent or may occur. The choice of antimicrobial agent depends on the suspected disease, previous results, and cost. In herd epidemics, antimicrobials may be added to the feed or water supplies at therapeutic levels for several days, followed by preventive levels for an extended period, depending on the infection pressure in the population. The feed and water supplies of in-contact animals also may be medicated in an attempt to prevent new cases from developing.

E. Control of G I infection diseases

Effective control of the common infectious diseases of the GI tract depends on practicing good sanitation and hygiene, developing and maintaining nonspecific resistance in the animal, and in certain cases, providing specific immunity by vaccinating the pregnant dam or susceptible animal.

- Effective sanitation and hygiene is achieved primarily by providing adequate space for animals and by regular cleaning of pens and efficient removal of manure from the immediate environment.
- Development and maintenance of nonspecific resistance depends on the genetic selection of animals that have a reasonable degree of inherent resistance and on the provision of adequate nutrition and housing, which minimizes stress and allows the animals to grow and behave normally.
- The development of infected but clinically healthy animals, which can shed pathogens for weeks or months, is a major problem with some infectious diseases of the GI tract, eg, salmonellosis. Ideally, these carrier animals should be identified by microbiologic means and isolated from the rest of the herd until free of the infection or culled.
- Certain diseases (eg, enterotoxigenic colibacillosis in calves and piglets) can be controlled by vaccination of the pregnant dam several weeks before parturition. This method depends on achieving a protective level of antibodies in the colostrum. There are exceptions but, in most cases, systemic immunity provides little protection against the infectious enteritides; effective immunity against GI disease depends on stimulation of local intestinal immunity after the neonatal period.
- During the neonatal period, protection can be provided through the local action of maternally derived antibodies. For example, secretory IgA progressively increases in sow's milk from the time of farrowing until weaning, which provides the piglet with daily protection during the nursing period.

L O 1.3 – Characterise Common viral diseases

Viral diseases are frequently found in different animals farms for which the development of nontoxic chemicals for therapeutic use is difficult. Antiviral drugs are generally only effective prophylactically or in early stages of disease when viral replication is occurring.

- Topic 1: Common Viral diseases

A. Lumpy Skin Disease

A.1. Definition

This is an acute disease of cattle, which is endemic in sub-Saharan Africa and Madagascar, is caused by lumpy skin disease virus, a capripox-virus. Outbreaks have also occurred in Egypt and Israel.

A.2. Etiology

The causal agent is a virus related to that of shee-pox and capripox it is known as Capripox bovis nodular or Neethling poxvirus.

A.3. Epidemiology

Although virus is present in the saliva of infected animals and transmission may occur through environmental contamination, lumpy skin disease is not particularly contagious. The principal method of transmission is by mechanical transfer through biting insects. As a consequence, disease outbreaks usually occur during the rainy season when insect activity is high and epidemics are often associated with heavy rainfall. New outbreaks may appear in areas far removed from an initial outbreak. It is unclear how the virus persists between epidemics but sub-clinically infected cattle are probably important. A wildlife reservoir, possibly the African Cape buffalo, may be involved. Suckling calves can get the disease from drinking contaminated milk.

A.4. Pathogenesis

Virus, which is transmitted mechanically by biting insects, rapidly disseminates through a leukocyte-associated viraemia. Many cell types including kerati-nocytes, myocytes, fibrocytes and endothelial cells become infected. Damage to endothelial cells, which results in vasculitis, thrombosis, infarction, oedema antinflammatory cell infiltration, accounts for the nodular skin lesions.

A.5. Clinical signs

Picture 21: Nodules on the skin of cattle affected by Lampy Skin Diseases

The incubation period is up to 14 days.

- Animals are weak, tired and stop eating;
- They have fever that sometimes goes down after 1-2 weeks but it goes up again;
- There is a persistent fever accompanied by lacrimation, nasal discharge and a drop in milk yield.
- Superficial lymph nodes become enlarged and there is oedema of the limbs and dependent tissues.
- Multiple circumscribed, firm, well defined skin nodules develop particularly on the head, neck, udder and perineum. Nodules also develop on the mucous membranes of the mouth and nares. These structures are composed of a central plug of necrotic tissue which sloughs leaving a deep ulcer with heal and scar.
- The skin nodules a firm, creamy-gray or yellow mass of tissue;
- Secondary bacterial infection or myiasis can exacerbate the condition. Recovery may take several months. Affected animals are often debilitated and pregnant cows may abort.
- The severity of the disease relates to the strain of virus and the breed of cattle. Domestic breeds (*Bos taurus*) are more susceptible than zebu (*Bos indicus*) cattle. Some animals have few skin lesions and no systemic reaction while others display the full spectrum of clinical signs. Although the mortality rate is usually less than 5%, the economic impact of the disease can be considerable.
- Discharge from eyes and nose becomes mucopurulent and keratitis may develop;
- Limbs may be oedematous and the animal is reluctant to move;

A.6. Diagnosis

- Generalized skin nodules in cattle in an endemic area are highly suggestive of lumpy skin disease.
- Intracytoplasmic inclusions may be demonstrable histologically in recently developed lesions.
- Capripoxvirus particles in biopsy material or desiccated crusts can be identified using electron microscopy.

- The virus can be isolated in lamb testis cell monolayers.
- Serological assay methods include virus neutralization.

A.7. Control

Although there is no specific treatment of lumpy skin disease giving an antibiotic injection may help to stop the damaged skin getting secondary infections.

The prevention and control of the disease may include the following actions:

- In endemic regions, vaccination is the method of control.
- Two modified live vaccines have been used specifically for the control of lumpy skin disease, one based on a South African strain of lumpy skin disease virus and the other on a Kenyan strain of sheep-pox virus.
- A recombinant vaccine providing protection against lumpy skin disease and rinderpest has been developed.
- A better method is preventive immunising by means of vaccination of calves of mother that have been never exposed to the disease; it could be done at any age.
- Calves of cows that have been vaccinated, should be vaccinated after 6 months of age; after that vaccination should be done annually.
- Imported cattle should be vaccinated before introduction into high-risk area;
- Strict quarantine to avoid introduction of infected animals into safe herd;
- Surveillance and eradication policies are appropriate control measures in countries bordering on endemic regions.
- Cattle must be spayed with insecticides during the risk period to limit biting insects.
- Local wound dressing to discourage fly worry and prevent secondary infection;
- When epizootic occurs in an enzootic area and lumpy skin disease has already spread extensively, slaughter policies are inappropriate and extensive vaccination campaigns are recommended.
- Proper disposal of dead animal or incineration; clean and disinfect premises and implements.
- In countries free from lumpy skin disease the import of livestock, carcasses, hide, skin and semen must be done under restrictions.

B. Foot And Mouth Disease (FMD)

B.1. Definition

Foot and mouth disease (Hoof and mouth disease) is a highly contagious disease of even-toed ungulates characterized by fever and the formation of vesicles on epithelial surfaces especially on mouth and hooves.

B.2. Etiology

FMD is caused by an aphtovirus of the family of Picornaviridae, which are icosahedral and non-enveloped; contain a molecule of single-stranded RNA. Virions are 30 nm in diameter. The capsid is composed of 60 identical subunits, each containing four major proteins.

There are 7 immunologically distinct serotypes :A,O,C,Asia1 and SAT(Southern Africa Territories)1,2,3.Within each serotype they are large number of strains that exhibit a spectrum of antigenic characteristics; therefore more than one vaccine strain for each serotype particularly O and A is required to cover antigenic density. Picornaviruses are resistant to ether, chloroform and non-ionic detergents. Individual genera differ in their thermal ability and pH stability.

Aphthoviruses are inactivated outside the PH range of 6,0 -9,0 and by dessicaion and temperature>56^{0}C;viruses can survive a considerable time when associated with animal proteins.

B.3. Epidemiology

Distribution

African swine fever has been reported in large number of countries in Africa;in south Sahara as enzootic;it has been found in Spain(1978) ;Belgium(1985) and other countries of Europe like France ang England**.**The disease has been reported in south America and Asia.

Foot-and-mouth disease virus serotypes geographical distribution

O, A and C South America

O, A and C Eastern European countries

O, A, C, SAT1, SAT2 and SAT3 Africa

O, A, C and Asia1 Asia.

Transmission

Large numbers of virus particles are shed in the secretions and excretions of infected animals.

Virus shedding begins during the incubation period, about 24 hours before the appearance of clinical signs. Transmission can occur by direct contact, by aerosols, by mechanical carriage by humans or vehicles, on fomites and through animal products such as meat, offal, milk, semen or embryos. Because of their large respiratory volume and the low dose of virus required to establish infection, cattle are highly susceptible and are often the first species in which clinical signs are evident. Infected groups of animals, particularly pigs, shed large quantities of virus in aerosols. Under favourable conditions of low temperature, high humidity and moderate winds, virus in aerosols may spread up to 10 km over land. Turbulence is generally less marked over water than over land. In 1981, virus was carried a distance of more than 200 km from France to the south coast of England.

Susceptible hosts

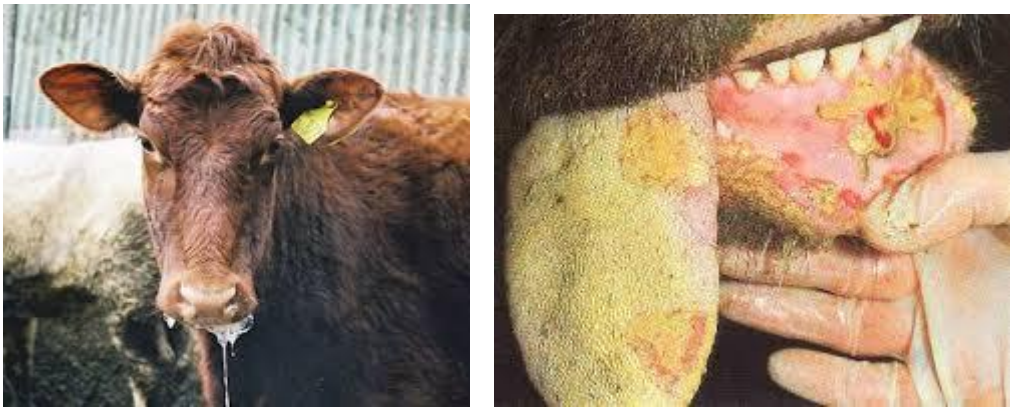
Cattle, sheep, goats, pigs and domesticated buffalo are susceptible to FMD. Several wildlife species including African buffalo, elephants, hedgehogs, deer and antelopes are also susceptible. Horses, dogs and cats are not affected by FMD. They can act as mechanical vectors as can man.

B.4. Pathogenesis

Although infection usually occurs through inhalation, the most efficient method of infection, virus can also gain entry to tissues through ingestion, insemination and inoculation, and through contact with abraded skin. Pigs are more resistant than ruminants to the virus and infection tends to occur through the feeding of contaminated, untreated swill.

Primary viral replication, after inhalation, takes place in the mucosal and lymphatic tissues of the pharynx. Viraemia follows primary multiplication with further viral replication in lymph nodes, mammary glands and other organs as well as the epithelial cells of the mouth, muzzle, teats, interdigital skin and coronary band. In these areas of stratified squamous epithelium, vesicle formation results from swelling and rupture of keratinocytes in the stratum spinosum.

B.5. Clinical signs



Picture 22: Profuse salivation and Ulcers from ruptured blisters in the mouth of cattle with FMD

The incubation period ranges from 2 to 14 days, but is generally shorter than a week.

General signs

- Infected cattle develop fever and inappetence and show a drop in milk production.
- Profuse salivation, with characteristic drooling and smacking of lips, accompanies the formation of oral vesicles which rupture, leaving raw, painful ulcers.
- Ruptured vesicles in the interdigital cleft and on the coronary band lead to lameness.
- Vesicles may also appear on the skin of the teats and udders of lactating cows.

- Infected animals lose condition. Mature animals seldom die. Calves may die from acute myocarditis. Although the virus does not cross the placenta, abortion probably relates to the pyrexial response.
- Although the ulcers tend to heal rapidly, there may be secondary bacterial infection which exacerbates and prolongs the inflammatory process.
- In pigs, foot lesions are severe and the hooves may slough. Marked lameness is the most prominent sign in this species.
- The disease in sheep, goats and wild ruminants is generally mild, presenting as fever accompanied by lameness which spreads rapidly through groups of animals.
- Human infection, usually mild, has been described on rare occasions in laboratory personnel working with the virus and in individuals handling infected animals.

Local signs and lesions

In the mouth:

- Hypersalivation(drooling) because of blisters in the mouth;
- Vesicles (blisters) developed in the mouth, on the tongue, dental pad, gums and lips;
- Quivering of lips.

On the feet

- The animals are very lame (because of rupture of blisters leaving wounds) usually all four feet are painful.
- Vesicles on the coronet (on all legs).

On the teats/udder

- Vesicles appear on teats/udder particularly of the lactating cows and sows
- The calves, lambs, kids and piglets may die without showing any vesicle

B.6. Diagnosis

Foot-and-mouth disease clinically resembles other vesicular diseases of domestic animals including vesicular stomatitis in cattle and pigs, swine vesicular disease and vesicular exanthema in pigs. Consequently, FMD requires laboratory confirmation.

- A confirmatory diagnosis is based on the isolation of FMDV from samples of tissue or vesicular fluid. Epithelium collected from an unruptured or recently ruptured vesicle is ideal for laboratory processing.
- Viral antigen demonstration may be carried out by ELISA.
- In persistent or subclinical infections, samples of oesophageal/pharyngeal fluid can be obtained with a probang (sputum) cup and examined by virus isolation ;

- Virus isolation is carried out in special cell lines such as primary bovine thyroid or kidney cells.
- Demonstration of specific antibody by virus neutralization or ELISA can be used to confirm a diagnosis in unvaccinated animals.

B.7. Prevention

Foot and mouth disease has a major effect on local and international trade arrangement.

- In countries that are free from FMD, the disease is notifiable.
- Affected and in-contact animals are slaughtered;
- After slaughtering, the carcasses are either burned or buried.
- Following an outbreak, movement restrictions are applied and infected premises must be thoroughly cleaned and disinfected.
- Mild acids such as citric acid and acetic acid and alkalis such as sodium carbonate are effective disinfectants.
- Reserves of inactivated virus are maintained in several countries to provide an adequate supply of vaccine at short notice in the event of a major outbreak of the disease. Although ring vaccination around affected premises may help to limit the spread of the disease, it may also allow the development of the carrier state in animals subsequently exposed to the virus.
- In countries where FMD is endemic, efforts are generally directed at protecting high-yielding dairy cattle by a combination of vaccination and control of animal movement. They are usually multivalent, containing three or more virus strains. Protection against antigenicaly-similar strains of virus is satisfactory and lasts for up to 6 months; thus vaccination must be done twice every year.

C. Rinderpest

C.1. Definition

Rinderpest also known as cattle plague is an acute contagious viral disease of wild and domesticated ruminants and pig, affecting mainly cattle and buffalo. The disease is characterized by sudden onset of fever, occular and nasal discharges, erosive stomatitis, gastro-enteritis, dehydration and death.

C.2. Etiology

Virus of Paramyxoviridae, genus of Morbillivirus, 150 nm or more in diameter and enveloped; they are single-stranded RNA.

The attachment proteins allow the virus to bind to cell surface receptors and the fusion protein causes the virus envelope to fuse with the host cell membrane. The labile virions are sensitive to heat, desiccation, lipid solvents, non-ionic detergents and disinfectants.

C.3. Epidemiology

Domestic cattle, buffalo and several wildlife species including giraffe, warthog, Cape buffalo and eland are highly susceptible to infection. Gazelles and small domestic ruminants are less susceptible. Asiatic breeds of domestic pig develop disease whereas infection in European breeds is sub-clinical. Transmission, which occurs through aerosols, usually requires close contact as the virus is labile and remains viable in the environment for short periods only.

Virus shedding in all secretions and excretions begins a few days before clinical signs develop. In endemic areas, the disease is usually mild and is restricted to young cattle in which maternally-derived immunity has declined. As there is no carrier state, maintenance of infection requires continuous transmission to susceptible animals. All ages of animals are affected in epidemics. Morbidity may reach 90% and mortality can approach 100%.

C.4. Pathogenesis

Following transmission through close contact or by aerosols (inhalation of the virus) replication occurs primarily in the respiratory tract, in the pharyngeal and mandibular lymph nodes. Viraemia develops within 3 days, resulting in spread to other lymphoid tissues and to the mucosae of the respiratory and digestive tracts. Leukopenia and immunosuppression follow necrosis in lymphoid tissues. Virus shedding, which continues throughout the acute phase of the disease, subsides a few days after body temperature returns to normal.

C.5. Clinical signs and lesions

Picture 23: Occulonasal discharge and Mucosal erosion of cattle affected by Rinderpest

- After an incubation period of 3 to 9 days, infected animals develop a fever and become anorexic and depressed.
- Mucosal erosions in the mouth and nasal passages become evident within 5 days.

- Profuse salivation is accompanied by mucopurulent oculonasal discharge and the muzzle appears dry and cracked;
- As the disease progresses the animal becomes photophobic and tends to go under trees to hide from sunlight.
- Pinpoints necrotic lesions which rapidly enlarge to form cheesy plaques appear on the gums, buccal mucosal and tongue.
- About 3 days after the appearance of the mucosal ulcers fever regresses and profuse diarrhoea develops.
- The dark fluid faeces often contain mucus, necrotic debris and blood. Dehydration and wasting soon become evident. Animal shows severe abdominal pain, thirst and dyspnea and may die from dehydration.
- Severely affected animals may collapse and die within 12 days of the onset of clinical signs. In surviving animals, convalescence lasts several weeks. Secondary infections and activation of latent protozoal infections are frequent complications. Pregnant animals may abort during the convalescent period.
- Gross pathologic changes are evident through GT tract and upper respiratory tract, either as areas of necrosis and erosion or congestion and haemorrhages. The later creating classic zebra-stripping in rectum. Lymph nodes may be enlarged.

C.6. Diagnosis

Clinical and pathological findings may be sufficient for diagnosis in endemic areas. They may also be adequate in individual animals in outbreaks which have been confirmed by laboratory tests. Lesions are found throughout the gastrointestinal tract with characteristic congestion and haemorrhage along the longitudinal folds of the large intestine, referred to as 'zebra striping'.

In regions where rinderpest is uncommon or absent, laboratory confirmation is required to differentiate it from bovine viral diarrhoea, infectious bovine rhinotracheitis, malignant catarrhal fever and foot-and-mouth disease. When investigating an outbreak, specimens for laboratory examination should be collected from several febrile animals which have not developed diarrhoea.

The presence of rinderpest virus, which produces cytopathic effects in cell cultures, can be confirmed by immunofluorescence.

Post mortem enteric lesions are characteristic but not pathognomonic. Congestion of the folds of the colonic mucosa often produces a zebra-stripe pattern.

C.7. Prevention

The preventive methods and control are depending on the presence or absence of the disease in the country.

- In countries free of rinderpest, control is based on different actions restriction of animal movement, quarantine of imported animals and slaughter of infected animals.
- In endemic areas, control is achieved by vaccination of domestic cattle and buffaloes with a modified live tissue-culture-based vaccine that induces immunity lasting at least 5 years. This stable freeze-dried vaccine is thermo-labile following reconstitution. Recombinant vaccinia and capripoxvirus vaccines expressing either haemagglutinin protein or fusion protein of rinderpest virus have high heat stability and have been used to protect cattle.
- Control of animal movement is the single most important measure for preventing disease transmission.

D. Bluetongue

D.1. Definition

Bluetongueis non-contagious arthropoborne viral disease primarily of domestic and wild ruminants is transmitted by biting insects, principally *Culicoides* species.

D.2. Etiology

Causal agent, bluetongue virus (BTV), belongs to a distinct serogroup in the *Orbivirus* genus in the family of Reovirudae. There are at least 24 serotypes worldwide.

D.3. Epidemiology

Distribution

Bluetongue is widely distributed between latitudes 53°N and 40°S, reflecting the distribution of *Culicoides* species. *Culicoides imicola* is the principal vector in Africa, Mediterranean Basin and the Middle East. In Australia, *Culicoides fulvus, Culicoides wadai* and *Culicoides brevitarsis* are involved in transmission. Other *Culicoides* species of importance in transmission are *C. varipennis* var. *sono-rensis* in North. America and *Culicoides insignes* in South America. Since 2006, BTV-8 has caused a severe epizootic in Northern Europe. Indigenous European *Culicoides* species including *C.dewulfi* and *C. obsoletus* complex appear to be capable of maintaining the epizootic from one vector season to the next.

Transmission

Female midges feeding on viraemic animals become infected and virus replicates in their tissues. *Culicoides* species can transmit virus in saliva within 7 to 10 days and they remain infected for life.

Temperatures of 18°C to 29°C along with high humidity favour insect activity, while temperatures greater than 12°C are required for virus replication within the vector. These factors account for the seasonal occurrence of the disease in many parts of the world. *Culicoides* species are most active at dawn and dusk. In localized areas within endemic regions, there may be an increased frequency of BT outbreaks. These areas are particularly suitable for the breeding of *Culicoides* species because of the accumulation of animal faeces in marshland.

Extension of disease to contiguous areas occurs through the movement of viraemic animals or insect vectors. Although the flight range of *Culicoides* species is limited, they may be transported over long distances by wind movement resulting in BT outbreaks in susceptible ruminant populations outside endemic regions. Such events may precipitate epidemics which are usually self-limiting unless the climate is suitable for vector activity throughout the year.

It is considered that four potential routes may operate for the virus to overwinter in ruminant populations, permitting viral recrudescence in spring: vertical transmission in ruminants (dam to offspring), prolonged subclinical Viremia in certain infected individual animals, vertical transmission in the vector with survival of infected offspring through the winter, and survival of infected adult midges.

