

**WOLLO UNIVERSITY
SCHOOL OF VETERINARY MEDICINE**

**VETERINARY GENERAL MEDICINE
(VetM3132)**

(For 3rd Year DVM students)

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Course Outline

- **Course Code: Vetm3132**
- **Lecture : 4 credit hours, Has no practical class**
- **The course includes: -**

1. General systemic states

- 1.1. Toxemia**
- 1.2. Septicemia, bacterimia and viremia**
- 1.3. Postpartum septic metritis**
- 1.4. Localized infections**
- 1.5. Dehydration, electrolyte and acid - base imbalance**
- 1.6. Hypothermia, hyperthermia and fever**
- 1.7. Allergy and anaphylaxis**

2. Practical antimicrobial therapeutics

- 2.1. Antimicrobial drugs and principle of treatment**

3. Disease of the newborn

- 3.1. Neonatal infections**
- 3.2. Omphalitis, omphalophlebits and omphaloarteritis**
- 3.3. Principle of prevention and control of neonatal infections in farm animals**

4. Diseases of digestive system

4.1. Diseases of oral cavity and esophagus

- Stomatitis
- Pharyngeal disorders (pharyngitis and paralysis of pharynx)
- Esophageal disorders(esophagitis, spasm of esophagus, paralysis of esophagus, stenosis of esophagus, esophageal dilation).

4.2. Diseases of the fore stomachs(proventriculus) of ruminant animals

- Acute and chronic hypotonia and atonia of the fore stomachs in ruminant animals(rumen, reticulum and omasum)
- Simple indigestion
- Ruminant acidosis and alkalosis

Course cont'd---

- Carbohydrate engorgement of rumen
- Traumatic reticuloperitonitis/pericarditis

4.3. Diseases of the true stomach of ruminants(Abomasum)

- Abomasitis
- Impaction of abomasum

4.4. Diseases of stomach in monogastric animals

- Acute and chronic gastritis

4.5. Diseases of intestine

- Acute and chronic enteritis

4.6. Diseases of stomach and intestine

- Acute and chronic gastroenteritis

4.7. Diseases of stomach and intestine with colic in equines

- Acute and chronic dilation of stomach
- Meteorism of intestine
- Spasm(enteralgia) of intestine

Course cont'd---

- Chemostasis and caprostasis of intestine
- Internal intestinal obstruction
- Strangulation of intestine
- Torsion of intestine
- Invagination of intestine
- Thrombo - embolic colic
- General principles of treatment of GIT diseases

5. Diseases of hepatobiliary system

5.1. Diseases of liver

- Hepatitis
- Toxic dystrophy of liver
- Cirrhosis

5.2. Diseases of gall bladder and bile duct

- Cholelithiasis

- Cholangitis and cholecystitis

5.3. Symptom complex associated with the diseases of hepatobiliary system

- Icterus(jaundice)
- Cholemia
- Portal hypertension

6. Diseases of peritoneum

- Peritonitis
- Ascites

7. Diseases of respiratory system and pleura

7.1. Diseases of the upper respiratory tract and sinuses

- Rhinitis
- Frontitis and highmoritis
- Laryngitis

Course cont'd---

- Tracheitis

7.2. Diseases of lower respiratory tract

- Bronchitis
- Hyperemia and edema of lung
- Pneumonia
- Emphysema
- Bronchopneumonia

7.3. Diseases of thoracic cavity

- Pleuritis
- Pneumothorax
- Hydrothorax

7.4. Prevention measures of respiratory diseases

8. Disease of cardiovascular system

8.1 Cardiac diseases

8.1.1. Diseases of pericardium

- └ Pericarditis
- └ Hydro pericardium

8.1.2. Diseases of myocardium

- └ Myocarditis
- └ Myocardosis
- └ Myocardiofibrosis

8.1.3. Diseases of endocardium

- └ Endocarditis
- └ Stenosis and defect of valves

8.2. Vascular diseases

- └ Arteriosclerosis
- └ Vascular thrombosis

8.3. Prevention measures of cardiovascular diseases

9. Disease of urinary system

9.1. Disease of kidney

- └ Acute and chronic nephritis
- └ Pyelonephritis
- └ Nephrosis
- └ Nephrosclerosis

9.2. Diseases of the bladder, ureters and urethra

- └ Pyelitis
- └ Urolithiasis
- └ Cystitis
- └ Paralysis of bladder
- └ Spastic paralysis of sphincter of bladder

9.3. Chronic haematuria in cattle

10. Disease of blood and blood forming

organs/Disease of

hematopoietic and hemolymphatic system)

- └ Anaemia
- └ Leukemia
- └ Leucopenia
- └ Haemophilia

11. Disease of nervous system

11.1. Disease of brain and its membrane

- └ Anaemia of brain
- └ Hyperemia of brain and its membrane
- └ Brain abscess
- └ Neoplasm of brain
- └ Encephalitis
- └ Meningitis
- └ Encephalomalacia

Course cont'd---

- Hydrocephalus

11.2. Diseases of spinal chord and its membrane

- Spinal meningitis
- Myelitis

11.3. Diseases of nervous system with functional disturbance

- Epilepsy
- Tetani
- Neurosis

12. Diseases of the musculoskeletal system

12.1. Diseases of muscles

- Myopathy

12.2. Diseases of bones and joints

- Osteodystrophy
- Osteomyelitis
- Arthritis and synovitis

13. Diseases of skin

13.1. Diseases of hair, epidermis and dermis

Course cont'd---

- └ Pitriasis
- └ Parakeratosis
- └ Hyperkeratosis
- └ Impetigo
- └ Urticaria
- └ Eczema
- └ Dermatitis
- └ Photosensitization
- └ Alopecia

13.2. Diseases of subcutaneous

- └ Oedema
- └ Emphysema
- └ Lymphangitis
- └ Haemorrhage
- └ Gangrene

13.3. Cutaneous neoplasmas

- └ Papilloma, Sarcoid, Carcinoma, Melanoma

Part 1. General systemic states

1.1. Toxemia

- Toxemia is a generic term for the **presence of toxins in the blood**.
- It is a **generalized disease state** caused by the **presence of toxins** derived from **bacteria** or **body cells**.
 - NB. But this does not include diseases caused by **plant toxins** (phytotoxins), **insect toxins**, or any other **organic** and **inorganic toxins**.
- Demonstration of the presence of toxins in the blood stream in every case of toxemia is difficult.
- However, for every diagnosis and treatment, observation of the major clinical features of toxemia is very important.

Etiology

- Toxemia can be caused by different kinds of toxins.
- These toxins can be classified as follows :-
 1. Antigenic (bacterial) toxins

Toxemia cont'd---

1. Antigenic(bacterial) toxins

➤ These are toxins produced by **bacteria** and a **few fungi** and to a lesser extent by some **helminthes**.

➤ Antigenic toxins can be

- | 1. Exotoxin
- | 2. Endotoxin
- | 3. Enterotoxins

1. Exotoxins

➤ They are protein in nature and are produced by bacteria during their multiplication and have the ability to diffuse into the surrounding.

➤ The most important bacterial exotoxins are those which are produced by *Clostridium* species.

➤ Toxemia due to the exotoxins of *Clostridium* species can be

➤ 1. By ingestion of already performed toxin. Example

Toxemia cont'd---

- 2. By absorption of already performed toxins from deep necrotic wounds. Example *C. tetani*
- 3. Others may be produced by heavy growth of bacteria in the intestine. Example enterotoxaemic clostridia(*Cl. perfringens*(A -E)).
- 4. Still others may be produced in the tissues (e.g. *Cl. chauvoei* of black leg and *Cl. novyi* type B and *Cl. novyi* type D in case of black disease in sheep and haemoglobinuria in cattle respectively).

2. Endotoxin

- Endotoxin is naturally lipopolysaccharides and found within the bacterial cells as part of the cell wall.
- They are released into the circulation when the bacterial cell wall breakdown(bacterial lysis).
- Small amounts of endotoxin which is absorbed into

Toxemia cont'd---

- But in cases of liver damage or if the amount of toxin absorbed is large, a state of **endotoxemia** is produced.
- The other reason is that when the intestinal wall is damaged as in the case of enteritis or in the case of acute intestinal obstruction, these endotoxin is absorbed into the body fluid and may cause **systemic intoxication**.
- Endotoxin may also be absorbed from another sites other than intestine like from udder in the case of mastitis, from peritoneum in the case of peritonitis, from abscesses and from other septic foci of different organs.
- The common bacterial species that are endotoxin producers include those of *Escherichia coli*, *Salmonella* species etc.
- Endotoxin gains access to the blood when there is severe localized infection, such as a coli form mastitis in

Toxemia cont'd---

➤ The most common causes of endotoxemia in horses are associated with diseases of the gastrointestinal tracts including: -

Colitis

Strangulation

Obstruction and ileus(atonia of GIT)

- Complications associated with foaling and grain overload.

3. Enterotoxins

- These are exotoxins that exerts their effect principally on the mucous of intestine.

- They cause disturbance of fluid and electrolyte balance.

- The most typical example of enterotoxin producer bacteria is the enterotoxigenic strain of *E. coli* which causes a hypersecretory diarrhoea in neonatal farm

The difference between **exotoxin** and **endotoxin**

Property	Endotoxin	Exotoxin
Produced by	Gram-ve bacteria mainly	Gm +ve bacteria mainly
Diffusability	No	Yes
Heat stability	Thermo - stable at 100 °C for 1 hour	Thermo- labile, destroyed at 60 °C
Nature	Lipopolysaccharides	Protein
Antigencity	Weak	Strong
Toxicity	Weak	Strong
Converted to toxoid by formalin	No	Yes
Action	Non-specific (no specific receptors)	Specific (binds to specific receptors)

2. Metabolic toxins

- are those toxins that are produced as a result the **disorder** of **protein, carbohydrate** and **fat metabolism** and / or dysfunction of liver due to **liver diseases**.
- Because many liver diseases may **disturb the normal detoxification functions** including **oxidation, reduction, acetylation** and **conjugation** with such substances as glycine, glucuronic acid, sulphuric acid and cysteine etc.
- The above toxic substances may accumulate in large amount to cause injury to tissues and illness in the organisms.
- However, metabolic toxins are produced by each of our cells as they go about their everyday metabolic processes.
- Normally toxic metabolites produced in the alimentary tract or tissues are excreted in the urine and feces, sweat and together with exhalation gases or detoxified in the liver.

Toxemia cont'd---

➤ Sometimes intoxication due to metabolic toxins is called **autointoxication**.