In endemic areas, infection of cattle is common and usually unapparent. The viraemia in cattle commonly lasts several weeks facilitating acquisition of virus by insect vectors. Consequently, cattle are considered to be important reservoirs of virus. During the viraemic phase, virus can be detected in the semen of a proportion of rams and bulls.

Venereal and transplacental transfers of BTV infection are generally considered to be uncommon but the strain of BTV-8 circulating in northern Europe appears to be capable of vertical transmission in the ruminant population. Embryos collected from infected ewes may transmit infection to recipient ewes but this can be prevented by washing the embryos extensively.

D.4. Pathogenesis

After experimental infection, the virus replicates initially in regional lymph nodes. It is then carried in blood or lymph to other lymphoid tissues, lungs and spleen where further replication takes place. Virus localizes and multiplies in the endothelium of small blood vessels producing vascular damage with stasis, exudation and tissue hypoxia.

The initiation and development of surface lesions in areas of tissue hypoxia relate to minor trauma and may be complicated by secondary bacterial infection.

Lesions are particularly evident in the oral cavity, around the mouth and on the coronet of the hoof. In the bloodstream, the virus is highly cell-associated, particularly with erythrocytes. It has been suggested that this may protect virus from antibody and allow the virus to persist. Infectious virus can be detected for 35 to 60 days after infection. Sporadic cases of clinical disease in cattle are thought to involve type I hypersensitivity reactions with participation of IgE as a result of previous exposure to BTV or related orbiviruses.

D.5. Clinical signs and lesions

Picture 24: Cyanotic tongue of the goat with Blue tongue disease

The clinical presentation, which is highly variable, ranges from subclinical infection to severe disease with high mortality. Severe disease is generally confined to Merinos and European mutton breeds. Nutritional status, exposure to sunlight and age also appear to influence the severity of lesions:

- The incubation period in sheep is up to 10 days.
- Affected animals are febrile and depressed with vascular congestion of the lips and muzzle. Oedema of the lips, face, eyelids and ears develops. Erosions and ulcers are evident on the oral mucosa.
- There is excessive salivation and a watery discharge that subsequently becomes mucopurulent and dries forming crusts around the nares.
- The tongue may be swollen and cyanotic.
- Lameness may result from coronitis and laminitis.
- Some animals develop torticollis.
- Abortion may occur and lambs may be weak or deformed at birth.
- Mortality rate may be up to 30% and, in some outbreaks, may be higher.
- Animals recovering from infection may lose part of their fleece some weeks later.
- Clinical cases in susceptible cattle are characterized by fever, stiffness, ulceration of the oral mucosa, 'burnt muzzle' and dermatitis.

- Cattle infected during pregnancy may abort or give birth to malformed calves.

D.6. Diagnosis

In areas where the disease is endemic, a presumptive diagnosis of BT may be based on clinical findings and post-mortem lesions:

- Diagnostic methods include identification of the virus or demonstration of BTV-specific antibodies in non-vaccinated animals.
- Samples suitable for virus isolation include unclotted blood from febrile animals or fresh spleen and lymph node collected at post-mortem. Virus may be isolated by intravenous inoculation of embryonated eggs or in cell culture.
- Antigen detection ELISA systems have also been described.
- Laboratory confirmation is based on virus isolation in embryonated chicken eggs or mammalian and insect cell culture.
- Serological tests for the detection of antibodies to the BTV.

D.7. Prevention

Populations of insect vectors may be reduced by the use of:

- Larvicides at breeding sites.
- Insecticides applied to susceptible animals may temporarily halt feeding by vectors.
- Live attenuated vaccines have been used successfully for many years and provide protection against virulent virus of homologous serotype.
- Polyvalent vaccines are essential in regions where a number of serotypes are present.
- Attenuated vaccines may produce viraemia and may be teratogenic when used in ewes during the first half of gestation. They should not be used during periods of vector activity because of the risk of transferring vaccinal virus to pregnant ewes and the possibility of genetic reassortment with field virus and reversion to virulence.
- Killed adjuvanted vaccines can induce protection but are more expensive to produce and require two inoculations.
- Recombinant virus-like particles, capable of inducing protective immunity, have been produced in insect cells infected with recombinant baculoviruses expressing BTV proteins. However, vaccines produced by this method are not yet available commercially.

E. Small Ruminant Pest

E.1. Definition

Small ruminant pest (Peste des petits ruminants) is acontagious viral disease of goat and less commonly in sheep and it is characterized by fever, erosive stomastitis, enteritis, pneumonia and death.

E.2. Etiology

Peste des petits ruminants virus is classified in the family Paramyxoviridae, genus Morbivirus which closely ressembles the rinder peste virus. The virus is destroyed at 50^0C during one minute; PH stable between 5.8-10.0 thus inactivated at PH<4.0 or 11.0>. Susceptible to most disinfectants e.g. Phenol and sodium hydroxide 2%.

E.3. Epidemiology

Peste des ruminants (PPR) represented one of the most economically important animal diseases in the area that rely on small ruminants. Outbreaks tend to be associated with contact of animal from endemic area.

Morbidity rates in susceptible population can reach 50-100%; mortality rates vary among susceptible animals but can reach 50-100% in more severe instance.

Both morbidity and mortality rates are lower in endemic areas and in adult when compared to young animals.

Distribution

PPR was first described in Côte d'Ivoire, but it occurs in most African countries south of the Sahara and north of the equator, and nearly all Middle Eastern countries up to Turkey It is also wide-spread in India and south- west Asia. Recent incursions into China (Tibet) and Morocco have caused serious disease outbreaks and disease has been reported to be moving southwards in East Africa

Transmission

- Mainly by aerosols by direct contact between animals living in close quarters;
- Fomites may be means of spreading infection, bedding, feed and water troughs;
- No carrier state;
- Seasonal variations: more frequent outbreaks during the rainy season or the dry cold season;

Source of infection: tears, nasal discharge, coughed secretion, and all secretions of incubating and sick animals.

Susceptibility

Goats (predominantly) and sheep;

Cattle and pigs develop unapparent infections and do not transmit disease

E.4. Pathogenesis

The pathogenesis of PPR is similar to that of rinderpest. Mucosal erosions and profuse diarrhoea are features of the condition. During the acute phase of the disease, virus is shed in all secretions and excretions.

E.5. Symptoms and lesions

Incubation period is 4-10 days;

Peracute form

It is characterised by:

- High fever, depression and death;
- Higher mortality.

Acute form

It is characterised by:

- Sudden rise of body temperature (40-41^{0}C); animals become depressed or restless, anorexic and develop a dry muzzle and dull coat; pyrexia can last for 3-5 days.
- Serous nasal discharge becoming mucopurulent;
- Within4 days of onset of fever, gums become hyperaemic, and erosive lesions develop in the oral cavity with excessive salvation;
- Necrotic stomatitis is common;
- Small areas of necrosis on the visible mucous membranes
- Congestion of conjunctiva;
- Severe watery, blood stained-stained diarrhea is common in later stages;
- Bronchopneumonia evidenced by coughing is a common feature; rales and abdominal breathing;
- Abortion may occur;
- Dehydration, emaciation, dyspnoea, hypothermia and death may occur within 5-10 days. Survivors undergo long convalescence.

Subacute form

It is characterised by:

- Frequent in some areas because of local breed susceptibility; form commonly seen in experimentary infected animals.

- Usually 10-15 days development with inconsistent signs; on or about 6th day post-infection, fever and serous nasal discharge is observed.
- Fever falls with onset of diarrhea and, if this is severe, may result in dehydration and prostration.
- Lesions associated with PPR are very similar to those observed in cattle affected with rinder pest, except prominent crusty scabs along the outer lips and severe interstitial pneumonia frequently occur with PPR.
- Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lip and adjacent gum near the commissures and the portion of the tongue.
- Lesions on the hard palate, pharynx and upper third of the oesophagus in severe case;
- Rumen ,reticulum and abomasum rarely have lesions;
- Small streaks of haemorrhages and sometimes erosion in the first portion of the duodenum and the terminal ileum;
- Congestion around the ileo-coecal valve, at the coeco –colic junction and in the rectum;
- 'Zebra stripes'of congestion in the posterior part of the colon;
- Small erosions and petechiae on the nasal mucosa, larynx and trachea;
- Bronchopneumonia is a constant lesion;
- Possibility of pleuritis and hydrothorax;
- Congestion and enlargement of spleen;
- Congestion, enlargement and oedema of most of the lymph nodes.

E.6. Diagnosis

Clinical diagnosis

A presumptive diagnosis is based on clinical, pathologic and epidemiologic findings and may be confirmed by laboratory examination.

Sample to be taken for laboratory may include:

- Swabs of the conjunctival discharges and from the nasal and buccal mucosa;
- For serologic needs ,clotted blood can be collected at the end of an outbreak;
- Upon necropsy aseptically collect the following tissues chilled on ice and transported under refrigeration: lymph nodes (especially the mesenteric and bronchial nodes), spleen and lung.

Differential diagnosis

Rinderpest, contagious caprine pleuropneumonia, contagious ecthyma, pasteurellosis, foot and mouth disease, heartwater, coccidiosis, blue tongue, mineral poisoning.

E.7. Prevention

When the disease appears in previously PR-free zones or countries:

- Rapid identification, humane slaughter and disposal of affected animal, carcasses burned and buried.
- Strict quarantine and control of animal movements;
- Effective cleaning and disinfection of contaminated areas of all premises with lipid solvent of low or high PH and disinfectants as described above;
- Monitoring of wild and captive animals.

Endemic outbreak situation:

- Vaccination
- Monitoring of wild and captive animals especially avoiding contact with sheep and goat;
- Exposed or infected animal should be slaughtered and carcases burned with deep burial.

F. Sheeppox and Goatpox

F.1. Definition

Sheepox and Goatpox are serious, often fatal, disease characterized by widespread skin eruption. Both diseases are closely related antigenically and physico-chemically. They are also related to the virus of lumpy skin disease.

F.2. Etiology

Sheeppox and goatpox viruses of sheeppox and goatpox are members of the genus Capripoxvirus. A range of capripoxvirus strains have been isolated from sheep and goats and there is evidence of recombination between strains. Although some strains are extremely pathogenic in both sheep and goats, other strains produce severe disease in only one of these species. Recent genetic studies suggest that sheeppox virus and goatpox virus are phylogenetically distinct viruses.

F.3. Epidemiology

Both of these diseases are endemic in south-eastern Europe, the Middle East, Africa and Asia. The virus particles are shed from skin lesions and in ocular and nasal discharges during the acute stages of the disease. Infection occurs through skin abrasions or by aerosol.

Biting insects may also transmit the virus mechanically. Housing or stocking animals facilitates transmission of the virus. Following infection, capripoxvirus strains induce immunity.

In endemic areas, where indigenous animals frequently have a high level of naturally acquired immunity, generalized disease and mortality are rare. In isolated flocks, outbreaks of severe disease may occur.

F.4. Pathogenesis

The virus replicates locally either in the skin or in the lungs. Spread to the regional lymph nodes is followed by viraemia and replication in various internal organs. Skin lesions, typical of poxvirus infection, appear about 7 days post infection. Lung lesions present as multiple nodular areas of consolidation.

F.5. Clinical signs

Picture 25: Lesions on the mouth and nose of the sheep affected by sheeppox

Following an incubation period of about 1 week, infected animals show the following symptoms and lesions:

- Fever, oedema of the eyelids, conjunctivitis and nasal discharge.
- Within a few days, macules which rapidly develop into papules appear on the skin and external mucous membranes.
- Scabs form overnecrotic papules.
- The severity of the clinical signs depends on the breed, age, immune status and nutritional status of the host animal as well as the strain and virulence of the infecting capripoxvirus
- Lesions in mild afebrile disease may be minimal and confined to the skin beneath the tail.
- Mortality rates with infections from some strains of capripoxvirus may be up to 50% even in indigenous breeds.
- The disease is most severe in young animals and in imported breeds. In some European breeds, which are extremely susceptible, mortality rates may approach 100%.
- Secondary bacterial infection or dissemination of the virus into other sites may result in a severe form of the disease.

F.6. Diagnosis

Diagnosis can often be made solely on clinical grounds:

- Skin biopsies or post-mortem specimens may be used for laboratory confirmation.
- Eosinophilic intracytoplasmic inclusions may be demonstrable histologically in epidermal cells.
- Electron microscopy can be used for the rapid identification of poxvirus particles in material from lesions.
- Capripoxviruses can be readily distinguished from parapoxviruses in differential diagnosis.
- Virus may be isolated in lamb testis or kidney cell monolayers.
- An antigen-trapping ELISA has been developed for the detection of capripoxvirus antigen.

F.7. Prevention

In endemic areas, control is based on annual vaccination:

- All capripoxviruses share a major neutralizing site, inducing good cross-protection against all field strains of virus.
- Several modified live vaccines are available including a Kenyan sheeppox strain used in sheep and goats, a Romanian strain used in sheep, and the Mysore strain used in goats.
- Inactivated vaccines are not recommended as they are less effective than modified live vaccines because cell-mediated immunity is the predominant protective response.
- Capripoxviruses are being employed as vectors for important ruminant viral vaccines.
- These vector vaccines may provide protection against diseases caused by capripoxviruses as well as diseases such as rinderpest and peste des petits ruminants.

G. Contagious ecthyma

G.1. Definition

Contagious ecthyma, is the important disease of sheep, also known as contagious pustular dermatitis or Orff occurs worldwide and is caused by a parapoxvirus. Goats, camels and humans are also susceptible to infection. The disease affects primarily the lips of young animals and it is severe in goat than in sheep.

G.2. Etiology

Poxvirus related to those of pseudo-cowpox and bovine popular stomatitis.

G.3. Epidemiology

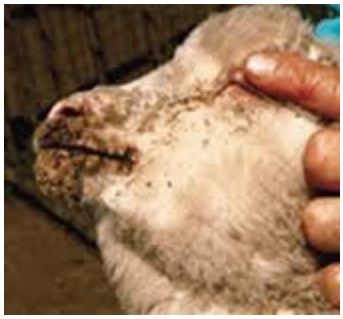
The virus is transmitted through direct or indirect contact. Under dry environmental conditions, the virus is stable and can survive in scab material for months. Infectivity is substantially reduced after exposure to adverse climatic conditions. In most flocks, infection is maintained by sheep with chronic lesions.

G.4. Pathogenesis

The virus, which is epitheliotropic, produces proliferative wart-like lesions following entry through skin abrasions such as those acquired from thistles. The virus replicates in epidermal keratinocytes, and infected cells release a vascular endothelial growth factor, which stimulates angiogenesis in the skin The virus encodes a number of factors that interfere with inflammation and the local immune response including a homologue of interleukin 10,a chemokine binding protein and a granulocyte-macrophage colony stimulating factor interleukin 2 binding protein.

Papular lesions progress to vesicles, pustules and eventually scab formation. Proliferation of cells underlying scabs produces verrucose masses. In the absence of secondary bacterial infection, lesions usually heal within 4 weeks.

G.5. Clinical signs



Picture 26: Lesions on the lips for goat suffering contagious ecthyma

It is characterised by:

- The disease primarily affects young sheep
- The incubation period is up to 7 days.
- Although lesions most often occur on the commissures of the lips and on the muzzle, they may also develop in the mouth and on the feet, genitalia and teats. Mild lesions may go unnoticed.
- Severely affected lambs with lesions in the buccal cavity often fail to eat, lose condition and may die.
- Outbreaks last for some months and vary in severity from farm to farm and from year to year. The disease does not usually recur until the birth of new susceptible lambs in the flock. Environmental management factors may influence the outcome of infection.

- In chronically infected sheep, lesions may be either mild or proliferative.
- Immunity to the virus requires a cell-mediated immune response and neonatal lambs are susceptible to infection despite receipt of colostrum's from previously infected ewes.

G.6. Diagnosis

Lesions of orf are readily recognized by their characteristic appearance and distribution:

- Virus present in lesion material can be identified by electron microscopy.
- Concurrent bacterial infection with oedema of the head and swelling of the tongue, which could be confused with bluetongue, has been described.
- Lesions in animals previously affected are usually less severe and heal more rapidly than those developing after first exposure.

G.7. Treatment and Prevention

There is no specific treatment for infection with orf virus:

- Antibiotic therapy reduces the effect of secondary bacterial infection in young lambs.
- In endemically infected flocks, control is based on the use of a fully virulent live vaccine derived from scab material or cell culture.
- Ewes should be vaccinated by scarification in the axilla at least 8 weeks before lambing. When close to lambing, they must be moved to a new grazing area in order to minimize exposure of lambs to infectious vaccinal scab material.
- Lambs should be vaccinated only if an outbreak occurs in a flock.
- If carried out effectively, thorough cleaning and disinfection of surfaces and equipment between periods of housing may reduce the amount of residual virus in buildings.
- Humans are susceptible to infection with orf virus. Typically, a single lesion occurs on hands, forearms or face. Care should be exercised when handling infected sheep and when using live vaccines.

H. Classical Swine Fever

H.1. Definition

Classical swine fever is a contagious febrile disease of pigs caused by a pestvirus of the family Flaviviridae.

H.2. Etiology

Classical swine fever is caused by a small, enveloped RNA virus in the genus of Pestvirus of the family Flaviviridae

H.3. Epidemiology

Distribution

Classical swine fever was first described in USA in 19th century, then in Europe termed Swine fever; both names are used to distinguish it from Africa swine fever. The virus is moderately fragile and does not persist in the environment or spread long distances by the air borne route. It can survive for prolonged periods in a moist, protein-rich medium such as meat, other tissues, and body fluids particularly if kept cold or frozen. Classical swine fever is distributed worldwide. It is endemic in parts of Latin America, Caribbean islands, Asia. It has been reported in South Africa. Australia, New Zeeland, Canada, and USA are free from Classical swine fever.

Transmission

The source of infection is the pig; movement of infected pig spreads the disease. Mechanical transmission on vehicles, and equipment as well as personal (notably veterinarians) travelling between pigs farms are also significant means of spread within an infected areas.

Susceptible hosts

Classical swine fever virus infects only suidae (pigs and wild boar) but experimental infection can be induced to other species.

H.4. Clinical symptoms and lesions

Picture 27: Hemorrhage and cyanosis in the skin of pig affected by Classical Swine Fever

Severe acute form is characterized by:

- Fever, inappetance, and depression.
- The incubation period typically 3-7 days with death at 10-20 days after infection; fever (>41^{0}C) persists until the terminal stage of the disease.
- Constipation is common followed by diarrhea.

- The principal lesion is generalized vasculitis seen in live pig as haemorrhages and cyanosis in the skin notably in extremities. There may also be a generalized erythema.
- Vasculitis in CNS may produce incoordination or even convulsion.
- At necropsy 'the principal findings are widespread petechial and ecchymotic haemorrhages, especially in lymph nodes, bladder, and larynx. Infarction may be seen, notably in spleen.

In chronic disease:

Pigs often survive for up to3 months. After an initial acute febrile phase, they may show apparent recovery but then relapse, with anorexia, depression, fever, and progressive loss of condition. Histologically, there is atrophy of the thymus and lymphoid depletion.

H.5. Diagnosis

Clinical signs are not necessarily typical, differential and laboratory diagnosis generally follows. Differential diagnosis includes other febrile haemorrhagic diseases of pigs such as: African swine fever, bacterial septicaemia (salmonellosis, erysipelas), anticoagulant poisoning (coumarin derivatives).

Serologic tests for antibody area required for antibody detection, in this case clotted blood is taken.

Antigen detection can be performed using direct immunofluorescence on frozen tissues section particularly of tonsil.

H.6. Prevention

No treatment is possible; and preventives measures include the following:

- Affected pigs mu be slaughtered and the carcasses buried and incinerated;
- In countries where the virus is endemic, infected animals are destroyed, and vaccination is used to prevent further spread of the virus.
- Countries free of classical swine fever implement measures to avoid outbreaks of the disease.
- I n wild boar outbreaks, emergency vaccination using baits with modified live vaccines has been used successfully in Germany and other European countries.

I. African Swine Fever

I.1. Definition

African swine fever (ASF) is an economically important contagious viral disease of pigs, characterized by fever, haemorrhages in many tissues and a high mortality rate.

I.2. Etiology

The virus is from Asfarviridae family.

It is DNA virus transmitted by arthropods. Virions are 175 to 215 nm in diameter and consist of a membrane-bound nucleoprotein core inside an icosahedral capsid surrounded by an outer lipid-containing envelope modification of mRNA. African swine fever virus is stable in the environment over a wide range of temperature (4°C to 20°C) and pH values. The virus may persist for months in meat. Infectivity can be destroyed by heating and by treating with lipid solvents and some disinfectants such as orthophenylphenol, formalin and halogen compounds. African fever virus(ASFV)in body fluids and serum is inactivated in 30 minutes at 60^0C.The virus in unprocessed meat can remain viable for several weeks.

I.3. Epidemiology

Distribution

It is endemic in sub-Saharan Africa, Madagascar and Sardinia. Outbreaks have occurred in Belgium, Italy, The Netherlands, Russia, Malta, Brazil, Cuba, Haiti and the Dominican Republic. The Iberian Peninsula was declared free of the disease in 1995, almost 30 years after initial introduction into theregion, but ASFV reappeared in 1999 in Portugal. It has been eradicated from South America and the Caribbean.

Susceptible hosts

Domestic and wild pigs are the only species susceptible to infection.

Transmissions

In Africa, ASFV is maintained in a sylvatic cycle involving soft ticks of the genus *Ornithodorus* and unapparent infection of warthogs and bush pigs.

Adult warthogs with persistent unapparent infection rarely develop viraemia. In contrast, young warthogs develop viraemia and are a major source of virus for soft ticks. Replication of virus occurs in the ticks and both transovarial and transstadial transmission have been described. Soft ticks feed for short periods on hosts before dropping off and sheltering in crevices in walls or cracks in the ground.