➤ Metabolic toxins includes

 | Phenols

 | Cresols

 | Amines

 | Ketone

 | Lactic acidemia etc

➤ These toxins may accumulate as a result of

➤ 1. Incomplete elimination of toxic materials normally produced by body metabolism.

➤ 2. Their higher production in the body of organism due to abnormal metabolic process in the cells.

➤ 3. Obstruction of the lower alimentary tract(large intestine) may increase the absorption of phenols, cresols and amines which are

Toxemia cont'd---

normally excreted with feces in ruminant animals.

NB. In mono gastric animals the above metabolic products are not absorbed through the mucous of large intestine. But if there is regurgitation of fecal masses to small intestine, there will be

autointoxication by the above metabolic toxins.

➤ The production of toxins by abnormal metabolism includes the production of histamine and histamine like substances which are mediators of inflammatory process and causes tissue damages. .

➤ The most common toxemia in animals which are caused by abnormal metabolic process are

- | 1. Ketonaemia
- | 2. Lactic - acidaemia

1. Ketonaemia

➤ It is metabolic toxemia caused by disproportionate

Toxemia cont'd---

hypoglycemia mostly in large and small ruminants and pigs.

2. Lactic - acidaemia

➤ It is metabolic toxemia caused by acute ruminal impaction of rumen by easily digestible carbohydrate rich animal feeds.

Pathogenesis

➤ Pathogenesis of bacterial and metabolic toxins is different.

➤ Bacterial toxins are specific toxins and are extracted from them and their mode of action is well studied.

➤ 1. For example the endo toxin of *E. coli* causes

➤ Shock by producing biochemical mediators like cytokines, TNF - α , prostaglandins and vasoactive amines.

➤ Endotoxin increases heart rate and cardiac out put in the early stage endotoxemia.

➤ This stage of endotoxemia is known as **hyper**

Toxemia cont'd---

- As the result the mucous membranes are **red** and **congested**.
- Shunting of blood continues in organs such as the GIT tract and kidney.
- As the result there is ileus and reduction of urine production in intestine and kidneys respectively.
- However uncontrolled endotoxemia leads the hyper **dynamic stage of endotoxemia** to change to **hypo dynamic stage of endotoxemia**.

During hypo dynamic stage of endotoxemia:-

- Cardiac out put decreased results in systemic hypotension
- Central venous out put decreased
- Hypothermia and rapid irregular pulses
- Prolonged capillary refill time
- As the result the visible mucous membrane becomes pale to cyanotic.
- Acidaemia and hypoxemia provide clinical evidence of the advanced stage of endotoxemia.

Toxemia cont'd---

occurs.

- Endotoxemia causes an acute and severe neutropenia, which precedes neutrophilia and haemoconcentration.
- Neutropenia is due to margination and sequestration of leukocytes while hemoconcentration is due to movement of fluid from vascular to vascular space.
- The action of endotoxemia on thermoregulation is that they activate in production biological active molecules in the family of poly peptides.
- These poly peptides are **interleukin - 1 and interleukin - 6**.
- They are the key mediators of various infectious, inflammatory and immunological challenges.
- Both these mediate cause hyperthermia followed by hypothermia.
- Endotoxemia causes ileus in GIT tracts of animals.

Effect of endotoxemia in different systems of organism

1. Carbohydrate metabolism

2. Protein metabolism

- There is an increase in tissue protein breakdown and rise in blood Non-Protein Nitrogen (NPN) compounds.
- However, in spite of this tissue breakdown, there is an increase in total serum protein level as a result of stimulation of antibody production.

3. Mineral metabolism

- Hypoferremia (reduced iron levels) and hypozincemia (reduced Zinc levels) are observed.

4. Combined effects of endotoxemia on body system

- There is dilatation and sometimes damage to capillaries so that effective circulating blood volume is decreased.
- Weakness of the myocardium can also occur.
- The decrease in circulating blood volume in combination with diminished cardiac output leads to fall in blood pressure and the development of circulatory failure.

A decrease in liver function and damage to renal

Toxemia cont'd---

- As the result appetite falls.

5. Hypersensitivity

- It is a secondary effect produced by some toxins which is creation of a state of hypersensitivity at the first infection.
- So that a second infection, or administration of the same antigen causes **anaphylaxis** or an **allergic phenomenon** like that of **purpura hemorrhagica**.

6. Abortion

- Endotoxemia may cause abortion very often in pregnant cows because it activates in production of prostaglandin -F2 α which has lytic effect on corpus leutum of ovary.

Clinical findings of toxemia

- The clinical feature of toxemia in most non-specific toxemias is approximately the same.
- It differs with the amount of toxin produced, type of toxin, the speed and severity of the toxin.
- The clinical findings depends on the form of toxemia

Toxemia cont'd---

- 1. Acute toxemia
- 2. Endotoxemia with out bacterimia and septicemia
- 3. Endotoxemia with bacterimia and septicemia
- 4. Chronic toxemia
- 5. Toxemia due to localized infection

➤ **In acute toxemia** the clinical findings are hyperthermia at the early stage of but later the temperature may be normal or sub normal.

➤ The common syndromes of in this form of toxemia are depression, anorexia and muscular weakness.

➤ **In endotoxemia with out bacterimia and septicemia** toxin formation or liberation in to circulation is rapid and the toxicity of the toxin is high enough and cause toxic shock-syndrome which results cardiovascular collapse.

➤ The remarkable clinical findings during this form of endotoxemia are: -

- Severe peripheral vasodilatation with consequent fall in blood pressure
- Pallor of mucosa
- Hypothermia
- Tachycardia

Toxemia cont'd---

- }\ Muscle weakness

- Very often **endotoxemia may associated with bacterimia or septicemia** due to infection with Gram negative bacteria, especially *E. coli*.

- This cause to develop severe endotoxemia with clinical signs of :-

- }\ Depression

- }\ Hyperthermia followed by hypothermia

- }\ Tachycardia followed by decreased cardiac out put

- }\ Decreased systemic blood pressure

- }\ Cool skin and extremities

- }\ Diarrhea

- }\ Congested mucosa with an increased capillary refill time

- }\ Muscular weakness leading to recumbency.

- }\ In this form of endotoxemia renal failure is common which is characterized by **anuria**.

- **In chronic toxemia form** the major clinical forms are inappetance, separation from the herd, lethargy, slow

Toxemia cont'd---

➤ **In endotoxemia associated with localized infection**, in addition of the general clinical signs of toxemia there is clinical effects of the space occupation by the lesion.

Clinical pathology of endotoxemia

Hematology

- There is a change in total and differential leukocyte numbers.
- This change varies depending of the severity of endotoxemia.
- In mild form of toxemia there is leukocytosis and neutrophilia.
- But in sever form endotoxemia neutropenia and lymphopenia are the usual hematological findings.

Serum biochemistry

- Low concentration of glucose in plasma.
- High concentration of non protein nitrogen(NPN) in

Toxemia cont'd---

concentration in blood because of there is an increased capillary permeability.

➤ But the concentration of non – protein nitrogen(NPN) usually increased because there is a decreased glomerular filtration rate.

➤ The presence of NPN in blood is called azotemia.

Necropsy findings

➤ Gross findings at necropsy are limited to those of the lesion which produces the toxin.

➤ For example in case of toxemia caused by absorption of toxins from disease conditions like mastitis, peritonitis, localized abscesses or enterotoxaemia, gross pathological lesions can be observed in these areas.

➤ But microscopically, there is degeneration of the parenchyma of the liver, the glomeruli and tubules of the kidney.

Toxemia cont'd---

Diagnosis

- Theoretically the diagnosis of toxemia should depend on the isolation of the toxin causing toxemia.
- But practically diagnosis is based on observation of the common clinical signs for early treatment.

Treatment of endotoxemia

- └ The principles of treatment of toxemia should be directed towards
 - └ Removal of the foci of infection
 - └ Administration of antimicrobial agents
 - └ Aggressive fluid and electrolyte therapy to combat the relative hypo volemia, hypoglycemia and electrolyte and acid - base disturbance
 - └ Using of anti inflammatory preparates like that of glucocorticoides

Toxemia cont'd---

- Supportive treatment should also be given to counteract the effects of toxemia.
- So that intensive fluid and electrolyte therapy by intravenous infusion is essential until the animal starts to eat and drink.
- Administration of glucose, amino acids and vitamins particularly B-complex group may be important.
- The use of glucocorticoides is usually included in the treatment of toxemia especially when there is septic shock. E.g. **dexametazone**.
- The other most important part of treatment of toxemia is administration of broad-spectrum antibiotics. E.g. ampicillin, chloramphenicol, sulphamethazine, tetracyclines and other drugs as well.

1.2. Septicemia, bacterimia and viremia

- Septicemia is the acute invasion of the systemic

Toxemia cont'd---

possible localization in various body systems or organs if the animal survives.

- Septicemia is the common cause of morbidity and mortality in new born farm animals which have not received a sufficient quantity of **colostrum** in the first 12 - 24 hours after birth.
- Bacterimia is different from septicemia in that bacterimia is not accompanied by sepsis or septic shock and the bacteria is present in the blood stream for only transitory period and do not reproduce in it and do not produce clinical signs.
- Viremia is the invasion of the systemic circulation by pathogenic viruses with possible localization in various body tissues and in which the lesions produced are characteristic of the specific virus.

Etiology

- Many different infectious agents can result in septicemia or viremia.

Diseases of animals that cause septicemia	Species and age of animals
Anthrax, pasteurellosis and salmonellosis	All species and all age groups of animals
Most gram negative bacteria	Neonatal
<i>E. coli</i>, <i>Salmonella</i> species and rarely Gram positive bacteria and mixed infection	Calves
<i>E. coli</i> and <i>Streptococcus suis</i> type 1 which is associated with localization in joints, endocardium and meninges	Pig lets
Septicemia with localization infection and caused by <i>E. coli</i>, <i>Actinobacillus equi</i>, <i>Klebsiella pneumoniae</i>, α - haemolytic <i>Streptococcus</i> and <i>Salmonella</i> species	Foals

<i>Pasteurella multocida, Mannheimia haemolytica, Yersinia pseudotuberculosis.</i> Acute and chronic bovine virus diarrhoea(BVD) and bovine malignant catarrh fever.	Lambs
<i>Histophilus somni</i> (formerly <i>Haemophilus somnus</i>)	Sheep
Hog cholera, African swine fever virus and <i>Erysipelothrix rhusiopathia</i>	Pigs
African horse sickness virus(AHSV) and <i>M. haemolytica</i>	Horse , donkey and mules

Epidemiology

- Epidemiology of systemic infections caused by bacteria, viruses, Rickettsiales, fungi and other pathogens occur in animals of all ages and under many circumstances.
- The risk factors for each infectious diseases are categorized according to:-
 - Animal risk factors
 - Environmental risk factors
 - Pathogen risk factors

Septicemia cont'd---

Pathogenesis

- The exotoxins or endotoxin produced by the infectious agents(bacteria) initiate a profound toxemia and high fever.
- Localization of certain pathogens occurs in many organs and may produce severe lesions in animals which survive the toxemia.
- Direct endothelial damage and hemorrhages may also be caused.
- The same general principles apply to viremia, except that toxins are not produced by viruses.
- It is more likely that the clinical manifestations are the result of direct injury of the cells invaded by the virus.
- Transplacental infection can occur resulting in fetal mummification, abortion, or infection of the fetus which may be carried to term.