As ticks are able to maintain and transmit the virus to pigs for years, the presence of ticks in a particular region makes the eradication of ASF difficult. The principal tick species involved in transmission are O. porcinus (*O.moubata*) in Africa and *O.erraticus* in Spain and Portugal. Experimentally, several other *Ornithodorus* species support virus replication.

Virulent strains of ASFV, producing high mortality in infected animals, are widely distributed in Africa. Many isolates from other parts of the world are less virulent and mortality rates are usually below 50%.

Following infection of domestic pigs with virulent virus, body fluids and tissues contain large quantities of virus until death or recovery occurs. Ingestion of uncooked meat from infected warthogs or domestic pigs is a major method of transmission. Spread can also occur by direct contact usually through oral or nasal secretions.

Occasionally, animals become infected by contact with blood shed as a result of fighting. Indirect transmission can occur through contaminated transport vehicles, fomites and footwear. Feeding uncooked swill is an important mechanism of spread of ASF internationally, with outbreaks often starting in herds close to airports and ports.

Pigs which have recovered from clinical disease may remain infected for long periods. Carrier pigs are considered to be important sources of virus dissemination. Although recovered pigs are clinically unaffected by challenge with genotypically related ASFV, the challenge virus may replicate and spread to other pigs.

I.4. Pathogenesis

Domestic pigs are usually acquired via the oronasal route. The virus replicates initially in pharyngeal mucosa, tonsils and the regional lymph nodes. Infection then spreads by the bloodstream to other lymph nodes, bone marrow, spleen, lung, liver and kidneys. Secondary replication in these sites results in prolonged viraemia.

Although the virus replicates primarily in cells of the lymphoreticular system, it can also infect megakaryocytes, endothelial cells, renal epithelial cells and hepatocytes.

I.5. Clinical signs and lesions

Picture 28: Typical reddening of the ears of pig with ASF (African Swine Fever)

The clinical signs of ASF which range from unapparent to Peracute: relate to the challenge dose and virulence of the virus and to the route of infection.

- The incubation period, which may extend from 4 to 19 days, is typically 5 to 7 days in acute cases.
- Animals with peracute disease die suddenly without premonitory clinical signs. Fever, inappetance, depression and recumbency are features of disease. Cutaneous hyperaemia and, in some cases, haemorrhages may be evident. Other signs include dyspnoea, conjunctivitis, diarrhea, bleeding from the nose and rectum, and abortion.
- The mortality rate is high.
- Subacute disease has a course of 3 to 4 weeks. Clinical signs include pneumonia, swollen joints, emaciation, depression and inappetance.
- Mortality rates, which are variable, depend on the age and general health of infected pigs. Animals may recover clinically or may develop a chronic form of the disease, which usually occurs in regions where ASFV is endemic.
- The immune mechanisms responsible for recovery and protection from ASFV are poorly understood. Cell-mediated immunity is considered to be an important component of the immune response.
- Lesions include splenic enlargement, swollen haemorrhagic gastro hepatic and renal lymph nodes, subcapsular petechiation in the kidneys, petechial and ecchymotic haemorrhages on serosal surfaces, oedema of the lungs and hydrothorax. The widespread haemorrhages result from disseminated intravascular coagulation, endothelial damage and destruction of megakaryocyte. Leukopenia is marked.
- Because the virus does not appear to replicate in T and B lymphocytes, it has been suggested that the lymphopenia and necrosis of lymphoid organs follows apoptosis of lymphocytes induced by proinflammatory cytokines.
- Lesions in chronic disease include pneumonia, fibrinous pleuritis and pericarditis, pleural adhesions and hyperplasia of lymphoreticular tissues.

I.6. Diagnosis

Laboratory confirmation of ASF is mandatory because the clinical signs and lesions, which occur in some other important pig diseases such as classical swine fever, erysipelas and septicaemic salmonellosis, are similar:

- Suitable samples for laboratory examination include blood, serum, tonsil, spleen and lymph nodes.

- Direct immunofluorescence, which is fast and economical, can be carried out on impression smears or cryostat sections. However, the sensitivity of the test is only 40% in pigs with subacute or chronic ASF because of the blocking action of bound antibody in antigen-antibody complexes.
- Antibodies persist for long periods in recovered animals. Serological testing may be the only means of detecting animals infected with strains of low virulence.
- Techniques for detecting antibodies to ASF include ELISA and immunoblotting.

I.7. Prevention

A successful vaccine is not yet available and the following are prevention methods:

- Inactivated vaccines do not induce protection. Although live attenuated vaccines induce protection against challenge with homologous virus strains in some pigs, a proportion of these animals become carriers and may develop chronic lesions.
- Countries maintain disease-free status by prohibiting importation of pigs and pig products. Waste food scraps from aircraft and ships must be boiled before inclusion in pig feed. In the face of an outbreak of ASF in countries free of infection, an eradication policy is implemented. The occurrence of low virulence strains renders eradication difficult.
- Restriction of pig movement, serological monitoring of carrier pigs and prevention of contact between domestic pigs and warthogs or ticks are important control measures in countries where the disease is endemic.
- Eradication of tick species which act as vectors of ASFV is an essential part of a control programme.

J. Fowlpox

J.1. Definition

Fowlpox is the disease which affects domestic poultry, including chickens and turkeys, is caused by infection with fowlpox virus. The infection is slow-spreading and characterized by proliferative skin lesions and diphtheritic lesions in the upper digestive and respiratory tracts. Fowl pox has a worldwide distribution.

J.2. Etiology

Avipoxvirus family of Poxviridae, resistant and may survive for several years in dried scabs.

J.3. Epidemiology

Fowlpox, pigeonpox and turkeypox viruses are closely related and are not strictly host-specific. Several avian species are susceptible to infection with fowlpox virus.

Transmission occurs by contact and by mechanical transfer on the mouthparts of biting arthropods, particularly mosquitoes. Virus enters the body through abrasions on unfeathered skin, on oral mucosa or on respiratory mucosa. Aerosols generated from scab material may result in transmission by inhalation. There is evidence that the virus may persist in some birds with reactivation occurring as a result of stress or immunosuppression.

J.4. Pathogenesis

Virus multiplication occurs at the site of introduction and may be confined to that site when the strain of infecting virus is of low virulence. Infections caused by virulent strains result in viraemia with replication in internal organs. The route of introduction influences the distribution and severity of lesions. Factors such as malnutrition, debilitation and stress may contribute to the severity of the disease.

J.5. Clinical signs

The incubation period is up to 14 days. Two forms of fowlpox have been described: a **cutaneous form** (dry pox) and a **diphtheritic form** (wet pox).

Picture 29: Cutaneous and Diphteric form in chicken suffering Fowlpox

In the cutaneous form:

Nodular lesions develop on the comb, wattles and other unfeathered areas of skin. Progression to vesicle formation is followed by ulceration and scab formation. Healing occurs within 2 weeks. In severely affected birds, lesions may involve both feathered and unfeathered areas of skin and involvement of the eyelids may lead to complete closure.

In the diphtheritic form of the disease:

Yellowish necrotic lesions (cankers) develop on the mucous membranes of the mouth, oesophagus and trachea:

- Oral lesions may interfere with eating. Tracheal involvement may lead to laboured breathing and rales.
- The mortality rate, which is higher in birds with the diphtheritic form than the cutaneous form, may approach 50% in severe outbreaks, particularly when accompanied by secondary bacterial or fungal infection.
- Economic losses are largely due to a transient drop in egg production in laying birds and reduced growth in young birds.

J.6. Diagnosis

- Typical poxvirus particles can be demonstrated by electron microscopy in material from lesions.
- Virus may be isolated on the chorioallantoic membrane of 9 to 12-day-old embryonated eggs.
- Suitable serological tests include ELISA, virus neutralization, agar gel precipitation and passive haemagglutination.

J.7. Treatment and control

There is no specific treatment. Control of secondary bacterial infection is desirable.

- In endemic areas, improved management and hygiene along with regular vaccination have reduced the effect of the disease on commercial poultry production. Modified live fowlpox or pigeonpox virus vaccines, produced in tissue culture or chick embryo, are available commercially.
- Chickens are usually vaccinated at about 1 month of age.
- Recombinant vaccines employing fowlpox and canarypox viruses are being developed for use not only in birds but also in mammalian species.

K. Gumboro Disease

K.1. Definition

Gumboro disease or Infectious bursal disease is a highly contagious disease of young chickens which is caused by infectious bursal disease virus (IBDV). The causal agent was first isolated in Gumboro, Delaware, and the disease was originally known as Gumboro disease.

K.2. Etiology

Birnaviruses are so named because their genomes contain two segments (A, B) of linear, double-stranded RNA. Genetic reassortment has been described. The icosahedral virions are about 60 nm in diameter.

Virions, which are non-enveloped, are stable over a wide pH range and at a temperature of 60°C for 1 hour. They are resistant to treatment with ether and chloroform.

K.3. Epidemiology

Infection, which is usually acquired by the oral route, occurs when maternally-derived antibody levels are waning at 2 to 3 weeks of age. Virus is shed in the faeces for up to 2 weeks after infection and can remain infectious in the environment of a poultry house for several months.

Spread to other poultry units can occur by fomites. Neither a carrier state nor vertical transmission has been demonstrated.

K.4. Pathogenesis

Within hours of ingestion, virus can be detected in macrophages and lymphoid cells in the caeca, duodenum and jejunum. Virus reaches the liver via the portal circulation and infects Kupffer cells. Infection spreads to the bursa of Fabricius where rapid replication results in a pronounced secondary viraemia and dissemination to other tissues. The main target cells are B lymphocytes and their precursors in the bursa.
Depletion of B lymphocytes in early life results in impaired humoral immune responses, lowered resistance to infectious diseases and ineffective responses to vaccines.
In chickens older than 3 weeks of age, viral infection only marginally affects immune competence because many B lymphocytes are distributed throughout the tissues before bursal damage occurs.

K.5. Clinical signs and lesions

Picture 30: Gumborodisease in young chicken

The severity of clinical signs is influenced by the virulence of the virus, the age of chicks at the time of infection, the breed of the chicks and the level of maternally-derived antibody. Chicks develop an acute form of the disease, usually between 3 and 6 weeks of age, following a short incubation period (3-4 days):

- Affected birds are depressed and inappetent and show evidence of diarrhoea and vent pecking.

- At necropsy the cloacal bursa is swollen, oedematous, yellowish and occasionally haemorrhgic especially in birds that have died of disease; congestion and haemorrhage of the pectoral, thigh and leg muscle is common;
- Morbidity ranges from 10% to 100% with amortality rate up to 20% or, occasionally, higher.
- The course of the disease is short with surviving birds recovering in about 4 days.
- Many outbreaks are mild, detectable only by impaired weight gains.
- Although infections before 3 weeks of age are usually subclinical, severe depression of antibody responses may result.
- In general, the earlier in life infection occurs, the more pronounced the immunosuppression.
- Clinical signs in these birds are usually vague.
- Suboptimal growth, predisposition to secondary infections and poor response to vaccination may be encountered.

K.6. Diagnosis

In acute disease, clinical signs and a swollen oedematous bursa at post-mortem are often sufficient for diagnosis.

Confirmation and identification of subclinical infection require laboratory tests:

- Viral antigen can be detected in smears or frozen sections ofthe bursa using immunofluorescence.
- Macerated bursal tissue is suitable for detection of viral antigen by ELISA or by gel diffusion tests.
- Specimens of bursa, spleen or faeces are suitable for virus isolation. Most strains grow on the chorioal-lantoic membrane of embryonated eggs. Birds that have recovered from the acute disease develop high antibody titres as mature peripheral B lymphocytes are largely unaffected.

K.7. Prevention

- Depopulation, thorough cleaning and effective disinfection programmes are required following an outbreak of disease in a unit.
- Live vaccines can be administered by aerosol or in drinking water.
- Birds selected for breeding are usually vaccinated at 4 to 10 weeks of age with a live vaccine and again close to laying, with an inactivated oil-adjuvanted vaccine, to ensure high levels of maternally-derived antibody in chicks.
- Vaccines used in parent stock should contain both classical and variant strains of IBDV.
- Chicks can be actively immunized after maternally-derived antibody levels decline at about 4 weeks of age.
- In high-risk flocks, vaccination may begin at 1 day of age to protect birds with little or no maternally-derived antibody, followed by booster inoculations at 2 and 3 weeks of age.

- Partially attenuated vaccines termed 'intermediate' and 'intermediate plus' ('hot') are generally used in this way in broilers and commercial layer replacements as they are capable of overcoming low levels of maternally-derived antibody.

L. Newcastle disease

L.1. Definition

Newcastle disease (Avian pneumoencephalitis) is a contagious bird disease affecting many domestic and wild avian species; it is transmissible to humans. Its effects are most notable in domestic poultry due to their high susceptibility and the potential for severe impacts of an epizootic on the poultry industries. It is endemic to many countries.

Exposure of humans to infected birds (for example in poultry processing plants) can cause mild conjunctivitis, but the Newcastle disease virus (NDV) otherwise poses no hazard to human health.

L.2. Etiology

The etiologic agent is paramyxovirus, RNA virus; ND virus is very resistant, live in dust for 255 days

It is readily cultivated in developing chick embryo**.**

The most important avian paramyxovirus is Newcastle disease virus (NDV), also designated avian paramyxovirus

L.3. Epidemiology

Distribution

This disease occurs in poultry worldwide. Newcastle disease was first described in 1926 when severe outbreaks were reported in Newcastle, England, and in Java. Other major outbreaks of the disease occurred in the Middle East during the late 1960s and in the 1970s when pigeons were the species primarily affected. Infection with NDV is probably endemic in wild birds especially water fowl. Strains oNDV differ in their virulence. On the basis of virulence and tissue tropism in poultry, isolates are categorized into five groups or pathotypes.

Transmission

Usually transmission occurs by aerosols or by ingestion of contaminated feed or water. The relative stability of the virus permits mechanical transfer of infective material through the movement of personnel and equipment. Virus, which can survive in carcasses for some weeks, is present in all organs of acutely affected birds and in eggs. Captive and wild birds can contribute to the spread of infection. Pigeons are susceptible to all strains of NDV and may play a role in the transmission of Newcastle disease. Pigeon and imported psittacine species are the main source of transmission. Infected birds shed virus in exhaled air, respiratory discharge and feces; virus may be present in eggs laid during clinical disease and in all parts of carcass during acute virulent infection.

Susceptibility

A wide range of avian species including chickens, turkeys, pigeons, pheasants, ducks and geese are susceptible. Infection with NDV is probably endemic in wild birds especially water fowl. Strains oNDV differ in their virulence.

L.4. Pathogenesis

Viral replication, which occurs initially in the epithelia of the respiratory and intestinal tracts, is followed by haematogenous spread to the spleen and bone marrow. Secondary viraemia results in infection of other organs including lungs, intestine and CNS.

L.5. Clinical signs and lesions

Picture 31: Torticollis in young chicken affected by Newcastle disease

It is characterised by:

- The incubation period is usually about 5 days.
- When present, signs in these flocks include listlessness, weakness and a decrease in egg production. Viscerotropic strains tend to produce respiratory signs such asgasping,coughing , sneezing and rales;
- Oedema of the head and neck and greenish diarrhoea.

- The mortality rate in fully susceptible flocks may be close to 100%.
- Birds that survive the acute phase may develop neurological signs.
- Infection with neurotropic strains results in respiratory disease followed by nervous signs such as wing paralysis, leg paralysis, and torticollis and muscle spasms.
- Infection in turkeys, which usually involves the respiratory and central nervous systems, is less severe than that in chickens.
- Pigeons infected with ' pigeon' paramyxovirus 1 present with neurological signs and diarrhoea, and mortality in affected birds may approach 10%.
- Humans may develop a transitory conjunctivitis if exposed to high concentrations of NDV.
- Eggs may be abnormal in colour, shape or surface and have watery albumen.
- Lesions may include: petechia on the serous membrane; haemorrhages of the proventricular mucosa and intestinal mucosa accompanied with necrotic hemorrhagic areas on the mucosa surface of intestine; splenic necrosis.

L.6. Diagnosis

A presumptive clinical diagnosis may be made when the characteristic signs and lesions associated with virulent strains are present. Laboratory confirmation by isolation and identification of the virus is necessary.

Tracheal and cloacal swabs from live birds are suitable for virus isolation. Suitable post-mortem specimens for laboratory examination include faeces, intestinal contents and portions of trachea, intestine, spleen, brain and lung.

Demonstration of viral antigen in tracheal sections or impression smears using immunofluorescence is a less sensitive technique than virus isolation.

L.7. Prevention

National control policies for Newcastle disease differ from country to country and range from compulsory vaccination to slaughter of infected flocks:

- General control measures include locating poultry farms several kilometres apart, bird-proofing of houses and feed stores, controlled access to farms, thorough cleaning and disinfection of vehicles and equipment and restriction of movement between poultry farms.
- A combination of vaccination and slaughter policies is frequently employed. Vaccination is particularly important for birds in breeder flocks.

- The presence of maternally-derived antibodies interferes with the efficacy of live vaccines. In order to avoid this undesirable effect, vaccination should be delayed until 2 to 4 weeks of age when most birds will be susceptible, or alternatively live vaccine can be administered to day-old chicks by conjunctival instillation or by a coarse spray with large droplets in order to 'seed' a flock with vaccinal virus. This method, which may result in respiratory disease in fully susceptible birds, establishes active infection in some birds that persists until maternally-derived immunity has decreased sufficiently in the rest of the birds to allow infection to develop. Revaccination is normally carried out 3 to 4 weeks later. A schedule of vaccination employing both live and inactivated vaccines gives good results.
- Several recombinant vaccines employing different vector viruses have been developed.
- B1 and Lasota vaccines are widely used and administered to poultry by mass application in drinking water or by spray.
- People who are exposed to large quantity of virus such laboratory workers and vaccination teams must handle live vaccines carefully because a transitory conjunctivitis can be produced.

M. Rabies

M.1. Definition

Rabies is the viral infection, which affects the central nervous system of most mammals including humans; it is invariably fatal. However, mammalian species vary widely in their susceptibility. Most clinical cases are due to infection with rabies virus (genotype 1).

A number of other neurotropic lyssaviruses, closely related to the rabies virus, produce clinical signs indistinguishable from rabies.

M.2. Etiology

Rhabdoviridae have characteristic rod shapes, a linear, non-segmented, single-stranded RNA genome. Rhabdoviruses of vertebrates are bullet-shaped or cone-shaped while those infecting plants are generally bacilliform. This large family contains viruses of vertebrates, invertebrates and plants. Replication occurs in the cytoplasm (with the exception of nucleorhabdoviruses).

Newly synthesized nucleocapsids acquire envelopes from the plasma membrane as virions bud from the cell. Virions (100 to 430nm × 45 to 100 nm) are stable in the pH range 5 to 10. They are rapidly inactivated by heating at 56°C, by treatment with lipid solvents.

M.3. Epidemiology

Strains affecting a particular species are transmitted more readily to members of that species than to other animal species. In a given geographical region, rabies is usually maintained and transmitted by particular mammalian reservoir hosts. Two epidemiologically important infectious cycles are recognized, urban rabies in dogs and sylvatic rabies in wildlife. More than 95% of human cases are the result of bites from rabid dogs.

Racoons, skunks, foxes and bats are important reservoirs of rabies virus in North America. In continental Europe, the principal reservoir is the red fox. The vampire bat is an important reservoir of the virus in Central and South America and in the Caribbean islands.

In developed countries, the control of stray dogs and the use of vaccination programmes have reduced the importance of urban rabies and have focused attention on wildlife reservoirs. Species susceptibility to rabies virus is important epidemiologically.

Domestic animals and humans are considered to be moderately susceptible to the virus, whereas foxes, wolves, coyotes and jackals are considered to be highly susceptible. Although virus may be transmitted through scratching and licking, transmission usually occurs through bites. Infected animals may excrete virus in their saliva for some time before the onset of clinical signs.

M.4. Pathogenesis

Following introduction into the tissues, virus enters peripheral nerve endings. The virus travels via peripheral nerves to the spinal cord and ascendant to the brain. After reaching the brain, the virus travels via peripheral nerves to the salivary glands. If an animal is capable of transmitting rabies via its saliva, virus will be detectable in brain.

Virus is shed intermittently in the saliva; the presence of virus in saliva, especially in carnivores, is an important factor in rabies transmission. Clinical signs develop following neuronal damage caused by viral replication.

M.5. Symptoms

Picture 32: A dog affected with rabies

The period between infection and the first symptoms is normally two to twelve weeks but can be as long as two years. Soon after, the symptoms expand to slight or partial paralysis, cerebral dysfunction, anxiety, insomnia, confusion, agitation, abnormal behaviour, paranoia, terror, hallucination.

Production of large quantities of saliva and tears coupled with an inability speak or swallow are typical during the later stages of the disease this can result hydrophobia, where the victim has difficulty swallowing because the throat and jaw become paralyzed.

In general the most reliable signs regardless of species are acute behavioral changes and unexplained progressive paralysis and:

- Sudden anorexia;
- Sign of apprehension or nervousness
- Irritability
- Hyperexcitability (including priapism);
- Animal may seek solitude;
- Ataxia;
- Alerted phonation and change in temperament are apparent;
- Uncharacteristic aggressiveness may develop;
- A normally docile animal may suddenly become vicious;
- Commonly, rabid wild animals may lose fear of humans, and species that are normally nocturnal may be seen wandering about during the daytime.

The clinical course may be divided into 3 general phases: Prodromal phase, acute excitative phase, and paralytic phase (end stage).

Prodromal phase:

It lasts 1-3 days, animals show only vague no specific signs which intensify rapidly. The disease progresses rapidly after the onset of paralysis and death is virtually certain a few days thereafter. Some animals die without marked clinical signs.

Furious form:

This form is also called furious phase and this is the classic "mad-dog syndrome" although it may be seen in all species.

There is rarely evidence of paralysis during this stage; the anima becomes irritable and, slightest provocation may viciously and aggressively uses its teeth, claws or hooves.