Septicemia cont'd---

Neonatal septicemia

➤ Neonatal septicemia is common in all farm animal species from few hours up to several days of age.

➤ The following features are common in neonatal septicemia:-

- ↓ Recumbency

- ↓ Depression

- ↓ Absence or marked depression of the suck reflex

- ↓ Dehydration

- ↓ Fever

- ↓ Diarrhea

- ↓ Congested mucus membranes

- ↓ Weakness and rapid death.

Septicemia cont'd---

- Septic polyarthrititis is common and is characterized by heat, pain, synovial distention and lameness.
- Pneumonia is often observed and is characterized by dyspnea and abnormal lung sounds.

Clinical pathology

Blood culture

- It is used for isolation of the causative bacteria/ virus from the blood stream.
- Isolation should be attempted by culture or animal(cell) inoculation.

Procedure

- Collect 10 - 30 ml of blood aseptically from the major vein or artery vessels before **the onset of fever** for culturing in artificial media.
- Or at the **highest stage** of fever for animal inoculation.
- Inoculate the blood sample into culture broth in ratio

Septicemia cont'd---

- Incubate the inoculated broth and examined daily.
- Growth of bacteria is manifested by the change in turbidity and/ or haemolysis.

Leukogram(Haemogram)

- The presence of leukocytosis or leukopenia is an aid in diagnosis of septicemia.
- The type and degree of leukocytosis or leukopenia has prognostic significance in diagnosis of septicemia and viremia.

Immunoglobulin status

- Low levels of serum protein and Immunoglobulins are associated with failure of passive transfer of colostral.
- Immunoglobulins in newborn farm animals with subsequent septicemia due, most commonly, to Gram-negative bacteria.

Serology

- There are several serological tests that are important to diagnose many infectious diseases that causes

Septicemia cont'd---

Necropsy findings

- The lesions will reflect the specific disease causing the septicemia.
- Sub-serous and sub-mucosal hemorrhages may be present, together with **embolic foci** of infection in various organs accompanied by the lesions typical of the specific pathogen.

Diagnosis

- Diagnosis of septicemia can be tentative and confirmatory
- Tentative diagnosis depends on
 - └ Observation of major clinical signs
 - └ Presence of petechiae and occasionally ecchymotic haemorrhages in individual organs.
- But the confirmatory diagnosis is based on isolation and identification of the pathogen from blood.

Treatment

Septicemia cont'd---

- Treatment during septicemia must be focused on using broad spectrum antibiotics and giving of supporting treatments.
- But if there is failure of transmission of passive(colostral immunity) in neonates it is possible to provide them immunoglobulin by injection of plasma from the mother.

1.3. Postpartum septic metritis in cattle

- The postpartum period is the time starting from parturition to the time of complete uterine involution (return of the uterus to its normal size and function).
- Postpartum septic metritis occurs primarily in dairy cows within a few days(2 - 10 days) after parturition.

It is characterized clinically by: -

- └ Severe toxemia
- └ Copious foul smelling uterine discharge with or

Septicemia cont'd---

Etiology

- The cause of postpartum metritis is multifactorial.
- It is usually assumed that the cause is a combination of: -
 - ↳ Impaired neutrophil function, often with retained fetal membranes (RFM)
 - ↳ Infection of uterus(endometritis, perimetritis and metritis).
- A mixed bacterial flora is common in septic metritis which includes:
 - 1. *Actinomyces pyogenes* , *Corynebacterium pyogenes*, *Bacteriodes* species and *Fusobacterium necrophorum* are commonly predominate as mixed flora in cows with retained placenta and post partum metritis particularly after 5 – 7 days of post partum.
 - 2. *E. coli* is commonly predominate in cows with

Septicemia cont'd---

placenta and post partum metritis includes:-

- \ *Staphylococcus* species
- \ *Streptococcus* species
- \ *Pseudomonas aeruginosa*
- \ *Proteus*
- \ Occasionally *Clostridium* species are also present.

NB. The presence of *Cl. tetani* may result in tetanus if the condition helps to proliferate.

Epidemiology

- Postpartum septic metritis (PPSM) is common in mature dairy cows within 2 - 4 days after parturition.
- Factors strongly associated with an increased incidence of metritis include:
 - \ Large herd size

Septicemia cont'd---

└ Dystocia

└ Retained fetal membrane

└ Over conditioning and under conditioning of cows

➤ It is most common in cows when there is RFM for more than 24 hours after parturition.

➤ Retention of fetal membranes is commonly associated with abortion, dystocia and multiple births.

➤ In one study, RFM was found 9.5% in single and 46% in twin births.

➤ Metritis is 25 times likely to occur in cows with RFM than without.

➤ Forceful extraction of RFM is considered as a major predisposing factor for postpartum septic metritis.

➤ Uncomplicated cases of RFM in cattle have no significant effect on subsequent fertility and the calving-to-conception interval

Septicemia cont'd---

➡ Other less common risk factors associated for RFM include:

Placentalitis

Vitamin A and E deficiency

Selenium deficiency

Old age of cows

- Prolonged gestation period

Uterine prolapse

Hormone induced parturition

↳ Fetal anasarca(generalized fetal edema)

1 Fetotomy

- The factors that are associated with RFM are indirectly associated with the development of postpartum metritis.

Recent works indicate that the fundamental cause of

immune system to recognize the placenta as foreign tissue is impaired.

Pathogenesis

➡ All this results in the formation of: -

- Marked accumulation of foul – smelling fluid in the uterus.

- As the result there is production of different toxins which may absorb in to the body of the organism and cause sever toxemia.

- Sever toxemia causes irreversible fatty degeneration of liver in fat cows.

Septicemia cont'd---

- Body temperature is usually elevated from 39.5 °C - 41°C.
- The heart rate is also increased up to 96 - 120 beat/min.
- Respiratory rate increases from 60 - 72 breath/min
- Ruminal movements may be reduced(hypotonia or be absent(atonia).
- Discharge of a foul smelling fluid which has dark brown up to red colour.
- Rectal examination usually reveals that the uterus is large, flaccid.
- In large cows the uterus is located far from pelvic brim, and it may extend up to the ventral part of abdominal cavity which makes it difficult to palpate it.
- Tenesmus occurs most commonly when the fetal membranes are retained.

Septicemia cont'd---

Clinical pathology

1. Hematology

- Leukopenia, neutropenia and degenerative left shift may occur as a result of this disease.
- The degree of change depends on the severity of the disease and reflects the absorption of endotoxin.

2. Vaginal/ uterine fluid

- Microbiological examination of samples taken from the vagina may reveal a mixed bacterial flora including *E. coli*, *Proteus spp*, *Actinomyces pyogenes*, *Staphylococcus spp.*, *Streptococcus spp.*, *Corynebacterium spp* and other anaerobic bacteria with time since parturition.
- In general, *E. coli* predominates in the first 5 days after parturition.
- Where as *A. pyogenes* and *F. necrophorum*

Septicemia cont'd---

NB. Uterine lochia of cattle with retained placenta had much endotoxin concentration in the first 2 days post partum than did lochia of healthy cattle or cattle that had under go a dystocia but did not have retained placenta.

3. Other samples and tests

➤ Ketonuria may occur in over conditioned animals as a result of excessive fatty degeneration which results in ketosis.

Necropsy findings

➤ The uterus of affected cows is enlarged and flaccid and it may contain several litters of dark brown foul-smelling fluid with decomposed fetal membranes.

➤ The uterine mucosa is necrotic and hemorrhagic. The uterine wall also becomes edematous and thickened.

➤ In sever cases, fibrin may be present on serosal surface of uterus.

➤ The liver may be enlarged and fatty

Septicemia cont'd---

Diagnosis

- Diagnosis of PPSM can be tentative and confirmatory.
- Tentative diagnosis is based on history, clinical and necropsy findings.
- Confirmatory diagnosis is based on pathognomonic signs and isolation and identification of specific pathogenic agents.
- Typical pathognomonic signs are
 - Delayed post partum involution of uterus called **sub involution of uterus** with dark brown foul-smelling fluid.
 - Enlargement of uterus due to accumulation of exudates during rectal palpation.

Septicemia cont'd---

Differential diagnosis

- PPSM must be differentiated from other diseases which cause toxemia, anorexia, reduced ruminal motility and milk production.
- Common diseases which can be confused with postpartum septic metritis includes: -

1. The fat cow syndrome

- This occurs in cows with excessive body condition and is characterized by anorexia, ketonuria, marked loss in milk production, decreased ruminal movements and delayed uterine involution.

Septicemia cont'd---

2. Left-sided displacement of the abomasum (LDA)

➤ This is characterized by inappetance, anorexia, ketosis, ketonuria and other related signs.

3. Right-sided dilatation and displacement of the abomasums (RDA)

➤ This results in inappetance to complete anorexia, drop in milk production and decreased ruminal motility.

4. Acute diffuse peritonitis

➤ This condition is characterized by anorexia, toxemia, grunting, rumen stasis, fever and the presence of inflammatory exudates in the peritoneal fluid.

5. Per acute and acute mastitis

➤ This occurs in cows within a few days after parturition and is characterized by severe toxemia, swelling of the affected quarters and abnormalities of milk.

6. Simple indigestion

➤ This is common in high yielding dairy cows which are offered large amounts of high-energy palatable diets shortly before parturition.

Septicemia cont'd---

- Affected cows are inappetent, sometimes anorexic and rumen motility is decreased.

Treatment

- Treatment of PPSM can be done using one of the methods or in combination them.

- The methods are

- 1. Conservative therapy
- 2. Therapy using antimicrobial drugs
- 3. Intrauterine medication
- 4. Using ancillary(supportive) treatment

1. Conservative therapy

- Uncomplicated cases of retained fetal membranes without any evidence of clinical toxemia require no parenteral and intravenous treatment.

Septicemia cont'd---

- As the placenta will usually be expelled within 4 – 6 days.
- Cows with **retained fetal membranes and tenesmus** should be examined vaginally to ensure that there is no evidence of injury to the vagina or cervix.
- In cows with tenesmus, if the placenta is detached and loose it should be removed by careful traction.

NB. Forceful removal of the placenta should be avoided.

2. Therapy using antimicrobial drugs

- Treatment of animals with antibiotics during RFM depends on severity of cases and it must be:
 - 1. Cows with **retained fetal membranes** but without **systemic illness** should be monitored but treatment with antimicrobial agents must not be indicated.
 - 2. Cows with **retained fetal membranes** complicated by **septic metritis and toxemia** should

Septicemia cont'd---

agents daily for several days or until recovery occurs.
Some of commonly administered broad-spectrum antimicrobials

- 1. Intramuscular administration of **procaine penicillin (22000 IU/kg BW)** every 24 hours.
- 2. Subcutaneous administration of **ceftiofur (2.2 mg/kg BW)** every 24 hours.
- 3. Intramuscular administration of **ampicillin (10 mg/kg BW)** every 24 hours
- 4. Intravenous administration of **oxytetracycline (11 mg/kg BW)** every 24 hours.

3. In severely affected cases :-

- Large amounts of balanced isotonic fluids, electrolytes and glucose must be administered by continuous intravenous infusion.
- The uterus should always be examined by palpation

Septicemia cont'd---

- ‖ The degree of uterine involution
- ‖ The thickness of the uterine wall
- ‖ The volume of the uterus
- ‖ The nature of the luminal contents and the degree of attachment of the placenta to the cotyledons.
- Uterine fluids should be drained by creating a siphon.

3. Intrauterine medication

- The necessity for intrauterine medication nowadays it is controversial.
- But we can treat animals with PPSM that have sepsis in lumen of uterus by:
 - ‖ Intrauterine administration of antibiotic suspensions with intrauterine catheter.
 - ‖ Administration of intrauterine antibiotic bolus.
 - ‖ Intrauterine administration of antiseptics like povidone iodine, chlorhexidine, KMnO_4 solution and hypertonic NaCl solution.

Septicemia cont'd---

NB. If the tonus of uterus is low and does not drain the antiseptics that is found in lumen of uterus, stimulate it by injection of oxytocin(10 – 40 IU intramuscularly or 2.5 – 10 IU intravenously).

4. Using ancillary(supportive) treatment

➤ The infusion of **collagenase solution**(200 000 IU dissolved in 1 L of 0.9% NaCl containing 40 mg calcium chloride and sodium bicarbonate) into the umbilical arteries within 12 hours of parturition is an effective treatment for retained placenta.

➤ Collagenase injection therefore provides an effective method for preventing septic metritis in cattle with retained placenta.

➤ However, the collagenase solution is expensive, not widely available and the technique is difficult in some animals.

➤ There are many hormonal preparates that are used to control PPSM in cattle.

Septicemia cont'd---

Prostaglandins F2 α in dose 500 μ gs per animal intramuscularly.

Oxytocin in dose 30IU per animal intramuscularly.

1.4. Localized infections

Localized infections are common in farm animals and many are bacterial infections secondary to traumatic injuries.

Because most of them have a surgical outcome, by incision and drainage or by excision or amputation.

Here we should understand different localized infections like

Abscesses

Phlegmon

Cellulites

Abscesses

They are localized infections with circumscribed aggregation of bacterial debris, exudates and necrotic tissue.

They may be firmly walled off by a dense fibrotic wall

Localized cont'd---

- | Metastatic abscess
- | Non - metastatic abscess

Phlegmon

- When the infectious material is purulent but diffusely spread through tissues, it is known as **phlegmon**.
- But when it is inflammatory but not purulent the same lesion is called **cellulites**.

Etiology

- Abscesses and similar aggregations of pyogenic material occur in different locations of the body.
- The most important ones include: pharyngeal, retropharyngeal, hepatic, splenic, pulmonary, spinal cord and sub-cutaneous abscesses.
- More widespread accumulations of necrotic/toxic pyogenic debris occur as pericarditis, pleurisy, peritonitis, metritis, mastitis, meningitis and pyelonephritis.
- Common skin contaminant bacteria causing localized infections include:

Localized cont'd---

\ *Fusobacterium necrophorum*

\ *Streptococcus*

\ *Staphylococcus* and rarely in clostridial infections

\ *Corynebacterium pseudotuberculosis* which causes caseous lymphadenitis in sheep and goat and ulcerative lymphangitis in horses

\ *Rhodococcus equi* which causes pulmonary and subcutaneous abscesses in horses and cervical lymphadenitis in pigs

\ *Pseudomonas mallei* which causes glanders in horses

Pathogenesis

Portal of entry

Most localized infections begin as:

- 1. Penetrating wounds of the skin, caused accidentally or neglectfully because of failure to disinfect the skin adequately before an injection or

Localized cont'd---

- 2. Metastatic implantation from another infectious process, especially endocarditis, carried by blood or lymph, is the next most common cause.
- Pyogenic bacteria enters to the organism through the above portal of entry.
- Deposition of bacteria in tissues is sufficient to establish infection there in most instances.
- Conditions that favor abscess development include: -
 - └ Ischemia
 - └ Trauma
 - └ The presence of a hematoma
- A continuing process of pus formation results in enlargement to the stage of pointing and rupturing of an abscess, or spread along the path of **least resistance** into a nearby cavity or vessel, or discharge to the exterior through a **sinus**.
- Continuing discharge through a **sinus** indicates the persistence of a septic focus, usually a **foreign body**, such as a grass



Dr. Jaime Ruiz



Localized cont'd---

We can determine the species of bacteria that causes abscess by the colour , consistency and odor of the pus.

For example: -

- 1. Staphylococci produce large quantities of thick yellow pus.
- 2. Streptococci produce less pus and more serous-like exudates.
- 3. Pus associated with *A. pyogenes* is deep- colored, yellow or green in color and very thick and tenacious.
- 4. The pus of *F. necrophorum* is very foul-smelling and usually accompanied by the presence of gas.

Clinical findings

➤ Clinical signs of abscesses and other localized aggregations of pyogenic lesions differ according to their causes.

➤ General clinical signs may include:

Localized cont'd---

the back, or gait abnormality, including severe lameness

‖ Weight loss, which can be dramatic in degree and rapidity

‖ Obstruction of lymphatic and venous drainage, which can cause local swelling and edema.

Clinical pathology

Haemogram(Leucogram)

➤ A complete blood count is helpful in supporting a diagnosis of local abscess.

➤ Unless the infection is completely isolated by a fibrous tissue capsule or is small in size relative to the size of the animal there will be a **leukocytosis** with a left shift and an elevation of poly morph nuclear leukocytes in acute lesions.

➤ But in chronic causes lymphocytes and monocytes

Localized cont'd---

Sample of lesion for culture and staining

- Attempts to identify the presence of an infectious agent and to establish its localized infections identity are usually undertaken but care is necessary to avoid spreading infection from a site in which it is presently contained.
- Techniques used include: -
 - └ Paracentesis
 - └ Careful needle aspiration from an abscess
 - └ Blood culture (with the chances of isolation of bacteria being very small unless there is phlebitis or endocarditis)
 - └ Aspiration of cerebrospinal fluid.
- The isolation of bacteria from a well contained abscess may be difficult because of the paucity of organisms.

Necropsy findings

- The presence and location of the local infection can be demonstrated at necropsy.

Localized cont'd---

history, clinical signs and clinical pathology.

➤ But internal local infections are difficult to diagnose them clinically.

Treatment

➤ Treatment of local infection can be done in combination of

 \ 1. Drainage of abscesses

 \ 2. Using of anti microbial agents

1. Drainage of abscesses

➤ Surgical opening, flushing and drainage of the lesion and aspiration of the contents of the lesion.

➤ The site is prepared surgically and the abscess is drained, flushed and topically medicated.

➤ But if the abscess has not yet pointed with a soft spot, hot fomentations and hydrotherapy may aid in the maturation of a superficial abscess.

Localized cont'd---

- An analgesic may be required during this stage of therapy.

2. Using of anti microbial agents

- Antimicrobial agents given parenterally can be used for the treatment of deep abscesses not readily accessible to surgical drainage.
- Ideally, a sample of the contents of the abscess should be cultured and antimicrobial susceptibility determined.
- The agent must achieve high plasma concentrations to facilitate penetration into an abscess and daily treatment for several days is usually necessary.
- 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.
- Because capsule prevents diffusion of the drug into

florfenicol or macrolides, are theoretically advantageous in penetrating into abscesses.

1.5. Disturbances of free water, electrolytes and acid-base balance

➤ There are many diseases of farm animals that can disturb body fluids (free water), electrolytes and acid - base balance.

➤ A disturbance of body water balance in which more fluid is lost from the body than is absorbed results in reduction of circulating blood volume is called **dehydration**.

➤ In contrast, the rapid ingestion of large quantities of water is called **over hydration**.

➤ Electrolyte imbalances occur commonly as a result of

↳ Loss of electrolytes

Disturbance cont'd---

➤ Common electrolyte imbalances include

- ↳ Hyponatremia
- ↳ Hypokalemia
- ↳ Hypocalcemia
- ↳ Hypochloremia

➤ Acid-base imbalances, either acidemia or alkalemia, occur as a result of: -

- ↳ 1. The addition of acid and depletion of alkali reserve or
- ↳ 2. The loss of acid with a relative increase in alkali reserve

➤ Under most conditions, the above disturbances of fluid and electrolyte balance will occur simultaneously, in varying degrees, depending on the initial cause.

1.5.1. Dehydration

Etiology

➤ There are two major causes of dehydration: -

- ↳ 1. Inadequate water intake and/or

1. Inadequate water intake

➤ The most common reasons of inadequate water intake are

- ‖ Deprivation of water
- ‖ Lack of thirst due to toxemia
- ‖ Inability to drink water because esophageal obstruction

➤ The most common cause of dehydration is when excessive fluid is lost from the organism.

➤ Excessive fluid is lost from the organism through: -

- ‖ Diarrhea which is the most common reason for excessive fluid loss
- ‖ Vomiting
- ‖ Polyuria
- ‖ Loss of fluid from extensive skin wounds and copious

Disturbance cont'd---

- In acute carbohydrate engorgement in ruminants
- Acute intestinal obstruction and diffuse peritonitis in all species
- Dilatation and volvulus of the abomasum.

Pathogenesis

➤ Two factors are involved in the pathogenesis of dehydration

- 1. Depression of tissue fluid levels with resulting interference in tissue metabolism
- 2. Reduction in the fluid content of the blood

1. Depression of tissue fluid levels

➤ The initial response to negative water balance is the withdrawal of fluid from the tissues and the maintenance of normal blood volume.