The posture and expression is one alertness and anxiety, with pupils dilated. Such animals lose caution and fear of human and other animals; carnivores with this form of rabies frequently roam extensively, attacking other animals including people and any moving object.

They commonly swallow foreign objects e.g: feces, straw, sticks and stones. As the disease progresses, muscular incoordination and seizures are common. Death results from progressive paralysis.

Paralytic form:

It is characterised by Ataxia and paralysis of throat and masseter muscles, often with profuse salivation and inability to swallow. Dropping of lower jaw is common in dogs. These animals may not be vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, coma and death follow in few days.

M.6. Diagnosis

In endemic areas, suspect domestic carnivores which have bitten humans should be isolated and observed for up to 7 days. The brains of animals that develop clinical signs should be examined for the presence of virus.

Rabies virus is particularly abundant in cerebrum, cerebellum and medulla. Rapid laboratory confirmation is essential for the implementation of appropriate treatment of human patients.

The preferred method of diagnosis is the direct fluorescent antibody test (FAT) on acetone-fixed brain tissue smears.

M.7. Prevention

Comprehensive guidelines for control in dogs have been prepared internationally by the World Health Organization and in USA by the National Association of State Public Health Veterinarians (NASPHV).They include the following:

- Notification of suspected cases and **euthanasia** of dog with clinical signs and dogs bitten by suspected rabid animal.
- Reduction of contact rates between susceptible dogs by leash laws, dg movement control and quarantine.
- Mass immunization of dogs by campaigns and by continuing vaccination of young dogs.
- Straying dog control and euthanasia of unvaccinated dog.
- Dog legislation;
- In most countries that are free of rabies the prevention relies on:
- Rigorous quarantine measures to prevent the introduction of disease.
- Movement of vaccinated domestic carnivores is permitted between some countries provided that strict identification and testing procedures are in place.
- In countries where rabies is endemic, control methods are aimed mainly at reservoir species. Urban rabies can be effectively controlled by vaccination and restriction of dog and cat movement and by the elimination of stray animals.
- Control of sylvatic rabies requires special measures. Regional depopulation of reservoir species, which has rarely been successful, is ecologically unacceptable.
- Vaccination of red foxes with live oral vaccines, delivered in baits, has eliminated sylvatic rabies from several regions of Western Europe.
- A number of modified live rabies virus vaccines have been used including SAD B19, SAG-1 and SAG-2. However, as a result of concerns about the safety of live vaccines, a vaccinia-rabies virus glycoprotein (VRG) recombinant vaccine was developed and has proved effective for vaccinating foxes, coyotes and racoons.
- The rapid increase in racoon rabies in the USA has proved difficult to control through vaccination and several challenges remain for successful eradication in North America including development of practical vaccination methods for bats.

M.8. Management of suspected rabies cases

In place where terrestrial wildlife or bat rabies is known to occur, any animal bitten by a wild carnivorous mammal (or a bat) not available for testing should be regarded as having been exposed to rabies.

Any unvaccinated dog, cat must be euthanized; if the owner is unwilling to do this, the animal should be placed in strict isolation (no human or animal contact) for 6 months and vaccinated before release.

Zoonotic risk

Any domestic dog, cat whether vaccinated against rabies or not, that exposes (bites or deposits saliva in a fresh wound or on a mucous membrane) a person should be confined for 10 days; if the animal develops any sign of rabies during that period it should be euthanized as soon as possible and its brain promptly submitted for rabies diagnosis.

When a person is exposed to an animal suspected of having rabies, the risk of rabies transmission should be evaluated carefully. Risk assessment should include consideration of the species of animal involved, the prevalence of the rabies in the area.

Wild carnivores and bat present a considerable risk where the disease is found regardless whether abnormal behaviour has been observed. Direct contact with bat could be considered as risk for virus exposure.

N. Canine Distemper

N.1. Definition

This highly contagious disease of dogs and other carnivores has a worldwide distribution. Canine distemper virus (CDV), a pantropic morbillivirus, produces a generalized infection involving many organ systems.

N.2. Etiology

Paramyxovius, fragile enveloped virus sensitive to lipid solvents such as ether and most disinfectants including phenols, and quaternary ammonium compounds. It is relatively unstable outside the host.

N.3. Epidemiology

The disease is found in canidae (dogs, foxes, and wolves); outbreaks of disease have been documented in several wildlife species including foxes, skunks, racoons, black-footed ferrets and lions. The virus is relatively labile, requiring transmission by direct contact or by aerosols.

In urban dog populations, the virus is maintained by infection of susceptible animals. Infection spreads rapidly among young dogs, usually between 3 and 6 months of age, when maternally-derived immunity declines.

The number of dogs in populations in rural areas is often too low to maintain continuous infection with the result that, irrespective of age, unvaccinated dogs are susceptible and significant outbreaks of the disease can occur.

N.4. Pathogenesis

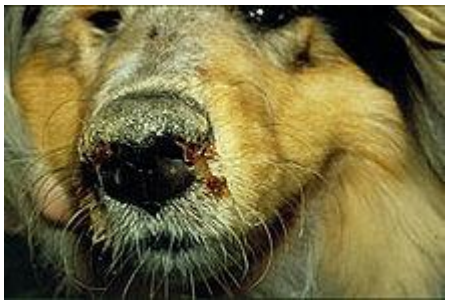
The virus, which replicates in the upper respiratory tract, spreads to the tonsils and bronchial lymph nodes. A cell-associated viraemia follows with spread to other lymphoreticular tissues. Viral replication produces lymphocytolysis and leukopenia resulting in immunosuppression which facilitates a secondary viraemia to develop.

The extent of spread to tissues and organs is determined by the rapidity and effectiveness of the immune response. In the absence of a sufficiently vigorous response, dissemination and replication of CDV occurs in the respiratory, gastrointestinal, urinary and central nervous systems. Spread to the skin may also occur.

Virus infects both neurons and glial cells within the CNS and may persist there for very long periods. Old dog encephalitis is apparently associated with prolonged persistence of the virus in the brain, possibly as a result of non-cytolytic spread from cell to cell without budding from the cell membrane, thus evading immune detection.

This mechanism appears to be analogous to that causing subacute sclerosing panencephalitis of children which is associated with persistent infection with defective measles virus. The presence of viral antigen in these conditions stimulates a low-grade prolonged inflammatory response eventually leading to the development of neurological signs.

N.4. Clinical signs and lesions



Picture 33: Hyperkeratosis of the dog's nose with Canine Distemper

It is characterised by:

- The incubation period is usually about 1 week but may extend to 4 weeks or more when nervous signs appear without prior evidence of infection.
- The severity and duration of illness are variable and are influenced by the virulence of the infecting virus, the age and immune status of the infected animal and the rapidity of its immune response to infection.

- The pyrexic response to infection is biphasic although the initial elevation of temperature may not be noticed. During the second period of pyrexiaoculonasal discharge, pharyngitis and tonsillar enlargement become evident.
- Coughing, vomiting and diarrhoea are often consequences of secondary infections.
- A skin rash and pustules may be present on the abdomen.
- Some affected dogs have hyperkeratosis of the nose and footpads, referred to as 'hardpad'. Acute disease, which may last for a few weeks, is followed either by recovery and life-long immunity or by the development of neurological signs and, eventually, death.
- Common neurological signs include paresis, myoclonus and seizures.
- A grave prognosis is indicated in animals displaying neurological disturbance. Residual neurological deficits are common in dogs that survive.
- Old dog encephalitis, characterized by motor and behavioural deterioration, is invariably fatal.
- As lesions; thymic atrophy is a consistent postmortem finding in infected young puppies, hyperkeratosis of the nose and footpads is often found in dogs with neurologic manifestations.
- In case of acute and peracute death, exclusively respiratory abnormalities may be found.
- Depending on the degree of secondary bacterial infection, bronchopneumonia, enteritis, and skin pustules may also be present.
- Histologically, canine distemper virus produces necrosis of lymphatic tissues, interstitial pneumonia.

N.6. Diagnosis

- A febrile, catarrhal illness with neurological sequelae in young dogs is highly suggestive of canine distemper.
- Viral antigen may be demonstrated by immunofluorescence in conjunctival or vaginal impression smears or in smears of cells from the buffy coat.
- Cryostat sections of lymph nodes, urinary bladder and cerebellum are also suitable for the demonstration of viral antigen.
- Eosinophilic inclusions can be demonstrated in nervous and epithelial tissues.
- Virus isolation may prove difficult. Urinary bladder and brain are suitable post-mortem specimens for virus isolation.

N.7. Prevention

Successful immunization of pups with canine distemper modified live virus (MLV) vaccine depends on the lack of interference by maternal antibody .To overcome this barrier pups are vaccinated with MLV vaccine when 6 weeks old at 3 to 4 weeks intervals until 16 weeks old.

Many varieties of attenuated distemper vaccine are available and should be used according to manufactures' directions.

L O 1.4 – Characterise Common fungal diseases

Fungal diseases are caused by microscopic fungi; they involve skin or mucous membrane and systemic cases are found in different animal species.

- Topic 1: Common fungal diseases

A. Ringworm

A.1. Definition

Ringworm (Dermatophytosis) is the disease caused by dermatophytes which affect skin, hair, feathers, horn, hooves, claws and nails of many animal species.

Dermatophytes are susceptible to common disinfectants, particularly those containing cresol, iodine, or chlorine. They survive for years in in the inanimate environment.
It is a zoonosis and most human infections are caused by *Microsporum canis* contracted from infected cats.

A.2. Etiology

Dermatophytes Geophilic dermatophytes inhabit and replicate in the soil in association with decomposing keratinous materials such as hairs or feathers. Animals can acquire infection with geophilic dermatophytes from soil or from contact with infected animals. Zoophilic and anthropophilic dermatophytes are obligate pathogens which are unable to replicate in soil.

Their existence as pathogens of keratinized structures usually corresponds with an inability to reproduce sexually. Dermatophytes growing on keratinized structures rarely produce macroconidia and consequently rely on the production of arthrospores for transmission. Each zoophilic species tends to parasitize a particular animal species

A.3. Epidemiology

Dermatophytoses often affect the young. Extent and severity are influenced by environmental factors. Crowding of animals or assembling of large number is often associated with increased prevalence. Infected individuals of then same species the important dermatophytoses of animals.

Dermatophyte hosts geographical distribution:
Microsporum canis (var. *canis*) Cats, dogs Worldwide;

M. canis var. *distortum* Dogs New Zealand, Australia,North America;

M. canis (syn. *M. equinum*) Horses Africa, Australasia, Europe, North and South America;

M. gallinae Chickens, turkeys Worldwide;

M. gypseum Horses, dogs, rodents Worldwide;

M. nanum Pigs North and South America, Europe, Australasia;

M. persicolor Field voles Europe, North America;

Trichophytonequinum Horses Worldwide;

Dermatophytes are disseminated by direct and, owing to their persistence on fomites and premises, indirect contact.

A.4. Pathogenesis

Dermatophytes invade keratinized structures such as the stratum corneum of the epidermis, hair follicles, hair shafts and feathers. Lesion development is influenced by the virulence of the dermatophyte and the immunological competence of the host.

Young, aged, debilitated and immunosuppressed animals are particularly susceptible to infection, which occurs either directly by contact with an infected host or indirectly through infected epithelial debris in the environment. Infective arthrospores adhere to keratinized structures and germinate within 6 hours. Minor trauma such as gentle rubbing of the skin or bites from arthropods may facilitate infection.

Damp skin surfaces and warmth favour germination of spores. Metabolic products of hyphal growth may provoke a local inflammatory response. Hyphae grow centrifugally from the initial lesion towards normal skin, producing typical ringworm lesions. Alopecia, tissue repair and nonviable hyphae are found at the centres of lesions as they develop.

Growth of hyphae can result in epidermal hyperplasia and hyperkeratosis. Secondary bacterial infection sometimes follows mycotic folliculitis.

A.5. Symptoms and lesions

Picture 34: Head Ringworms in cattle

The manifestations of dermatophyte infections can vary and may be summarized as:

- Subclinical or inapparent infections;
- Classical round ringworm lesions;
- Serious generalized lesions that may be complicated by mange mites or by secondary bacterial infection, in particular by *Staphylococcus aureus* or *S. Pseudintermedius*
- Nodular or tumourous lesions called kerions, seen most commonly in dogs.

Dermatophytosis in dogs and cats

Microsporum canis is the usual cause of dermatophytosis in dogs and cats.

Dermatophytosis is a comparatively common clinical condition in both companion and farm animals. Because of the zoonotic nature of the dermatophytoses, affected animals should be handled with care.

Clinical features of the disease include:

- classical ringworm lesions,
- miliary dermatitis,
- pseudomycetomas ,
- onychomycosis and,
- Rarely, generalizedlesions in immunosuppressed animals.
- Inapparent infections are known to occur and cats may also carry arthrospores physically in their coats.
- The disease usually presents as areas of alopecia, scaling and broken hairs surrounded by inflammatory zones.
- Less commonly encountered lesions include folliculitis and onychomycosis.
- Lesion distribution on the muzzle may relate to certain behavioural activities such as compulsive digging in soil, rat-catching and attacking hedgehogs.
- Generalized infection is uncommon in dogs and is often associated with conditions such as hyperadrenocorticism and immunosuppression.

Dermatophytosis in cattle

Clinical features of the disease include:

- *Trichophyton verrucosum* is the usual cause of ringworm in cattle.
- Calves are affected most commonly and often develop characteristic lesions on the face and around the eyes.
- In heifers and cows, lesions may be present on the neck and limbs.

- Oval areas of affected skin are alopecic with greyish white crusts.
- Infection is most common in winter months, with a number of animals usually affected.
- Bovine dermatophytosis is usually self-limiting. However, individual valuable animals may require treatment.
- Topical preparations such as 5% lime sulphur, natamycin may be effective.
- Individual lesions can be treated with fluconazole, itraconazole or terbinafine.
- Commercial vaccines containing an attenuated strain of *T.verrucosum* (LTF-130 or CCM 8165) have been used successfully for the control of bovine dermato- phytosis in Europe and Russia.

Dermatophytosis in pigs

Clinical features of the disease include:

- Dermatophytosis in pigs is uncommon and is usually caused by *Microsporum nanum*.
- The condition, which can be endemic in a herd, may not be recognized, particularly in pigs with pigmented skin.
- All ages are susceptible and lesions can occur anywhere on the body surface as thick brownish crusts. Ringworm in pigs is not of economic importance.

Favus in poultry

Clinical features of the disease include:

- Gallinaceous birds are occasionally infected with *Microsporum gallinae*, the cause of avian ringworm or favus. White patchy crusts develop on the comb and wattles.
- If the disease is severe, feather follicles may be invaded and affected birds may show signs of systemic illness.

A.6. Diagnosis

Laboratory investigation of dermatophytosis is often necessary because diagnosis on clinical grounds can be difficult:

- As dermatophyte species tend to parasitize particular hosts, the animal species affected may indicate the dermatophyte most likely to be involved.
- Specimens suitable for laboratory examination include plucked hair, deep skin scrapings from the edge of lesions, scrapings from affected claws, and biopsy material from pseudomycetomas. Suitable material from cats can also be collected on a large sheet of paper by brushing the coat with a clean toothbrush.

- Hairs and skin scrapings treated with KOH should be examined microscopically for the presence of arthrospores.The arrangement of arthrospores on hair shafts is typically ectothrix. Mites, such as *Demodex* species, may also be detected in these specimens.
- Histological sections of skin or pseudomycetomas can be stained by the methenamine silver techniques to demonstrate fungal structures.
- In cats and dogs with suspicious lesions, examination with Wood's lamp should always be carried out because *M. canis* infections are comparatively common in these species. A characteristic apple-green fluorescence from infected hairs is evident in more than 50% of affected dogs and cats.

Detection of fluorescence depends on factors such as stage of infection and the characteristics of the infecting strain.

A.7. Treatment

Because the dermatophytoses are zoonoses, treatment and control are particularly important in domestic carnivores:

- If lesions are limited in extent, treatment with preparations such as lime sulphur or miconazole shampoo may be effective .A solution of 0.2% enilconazole is approved in most countries for use in dogs, cats, and cattle.
- Clipping of the haircoat is advisable, particularly if lesions are extensive. The clippings, which contain numerous infective arthrospores, must be disposed of carefully.
- Itraconazole, Fluconazole or Terbinafine, administered orally, are the drugs of choice for systemic therapy.
- Because they are potentially teratogenic, azole drugs should not be given to pregnant animals. Although Griseofulvin has been used for many years for the treatment of dermatophytosis, it is used less frequently because of the risks of teratogenicity. In addition, Griseofulvin can induce neutropenia and should not be given to cats with feline immunodeficiency virus infection.

A.8. Prevention

- Animals with suspicious lesions should be isolated.
- Early laboratory confirmation is essential.
- In-contact animals should be examined under a Wood's lamp and closely monitored for skin lesions.
- Contaminated areas should be vacuum-cleaned to remove infected skin debris and hairs.

- Contaminated bedding should be burnt and grooming equipment should be disinfected with 0.5% sodium hypochlorite.
- A number of vaccines for use in dogs and cats have been produced over the years but a vaccine of proven efficacy still appears to be lacking.

B. Candidiasis

B.1. Definition

Candidiasis is a localized mucocutaneous disease caused by species of the yeast-like fungus candida; most commonly *Candida albicans* .It is distributed worldwide in a variety of animals as a normal inhabitant of the nasopharynx, GI tract, and external genitalia of many species of animals and is opportunistic in causing disease.

B.2. Etiology

Yeasts are eukaryotic, round or oval, single-celled organisms. Yeasts grow aerobically on Sabouraud dextrose agar, and the species capable of tissue invasion grow well at 37°C. Colonies, which are usually moist and creamy in texture, resemble large bacterial colonies. The yeast cells are round to oval and 3.5 to 8.0 µm in diameter. A daughter cell is formed as a bud, on a narrow neck, from the mother cell.

Yeasts are found in the environment, often on plants or plant materials. They may also occur as commensals on the skin or mucous membranes of animals. They cause opportunistic infections which are categorized as exogenous, when derived from the environment, or endogenous, when resulting from overgrowth of commensals.

Immunosuppression or factors such as prolonged antimicrobial therapy which disturb the resident flora on mucosal surfaces may facilitate yeast overgrowth leading to tissue invasion. Yeasts of importance in animal disease are *Candida* species (particularly *Candida albicans*), *Cryptococcus neoformans* and *Malassezia pachydermatis*.

Macrorhabdus ornithogaster (formerly referred to as 'megabacteria') is yeast found in the proventriculus of several avian species. It is associated with 'going light' in budgerigars, a fatal disease characterized by progressive weight loss. Other yeasts, such as *Trichosporon beigelii*, and the yeast-like mould *Geotrichum candidum* rarely cause infection.

Candida albicans is the species most often implicated in animal disease. It grows aerobically at 37°C on a wide range of media including Sabouraud dextrose agar.

Colonies are composed of budding oval cells approximately 5.0 × 8.0 µm. In animal tissues, *C. albicans* may exhibit polymorphism in the form of pseudohyphae or hyphae. On certain media, it characteristically produces thick-walled resting cells known as chlamydospores (chlamydoconidia). Most *Candida* species have a similar colonial appearance.

Colonies, which are whitish, shiny and convex, are 4 to 5 mm in diameter after incubation for 3 days.

The thick-walled resting cells of *Candida albicans* are called chlamydospores (chlamydoconidia).

These resting cells are formed from pseudohyphae when submerged colonies grow in cornmeal agar. The smaller cells are blastoconidia (arrow).Germ tubes form within 2 hours when cells of *Candida albicans* are incubated in serum at 37°C.

B.3. Epidemiology

Candida species occur worldwide on plant materials and, as commensals, in the digestive and urogenital tracts of animals and humans. Thrush of the oesophagus or crop in young chickens may be associated with prolonged antibiotic administration, debilitating conditions such as intestinal coccidiosis, or unsanitary, overcrowded housing conditions.

In addition, a number of *Candida* species have been isolated from cases of bovine mastitis. Mycotic mastitis occurs sporadically either as a consequence of contaminated intramammary preparations or from heavy environmental contamination. Usually one quarter is involved and spontaneous elimination of the infection frequently occurs. Rarely, yeast cells may be shed for up to 12 months.

Mycotic stomatitis has been reported in pups, kittens and foals. *Candida albicans* has been implicated in gastro-oesophageal ulceration in pigs andfoals.

Rarely, disseminated candidiasis may occur in pigs, calves, dogs and cats.Bovine abortion caused by *Candida* species has been recorded.
Candida albicans is isolated from environmental sources less frequently than other *Candida* species, suggesting adaptation towards a parasitic rather than a saprophytic existence.

B.4. Pathogenesis

Factors associated with candidal infections are disruption of mucosal integrity, indwelling, intravenous, or urinary catheter s administration of antibiotics and immunosuppressive drugs or diseases. The most affected animals are birds with superficial infection and cattle, calves, sheep and foals with systemic candidiasis.

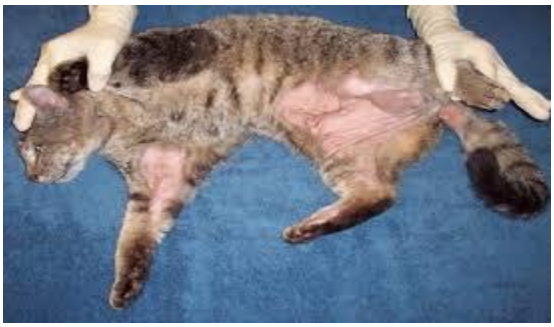
Candida albicans, the principal yeast involved in animal disease, possesses a number of putative virulence factors. The organism has surface integrin-like molecules which allow adhesion to matrix proteins. In addition, surface structures can bind fibrinogen and complement components. Production of proteases and phospholipases may aid tissue invasion. Phenotypic switching and biofilm formation, which have been demonstrated in *C. albicans*, may facilitate evasion of host defence mechanisms.

During the early stages of infection, phagocytic clearance mechanisms eliminate most of the yeast cells. Cells that are not cleared rapidly convert to hyphal forms. This transition from budding to hyphal forms probably facilitates tissue penetration and increases resistance to phagocytosis due to the larger size of the hyphae.

Phospholipases, concentrated in hyphal tips, appear to enhance invasiveness. The localized mucocutaneous form of candidiasis is associated with overgrowth of resident *C. albicans* in the oral cavity or gastrointestinal and urogenital tracts.

Predisposing factors include defects in cell-mediated immunity, concurrent disease, and disturbance of the normal flora by prolonged use of antimicrobial drugs and damage to the mucosa from indwelling catheters. Affected mucosa is thickened and often hyperaemic. Haematogenous spread may occur following vascular invasion by hyphae or pseudohyphae, producing systemic lesions.

B.5. Symptoms and lesions



Picture 35: Candidiasis in cats

Clinical features of the disease include:

- Calves with forestomach candidiasis have watery diarrhea, anorexia, and dehydratation with gradual progression to prostration and death.
- Affected chicks listless and have reduced feed intake and growth rate.
- Porcine candidiasis affects the oral, oesophageal and gastric mucosa, with diarrhea and emaciation.

Lesions include:

- Gross lesions of the skin and mucosae are single or multiple, raised, circular, and white mass covered with scabs.
- The organism can penetrate keratinized epithelium and cause marked keratinous thickening of the mucosa of the tongue, oesophagus, and rumen.
- In birds, the crop and oesophageal lesion are white, circular ulcers, with raised surface scabs that produce thickening of the mucosa; an easily removed pseudomembane is common.

B.6. Diagnosis

- The wood's light (ultraviolet light) in population screening of cat colonies where *M.canis* is the only concern.
- Skin scaping and hair are examined microscopically for the presence of hyphae and arthroconidia
- The scraping should include material from the margin of any lesion and the full thickness of keratinized epidermis.

B.7. Treatment

Combined topical and systemic treatment is often preferable:

- Two systemic agent available Griseofulvin and Ketoconazole (the later is more costly and less proven); both are given orally and are relatively tolerated.
- Terbinafine may be a useful alternative;
- Antifungal orchard spray is effective on ringworm of large and small animals; affected areas are first clipped. In large animals two applications at biweekly intervals are recommended; with dogs weekly dips can be repeated to effect. Thiabendazole is used on small and large animals;
- Fluconazole(5g/kg,PO, once a day for 4-6 weeks) is also used to successfully treat disseminated candidiasis in foals;
- Itraconasole and AmphotericinB lipid complex are considered the treatment of choice in dogs.
- Povidone-iodine and Chlorexidine, available as solutions and ointments, are general antiseptics with antifungal action.
- Nystatin ointment or topical application of AmphotericinB or 1% iodine solution, may be useful in the treatment of oral or cutaneous candidiasis.

B.8. Prevention

Exposed animals are treated prophylactically and infected individuals should be isolated and treated:

- Successful vaccination is widely practiced on European cattle;
- A live attenuated strain appear to most immunogenic;
- Neither live attenuated vaccines nor killed products have been effective in preventing dermatophytosis in cats.

C. Aspergillosis

C.1. Definition

Aspergillosis is a fungal disease caused by several Aspergillus species found worldwide in almost all domestic animals and birds as well as in many wild species. It is primarily a respiratory infection that may become generalized; however, tissue predilection varies among species.

C.2. Etiology

Aspergillosis is caused by several aspergillus spp especially *Aspergillus fumigatus* and *Aspergillusterreus*. Members of the phylum *Ascomycota* .The hyphae are septate, hyaline and up to 8.0 µm in diameter. Unbranched conidiophores develop at right angles from specialized hyphal foot cells. The tip of the conidiophore enlarges to form a vesicle which becomes partially or completely covered with flask-shaped phialides.

The phialides produce chains of round pigmented conidia (phialoconidia) which may be smooth or rough and are up to 5.0 µm in diameter Aspergilli are aerobic and grow rapidly, forming distinct colonies after incubation for 2 to 3 days. The colour of the obverse side of colonies, which may be bluish green, black, brown, yellow or reddish, varies with individual species and with cultural conditions. *Aspergillus fumigatus*, a thermotolerant species, grows at temperatures ranging from 20°C to 50°C.

C.3. Epidemiology

Respiratory infections may occur following inhalation of spores. Less commonly, infection can result from ingestion of spores or following tissue trauma. Systemic infection is invariably associated with immunosuppression.

Aspergilli are common soil inhabitants and are also found in large numbers in decomposing organic matter.Spores of *Aspergillus* species are present in dust and air.

The disease is found worldwide and almost all domestic animals and birds as well as in many wild species.

C.4. Pathogenesis

Infection with *Aspergillus* species, mainly *Aspergillus fumigatus*, has been recorded in many species of animals.

Aspergillosis, which is primarily a respiratory infection, follows spore inhalation. Because the spores of *Aspergillus fumigatus* are small, they can pass through the upper respiratory tract and may be carried to the terminal parts of the bronchial tree. Germination of inhaled spores and hyphal invasion of tissues depend on a number of factors. A true virulence factor has not been demonstrated but rather a number of factors appear to combine to facilitate disease production. *Aspergillus* spores adhere to collagen, fibrinogen, fibronectin and laminin. Pathogenic *Aspergillus* species grow well at 37°C and produce a wide variety of extracellular enzymes. Proteases with elastase, fibrinolytic and anticoagulative properties are considered to be important. In addition, a metabolite of *Aspergillus fumigatus*, gliotoxin, inhibits both the activity of cilia and phagocytosis by macrophages.

Immune competence of the host largely determines the outcome of infection. Factors that may modify immune competence include corticosteroid therapy and long-term treatment with antimicrobial drugs. Interference with both neutrophil and monocyte function may predispose to tissue invasion.

Hyphal invasion of blood vessels leads to vasculitis and thrombus formation. Mycotic granulomas may develop in the lungs and occasionally in other internal organs.

C.5. Clinical signs and lesions

In birds

The disease is primarily bronchopulmonary with dyspnea, gasping and polypnea accompanied by somnolence anorexia and emaciation. Mycotic tracheatis, torticolis and disturbances of equilibrium are seen when infection disseminate to the brain. Yellow nodules of varying size and consistency or plaque lesions are found in the respiratory passages, lungs' air sacs, or membranes of body cavities.

In ruminants

The disease may be asymptomatic, appear in bronchopulmonary form, cause mastitis, or cause placentitis and abortion. Mycotic pneumonia may be rapidly fatal. Signs include pyrexia, rapid, shallow, stertorous respiration; nasal discharge and moist cough.

The lungs are firm, heavy, and mottled and do not collapse. In subacute to chronic mycotic pneumonia, the lungs contain multiple discrete granulomas, and the disease grossely ressembles tuberculosis.

In the absence of pneumonia, infected cow generally have no signs except for abortion; a dead foetus is aborted 6-9 month of gestation and the foetal membranes are retained. Lesions are found in the uterus, foetal membranes, and often the foetal skin.

In the uterus, the intercaruncular are grossely thickened, leathery, dark red to tan and contain elevated or eroded forci covered by yellow-gray adherent pseudomembrane. Maternal caruncles are dark red to brown, and the adherent foetal cotyledons are markedly thickened. Cutaneous lesions in aborted foetuses consist of soft, red to gray, elevated discrete forci that ressembles ringworm.

In dogs

Aspergillosis is typically localized in the nasal cavity or paranasal sinuses. The sign include; nasal pain, ulceration of the nares, sneezing, unilateral or bilateral sanguinopurulent nasal discharges, frontal sinus osteomyelitis and epistaxis.

Gross lesions vary considerably with the site of infection, but the mucosa of the nasal and paranasal sinuses may be covered by layer of necrotic material and white to gray white fungal growth. The clinical signs of disseminated aspergillosis may include lethargy, lameness, anorexia, weight loss, pyrexia, hematuria, urinary incontinence, generalized lymphadenopathy and neurologic deficits. Lesions are frequently found in the kidneys, spleen and vertebrae.

C.6. Diagnosis

The diagnosis may include:

- Visualization of fungal plaques by rhinoscopy together with serologic evidence of disease is often how diagnosis is made
- Immunofluorescent procedures can be used to identify hyphae in tissues section**.**
- Certain specific clinical conditions such as guttural pouch mycosis may suggest the involvement of *Aspergillus* species.
- Endoscopic examination can be used to detect lesions in thenasal cavity and guttural pouch.

C.7. Treatment

Different drugs can be used**:**

- The use of Clotrimazole is generally considered the first line treatment via the nares as single infusion;
- Drugs given systemically may include Ketoconazole, Itraconazole, Fluconazole and Posaconazole

- Administration of Itraconazole through tubes inserted surgically in the frontal sinuses and nasal chambers may be used together with systemic treatment with fluconazole or Voriconazole which should continue for 6 to 8 weeks. Voriconazole is probably the most effective antifungal for treating aspergillosis but its cost is very high compared to the other choices.

C.8. Prevention

Strict hygiene and routine fumigation of incubators are effective control measures.

Learning Unit 2 – Perform Diagnosis of infectious disease

Infectious diseases cause losses and decrease animal production; this can be one of limitations of development of population; furthermore some of them are zoonosis; thus a good diagnosis is very important in order to fight against them efficiently.

L O 2.1 – Conduct specific examination

The examination of a patient consists of a general inspection done from a distance (the distant examination, and the particular distant examination of body regions), followed by a close physical examination of all body regions and systems. Only the major body systems that are routinely examined are presented here as part of the general examination.

- Topic 1. Examination of apparatus.

A. Digestive system

A.1. Auscultation (rumen sound)

There are two types of auscultation method:

- **Direct listening** to the sounds produced by organ movement is performed by placing the ear to the body surface over the organ.
- **Indirect auscultation** by a stethoscope is the preferred technique. Auscultation is used routinely to assess heart sounds (heartbeat, frequency and rythm, pulse rate), lung sounds (respiratory rythm, frequency and sound) and gastrointestinal sounds (frequency and sound).

Condition that can occur in Rumen are: secondary free gas bloat, frothy bloat, ruminal acidosis, cold water ruminal atony, neoplasia of rumen, rumen collapse syndrome, rumen foreign body, rumen impaction, vagal indigestion.

Ausculation of the rumen help in recognizing the rate, quality and rhythm of the normal ruminal movements. The ruminal movements arise from the churning action of the organ. The rate of ruminal movement in health animals is 2-5 in cattle, 3-6 in sheep, 2-4 movements every two minutes in goats. It decreases in cases of rumen atony; diseases of reticulum, omasum and abomasum; impaction and late stage of tympany; also in severe feverish conditions and in traumatic reticuloperitonitis. Increased rate is seen in early stage of digestive disorders such as tympany and the form of vagus indigestion with hypermotility.

Absence of movements occurs in the following:

- Severe dilation of the rumen with gases (tympany) or with food (impaction).
- Toxic conditions.
- The quality could be described as strong in healthy animals, weak in cases of ruminal atony and very strong as in early stages of digestive disorders such as tympany and vagus indigestion with hypermotility.

A.2. Percussion (rumen sound)

Percussion and simultaneous auscultation of the left and right sides of the abdomen is a useful technique for examination of the abdomen of large animals.

- This is a valuable diagnostic aid for the detection and localization of a gas-filled viscus in the abdomen of cattle with left-side displacement of the abomasum, right-side dilatation and volvulus of the abomasum, cecal dilatation and torsion, intestinal tympany associated with acute obstruction or paralytic ileus, or pneumoperitoneum.
- To elicit the diagnostic 'ping', it is necessary to percuss and auscultate side by side and to percuss with a quick, sharp, light and localized force.
- The obvious method is a quick tap with a percussion hammer or similar object.
- Another favoured method is a 'flick' with the back of a forefinger suddenly released from behind the thumb.

A gas filled viscus gives a characteristic clear, sharp, high-pitched 'ping' which is distinctly different from the full, low pitched note of solid or fluid-filled viscera.

A drum like sound can be heard, and this type of sound is heard from bloated rumen, abomasum and intestine and is called “**Tympanic sound**”.

A.3. Observation

Buccal cavity and mucous membranes

Examine the mouth and appetite; oral lesions, salivation, feed intake should be noted. Ability to prehend, masticate and swallow food is also observed.

Excessive salvation, abnormalities of mucosa (the colour of the mucous membrane) is a good indicator of health, as it shows the condition of the blood.

Mucous membrane is found, on the gums, inside the mouth, and at the entrance to the anus. In healthy animals, it should show a salmon pink colouring) local lesions may be; haemorrhages, discoloration of jaundice and cyanosis or anaemia;

The colour of mucous membrane may change occurs in various diseases.

Anaemic mucous membranes:

- Blood loss anaemia.
- Parasitic infestations leading to haemolysis.
- Tumours or leucosis.
- Iron deficiency anaemia.
- Long-standing infectious diseases.
- Exposure to X-rays and some medications.

Congested mucous membranes:

- High environmental temperatures and exercise.
- Any disease resulting in fever.
- Diseases of the heart, brain and its membranes.

Yellowish or icteric mucous membranes:

- Icterus of jaundice occurs due to increase of blood bilirubin concentration (blood parasites, leptospirosis, hepatitis, cholangitis, cholecystitis and cholangiohepatitis).
- Infectious anaemia and contagious pleuropheumonia of horses.
- Chronic gastric dilatation.

Cyanosed mucous membranes:

- Bluish discoloration of visible mucous membranes resulting from presence of reduced haemoglobin in blood capillaries.
- Myocarditis, pericarditis.
- Plant and mineral intoxications.

Faeces

The aspect of faeces, odour, frequency of defecation and volume; consistency and covering are also observed:

- Observe the surface of the faeces, where mucus and blood always exists
- The colour of the faeces surface: Brown faeces indicate the presence of blood and very clear indicate presence of mucus.

- The odour of the faeces: Fishy smell generally indicates bleeding, and abnormal constituents in faeces

Discharges

Observation of aspect, colour and volume of discharges can be useful. The technician can also observe the following:

- Salivation; normal or excessive. Excessive salivation or frothing at the mouth denotes painful conditions of the mouth or pharynx or is associated with tremor of the jaw muscles due to nervous involvement.

B. Circulatory apparatus

The heart is suspended by great vessels and located on the left median mediastinum of ventral thorax. The left side of the heart apex reaches the chest wall. After locating the heart the following should be noted through physical examination (palpation, percussion, auscultation) the following would be examined:

- Heart rate
- Abnormal variation in heart rate
- Heart sounds
- Normal heart sounds (dub-lab)
- Adventitious heart sounds (murmurs)
- Pericardial frictional sounds
- Venous pulsation (jugular pulsation)

Auscultation (heartbeat, frequency, rythm)

Auscultation (based on the Latin verb auscultare "to listen") is listening to the internal sounds of the body, usually using a stethoscope. Auscultation is performed for the purposes of examining the circulatory and respiratory systems (heart and breath sounds), as well as the alimentary canal. The term was introduced by René Laennec in 1819 and he invented the instrument called “stethoscope”.

The heart sounds consist mainly of two separate noises occurring when the two sets of heart valves close. Either partial obstruction of these valves or leakage of blood through them because of imperfect closure results in turbulence in the blood current, causing audible, prolonged noises called murmurs. In certain congenital abnormalities of the heart and the blood vessels in the chest, the murmur may be continuous. Murmurs are often specifically diagnostic for diseases of the individual heart valves; that is, they sometimes reveal which heart valve is causing the ailment.

Likewise, modification of the quality of the heart sounds may reveal disease or weakness of the heart muscle.

Auscultation is also useful in determining the types of irregular rhythm of the heart and in discovering the sound peculiar to inflammation of the pericardium, the sac surrounding the heart.

Pulse rate

Pulse is defined as the regular expansion and contraction of the arterial wall caused by the flow of blood through it at every heartbeat. Pulse gives information with regard to the cardio-vascular abnormalities.

It is influenced by exercise, excitement, annoyance, relative humidity, environmental temperature.

Pulse can be adapted from the number of heart beats per minute by using stethoscope in less manageable animals. The rhythm of pulse should also be noticed while taking pulse. The pulse rate can rise rapidly in nervous animals or those which have undergone strenuous exercise. In such cases the pulse should be checked again after a period of rest lasting 5 to 10 minutes.

Pathological changes of pulse rate

Bradycardia (slow pulse rate): accompanied with brain diseases attended by great depression as: chronic and Subacute hydrocephalus, brain tumors, poisoning from alcohol and lead.

Tachycardia (Fast pulse rate): In most severe diseases especially when attended by fever, in most painful conditions: severe injuries, fractures and abscesses and in mental excitement as: fear or anxiety.

Observation of mucous membrane

The abnormalities of color of mucous membrane are cause by different factor like:

- Pallor of the mucous membranes may indicate anaemia caused by direct blood loss or by haemolysis, A blue tinge may indicate cyanosis caused by insufficient oxygen in the blood,
- A yellow colour is a sign of jaundice, the mucosae may be bright red (sometimes described as being ‘injected mucous membranes’) in febrile animals with septicaemia or viraemia.
- Bright red colouration of the conjunctiva is often seen in cases of bovine respiratory syncitial virus infection.
- A cherry-red colouration may be a feature of carbon monoxide poisoning.
- A greyish tinge in the mucosae may be seen in some cases of toxaemia – such membranes are sometimes said to be ‘dirty’.
- High levels of methaemoglobin, seen in cases of nitrate and/or nitrite poisoning, may cause the mucosae to be brown coloured.

Capillary refill time (CRT) - reflects perfusion of peripheral tissues. Press on an area of mucous membrane. The gums will "blanch" white as they are pressed and become pink again when pressure is released. Normal: < 2 seconds and Prolonged CRT (> 2 seconds) may indicate compromised circulation due to cold, shock, cardiovascular disease, anemia or other causes and must be further evaluated by a supervisor before admitting the animal for surgery.

C. Respiratory apparatus

Auscultation (Respiratory rhythm, frequency and sound)

Auscultation: Use stethoscope, listen to the respiration sound in the trachea or lung area.

Inspiratory or expiratory movements of the chest wall or flank can be counted. In cold weather, exhaled breaths can be counted. If the animal is restless the clinician should count the rate of breathing for a shorter period and use simple multiplication to calculate the respiratory rate in breaths/minute. Mouth breathing is abnormal in cattle and is usually an indication of very poor lung function or a failing circulation

During auscultation, the stethoscope should be moved systematically to cover the whole of thoracic lung fields with the aim of identifying any abnormal sounds present, their location and their occurrence in relation to the respiratory cycle.

The location of an abnormal sound is deduced from the position of maximal intensity. Particular attention should be given to the apical lobe if bacterial pneumonia is suspected or the diaphragmatic lobe if lungworm is suspected.

Auscultation also reveals the modification of sounds produced in the air tubes and sacs of the lungs during breathing when these structures are diseased.

Abnormal lung sounds

- 'Wheezes' (continuous high pitched hissing heard more often on expiration) - occur with small airway diseases such as asthma
- 'Rales/crackles' (course to fine - discontinuous, nonmusical, brief sounds heard more commonly on inspiration) – may be heard when fluid in the lungs
- 'Rhonchi' (musical sounds-low or high pitched) or - 'Wheezes'(continuous high pitched hissing heard more often on expiration)
- 'Dull' lung sounds may indicate pneumonia, or consolidation

- Absence of breath sounds may indicate pleural space disease (pleural effusion) or space-occupying lesions

Observation of discharges

Examine the nose and nasal sinuses; lesions, discharges should be noted. Classify discharge: (serous, purulent, hemorrhagic, mucoid or mucopurulent).

Sneezing and nasal discharge can appear together or can occur as separate problems. They are associated with disorders of the nasal cavity, nasal sinuses, or both. While an occasional sneeze is perfectly normal, repeated bouts of vigorous sneezing suggests irritation of the nasal cavity caused by:

- viruses (especially in cats)
- air-borne irritants such as powder, dust, fumes, or aerosol sprays
- foreign material such as plant fragments
- nasal mites in dogs (rare)
- conditions that cause nasal discharge

Nasal discharge may be caused by a number of different conditions such as:

- infection of the nasal cavity or nasal sinuses by viruses, fungi, or bacteria
- masses such as polyps or tumors
- inflammation caused by foreign material
- irritation caused by nasal mites (dogs)
- allergies
- conditions extending from the mouth such as abscessed tooth roots or tumors
- disease deep in the airways

D. Uro-genital apparatus

The manifestations of the urinary tract diseases include abnormal constituents and appearance of urine, changes in the volume of daily urine flow and frequency, pain and dysuria and uremia.

Observation of Uro-genital mucous membrane aspect

Local lesions, hemorrhages, discoloration and cyanosis (note that the pink color is normal coloration).

Observation of Urine coloration

Normal urine color ranges from pale yellow to deep amber; the result of a pigment called urochrome and how diluted or concentrated the urine is.

Pigments and other compounds in certain foods and medications can change your urine color. Many over-the-counter and prescription medications give urine brilliant tones, such as red, yellow or greenish blue.

Discolored urine is often caused by medications or food dyes. In some cases, though, changes in urine color can be caused by specific health problems.

The color categories here are approximate, because what looks like red to you might look like orange to someone else.

Red or pink urine

Despite its alarming appearance, red urine isn't necessarily serious. Red or pink urine can be caused by:

- Blood: Factors that can cause urinary blood (hematuria) include urinary tract infections, an enlarged prostate, cancerous and noncancerous tumors, kidney cysts, long-distance running, and kidney or bladder stones.
- Medications: Rifampin (Rifadin, Rimactane), an antibiotic often used to treat tuberculosis, can turn urine reddish orange — as can phenazopyridine (Pyridium), a drug that numbs urinary tract discomfort, and laxatives containing senna.