➤ The fluid is drained primarily from the intracellular and interstitial fluid spaces

Disturbance cont'd---

muscle and skin.

➤ The loss of fluid from the interstitial and intracellular spaces results in:

‣ Loss of skin elasticity

‣ Dryness of skin and mucosa

‣ Reduction and retraction of the eyeball due to reduction in the volume of postorbital fat deposits

2. Reduction in the fluid content of the blood

➤ The secondary response for continued negative water balance is reduction in the fluid content of the blood.

➤ This cause in reduction of the volume of circulating blood .

➤ Reduction of volume of circulating blood is called **volume depletion**.

➤ Depletion in volume increases the concentration of the blood which is called **haemoconcentration**

Disturbance cont'd---

- Because of the haemoconcentration there is an increase in the viscosity of the blood which impedes blood flow and further exacerbates the peripheral circulatory failure.
- In deprivation of water and electrolytes or in deprivation of water alone, or inability to consume water in normal animals (eg. in esophageal obstruction), the dehydration is minimal.
- Because the kidney compensates effectively by decreasing output and increasing concentration.
- In addition, water is preserved by reduced fecal output and an increased absorption, which results in dehydration of the contents of the rumen which intern results in scant dry feces.
- In the diarrheic calf the kidney compensates very effectively for fecal water loss, and the plasma volume

Disturbance cont'd---

Clinical findings

- The first and most important clinical finding in dehydration is
- Dryness and wrinkling of the skin, giving the body and face a shrunken appearance.
- As the result the eyes recede into the sockets and the skin subsides slowly after being picked up into a fold.
- The dehydration is usually much more marked if water and electrolyte losses have been occurring over a period of several days.
- But in per acute and acute losses may not be obvious clinically.
- Because major loss will have occurred from the intravascular compartment and only minor shifts have occurred from the interstitial spaces.
- The best indicator of hydration status in calves has

Disturbance cont'd---

measured in millimeters.

➤ This distance is multiplied by constant of 1 . 7 to provide an estimate of the degree of dehydration as a percentage of euhydrated body weight

➤ 2. The second best indicator of hydration status in calves is the elasticity of the skin of the neck and lateral thorax, which are assessed by pinching the skin between the fingers, rotating the skin fold 90° and noting the time required after release of the skin fold for the skin fold to disappear (normally < 2 s).

➤ The best methods for assessing hydration status in adult cattle and other large animals has not been determined but it is likely that eye recession and skin tent duration in the neck region provide the most accurate and sensitive methods for estimating hydration status.

➤ In dehydration loss of body weight and muscular

Disturbance cont'd---

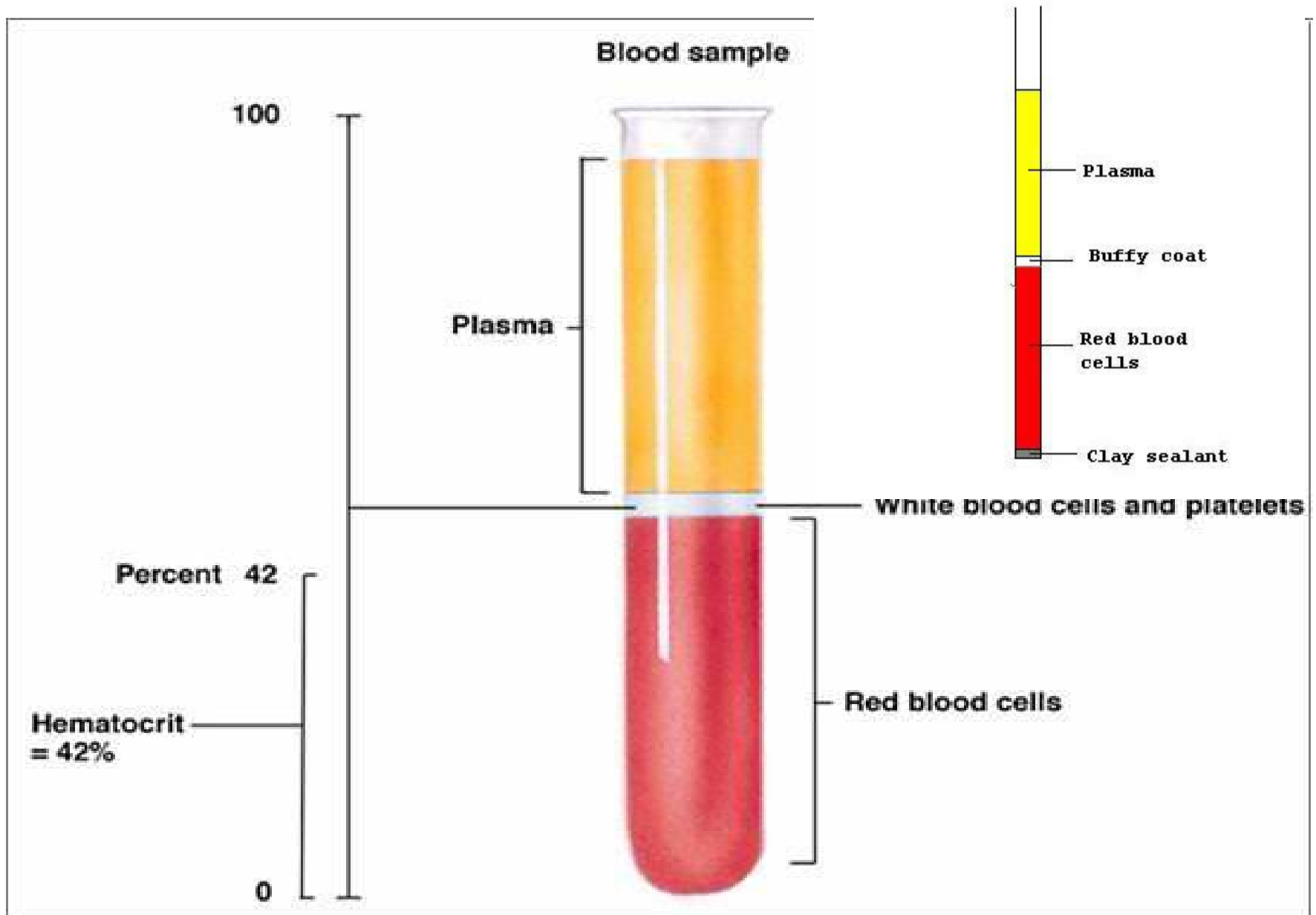
Clinical pathology of dehydration

- Dehydration is characterized by: -
 - └ Increasing the packed cell volume(PCV)
 - └ Increasing serum protein concentration

1.5.2. Electrolyte imbalance

- Most electrolyte imbalances are due to a net loss of electrolytes associated with diseases of the alimentary tract.
- But some causes like sweating, exudation from burns, excessive salivation and vomiting also result in electrolyte losses.
- But are of minor importance in farm animals, with the exception of **sweating** in the horse and **dysphagia** in ruminants.
- The electrolytes of major concern are: -

Hematocrit



Disturbance cont'd---

\ Potassium(K)

\ Calcium and phosphorus(Ca, p)

1. Hyponatremia

➤ **Hypo** means below norm

➤ **Natremia** means sodium ion(Na^+)in the blood

➤ So that **hyponatremia** means the existence of sodium ion in the blood and in extracellular fluid below norm.

➤ We know that sodium is the most abundant ion in the extracellular fluid and is chiefly responsible for maintenance of the osmotic pressure on the extracellular fluid.

Etiology and pathogenesis

➤ The most common cause of hyponatremia is increased loss of sodium through the intestinal tract in **enteropathies (intestinal diseases)**

Disturbance cont'd---

- There are 3 types of dehydration

- These are

- 1. Hypotonic dehydration

- 2. Isotonic dehydration

- 3. Hypertonic dehydration

- For example in calves with **acute diarrhea** due to **enterotoxigenic *E. coli***, the sodium concentration of the intestinal fluid secreted in response to the enterotoxin is similar to that of plasma, and hyponatremia usually occurs.

- This type of hyponatremia is called **hyponatremia due to isotonic dehydration**.

- Hyponatremia can be occurs when there is parallel loss of water and sodium.

- This type of hyponatremia is called **hyponatremia due to hypotonic dehydration**

Disturbance cont'd---

- **Hypertonic dehydration** is uncommon and occurs when there is a loss or deprivation of sodium.
- This occurs in animals which are unable to consume water because of an esophageal obstruction.
- But **hypertonic dehydration** never causes hyponatremia.
- Animals affected with diarrhea of several days duration continue to lose large quantities of sodium and the hyponatremia may become severe.
- Hyponatremia can also become severe when **sodium-free water** or **5% dextrose** are used as the only fluid therapy in animals already hyponatremic.
- Hyponatremia causes an increase in the renal excretion of water in an attempt to maintain normal osmotic pressure.
- This results in a decrease in the extracellular fluid space, leading to a decreased circulating blood volume, hypotension, peripheral circulatory failure and

Disturbance cont'd---

Etiology and pathogenesis of hyponatremia

**Acute diarrhea
e.g. horses,
calves**

**Enterotoxigenic *E. coli*,
e.g. calves, piglet**

**Increased loss of
sodium
normally secreted
in intestinal fluids**

**Produces intestinal fluid
with
a concentration of
sodium similar to
plasma**

**Exacerbation by
treatment with sodium-
free fluids,
e.g. 5% dextrose**



Chart cont'd---

HYPONATREMIA

```
graph TD; A[HYPONATREMIA] --> B[Increased renal excretion of fluid of low specific gravity may accentuate dehydration]; A --> C[Muscle weakness<br/>Mental depression]
```

Increased renal excretion of fluid of low specific gravity may accentuate dehydration

Muscle weakness
Mental depression

Chart cont'd---

Types of dehydration

**HYPERTONIC
DEHYDRATION**
e.g. simple
deprivation of
water

**Hypotonic
DEHYDRATION**
e.g. loss of isotonic
fluid as
in copious sweating,
Nephrosis,
simple enteritis

**Isotonic
DEHYDRATION**
e.g.
enterotoxigenic
colibacillosis,
salmonellosis
in the horse

**Simple
dehydration
without sodium
loss**

**Fluid
and sodium loss**

**Severe fluid plus
hypertonic sodium
loss**

**Mild
dehydration**

**Mild
dehydration
and
hyponatremia**

**Severe dehydration
plus severe
hyponatremia**

Clinical signs of hyponatremia

- There are no clinical signs that are characteristic of hyponatremia.
- But there is usually
 - ↳ Dehydration
 - ↳ Muscular weakness
 - ↳ Mental depression
- The above clinical signs also occur with other disturbances of both water and electrolytes and with acid-base imbalance.