Orange urine

Orange urine can result from:

Medications: Medications that can turn urine orange include the anti-inflammatory drug sulfasalazine (Azulfidine); phenazopyridine (Pyridium); some laxatives; and certain chemotherapy drugs.

Medical conditions: In some cases, orange urine can indicate a problem with your liver or bile duct, especially if you also have light-colored stools. Dehydration, which can concentrate your urine and make it much deeper in color, can also make your urine appear orange.

Blue or green urine

Blue or green urine can be caused by:

Dyes: Some brightly colored food dyes can cause green urine. Dyes used for some tests of kidney and bladder function can turn urine blue.

Medications: A number of medications produce blue or green urine, including amitriptyline, indomethacin (Indocin, Tivorbex) and propofol (Diprivan).

Medical conditions: Familial benign hypercalcemia, a rare inherited disorder, is sometimes called blue diaper syndrome because children with the disorder have blue urine. Green urine sometimes occurs during urinary tract infections caused by pseudomonas bacteria.

Dark brown or cola-colored urine

Brown urine can result from:

Medications: A number of drugs can darken urine, including the antimalarial drugs chloroquine and primaquine, the antibiotics metronidazole (Flagyl) and nitrofurantoin (Furadantin), laxatives containing cascara or senna, and methocarbamol — a muscle relaxant.

Medical conditions: Some liver and kidney disorders and some urinary tract infections can turn urine dark brown.

Extreme exercise: Muscle injury from extreme exercise can result in pink or cola-colored urine and kidney damage.

Cloudy or murky urine

Urinary tract infections and kidney stones can cause urine to appear cloudy or murky.

Observation of Vaginal discharges

Visually assess mammary glands for tumors, cysts, swelling, heat or discharge. Inspect vulva for size, inflammation, discharge (blood, pus), polyps, tumors or structural defects Color, odour, frequence and volume should be observed.

Normal Vulvar Discharges

Postparturient (post-farrowing) discharges and peri-estrous discharges are considered normal vulvar discharges in the animal.

The hormone changes, associated with onset of estrus and ovulation, contribute to vulvar discharges in per-estrous animals.

Abnormal Discharges

Blood: It is common to note the presence of fresh blood on the vulva. In contrast, the swollen vulva of a sow during estrus is subject to trauma by breeding or gestation crates.

Post-breeding Purulent Discharges: The presence of a purulent vulvar discharge at 16 to 20 days after breeding (or estrus) typically is indicative of a uterine infection (metritis or endometritis).

E. Musculo-skeletal system

Observation of status of aplomb

Examine the joints long bones and different muscles: arthritis, dislocation of the joints, rickets or osteocalcin, muscular atrophy, central or peripheral origin paralysis.

Rickets is caused by either a deficiency of Vitamin D, or inefficient reactions in the body to the vitamin. This could be caused by parathyroid glands not functioning properly and releasing the proper amount of calcium and phosphorus in bones.

Observation of Movements

Gait is denotes the locomotor processes of the animals. A locomotor disturbance of the animal is judged by the movements (Walking, running, trotting, circling etc.) There are certain diseases, which interfere with the process of locomotion.

Table 1: Abnormality of gait in domestic animals.

Gait	Disease
Lameness and disinclination to move	Laminitis, foot rot
Stiff gait	Arthritis
Walking in circles / head pressing	Gid (Coenuruses)/otitis/ listeriosis
Dragging of the hind leg	subluxation of medial patellar ligament

Observation of Hoofs trimming

Hoof trimming plays a large role in the prevention of lameness in dairy cattle, goats and sheep. Hoof trimming is done at least twice a year; however the presence of infectious diseases can increase the need of hoof trimming as it may alleviate some of pain associated with these diseases commonly digital dermatitis. Diseases causing bacteria are often present in manure slurry. For hooves with overgrown toes, a heel with compromised structure will be more susceptible to bacterial invasion as it will be in contact with more slurry.

Observation of standing position

The musculoskeletal system is important for the maintenance of posture and for locomotion.

The aim of the clinical examination is **to identify the site and the cause of the lameness**. Identification of the affected limb, the site of the lesion within the limb and assessment of the severity of the lesion, are primary aims of the initial part of the examination. Localization to a foot or an upper limb problem may be possible by observation.

Table 2: Abnormal posture in animals

Posture	Disease/disorders
Cow sit on sternum with head flexed to one side and resting on the flank	Milk Fever
Abducted elbow with extension of head and neck accompanied by mouth breathing in farm animals	Pneumonia
Arched back with tense abdomen	Peritonitis
Extension and rigidity of limbs, neck, ear and jaw	Tetanus
A male dog urinating like a bitch	Cystitis

F. Integumentary system

Structures or parts associated with skin as its appendages are hoofs, hairs, horns, quills, claws, nails, sebaceous glands and sweat glands. Discrete skin lesions range in type from urticarial plaques to the circumscribed scabs of ringworm, pox and impetigo. Diffuse lesions include the **obvious enlargements** due to subcutaneous edema, hemorrhage and emphysema. Enlargements of lymph nodes and lymphatics are also evident when examining an animal from a distance.

Elasticity

Skin turgor is the skin's elasticity: it is the ability of the skin to change shape and return to normal.

Skin elasticity is the sign of fluid loss (dehydration). Diarrhoea or vomiting can cause fluid loss and fever speeds up this process.

To check up the skin elasticity, the clinician grasps the skin between two fingers commonly on the limbs and abdomen. Skin with normal elasticity will snap rapidly to its normal position and the one with poor elasticity will take time to return to its normal position.

Oedema, lumps, abscess and other conditions such as nodules, tumors and scabies may reduce skin elasticity.

Sensibility

The term is used for both the physiological reaction to stimuli and for the pathological, abnormal or excessive sensitivity to stimuli. Irritability can be demonstrated in behavioral responses to both physiological and behavioral stimuli, including environmental, situational, and emotional stimuli.

Irritability may be associated with loss of function in a focal area or diffuse damage. It has been associated with dementia, stroke or concussion, neurochemical changes, e.g. serotonin depletion, or hormonal changes, e.g. in postpartum depression

Photosensitization is a condition in which areas of skin exposed to light and lacking sufficient protective hair, wool or pigmentation, are very sensitive to sunlight due to the presence of chemicals called photodynamic agents

Hyperesthesia is a condition that involves an abnormal increase in sensitivity to stimuli of the sense. Increased touch sensitivity is referred to as "tactile hyperesthesia", and increased sound sensitivity is called "auditory hyperesthesia". In the context of pain hyperaesthesia can refer to an increase in sensitivity where there is both allodynia and hyperalgesia.

Coloration of the skin

In some conditions, such as jaundice, the skin may provide through discolouration direct diagnostic evidence of a specific disease process. In other conditions, such as parasitism or severe mineral deficiency, a nonspecific general deterioration of skin health may occur causing a greater number of hairs than normal to enter the telogen or resting phase and a delay in their replacement, leaving the coat in poor condition with little hair.

Discoloration of the skin may arise due to anaemia, cyanosis, jaundice, and hyperaemia. In febrile conditions hair may be erect and in all chronic diseases with disturbances in nutrition, hair become rough, lusterless dry and coarse. Alopecia (Loss of hair) may occur due to diseases like ring worm, scabies, eczema, Iodine and Vit A deficiencies.

Oedema

Edema, (alternate spelling: oedema) formerly referred to as dropsy or hydropsy, is the swelling of the body's tissues due to excess interstitial fluid retention. Edema can occur locally, often affecting the extremities (peripheral edema), or generally, affecting the entire body (anasarca).

Edema itself is not a disease, but a symptom. Less severe forms of edema can result from lifestyle and general health factors such as:

- Staying in one position (sitting, standing or supine) for too long
- Increased sodium intake
- Hormonal changes due to menstruation
- Pregnancy

Edema can also occur as a side effect of several different medications, including:

- Vasodilators
- Calcium channel blockers
- Estrogen-based medication

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Certain diabetes medications

However, edema can also be a sign of severe underlying medical conditions, such as:

- Congestive heart failure
- Cirrhosis
- Kidney disease
- Chronic venous insufficiency
- Chronic lung diseases (pulmonary edema only)
- A damaged lymphatic system

Lumps

Raised skin lumps are very common, and in most cases they're harmless. They can result from a number of conditions, including infections, allergic reactions, skin disorders, and skin cancer.

Skin lumps can vary in appearance and number depending on the cause. They may be the same color as your skin or a different color. They may be itchy, large, or small. Some can be hard while others can feel soft and movable.

Swelling of the maxillae and mandibles occurs in osteodystrophia fibrosa; in horses swelling of the facial bones is usually due to frontal sinusitis; in cattle enlargement of the maxilla or mandible is common in actinomycosis. opisthotonos is an excitation phenomenon associated with tetanus, strychnin poisoning, acute lead poisoning, hypomagnesemic tetany, polioencephalomalacia and encephalitis. Lumps all around the body may be the manifestation of lumpy skin disease (LSD).

Phlegmons

Phlegmon is a medical term describing an inflammation of soft tissue that spreads under the skin or inside the body. It's usually caused by an infection and produces pus.

Phlegmon is frequently caused by bacteria; most often group ***A*** *streptococcus* or *Staphylococcus aureus*:

- Bacteria may enter via a scratch, insect bite, or injury to form a phlegmon just under the skin on your finger or feet.
- Bacteria in your mouth can cause an oral phlegmon or abscess, especially after dental surgery.

- Bacteria can also attach to the wall of an internal organ such as the stomach wall or the appendix and form phlegmon.

Non-infectious causes of phlegmon include acute pancreatitis, where inflammation is caused by leaking of pancreatic digestive enzymes into the surrounding tissues.

Skin abcess

In order to confirm that the swelling is an abscess a sterile needle should be carefully pushed through the skin into the swelling after first shaving the overlying skin and applying spirit or antiseptic to the site of the insertion. If the swelling is an abscess then pus will appear in the hub of the needle. If a cyst there may be clear fluid, if a haematoma serum, if a tumour then possibly nothing may appear.

Abscesses occur when an animal gets a small wound and bacteria gets inside. Small wounds heal over very quickly, but the trapped bacteria grow and form a pocket of puss. Puncture wounds from bites are a very common cause of abscesses in dogs and cats, because the small puncture is an easy delivery for bacteria and the opening is very small so it closes up quickly.

G. Nervous system

Sensibility

In moderate to severe spinal cord lesions, superficial sensation may be absent from cranial aspect of the lesion caudally. In severe spinal cord lesions, deep pain is absent from periosteum of all toes and tail.

Somme diseases (tetanus, rabies) may affect nervous system and the test on sensibility, reflexes, and movement's coordination must be carried out.

Reflexes

The crossed extensor reflex generally is considered an abnormal reflex except in the standing position. In the normal recumbent animal, the extension response is inhibited through descending pathways. Crossed extensor reflexes result from lesions in ipsilateral descending pathways. The crossed extensor reflex has been considered evidence of a severe spinal cord lesion. Animals that are still ambulatory may have crossed extensor reflexes, especially when the lesion is in the cervical spinal cord or the brainstem.

Encephalomyelopathies is manifested when animals presents as stiffness of the limbs and/or trunk. Patients lack sensory deficits, abnormal reflexes, or upper motor neuron signs, so that Parkinson's disease or a related disorder is suspected.

Movement's coordination

When, on being approached, an animal makes a normal response to external stimuli, such as movement and sound, the demeanor is said normal (bright). Normal reaction under these circumstances may consist of elevating the head and ears, turning towards and directing the attention at the source of stimuli, walking away and evincing signs of attack or flight.

The word ataxia means incoordination within the nervous system. The abnormal movement may occur in the legs, the head, the torso, or all three. There are three different forms of ataxia:

- **Proprioception;** the unconscious body awareness. When there is a proprioceptive abnormality, movement is difficult and quite abnormal.
- **Vestibular abnormality** or **vestibular syndrome**; it is another ataxia that occurs as result of abnormal function of inner ear and brainstem that causes disruption in body balance.
- **The cerebellum ataxia**; is localised in the part of the brain where fine motor movement is coordinated. These dogs often look normal at rest, but when they start to move, their limb movements can be quite exaggerated and they typically also have head tremors.

There are potentially many different causes of ataxia, depending on where the problem is located.

Spinal cord problems causing ataxia:

- Loss of spinal cord tissue (called degenerative myelopathy)
- A "stroke" to the spinal cord (called fibrocartilaginous embolus or FCE)
- Structural/developmental abnormality of the spine or spinal cord
- Tumors in the spine or spinal cord
- Infection in the vertebrae or intervertebral discs
- Inflammation of the spinal cord
- Trauma to the spinal cord
- Instability in the spine causing pressure on the spinal cord
- Narrowing of the spinal canal which protects the spinal cord

Vestibular causes of ataxia (inner ear):

- Middle or inner ear infection
- Geriatric vestibular disease in which the precise cause may never be identified
- Hypothyroidism
- Tumors in the ear or skull
- Trauma to the head/ear

Vestibular causes of ataxia (brainstem):

- Infection (e.g., canine distemper virus)
- Inflammation (the cause of which may or may not be uncovered)
- Thiamine deficiency (uncommon with current nutritional products)
- Metronidazole (an antibiotic) toxicity

Cerebellar causes of ataxia:

- Degenerative changes in the cerebellum
- Structural abnormalities (e.g., underdevelopment or malformation of the cerebellum or the surrounding skull)
- Brain tumor
- Infection or inflammation in the brain
- Metronidazole (an antibiotic) toxicity

Miscellaneous causes of ataxia:

- Too low or too high red blood cell count
- Low levels of potassium, calcium, or glucose (blood sugar)
- Heart/circulation and/or respiratory diseases

L O 2.2 – Perform Routine laboratory examination

- Topic 1: Sampling techniques

A. General principles for sample collection

Sample collection should follow the principles below:

- Samples should be taken from the affected site(s) as early as possible following the onset of clinical signs. This is particularly important in viral diseases as shedding of virus is usually maximal early in the infection. This is also true of enteric bacterial pathogens.
- It is useful to collect samples from clinical cases and in-contact animals, particularly if there has been an outbreak of disease. In-contact animals may be at an earlier stage in the infection with a greater chance of them shedding substantial numbers of microorganisms.
- Samples should be obtained from the edge of lesions and some macroscopically normal tissue included. Microbial replication will be most active at the lesion's edge.
- It is important to collect specimens as aseptically as possible; otherwise the relevant pathogen may be overgrown by numerous contaminating bacteria population of normal flora.
- The laboratory should be informed if treatment has commenced in order that counteractive measures may be taken to increase the possibility of isolating bacteria or that an alternative method of detection such as polymerase chain reaction (PCR) may be employed.
- When possible a generous amount of sample should be taken and submitted, such as blocks of tissue (approximately 2 cm3), biopsy material, or several millilitres of pus, exudate or faeces. For serology, at least 5 mL of blood should be obtained to allow a number of tests to be carried out if necessary and to allow the sample to be stored and tested with subsequent samples.
- Cross-contamination between samples must be avoided. This is essential where a highly sensitive amplification technique such as PCR is to be used for the detection of the aetiological agent.
- Precautions must be taken to avoid human infection where a zoonotic condition is suspected.

B. Sampling techniques

B.1. Faeces sampling

During sampling faeces you should consider the following:

- A faeces sample freshly voided or collected from the rectum is preferable to a rectal swab which often does not have enough faecal matter for agent detection.
- A faeces sample (about the size of the end of a thumb) may be forwarded to the laboratory without transport medium.

- Faecal swabs should be placed in medium such as buffered glycerol saline to avoid desiccation. Some organisms are shed intermittently and samples may need to be collected over several days.

B.2. Blood sampling

During sampling blood you should consider the following:

- Blood should be withdrawn using an aseptic technique into a dry syringe or vacutainer.
- If collected in a syringe, care must be taken not to cause haemolysis. The needle should be removed prior to expelling the sample carefully into a sterile dry tube. It is not acceptable to submit blood to the laboratory in a syringe.
- Glass tubes are fragile; blood clots often retract poorly in plastic bottles making it difficult to separate the serum.
- Whole blood should never be frozen prior to submission to a laboratory as the ensuing lysis of red blood cells makes it impossible to perform many serological assays.
- Serological tests are usually performed on the serum harvested from clotted samples.
- Paired samples are frequently required to make a definitive diagnosis on the basis of serology.
- Blood for the isolation of viruses or bacteria should be prevented from clotting. The laboratory should advise on the most appropriate anticoagulant.
- As a bacteraemia canbe intermittent, it is advisable to take more than one sample within a 24-hour period.
- The blood should be added aseptically and without delay to one of the special commercial blood-culture bottles.
- Blood samples for Tissues from outside the body cavities should be collected first followed by tissues from the thorax and then the abdomen. Sterile instruments should be used to collect tissue samples of at least 1 cm^3 which should then be placed in separate sterile screw-capped jars. If the laboratory is some distance away tissue may be forwarded in virus or bacterial transport medium. It is important to remember that virus transport medium usually contains antibiotics thus rendering the sample unsuitable for bacterial examination.
- Tissue for histopathological examination should be placed in at least 10 times its volume of neutral buffered 10% formalin.
- In cases of abortion the whole foetus and placenta should be submitted. If this is not possible then samples of tissue, a piece of affected placenta, foetal abomasal contents (ruminants) and uterine discharge (if applicable) should be forwarded to the laboratory. A clotted blood sample from the dam for serological examination may yield additional.

B.3. Samples from skin lesions

During sampling skin lesion you should consider the following:

- If intact pustules or vesicles are present, the surface should be disinfected with 70% ethyl alcohol, allowed to dry, and material aspirated from the lesion with a sterile syringe and fine needle. A swab may be taken from the raw surface of ulcers. A biopsy of wound tissue should be collected after the superficial area has been cleaned and debrided.
- In cases where ringworm is suspected, hair should be plucked from the lesion and the edge of the lesion scraped with a blunt scalpel blade until blood begins to ooze. Plucked hair, skin scrapings (including the scalpel blade itself) and any scab material that is present should be submitted.
- These specimens will also allow detection of mange or a bacterial infection, if present. In cases of orf the crust and scrapings from the edge of the lesion should be collected.
- In birds feather follicle skin is useful in the diagnosis of Marek's disease.

B.4. Swabs and discharges sampling

During sampling swabs and discharges you should consider the following:

- Fluids are preferable to swabs as the greater sample volume increases the likelihood of detecting the causal organism.
- Samples for agent isolation should be placed in sterile containers. Viruses and many bacteria are susceptible to desiccation especially if collected on a dry swab. Formulae for suitable transport media for viruses, chlamydia and other organisms must be respcted.
- Whenever possible the sample should be collected from the specific site of infection.
- The usual short cotton wool swabs are generally unsatisfactory for obtaining nasopharyngeal specimens of epithelial cells and mucus for the investigation of respiratory disease of large animals.
- Guarded swabs are necessary for certain bacteriological examinations where misleading results could be generated due to contaminants from adjacent sites that are colonized with bacterial culture should be kept cool and submitted to the laboratory as quickly as possible.

B.5. Urine samples

During sampling urine you should consider the following:

- Urine samples may be submitted for urinalysis, bacterial microscopy and culture or for a bacterial viable count to establish whether clinical bacteriuria is present.
- For bacteriological procedures the preferred methods of collection are by cystocentesis, by catheter or mid-stream urine sample.

B.6. Abscesses sampling

During sampling abescesse you should consider the following:

- If possible about 3 mL of pus should be collected together with scrapings from the wall of the abscess.
- Pus at the centre of an abscess is often sterile.
- Pus from recently formed abscesses will yield the best cultural results.
- Anaerobic bacteria can often be cultured from abscesses.

B.7. Eye sampling

During sampling an eye you should consider the following:

- A conjunctival swab may be taken gently holding the palpebrae apart.
- Scrapings may also be taken with a fine sterile spatula.
- The cells should be washed carefully into transport medium.

B.8. Bovine mastitic milk samples

During sampling mastitic milk you should consider the following:

- Milk samples should be collected from cows as soon as possible after the mastitis is first noticed and not from animals treated with either intramammary or systemic antibiotics. The udder should not be rinsed with water unless very dirty. If the udder and teats are washed, they should be dried thoroughly with a paper towel.
- Usually it is sufficient to wipe the teats vigorously, using 70% ethyl alcohol on cotton wool, paying special attention to the teat sphincters.
- Antiseptics should be avoided.
- The two teats furthest from the operator are wiped first and then the two nearest teats.
- The sterile narrow-necked collection bottle must be held almost horizontally.

- The first squirt of milk from each teat is discarded and then, for a composite sample, a little milk from each quarter is readily contaminated the nasal passages and upper trachea.
- The diagnostic laboratory should be consulted before collecting samples for the isolation of specific pathogens that require specialized media or culture conditions, for example, *Taylorella equigenitalis*, *Chlamydophila psittaci* or *Mycoplasma* species.
- The laboratory will either supply specialist swabs and transport media or recommend a reputable source, as appropriate.

C. <u>Sample Submission</u>

Submission of sample is a critical stage in sampling:

- Laboratories usually supply a variety of sample containers and transport media.
- The laboratory should supply sample submission forms. These forms must be completed by the veterinary practitioner and must give specific details of the tests required as well as clinical history, differential diagnoses, vaccination history, therapy, age of animal. The latter will enable the microbiologist to choose the most appropriate tests and to avoid unnecessary expenditure.
- A complete history will also allow the laboratory to identify a particularly urgent situation and expedite the processing of the sample. In certain instances owners may be concerned that information in relation to their animals is kept confidential.
- In such circumstances the clinician can assign a code name or number to the animal and the owner but should never omit clinical detail that will facilitate the selection of the appropriate tests and the interpretation of the results.
- The laboratory must be notified if the differential diagnosis includes a fungal infection or any agent that is potentially infectious for people.
- Many laboratories only set up certain tests on particular days or at a designated time each day. Clinicians need to familiarize themselves with the laboratory timetable in order for them to provide an efficient service to their own clients. An awareness of the time it takes to perform certain assays is essential. Some assays may take weeks to complete.
- Where certification is required, samples should be submitted in good time and allowance should be made for repeat testing and/or the collection of a second sample if necessary.
- The clinician should contact the laboratory in advance if they are not a regular client or if the tests required are not routine. It is essential to give the laboratory adequate time to prepare for the receipt of a sample which requires specialist testing.