2. Hypochloremia

- Hypo means below the norm
- Chloremia means chlorine ion(Cl^-) in the blood.
- So that hypochloremia means low level of chlorine in the blood and in the extracellular fluids.
- Hypochloremia occurs as a result of: -

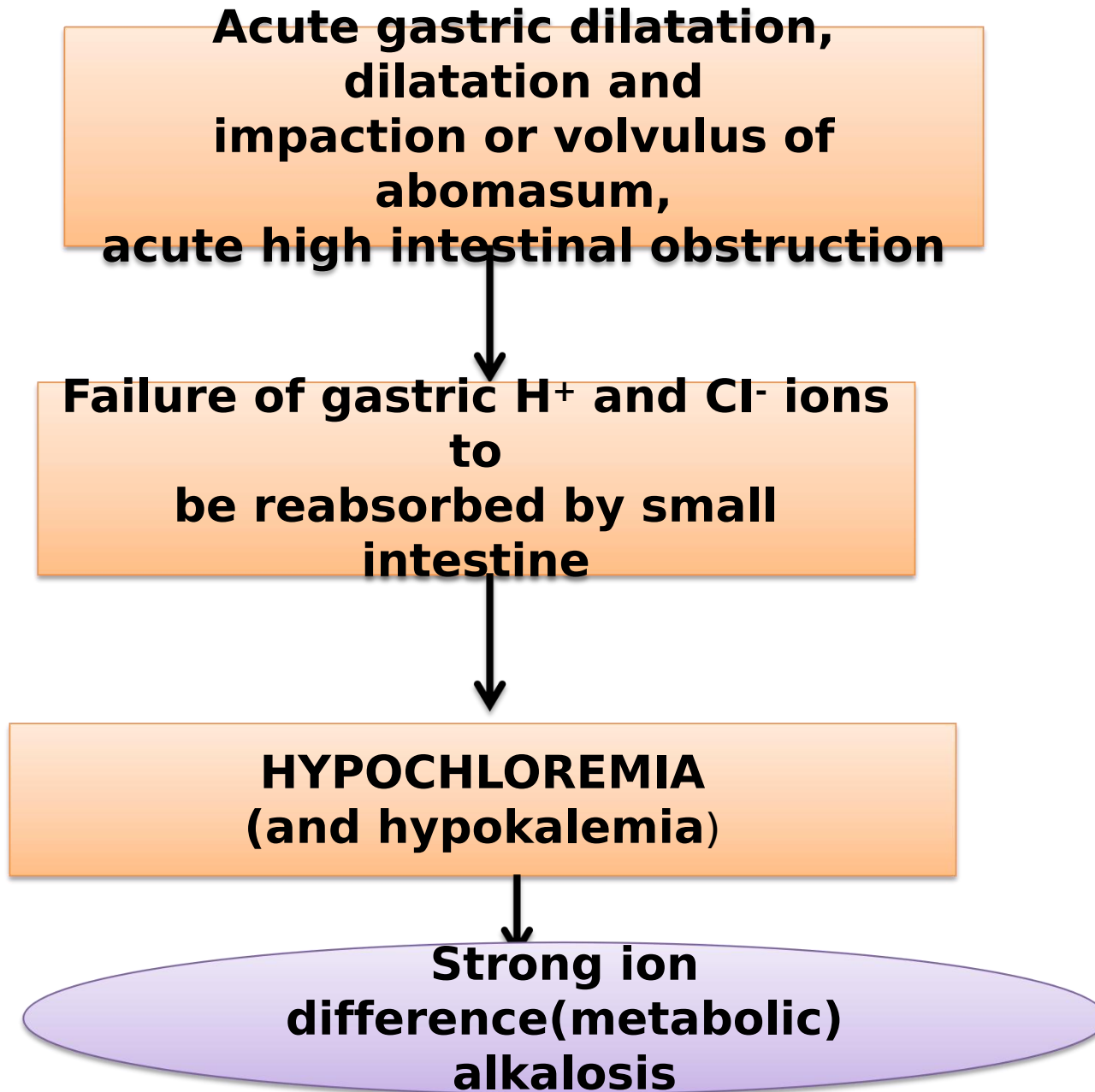
Disturbance cont'd---

] During acute intestinal obstruction, dilatation and impaction and volvulus of the abomasum and enteritis.

Pathogenesis

- Normally a large amount of chloride is secreted in the abomasum and stomach by the mucosal cells in exchange for bicarbonate, which moves into the plasma.
- The hydrogen, chloride and potassium ions secreted in gastric juice are normally absorbed by the small intestine.
 - Failure of abomasal function due to dilatation, impaction or torsion of the abomasum; and obstruction of the proximal part of the small intestine will result in the sequestration of large quantities of chloride, hydrogen and potassium ions which leads to a hypochloremia and hypokalemia.
- All this results in the formation of **hypochloric and hypokalemic metabolic alkalosis** in abomasum of ruminant animals.
- The importance of abomasum in production of

Etiology and pathogenesis of hypochloremia



Disturbance cont'd---

contents.

Clinical findings

➤ Clinical findings include:

- └ Anorexia
- └ Weight loss
- └ Lethargy
- └ Mild polydipsia
- └ Polyuria
- └ Lastly a marked metabolic alkalosis occurs, with hypokalemia, hyponatremia, azotemia and death.

3. Hypokalemia

- Hypo means below norm.
- Kalium(Latin word) meaning potassium.
- So that hypokalemia is the condition of lowered concentration of

Disturbance cont'd---

potassium ion in the blood and in extracellular fluids.

Etiology

- ‖ Hypokalemia may occur as a result of: -
- ‖ Decreased in dietary intake of potassium
- ‖ Increased renal excretion to balance the osmotic pressure
- ‖ Abomasal stasis
- ‖ Intestinal obstruction and enteritis
- ‖ Repeated administration of corticosteroids with mineral corticoid activity .
- ‖ Prolonged use of potassium-free solutions in fluid therapy for diarrheic animals may result in excessive renal excretion of potassium results in hypokalemia.

Pathogenesis

Disturbance cont'd---

decreased intracellular concentration of the ion

- The most common occurrence of hypokalemia in ruminants is in diseases of the abomasum which cause stasis and the accumulation of fluid in the abomasum.
- Potassium becomes sequestered in the abomasum along with hydrogen and chloride resulting in hypokalemia, hypochloremia and metabolic alkalosis.

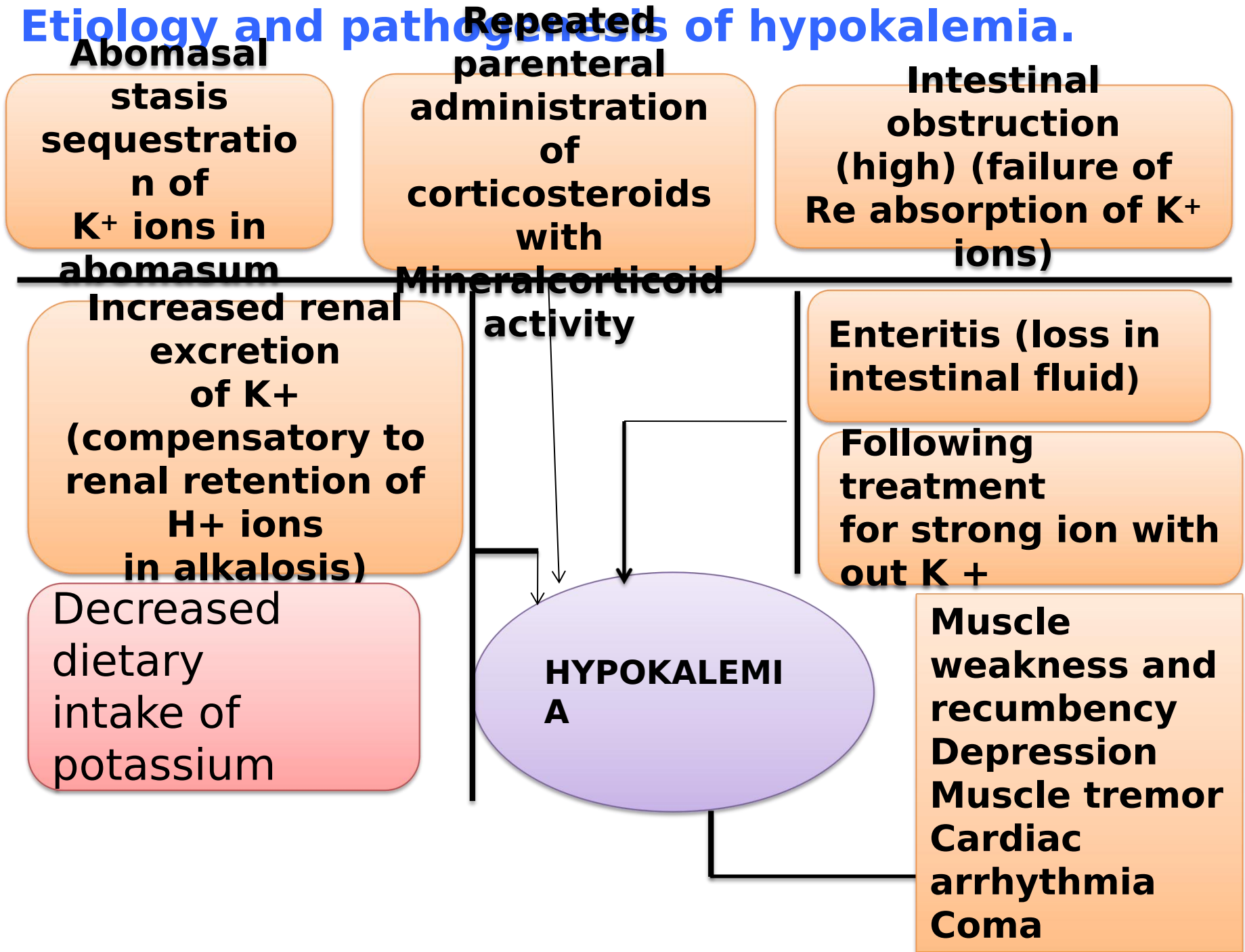
Clinical signs

- Hypokalemia can cause:

- ↳ Anorexia
- ↳ Muscle weakness and tremors
- ↳ Prolonged unexplained recumbency
- ↳ Inability to hold up the head
- ↳ If it is severe enough, coma.

- Hypokalemia causes muscle weakness by lowering the resting

Etiology and pathogenesis of hypokalemia.



Disturbance cont'd---
potential of membranes, resulting in decreased excitability of neuromuscular tissue.

➤ Thus, the differential diagnosis of the animal with muscle weakness should always include hypokalemia.

Diagnosis of hyponatremia, hypochloremia and hypokalemia

➤ Diagnosis can be done based on history, clinical signs and laboratory diagnosis of animal feed and water.

➤ It is also possible to diagnose the diseases by indirect determination of their concentration in serum and urine of animals.

Treatment of hyponatremia, hypochloremia and hypokalemia

➤ The treatment of hypochloremic, hypokalemic alkalosis requires correction of extracellular fluid volume and sodium and chloride deficits with: -

Disturbance cont'd---

➤ This is because providing adequate chloride ion allows: -

↳ Sodium to be reabsorbed without bicarbonate.

↳ Increased proximal reabsorption of sodium will decrease distal acid secretion because less sodium is presented to the distal nephron.

➤ Potassium should be administered intravenously or orally.

➤ The intravenous route is used only for the initial treatment of recumbent ruminants with severe hypokalemia and rumen atony, as it is much more dangerous and expensive than oral treatment.

➤ The most aggressive intravenous treatment protocol is an isotonic solution of KCl (1.15% KCl), which should be administered at less than 3.2 ml/ kg of BW per hour.

➤ Oral administration of potassium is the method of choice for treating hypokalemia.

➤ In appetent adult cattle should be treated with 30-60 g of feed

grade KCl twice with a 12-hour interval with the KCl

Disturbance cont'd---

intervals, for a total 24-hour treatment of 240 g KCl.

4. Hypocalcemia and hypophosphatemia

- Both these macro elements in adult animals causes a chronic disease called Osteodystrophy.
- The main reasons of osteodystrophy is disturbance in calcium, phosphorus and calcipherol (vitamin D).
- The disease appears in the form of dystrophic change in bone tissue that may have
 - └ Osteomalacis
 - └ Osteoporosis
 - └ Fibroses osteodystrophy
- Hypocalcemia very often common in pregnant and high productive cows.
- However very rarely it can be seen in other animals.
- Hypocalcemia or milk fever may occur in recently calved mature dairy cows that have been in appetent or anorexic for a few days.

Etiology

- Hypocalcemia and hypophosphatemia occurs due to

Disturbance cont'd---

- ↓ Decreased in dietary intake of these macro elements.
- ↓ In correct relation of calcium and phosphorus in animal feeds that must be 1.8: 1 – 2.0: 1
- ↓ Absence of motion, solar radiation and hypovitaminosis of vitamin D.
- ↓ Functional disturbance of thyroid hormones(parathyroidin and calcitonin).
- ↓ Functional disturbance of intestine , kidney and liver.

Pathogenesis

- During hypocalcaemia and hypophosphatemia the organism has compensatory mechanisms to keep its homeostasis.
- As the result the function of parathyroid gland increases.

The hormones acts on thyroid gland to produce

Disturbance cont'd---

- elements form bone tissues and to increase their reabsorption rate respectively.
- So that the number of osteoclasts increase to cause osteolysis.
- As the result osteodystrophy happens.
- Osteodystrophy can occurs during hypovitaminosis of Vitamin D.
- Because vitamin D(calcipherol) increase the rate absorption of calcium from intestine and its absorption in to bone tissues.

Clinical findings

- Clinical signs during hypocalcaemia and hypophosphatemia depends on stages of development of a disease .
- There are 3 stages of development of osteodystrophy.

They are

Disturbance cont'd---

\ 3. Third stage osteodystrophy

1. Primary stage of osteodystrophy

- At this stage the clinical signs of osteodystrophy are not observable.
- But symptoms like depression, rarely coma; animals start to eat like soils, their own faces, solid materials like sticks, bones etc.
- Hypotonia of rumen.
- Weak ton of heart but frequent pulse rate are the common clinical signs at this stage.

2. Secondary stage of osteodystrophy

- Uncoordinated movement of animals.
- Animals stands with difficulty when they stands.
- Palpation of bones of legs, maxillaries, ribs and joints painful.

Disturbance cont'd---

➤ At this stage the last bone of vertebral caudalis is absent as the result of osteolysis.

3. Third stage of osteodystrophy

➤ Characterized by progressive changes in bone tissues.

➤ Osteolysis continues to all vertebra of caudalis.

➤ All vertebra becomes soft and spongy during palpation .

➤ Rarely you can observe bending of vertebra in different directions.

➤ These are: -

↳ Up ward bending of vertebra called CIFORMSIS.

↳ Down ward bending of vertebra called LORDOSIS.

↳ Bending of vertebra to the right or left direction called ESCOLIOSIS.

➤ All bones specially the bones of legs, ribs, pelvic and

Disturbance cont'd---

➤ Lastly the animals are depressed, emaciated and there is disturbance all normal functions of internal organs.

Diagnosis of osteodystrophy

- It is easy to diagnose osteodystrophy when it appears clinically.
- The most difficult is to diagnose it early before it appears clinically.
- To prevent the disease, we must have a planned diagnose time at least 2 times a year to know the ration formulation and health of animals.
- But the best methods to diagnose osteodystrophy early are X - rays and ultrasonography.

Treatment

- Treatment of osteodystrophy depends on the causes of dystrophy.

Disturbance cont'd---

age, sex, body weight and productivity etc.

- Correct the ration if the ratio of Ca :P is not almost 2:1.
- If you can not correct the contents of calcium and phosphorus by using animal feeds , add to the ration premixes that are rich in calcium content like **lime stone (CaCO_3)**, **calcium phosphate($\text{Ca}_3(\text{PO}_4)_3$** and **bone meals**.
- If osteodystrophy is due to **hypophosphatemia**, you must correct the ration formulation first.
- If it is impossible to correct **hypophosphatemia**, by correcting the ration formulation, treat the animals with preparates that contain phosphorous.
- Hypophosphatemia is more safely treated by administration of orally **mono sodium phosphate ($\text{NaH}_2(\text{PO}_4)$** , **calcium phosphate ($\text{Ca}_3(\text{PO}_4)_2$** and

Disturbance cont'd---

- Oral administration also results in a more prolonged increase in plasma phosphorus concentration.
- Recommended dose is 200 g of feed grade monosodium phosphate (contains 50 g of phosphate) administered in gelatin boluses, drench, or by ororuminal intubation.
- If the **hypocalcemia** and **hypophosphatemia** **reaches** in climax stage like that of milk fever, you must administer preparates intravenous(IV).
- The most common preparates that can administer intravenously are:
 - 1. 10% calcium gluconate in dose up to 400ml for cattle.
 - 2. 10% calcium borogluconate in dose of 250 – 500 ml for cattle.
 - 3. A recommended treatment to an adult lactating

Disturbance cont'd---

strictly because even small drop of Ca Cl_2 can cause necrosis if it drops out of vein.

➤ Administer them of every day for 2 weeks.

5. Acid-base imbalance

➤ You know the PH of mammalian blood is maintained within the normal range of 7.35 - 7.45 by its buffer systems.

➤ There are 4 different buffering systems in mammalian blood.

➤ These are

1. Hemoglobin

2. Bicarbonate

3. Plasma protein

4. Phosphate

➤ Among them hemoglobin is the most important,

Disturbance cont'd---

- However the blood hemoglobin concentration is regulated on the basis of oxygen delivery instead of acid-base balance.
- And because rapid changes in hemoglobin concentration occur only with marked changes in hydration status or splenic contraction associated with exercise.
- The bicarbonate system has traditionally been considered to be the most important buffer in mammalian blood.
- Plasma proteins and phosphate buffers are not crucial as the two.
- The addition of relatively large amounts of acid or alkali to the blood is necessary before its buffering capacity is exhausted and its PH changed.
- Changes from normal acid-base balance towards

Disturbance cont'd---

1. Acidosis

- The acidity of the body fluids is a critical variable in normal metabolism.
- Even small changes in acidity can produce major disturbances of function.
- Because many physiological chemical reactions are strongly influenced by PH.
- In particular, most cellular enzymes function only within a narrow range of PH change, beyond which reaction rates fall sharply.
- Changes in the acid-base balance also affect several electrolytes- Na^+ , K^+ and Cl^- , in particular-which can further disrupt homeostasis.
- The normal range of PH is between 7.35 and 7.45, with an average of 7.40.
- When PH falls below 7.35, a condition of acidosis is

Disturbance cont'd---

slight variation to cause disease.

- Symptoms of disease develops when PH falls below 6.85.

- There are two mechanisms to develop acidosis

 - | 1. Metabolic acidosis

 - | 2. Respiratory acidosis

1. Metabolic acidosis

- Metabolic acidosis also called **non- respiratory acidosis**.

- In metabolic acidosis, the declining PH is the result of a fall in $[\text{HCO}_3^-]$ or a rise in $[\text{H}^+]$.

- Most often this is the result of increased production of acid metabolic products, as in prolonged hypoxia, where the shift to **glycolysis** produces lactic acid.

- It is also caused by renal failure as in the case of **glomerulonephritis** and **pyelonephritis**

Disturbance cont'd---

respectively.

- In either case, normal H^+ production exceeds excretion and thus PH falls.
- Direct loss of HCO_3^- can also produce acidosis, usually in chronic diarrhea, since there is significant secretion of bicarbonate into the lumen of the distal small intestine.

Etiology

- In general the causes of non - respiratory (metabolic) acidosis can be divided into three categories on the basis of pathogenesis.
 - └ Excessive loss of base(bicarbonate HCO_3^-)
 - └ Accumulation of endogenous or exogenous acid(H^+)
 - └ Combination of both of the above processes.
- Non - respiratory(metabolic) can be strong ion acidosis due to

Disturbance cont'd---

(hyper chloremia, hyper L-lactatemia, hyper D-lactatemia, and ketoacidosis.

‣ 2. An increasement of non -volatile buffer ion due to an increase in albumin, globulin and phosphate concentration.

➤ Some common specific causes to metabolic acidosis include:

‣ 1. Acute diarrhea in newborn animals

‣ 2. Acute enteritis in adult cattle and horses

‣ 3. Carbohydrate engorgement in ruminants and horses

➤ Example metabolic acidosis without dehydration, which is probably due to hyper **D-lactatemia**, has been described in neonatal goat kids and neonatal calves.

Disturbance cont'd---

6. The administration of excessive quantities of acidifying solutions

for the treatment of **metabolic alkalosis** also may cause acidosis.

Acute **intestinal obstruction** in the horse is commonly accompanied by metabolic acidosis, whereas in other species alkalosis occurs, at least initially.

1.1. Respiratory acidosis

➤ Respiratory acidosis occurs where there is retention of carbondioxide in the blood due to interference with normal respiratory exchange.