- It is inadvisable, for example, to submit a sample that needs to be passaged on a cell line that the laboratory does not use on a regular basis without prior discussion. In such circumstances the sample may have to be stored for days if not weeks, while cells are resuscitated from the freezer.
- In certain cases it may be necessary to forward the sample to a specialist laboratory. If samples are being submitted to a laboratory in another country an import licence may be required.
- Samples should be collected and delivered to the laboratory as early in the day as possible so that processing can commence on the same day. If a result is required urgently this must be indicated on the request form and the head of the laboratory should be notified in advance of the arrival of the sample. Prompt delivery to the laboratory will maximize the possibility of obtaining a diagnosis.
- Viruses only replicate in living cells and a delay in sample submission may result in loss of viability. The transit time needs to be minimized. Samples should be transported in contact with cold packs or wet ice.
- If transportation to the laboratory is delayed, most samples should be held in the refrigerator at +4°C rather than frozen.
- Serum harvested from clotted blood samples can be stored frozen for extended periods.
- The labelling of samples must be clear and unambiguous. Samples must be submitted individually in separate leak-proof containers that are clearly marked, indicating the identity of the sample (tissue, exudate, etc.), animal identification and the date of collection.
- Container caps should be screwed on tightly and taped, if necessary, to avoid leakage.
- All samples sent in the post must be labelled and packed in accordance with the regulations of the postal authorities.
- Glass tubes and other fragile containers must be adequately packaged to ensure they are not broken in the mail.
- Samples must always be surrounded by sufficient absorbent material to soak up the entire sample in the case of breakage or leakage.
- In general, triple packaging of diagnostic samples is required. The secondary or outer packaging must be a rigid container.
- The package should be clearly labelled with the words ‘biological substances directed into the bottle.
- The milk should be collected from the two near teats first and then from the two far teats, so that one’s arm is less likely to accidentally brush against a cleaned teat.

- Topic 2: Calfornia mastitis test (CMT)

The California Mastitis Test (CMT, also known as the California Milk Test) is a simple indicator of the Somatic Cell Count (SCC) of milk. It works by using a reagent which disrupts the cell membrane of somatic cells present in the milk sample; the DNA in those cells to reacting with the test reagent.

It is a simple but very useful technique for detecting subclinical mastitis on-farm, providing an immediate result and can be used by any member of farm staff. It is not a replacement for individual laboratory cell count sampling, but has several important uses.

A four-well plastic paddle is used, one well being used for each quarter of the cow to be tested. The foremilk is discarded, and then a little milk drawn into each well. An equal volume of test reagent is added and then the sample is gently agitated.

The reaction is scored on a scale of 0 (the mixture remaining unchanged) to 3 (an almost-solid gel forming), with a score of 2 or 3 being considered a positive result. This result is not a numerical result but is an indication as to whether the cell count is high or low; the CMT will only show changes in cell counts above 200,000.

The advantage of the CMT over individual cow cell count results is that it assesses the level of infection of individual quarters rather than providing an overall udder result, enabling the problem quarter(s) to be identified. It also provides a 'real-time' result; laboratory testing provides a historical result as it can take days for lab results to be returned.

A special reagent for the test is sold as 'CMT-Test', but domestic detergents ('washing-up liquid') can generally be substituted, being cheaper and more readily-available.

A. CMT kit components

- CMT paddle (with 4segments or wells);
- Bottle of CMT liquid (reagent).

B. Procedures

Milk samples from each quarter are collected in a clean CMT Paddle. The CMT paddle has four shallow cups marked A, B, C, and D to help identify the individual quarter from which the milk was obtained. The CMT solution should be reconstituted according to package instructions.

B.1. Steps of CMT

Step 1:

Take about 1 teaspoon (2 cc) milk from each quarter. This is the amount of milk that would be left in the cups if the CMT Paddle were held nearly vertical

Step 2:

Add an equal amount of CMT solution to each cup in the paddle.

Step 3:

Rotate the CMT Paddle in a circular motion to thoroughly mix the contents.

Do not mix more than 10 seconds.

Step 4:

Read the test quickly. Visible reaction disintegrates after about 20 seconds. The reaction is scored visually. The more gel formation, the higher the score.

B.2. Reading CMT results

N = Negative:

No infections. No thickening of the mixture.

200,000 SCC

T = Trace: Possible infections. Slight thickening of the mixture.

Trace reaction seems to disappear with continued rotation of the paddle. 200,000 to 400,000 SCC Example: If all four quarters read trace there is no infection. If one or two quarters read trace, infections are possible.

1 = Weak Positive Infected. Distinct thickening of the mixture, but no tendency to form a gel. If CMT paddle is rotated more than 20 seconds, thickening may disappear.
400,000 to 1,200,000 SCC.

2 = Distinct Positive Infected. Immediate thickening of the mixture, with a slight gel formation. As mixture is swirled, it moves toward the center of the cup, exposing the bottom of the outer edge. When motion stops, mixture levels out and covers bottom of the cup. 1,200000 to 5, 00,000 million SCC.

3 = Strong Positive Infected. Gel is formed and surface of the mixture becomes elevated (like a fried egg). Central peak remains projected even after the CMT paddle rotation is stopped. 8.1 million SCC.

Difficult to pour out easily in a well.

Note: Remember to rinse the CMT paddle after each test.

B.3. Interpretation of CMT results

Table 3: Interpretation of CMT results

CMT score	Somatic cell range	Interpretation
N(Negative)	0-200,000	Healthy quarter
T(Trace)	200,000-400,000	Subclinical mastitis
1	400,000-1,200,000	Subclinical mastitis
2	1,200,000-5,000,000	Serious mastitis infection
3	Over 5,000,000	Serious mastitis infection

B.4. Record keeping

Register is important for keeping obtained results; the recording operation must respect the quarters (left front, left back, right front, and right back) as they are corresponding to paddle wells named A, B, C, and D. This will help in identification of healthy quarter and sure regular checking of the herd.

- Topic 3: Milk ring test

A. Test procedure

The test is performed as follow:

- The test is performed by adding 30 µl of antigen to a 1 ml volume of whole milk that has been stored for at least 24 hours at 4°C.
- The height of the milk column in the tube must be at least 25 mm.
- If bulk tank samples from large herds are to be examined, the volume of milk should be increased to 3 ml.
- The milk samples must not have been frozen, heated or subjected to violent shaking.
- The milk/antigen mixtures are incubated at 37°C for 1 hour, together with positive and negative control samples.
- A strongly positive reaction is indicated by formation of a dark blue ring above a white milk column. Any blue layer at the interface of milk and cream should be considered positive as it might be significant, especially in large herds.
- The test is considered to be negative if the colour of the underlying milk remains homogeneously dispersed in the milk column. If the milk at the bottom of the tube becomes gradually whitened, the result is regarded as inconclusive and the test should be repeated.
- A sample of whole milk is mixed well with a drop of the stained Brucella antigen (a concentrated suspension of killed *B. abortus* stained with hematoxyilin) and incubated in a water bath at 70c for 40-50 min. If antibodies are present in the milk the bacilli are agglutinated to form a blue ring at the top. If antibodies are absent no colored ring is formed, and the milk remains uniformly blue.

- Topic 4: Tuberculin test

The primary screening test for TB in cattle in Great Britain is the **Single Intradermal Comparative Cervical Tuberculin** (SICCT) test, which is commonly known as the tuberculin “skin test”.

The tuberculin skin test, which is used throughout the world to screen cattle, other animals and people for TB, is the internationally accepted standard for detection of infection with *Mycobacterium bovis* (*M. bovis*), and is considered the best test currently available for detecting TB in live animals.

A. Procedure

The test involves injecting a small amount (0,1ml) of tuberculin (a sterile antigenic extract obtained from a culture of *M. bovis* or other organisms of the same family of bacteria) into the skin of the animal. In most cattle infected with *M. bovis*, this will cause the animal's immune system to react to the tuberculin and cause a localised allergic reaction (swelling) of the skin a few days after the injection.

Cattle are sometimes infected with other types of mycobacteria which may cause the animal to react to the test. So that we can distinguish between animals infected with *M. bovis* and those infected by other mycobacteria, we also inject the animal with tuberculin produced from *Mycobacterium avium*, an organism that can cause TB in birds. The size and nature of the reactions to both tuberculins ('avian' and 'bovine') is compared (hence the term single intradermal **comparative** cervical tuberculin test) to determine whether the test result is considered positive, negative or inconclusive.

In tuberculin test the **6 basic steps** are o followed:

- The animal is identified (by its ear tag) and its identification recorded.
- Two injection sites are selected in the middle third of the side of the neck, one above the other, separated by about 10-15cm. (if it is a small animal, the two sites will be on either side of the neck.)
- Hair is clipped around the sites to a radius of about 2 centimeters.
- A fold of skin at both sites is measured with calipers and the measurements recorded.
- Tuberculin is injected into the skin; the upper site is used for the avian tuberculin (or the left hand side on small calves).
- After 72 hours, the tester returns, confirms the animal identity, measures the same fold of skin at both sites and records the thickness of the skin fold.

B. Interpretation of results

Note that false positive and false negative reactions may occur and they may also look into before making any interpretation.

Circumstances (situation) in which false negative results may occur:

- Early of infection; tuberculin test may become positive only after 6 weeks of infection;
- Later stage of infection;
- In old animals;
- Test carried out just after parturition;
- Within 60 days of tuberculin test.

Circumstances in which false positive results may occur:

- Animal sensitized to other allergen like hydatid disease, bovine farcy;
- If the animal is infected with non-pathogenic mycobacteria (saprophytic mycobacteria).

- Topic 5: Sero-agglutination test

A corpuscular antigen - agglutinogen - is agglutinated when the specific antibody - agglutinin - is added. Agglutination can be read either visually or in the microscope. Presence or absence of clumping is noted. Antigen - antibody reaction can be visualized in different ways according to the type of the antigen, conditions of the reaction and the medium the reaction takes place in.

When an antibody combines with a corpuscular antigen (forming part of a cell - e.g. bacteria, virus, blood cell or inert part with bound antigen) the cells agglutinate that means they form clumps.

When an antibody combines with a non-corpuscular antigen (toxin, enzyme, microbial extract) a precipitate is formed, antigen - antibody complex is thrown out of solution. The test relies on reaction antigen-antibody making clumps. Below are the examples of agglutination procedures.

A. Slide agglutination test of brucellosis

Procedures:

- Take a clean dry glass slide;
- Place a drop of antigen over the middle area of the slide;
- Add one drop of test serum and mix properly with the help of clean glass rod or tooth pick;
- If the homologous antibodies are present the clumps of bacterial cells will occur through agitation;
- Clumps may be seen by naked eyes or by using a light microscope.

B. Tube agglutination test of brucellosis

Procedures:

- An equal amount of brucella antigen(brucella antigen is commercially available) and serum is mixed properly with agitation of the lack holding tubes;
- The tubes are then kept at 37^0C in the incubator for 24 hours;
- If agglutination occurs, the clumps of antigen and antibody complex will settle down leaving aclear supernatant;
- In case of no agglutination the turbid suspension remain the same.

• Topic 6: Blood smear

A smear is a preparations process where a specimen that is spread on a slide. You prepare a smear using the heat fixation process.

A. Principle

Animal blood is used to make a smear stained by one fast staining technique (alternative of May-Grünwald–Giemsa staining) in order to observe under the microscope the illustrated elements.

B. Protocol

You have to follow the procedures bellow:

- Place a small drop of fresh, well mixed anticoagulated blood on a clean glass slide approximately 2 cm from one end of the slide.
- Place a clean glass “spreader” slide in front of the drop of blood at an approximate 30-45° angle to the blood-film slide. The angle depends on nature of the blood-film (thin, thick).
- Back the “spreader” slide into the drop of blood.
- Let the blood spread along the contact line between the two slides; the spread is done by capillarity this should take place quickly.
- With a steady fluid movement, move the spreader slide down the entire blood-film slide, maintaining the angle without lifting the spreader slide. Blood from the drop will follow the spreader slide, placing a thin film on the other slide. The blood film should be 3 – 4 cm in length.
- Let the blood film air-dry with gentle agitations. Never blow dry.

L O 2.3 – Perform Differential Diagnosis and prognosis

A. Pathognomonic symptoms

Pathognomonic (rare synonym pathognomic) is a term, often used in medicine, that means "characteristic for a particular disease". A pathognomonic sign is a particular sign whose presence means that a particular disease is present beyond any doubt. Labelling a sign or symptom "pathognomonic" represents a marked intensification of a "diagnostic" sign or symptom.

While some findings may be classic, typical or highly suggestive in a certain condition, they may not occur uniquely in this condition and therefore may not directly imply a specific diagnosis. A pathognomonic sign or symptom has very high positive predictive value but does not need to have high sensitivity: for example it can sometimes be absent in a certain disease, since the term only implies that, when it is present, the doctor instantly knows the patient's illness. The presence of a pathognomonic finding allows immediate diagnosis, since there are no other conditions in the differential diagnosis.

B. Diagnosis (result interpretation)

Properly establishing and then understanding the performance of diagnostic tests is crucial to the design and operation of infection control and eradication campaigns incorporating these tests. Most diagnostic tests are imperfect, so that often a test result cannot be interpreted with certainty, and properly establishing test performance is technically demanding and expensive.

A test result is often classified into a category, such as positive or negative.

For bacterial culture

- A positive test may be the appearance of colonies on the medium that has a colony morphology consistent with that of the bacteria of interest within the appropriate timeframe.
- A negative test would be the failure of growth of any colonies with the appropriate morphology to appear by the time incubation is ended.

For a serologic test such as Enzyme-Linked Immunosorbent Assay (ELISA)

- A positive test result might be the appearance of a particular color marker of sufficient intensity (optical density) that it is above a cutoff value.
- A negative test result would be when the color intensity is below the cutoff value.

When test outcomes are classified as positive or negative, present or absent, the result can be wrong in two ways.

A positive result can be a true positive, correct, or a false positive, wrong.

A false positive occurs when the condition being tested for is actually not present in the animal, but the test indicates that it is

Fecal bacterial culture

A false positive could occur when organisms in contaminated feed or water are consumed by an animal, and then pass through the animal rather than infect it. Alternatively, false positive cultures could occur from accidental laboratory contamination or other error.

Serologic test such as ELISA

The false positive result could occur because the animal responded to an antigen in their environment that is immunologically similar to the target antigen from Map that is used in the test.

A negative test result can be a true negative or a false negative. A false negative test means that the condition is present in the animal but the test indicates that it is not.

In the case of fecal culture

A false negative could occur because the number of organisms in the fecal specimen is too few to be detected, but the animal is infected.

In the case of serologic tests

The test could be a false negative because an infected animal has not mounted the particular immune response that the test detects, or has mounted a weak immune response that is below the threshold of detection as a positive result.

The measure that is most useful when interpreting an uncertain test result is the predictive value, which is the likelihood that a test result is correct:

- The positive predictive value (PPV) is defined as the likelihood that a positive test result is a true positive

- The negative predictive value (NPV) is defined as the likelihood that a negative test result is a true negative

These predictive values depend on the prevalence of the condition being tested for in the population being tested, meaning both PPV and NPV are different when the test is used in an uninfected group compared to when the test is used in an infected group with a high prevalence:

- Intuitively, a positive test is more likely to be a false positive in a herd without any history of having any animals diagnosed with the infection.
- Similarly, a negative test is more likely to be a false negative in a herd with a history of having many animals with confirmed disease.

As a consequence, predictive values are not useful for comparing test performance across groups of animals with significantly different infection prevalences.

Because of the dependence of predictive values on the actual infection prevalence (as opposed to the apparent test prevalence) in the group being tested, they are generally not useful as a basis for comparing test performance even though they are often reported.

Instead, tests are most usefully compared on the basis of their respective epidemiological sensitivity and specificity values

- Sensitivity is defined as the probability that an animal with the infection will test positive (true positive,).
- Specificity is defined as the probability that an animal without the infection will test negative (true negative), respectively

It is important to note that test sensitivity depends on the spectrum of disease in the group of individuals in which the test is being used. Because the disease process is more advanced in an individual with advanced clinical disease, a test will usually be more sensitive in that animal than in an individual that was just infected and pathological changes are not as advanced.

Similarly, a test will work better in a herd with long-established disease than in a herd with recently introduced disease, since the long-infected herd will have both a higher prevalence and more individuals with advanced disease.

C. Prognosis

The information required for a reasonably accurate prognosis includes:

- The expected morbidity and case fatality rates for the disease;
- The stage of the disease;
- Whether or not a specific treatment or surgical operation is available or possible
- The cost of the treatment.

Prognosis (Greek: "fore-knowing, foreseeing") is a medical term for predicting the likely or expected development of a disease, including whether the signs and symptoms will improve or worsen (and how quickly) or remain stable over time; expectations of quality of life, such as the ability to carry out daily activities; the potential for complications and associated health issues; and the likelihood of survival (including life expectancy).

A prognosis is made on the basis of the normal course of the diagnosed disease, the individual's physical and mental condition, the available treatments, and additional factors.

Prognosis vs. Diagnosis

People often confuse the terms prognosis and diagnosis. The difference between the two is that while a prognosis is a guess as to the outcome of treatment, a diagnosis is actually identifying the problem and giving it a name, such as depression or obsessive-compulsive disorder.

Factors That Affect Prognosis

Different factors can affect the prognosis of each individual. These factors include:

- Age
- Gender
- Medical and/or family history
- How the disease or disorder is presenting
- Response to treatment
- Particular symptoms and how long they have been present
- Whether or not there are other illnesses or conditions present
- What treatment or treatments are being used

C.1. Favourable

A favorable prognosis means a good chance of treatment success. For example, the overall 5-year relative survival rate for testicular cancer is 95%. This means that most men diagnosed with the disease have a favorable prognosis. Prognosis depends on the stage of the cancer at diagnosis.

C.2. Unfavourable prognosis

An unfavorable prognosis means a bad chance of treatment success. For example; the overall 5-year relative death rate for testicular cancer is 5%. This means that men diagnosed with the disease have an unfavorable prognosis.

D. Decision making

Shared decision-making is a process where you are actively involved in making decisions around your healthcare alongside of your healthcare provider(s). It provides you with an opportunity to be informed about your treatment, share your concerns, wants or needs, and have a say in your treatment plan.

There are a lot of benefits that come with participating in shared decision-making. You may find that shared decision-making:

- Increases your treatment satisfaction
- Increases your confidence in treatment choice
- Can help improve your treatment adherence

Noted that the decision making for treatment of patient; should be supported by the animal owner.

Refer the cases

The clinician may refer or consult with other clinicians (formally or informally) to seek additional expertise about the patient health problems. The consult may help to confirm or reject the working diagnosis or may provide information on potential treatment options.

If the health problems are outside of clinician's area of expertise, he/she may refer the patient to the clinician who holds more suitable expertise. Clinician can also recommend the patient seek a second opinion from another clinician to verify their impressions of an uncertain diagnosis or if they believe this would be helpful to the patient.

Treat

Picking a medication is not easy. For decisions about medical treatments, prognosis, and treatment options; are key communicators and coordinators with providers during clinic visits where treatment decisions are made.

Decision-making in veterinary care is typically divided between the animal's owner and the treating veterinarian. And these veterinarians and animal owners are faced with difficult choices every day in caring for sick, injured, and dying animals.

To some extent, those choices are guided by the legal and professional ethical obligations of the veterinarian. While the animal owner faces fewer legal restrictions, her choices will be guided by a number of factors, including;

- emotional attachment,
- a sense of duty,
- an understanding of the animal's condition and the various options for treating it,
- The cost of the treatment choices, and her ability to pay for it.

Challenges may arise when no obvious “right” choice presents itself, especially where there may be disagreement between the animal's owner, the treating veterinarian, and other interested parties concerning what should be done. But perhaps even greater challenges occur when a treating veterinarian is certain that an animal's owner is making the wrong choice and sees little she can do to influence the outcome.

On the other hand, owner choices are often directed by the information they receive from veterinarians, who have superior understanding of the animal's condition, the available treatment options, and the likely outcome of various treatment choices. Veterinarians may therefore be able to manipulate owner choice by withholding or selectively presenting this information. In fact, an ethical dilemma that is sometimes cited by treating veterinarians is whether to present owners with the full range of treatment options.

E. Complementary exam

Complementary exams should be done periodically by a veterinarian and consist both physical examination to apply general inspection, palpation, percussion and auscultation methods used to detect clinical signs of abnormalities and clinical examination to check if the animals have further health problems.

General Physical examination can be carried out by taking vital sign such as; Temperature taking, Pulse taking, Respiration taking, Capillary Refill Time (CRT), Physical body condition, Normal demeanor, Abnormal demeanor.

Learning Unit 3 – Apply treatment for infectious diseases

L O 3.1 – Select appropriate treatment

In all cases the selection of treatment to be applied will based on the diagnosis and prognosis.

- Topic 1: Types of treatment

A. Specific treatment

Specific treatment is the causal treatment or treatment which is directed against the cause of a disease. This type of treatment is instituted when the etiology for the disease is known, but it is generally associated to symptomatic treatment.

B. Symptomatic treatment

Symptomatic treatment is any medical therapy that aims at treating symptoms of a disease, not its cause (its etiology). It is usually aimed at reducing the signs and symptoms for the comfort and well-being of the patient, but it also may be useful in reducing organic consequences or sequelae of these signs and symptoms of the disease. In many diseases, even in those whose etiologies are known (e.g., most viral diseases, such as influenza), symptomatic treatment is the only one available so far.

Examples of symptomatic treatments:

- Analgesics, for pain
- Anti-inflammatory agents, for inflammation caused by arthritis
- Antitussives, for cough

Parietive / supportive treatment can be provided

C. Economical treatment

Sommetimes the econmic treatment is suggested:

- When the disease is judged to incurable;
- When treated animal treated will apparently be healed but continues to spead disease(healthy carriers);
- The cost of the treatment; if success is dependent on prolonged and intensive therapy, the high cost may be prohibitive to the owner and economic treatment may be suggested.

L O 3.2 – Prescribe medicines

After diagnosis, if the treatment (specific treatment) must be carried out the following step is to prescribe the appropriate medicine by physician.

- Topic1: Identification of anti-infectious drugs

Anti-infectious drugs that are used in animal diseases treatment may be divided into 3 main groups; antibiotics, Sulfonamides and anti-inflammatory.

A. Antibiotics

Antibiotics are medicines that have activity against bacteria. They may function by either killing the bacteria (bactericidal) or by inhibiting the growth and proliferation of bacteria (bacteriostatic) allowing the animal's immune system to more effectively fight a bacterial infection. In either case, the bottom line is that an antibiotic treatment stops the growth of a bacterial infection so the host (i.e. the animal) can eliminate it. The animal can then recovers and return to health.

Antibiotics have broad spectrum (Tetracycline, Chloramphenicol) or narrow spectrum (macrolides acting on aerobic and anaerobic gram-positive bacteria) activity and they may be bactericide or bacteriostatic.

Classification

Classification of antimicrobial agents is based on their chemical composition and mode of action.

Table 4: Classification of antimicrobial agents

Antibiotics category	Brief Mode of action	Examples
Aminoglycosides	Inhibition of protein synthesis.	Gentamicin, tobramycin, amikacin, streptomycin, kanmycin
ß-Lactam Antibiotics	Inhibition of cell wall synthesis	✚ Penicillins: Natural: penicillin G, penicillin V; Penicillinase-resistant penicillin: methacillin, oxacillin, nafcillin; Extended-spectrum penicillin: ampicillin, amoxicillin, carbenicillin ✚ Cephalosporins: cephalothin, cefamandole, cefataxime ✚ Carbapenems: primaxin ✚ Monobactams: aztreonam
Chloramphenicol	Inhibition of protein synthesis	
Fluoroquinolones	Inhibition of nucleic acid synthesis	Enrofloxacin, ciprofloxacin, Danofloxacin, Difloxacin, Ibafloxacin, Marbofloxacin, Pradofloxacin, Orbifloxacin
Glycopeptides	Inhibition of cell wall synthesis.	Vancomycin, teicoplanin, avoparcin
Lincosamides	Inhibition of protein synthesis	Lincomycin, Clindamycin, and Pirlimycin
Macrolides	Inhibition of protein synthesis.	Erythromycin, Tylosin, Spiramycin, Tilmicosin, Tulathromycin
Polymixins	Inhibition of cell membrane function	Polymixin B, colistin (Polymixin E)
Rifamycins	Inhibition of nucleic acid synthesis.	Rifampin, Rifabutin, Rifapentine
Streptogramins	Inhibition of protein synthesis.	Virginiamycin
Tetracyclines	Inhibition of protein synthesis.	Chlortetracycline, oxytetracycline, demethylchlortetracycline, rolitetracycline, limecycline,

		clomocycline, methacycline, doxycycline, minocycline
Diaminopyrimidines (Trimethoprim)	Inhibition of other metabolic processes.	Trimethoprim, Aditoprim, Baquiloprim, Ormetoprim

Indication and uses

A veterinarian can prescribe a broad-spectrum antibiotic to treat a wide range of infections. A narrow-spectrum antibiotic is only effective against a few types of bacteria.

Some antibiotics attack aerobic bacteria, while others work against anaerobic bacteria. Aerobic bacteria need oxygen and anaerobic bacteria do not.

In some cases, a healthcare professional may provide antibiotics to prevent rather than treat an infection, as might be the case before surgery. This is the 'prophylactic' use of antibiotics. Technician commonly uses these antibiotics before bowel and orthopedic surgery.

Resistance

The overuse and misuse of antibiotics in all settings allows the development of resistant bacteria. While all uses of antibiotics can exert selection pressure for resistant organisms. This means that the antibiotic becomes less effective against that type of bacterium, as the bacterium has been able to improve its defenses.

These bacteria can then reproduce and spread their antibiotic-resistant genes to other generations, increasing their prevalence and leading to infections that cannot be healed by antibiotics. This is a growing matter of concern as antibiotic resistance is considered to be a serious future threat to animal welfare.

Association

The reasons for antibiotic overuse are myriad, including administration of antibiotics for nonbacterial or noninfectious syndromes, treatment of conditions caused by colonizing or contaminating organisms, and durations of therapy that are longer than indicated. Unnecessary use of antibiotics is particularly concerning because antibiotics may be associated with a number of adverse drug events (ADEs), including allergic reactions, end-organ toxic effects, subsequent infection with antibiotic-resistant organisms, and Clostridium difficile infections (CDIs).

Estimates of the incidence of antibiotic-associated ADEs in animals are generally unavailable

B. Sulfonamides

Sulfonamides are bacteriostatic anti-infectious but bactericidal action is evident at a high concentration; they are most effective in the early stages of acute infections when organisms are rapidly multiplying; they inhibit both gram-positive and gram-negative bacteria.

Classification

Highly Soluble Sulfonamides Used for Urinary Tract Infections

A few very water-soluble sulfonamides, eg, sulfisoxazole (sulfafurazole) and sulfasomidine, are rapidly excreted via the urinary tract (>90% in 24 hr) mostly in an unchanged form; because of this, they are primarily used to treat urinary tract infections.

Poorly Soluble Sulfonamides Used for Intestinal Infections

Some sulfonamide derivatives, such as sulfaguanidine, are so insoluble that they are not absorbed from the GI tract (<5%). Phthalylsulfathiazole and succinylsulfathiazole undergo bacterial hydrolysis in the lower GI tract with the consequent release of active sulfathiazole. Salicylazosulfapyridine (sulfasalazine) is also hydrolyzed in the large intestine to sulfapyridine and 5-aminosalicylic acid, an anti-inflammatory agent that might be used for management of ulcerative colitis in dogs.

Potentiated Sulfonamides

A group of diaminopyrimidines (trimethoprim, methoprim, ormetoprim, aditoprim, pyrimethamine) inhibit dihydrofolate reductase in bacteria and protozoa far more efficiently than in mammalian cells. Used alone, these agents are not particularly effective against bacteria, and resistance develops rapidly. However, when combined with sulfonamides, a sequential blockade of microbial enzyme systems occurs with bactericidal consequences. Examples of such potentiated sulfonamide preparations include trimethoprim/sulfadiazine (co-trimazine), trimethoprim/sulfamethoxazole (co-trimoxazole), trimethoprim/sulfadoxine (co-trimoxine), and ormetoprim/sulfadimethoxine. Sulfonamides are used in combination with pyrimethamine to treat protozoal diseases such as leishmaniasis and toxoplasmosis.

Topical Sulfonamides

Several sulfonamides are used topically for specific purposes. Sulfacetamide is not highly efficacious but is occasionally used to treat ophthalmic infections.

Mafenide and silver sulfadiazine are used on burn wounds to prevent invasion by many gram-negative and gram-positive organisms. Sulfathiazole is commonly included in wound powders for the same purpose.

Indication and uses

Sulfonamides are broad-spectrum antimicrobials inhibiting both gram-positive and gram-negative bacteria, as well as some protozoa, such as coccidia. They are considered ineffective against most obligate anaerobic and should not be used to treat serious anaerobic infections. However, they may affect aerobic organisms that contribute to the lowered oxygen tension in the microenvironment and, as such, they may be useful in certain diseases involving *Fusobacteria.*

Resistance

Resistance of animal pathogens to sulfonamides is widespread as a result of more than 50 years of therapeutic use and this limits their effectiveness; however, sulfonamides are still widely used in combination with other medications, as in the case of the potentiated sulfonamides. They are also utilized in herd management of disease and some individual animal applications. Cross-resistance between sulfonamides is considered complete.

Association

Sulfonamides are the oldest and remain among the most widely used antibacterial agents in veterinary medicine, chiefly because of low cost and their relative efficacy in some common bacterial diseases. The synergistic action of sulfonamides with specific diaminopyrimidines renders these drugs much more effective than sulfonamides alone.

C. Anti-inflammatory

Anti-inflammatory drugs may be included in microbial infection when they are accompanied by inflammation process; frequently anti-inflammatory agents are also antipyretic.

Classification

Steroid anti inflammatory

Glucocorticoid are natural hormones normal produced by the adrenal cortex in response to release of adrenocorticotropic hormone (ACTH) by the anterior pituitary gland.

Examples: Methylprednisolone, Dexamethazone, Prednisolone/prednisolone.

Non-steroidal anti inflammatory

The nonsteroidal anti-inflammatory drugs (NSAIDs) discussed under two classes, namely, inhibitors of prostaglandin (PG) synthesis and miscellaneous (locally applied) anti-inflammatory drugs.

Aspirin (Acetylsalicylate): Aspirin is a prototypical NSAID that is effective and inexpensive. Therapeutic uses: In general, aspirin is useful as an analgesic and an NSAID in dogs and cats, particularly in the control of osteoarthritis. However, it is not effective in treating colic.

Acetaminophen: Acetaminophen (paracetamol) is a para-aminophenol derivative with analgesic and antipyretic effects similar to those of aspirin, but it has weaker anti-inflammatory effects than does aspirin and other NSAIDs.

Phenylbutazone: The safety and efficacy profile in addition to its affordability makes it is the most commonly used NSAID in the horse and others species. Therapeutic uses: It is used to treat various forms of lameness as well as osteoarthritis and other painful conditions of the limbs including soft tissue or nonarticular rheumatism.

Ibuprofen and indomethacin: These two drugs are not routinely used in veterinarymedicine due to its low safety profile.

Diclofenac: Diclofenac is a nonsteroidal anti-inflammatory drug (NSAID) taken or applied to reduce inflammation and as an analgesic reducing pain in certain conditions. It is supplied as or contained in medications under a variety of trade names. Diclofenac Sodium Injection for veterinary use is for the treatment of various bacterial infections in sheep, swine, cattle, goats, and calves.

Novalgin: Dipyrone (Novalgin) is a readily available, relatively cheap and highly efficacious analgesic that is widely prescribed in Africa, Europe and South America. It is indicated in different situations: Catarrhal-spasmatic colic, meteorism and intestinal constipation in horses; spasms of the uterine cervix during birth; pains of urinary and biliary origin, esophageal obstruction; joint and muscular rheumatism; for preparation of surgical and obstetrical interventions.

Paracetamol: Paracetamol (synonym: acetaminophen) is an antipyretic and analgesic substance of thepara-aminophenol group of the non-steroidal anti-inflammatory drugs. It is used in pigs as a 10% medicated premix at a dosage rate of 15 to 30 mg/kg bw/day for 3 to 5 consecutive days in the treatment of painful disease states associated with pyrexia.

Indication and uses

NSAIDs are usually used for the treatment of acute or chronic conditions where pain and inflammation are present.

Aspirin, the only NSAID able to irreversibly inhibit COX-1, is also indicated for antithrombosis through inhibition of platelet aggregation. This is useful for the management of arterial thrombosis and prevention of adverse cardiovascular events like heart attacks. Aspirin inhibits platelet aggregation by inhibiting the action of thromboxane.

NSAIDs are useful in the management of post-operative dental pain following invasive dental procedures such as dental extraction. When not contra-indicated they are favoured over the use of paracetamol alone due to the anti-inflammatory effect they provide.

Resistance

It has been hypothesized that NSAIDs may delay healing from bone and soft-tissue injuries by inhibiting inflammation. On the other hand, it has also been hypothesized that NSAIDs might speed recovery from soft tissue injuries by preventing inflammatory processes from damaging adjacent, non-injured muscles.

There is moderate evidence that they delay bone healing. Their overall effect on soft-tissue healing is unclear

Association

When used in combination with paracetamol the analgesic effect has been proven to be improved. There is weak evidence suggesting that taking pre-operative analgesia can reduce the length of post-operative pain associated with placing orthodontic spacers under local anaesthetic.

Combination of NSAIDs with pregabalin as preemptive analgesia has shown promising results for decreasing post-operative pain intensity.

Note: Antifungal may be used topically (eg: Niconazole, Enilconazole, Nystatin) for cutaneous or mucosal fungal diseases while others may be used systemically (Itraconazole, Fluconazole, Ketoconazole) for systemic fungal diseases like aspergillosis. Systemic azole should not be given to pregnant animals because they are teratogenic. Griseofulvin is used for many years, but now days it is less used due to its teratogenicity action.

- Topic 2: Select medicines

The decision to use antimicrobial drugs involves the determination of whether there is an infectious agent present. Central to the decision to use antimicrobial drug is the demonstration that an infectious agent is the part of the disease process under consideration.

A. Factors of medicines selection

An appropriate selection of antimicrobial agents is very important and among the factors to be considered are the followings:

- microorganism ,results of sensitivity test,
- pathogenicity of organism, pathologic lesions, acuteness of infection,
- pharmacokinetics of the drug indicated, expense, potential drug toxicity,
- Organic dysfunction (especially kidney and liver function) and possible interaction with the drug administered concurrently.

B. Criteria of selection

Animal species: This must be taken into account because some medicines are more indicated in some species while prohibited for others;

Age: young animals are not in the same physiological status as adult ones, so age should be considered when selecting a medicine

Physiological status: some medicine cannot be administered in pregnant animals because their abortive effect.

Weight: the can influence the selection of medicine in the fact that dose calculations of a medicine to be administered; will be based of weight.

Symptoms and disease: the symptoms and disease will give a veterinarian an idea on which medicine to select.

- Topic 3: Prescription of medicines

Prescription is an order given by a physician for the preparation and administration of a medicine. The written directive is given to owner of a patient and he must submit it to a pharmacist, the later must give the deliver the medicine according the given instructions.

The main parts of a prescription are: inscription, subscription and instructions

Inscription: is the main part of a prescription containing the names and amounts of the drugs ordered;

Subscription: directions for mixing the ingredients and designation of the form (powder, solution etc) in which the drug is to be made;

Instructions: directions to the patient uses (owner of patient) regarding the dose and times of drug distribution.

L O 3.3 – Administer prescribed medicine

The good choice of route of administration is very important, frequently manufacturers indicate the appropriate route of administration; and the follow up is compulsory.

- Topic 1: Administration routes

The frequent routes of administration of anti-infectious are: Topic, Oral, Parenteral and inhalation routes

A. Oral administration

There are large numbers of pharmaceutical preparations available for oral administration. Solid dosage forms (powders, tablet, capsules, pills, etc.) and liquid dosage forms (syrups, emulsion, mixture, drench, electrolytes, etc.)

B. Parenteral administration (IV, IM, SC, Id, epidural, subconjunctival)

It refers to a drug administration by injection directly in to the tissue fluid or blood without having to cross the intestinal mucosa.

- **Intravenous route (IV):** Gives swift, effective and highly predictable blood concentration and allows rapid modification of dose and is used for emergency treatment. In most animals (horse, cattle, sheep and goat) usually given through jugular vein, in pig-ear veins, in the dog and cat-cephalic vein and recurrent tarsal vein.
- **Intramuscular (IM) route:** Absorption occurs either haematogenous or via lymphatic and is usually fairly rapid except for long acting preparation.
- **Subcutaneous (SC) route**: Preferred when slow and continuous absorption of drug is required. The injected drug disperses through the loose connective tissues. They dissolve in tissue fluid before it can enter either capillaries or lymphatic. Intradermal route (ID): Used for testing hypersensitivity test and for vaccination.
- **Epidural route**: Refers to deposition of drug up on or outside the dura matter. E.g. Introduction of local anesthetics between the first and second coccygeal vertebra to eliminate straining.
- **Subconjunctival:** Disposition of a pharmaceutical preparation beneath the conjunctiva.

C. Topical or local application

It refers to external application of drug to the body surface for localized action at accessible site, such as skin, eyes, body orifices, body cavity.

D. Inhalation

It refers to the drugs that have aerosol form are administered using respiratory tract. Drug administration by inhalation through the mouth must be atomized into small droplets than those used in nasal route, so that the drug should pass through windpipe (trachea) and into the lungs.

- Topic 2: Administration techniques

Different techniques in drug administration can be applied; some of them are the following:

- **Oral medication:** Tablets are the most commonly used oral form and Liquid preparations for oral administration may be purchased in several different forms (e.g., mixtures, emulsions, syrups, or elixirs).
- **Mixing the medicine with favourite feeds:** powder forms are can be mixed in foods
- **Mixing the medicine with water:** Mixtures consist of aqueous solutions (i.e., water) and suspensions for oral administration. Note that powder forms (water soluble) are also mixed in water.

- **Different injection where needle of injection and syringes are required:** Two forms of parenteral injection that are available are injections and implants. Injections are available as single-dose vials, multidose vials, ampules, or large-volume bottles that may be used to administer intravenous (IV) infusions

- Topic 3: Dosage

Dosage and other important information are given by manufacturer on notice. Because of the variety of dosage forms in veterinary medicine and the diversity of animal and bird species treated, drug or dosage delivery sometimes requires the development of specific devices to ensure fast, safe, effective and low cost efficient treatment.

Formulation of Drug Dosage Forms of Animals; are listed as following:

- Oral dosage forms: Refers to administration of drug through the mouth. The most commonly used preparations are solid oral dosage forms such as tablets, capsules, granules, powder, paste and boluses.
- Parenteral dosage forms: the most common parenteral dosage forms are stable aqueous solutions and subcutaneous implants.
- External dosage forms: Ointment- semisolid preparation for external application; Cream- a viscous semisolid, consisting of oil in water emulsion or water in oil emulsion; Dusting powder e.g., popular antibacterial agent applied on animal wounds; Lotion- an aqueous solution or suspension for local application. Spray-a drug applied in liquid form by pressure.
- Inhalation dosage forms: gaseous and volatile liquid anaesthetic agent (drugs), given by inhalation, e.g., Halothane

L O 3.4 – Follow up patient

- Topic: 1: Regular monitoring

A. Secondary effects

In medicine, a side effect is an effect, whether therapeutic or adverse, that is secondary to the one intended; although the term is predominantly employed to describe adverse effects, it can also apply to beneficial, but unintended, consequences of the use of a drug.

As the drugs are poisons, they are able to produce several undesirable effects called second effects or side effects (eg: allergy, anaphylactic shock etc).

B. Complications

Complications can happen in some cases; thus a regular monitoring should be compulsory and management is applied if necessary. A complication in medicine, or medical complication, is an unfavourable result of a disease, health condition, or treatment.

Complications may adversely affect the prognosis, or outcome, of a disease. Complications generally involve a worsening in severity of disease or the development of new signs, symptoms, or pathological changes which may become widespread throughout the body and affect other organ systems. Thus, complications may lead to the development of new diseases resulting from a previously existing disease. Complications may also arise as a result of various treatments.

C. Recovering process

The follow up is obliged until the animal is recovered otherwise other decision may be taken accordingly.

Complications That May Arise During Recovery

Excitement

Excitement during recovery is not common but may be seen in animals that were not adequately sedated prior to induction and in the sled dog breeds, sight-hounds and Dobermans. Not only can the animal injure itself as it thrashes around in its cage but the noise can agitate other patients and it can be very distracting for staff. Excitement may also be seen in painful animals so it is important to ensure that the animal is pain free before considering giving a sedative.

Pain

It is to be expected that pain will result following a surgical procedure. Analgesia should be provided before the patient becomes aware of the pain if possible. This makes the pain easier to control and reduces the chance of an excited recovery. How do you recognise pain in the recovering animal?; Agitation, vocalisation, reluctance to move, aggression are all signs that may reflect pain.

There are many factors that should be checked in the recovering animals to ensure that the animal comes around from anaesthesia with minimal chance for complications. Good observational skills play a large part in ensuring that recovery is smooth and free from problems.

- Topic 2: Post treatment check up

After the treatment has finished, you will have regular checkups. These will allow the health care technician to monitor the animal patient health and wellbeing

The care plans usually are:

- Provide a summary of the treatment set out a clear schedule for follow-up care,
- details for the health professionals involved in the treatment and any screening tests
- list any symptoms to watch out for, complications and possible long-term side effects
- identify any medical or psychosocial problems that may develop after treatment and ways to manage them
- Suggest ways to adopt a healthier lifestyle after treatment.

What do post treatment check-ups involve

During check-ups veterinarian may:

- assess animal patient recovery
- monitor and treat any ongoing side effects
- talk to the owner about any late treatment side effects to watch out for
- check any new symptoms
- do a physical examination
- ask if the owner have any concerns or questions
- discuss the general health and give healthy lifestyle advice

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Appendices

Appendix 1: Average resting temperatures.

Species	Normal temperatures	Critical points
Cattle	38.5^0C(101.5^0F)	39.5^0C(103.0^0F)
Sheep	39.0^0C(102^0F)	40^0C(104^0F)
Goat	39.5^0C(103.0^0F)	40.5^0C(105^0F)
Pig	39^0C(102^0F)	40.0(104^0F)
Horse	38^0C(100.5)	39^0C(102.0^0F)

Appendix 2: Resting pulse rates

Species	Pulse rates
Adult cattle	60-80/minute
Young calves	100-120/minute
Sheep and goat	70-90/minute
Adult horses	30-40/minute
Foals up to 1 year	70-80/minute

Appendix 3: Respiratory rates

Species	Respiratory rates
Cattle	10-30/minute
Sheep and pig	10-20/minute
Goat	25-35/minute
Horse	8-16/minute

Appendix 4: The normal colour of mucous membrane of different animals

Animal	Colour of mucous membrane
Cattle, sheet and goat	Pale pink
Horse	Pale roseate
Pig	Reddish
Dog	Pale roseate
Cat	Pale pink