➤ As the result of increasing in the level of CO_2 in the plasma lowers the PH of the blood.

➤ Thus pneumonia, severe pulmonary emphysema, depression of the respiratory center and congestive

Disturbance cont'd---

- Metabolic acidosis is characterized by a low arterial blood PH and a reduced plasma bicarbonate concentration.
- Because it is formed following the loss of bicarbonate or the addition of hydrogen ions.
- But initially extra and intra cellular buffering and respiratory compensation minimize the change in PH until the kidney can excrete sufficient hydrogen ions to correct the acid-base imbalance.
- In general, the body will tolerate a PH range of 7.0 - 7.6, although survival has been reported at PH values beyond these limits for short periods(mostly neonates).
- In respiratory acidosis, the increased CO₂ tension of the blood(**hypercapnia**) causes an increase in the depth and then the rate of respiration by stimulation of

Disturbance cont'd---

activation of the sympathetic nervous system in response to acidemia

causes: -

- | 1. Increased cardiac contractility
- | 2. Increased heart rate
- | 3. Increased cardiac output to composite hypoxemia

➤ In acidemia, the myocardial response is not depressed until the blood PH is decreased to below 7.0 - 7.14.

➤ The increased carbon dioxide tension of the blood and depletion of bicarbonate causes an increase in the depth and then the rate of respiration by stimulation of the respiratory center.

➤ However, when **hypovolemic shock** is severe enough, there is often depressed respiratory function, resulting in the additional accumulation of hydrogen ions, and so the acidemia is accentuated.

➤ Lastly acidemia causes varying degrees of depression

Etiology and pathogenesis of acidemia

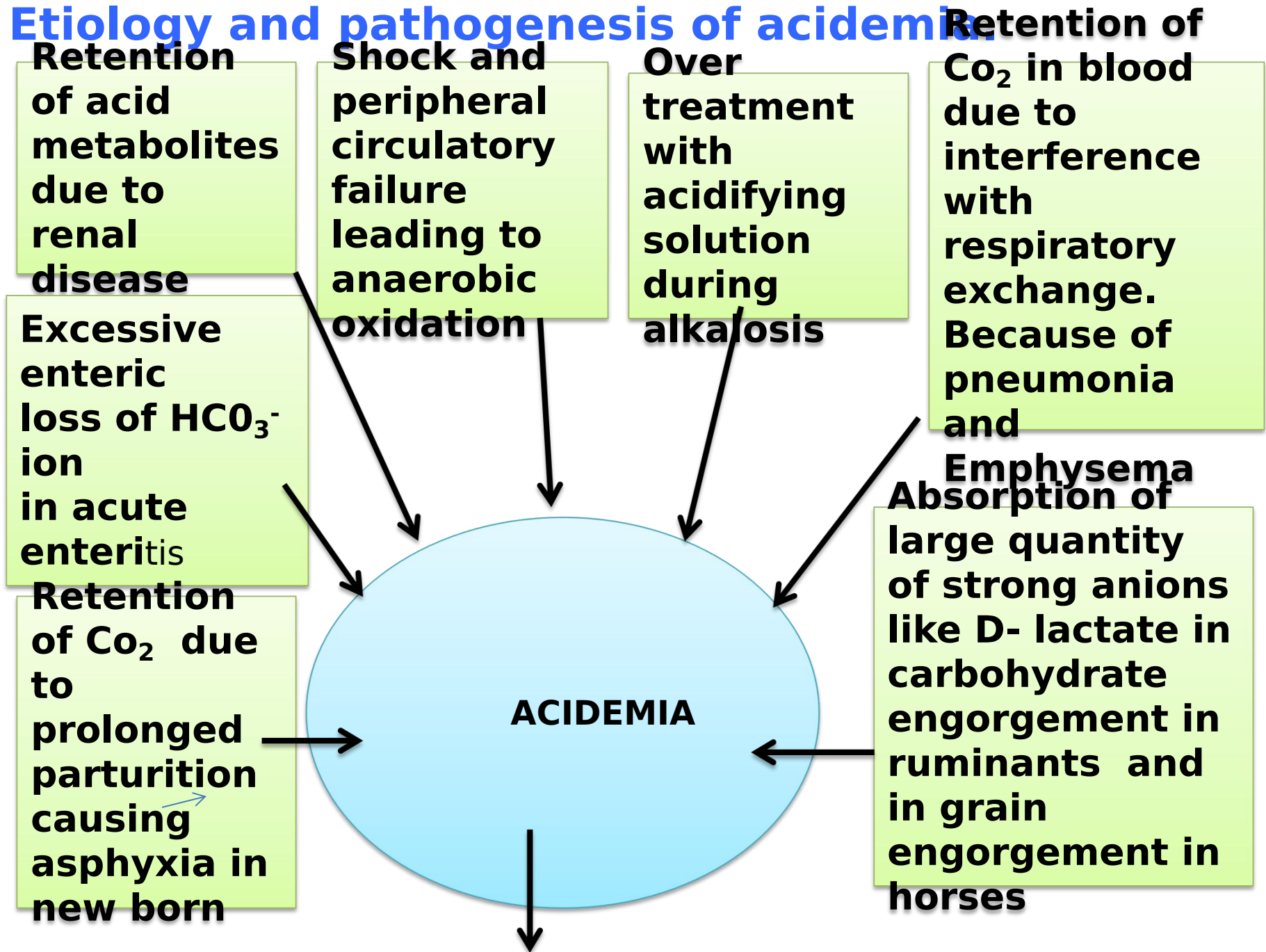


Diagram cont'd---

UNCOMPENSATED
when
respiratory
center
depressed by
hypovolemic
shock or
enterotoxigenic

Hypoventilation

**Respiratory
rate slow
and shallow
Increase in
CO₂**

**Weakness, lassitude,
terminal coma.**

**Tachycardia. Decrease in
blood**

pressure and pulse amplitude

**Compensated
Increased depth and
rate of respiration**

Hyperventilation

**Decrease in
CO₂**

**Interacellular
acidosis and
decreased
activity of Na - K
ATPase pump**

**Intracellular
swelling and
movement
of K out of cells
into extracellular
space**

Hyperkalemia

**Bradycardia,
arrhythmia**

Clinical findings

➡ The major clinical manifestations of metabolic acidosis are: -

Mental depression

◦ Varying degrees of muscular weakness.

➡ In severe acidosis, affected animals may be in **lateral recumbency** and appear to be in a **state of coma**.

➤ The major clinical manifestations in respiratory acidosis are:-

- Increasing of the depth and rate of respirations as CO_2 tension is increased .

➤ So that there is usually **tachycardia**, which becomes worse as the acidosis becomes more severe, and the **amplitude of the pulse and blood pressure** are both decreased.

➡ A syndrome of metabolic acidosis with minimal signs of dehydration or diarrhea has been described in calves

Disturbance cont'd---

respiratory function.

- Their respiratory rate will be much slower and the depth of respiration much more shallow than normal.
- In general affected calves are: -
 - ↳ Depressed
 - ↳ Weak and ataxic
 - ↳ The suck and menace reflexes may be weak or absent
 - ↳ Some calves appear comatose

Clinical pathology of acidosis

- The abnormal laboratory findings during acidosis include
 - ↳ A reduced venous blood PH
 - ↳ Increased CO_2 concentration
 - ↳ Reduced bicarbonate ion concentration
 - ↳ Marked hyper D-lactatemia
 - ↳ Elevated blood urea nitrogen
 - ↳ Increased anion and neutrophilic leukocytosis with a left shift.

Disturbance cont'd---

Diagnosis

- History, clinical findings and some what necropsy findings give tentative diagnose of a disease.
- Examples are : -
 - Animals affected with acute diarrhea due to infectious enteritis are likely to be in a state of **metabolic acidosis and hyponatremia**.
 - In intestinal obstruction of the horse, there are varying degrees of **dehydration and metabolic acidosis**.
 - Obstruction of the upper intestinal tract, or abomasal stasis, is characterized by varying degrees of **dehydration, and metabolic alkalosis with hypochloremia and hypokalemia**.
- So that a combination of history, clinical assessment and the available laboratory evaluation will allow the clinician to make the most rational approach to treatment.

Treatment of acidosis

1.2. Alkalosis (Alkalemia)

➤ It is a change of the PH of the body fluid toward alkaline PH.

➤ Alkalosis is caused by

└ An increased absorption of alkali

└ Excessive loss of acid

└ A deficit of CO₂

➤ There are two types of alkalosis: -

└ 1. Respiratory alkalosis

└ 2. Metabolic alkalosis

1. Respiratory alkalosis

➤ Respiratory alkalosis is formed as the result of excessive pulmonary CO₂ clearance.

➤ It may arise as a result of hyperventilation that attempts to compensate for hypoxia.

Disturbance cont'd---

- Respiratory alkalosis is formed during high fever, brainstem damage, or pulmonary embolism are other conditions in which hyperventilation occurs.

2. Metabolic alkalosis

- Alkalosis which is formed as the result of metabolic disorders that causes the PH of body fluids toward basic PH.

Etiology and pathogenesis

- Alkalemia(alkalosis) is caused by different diseases that results in :

- └ An increased absorption of alkali
- └ Excessive loss of acid
- └ Deficit of carbon dioxide

- For example abomasal atony due to dilatation, impaction or torsion of the abomasum is one of the commonest causes of **metabolic alkalosis**.

Disturbance cont'd---

hydrochloric acid and potassium into the abomasum, with failure of evacuation of the abomasal contents into the duodenum for absorption.

Disturbance cont'd---

Etiology and pathogenesis of alkalemia(

**Excessive loss of acid.
e.g. gastric
abomasal dilatation**

**continuous
secretion of**

**HCl and K^+ ions
into
abomasum and
failure**

**Hypochloremia
a
Hypokalemia**

**Overdosing
with
alkali,
usually
bicarbonate**

**Deficit of CO_2 ,
e.g.
excessive
breathing
in
excitement**

**ALKALEMIA
(Alkalosis)**

**Slow shallow respirations
because of lack of CO_2 stimulation. Depression
of ionized fraction of serum calcium may lead
to tetany**

Hyperpnea terminally

Disturbance cont'd---

Clinical findings of alkalosis

- The clinical signs of alkalosis are not characteristic enough to be recognized reliably.
- Alkalosis results in slow, shallow respirations in an attempt to preserve CO_2 .
- Muscle tremors and tetany with tonic and clonic convulsions may occur because of depression of ionized fractions of serum calcium.
- Hyperpnea and dyspnea may also occur in the terminal stages of alkalosis.

Diagnose of alkalosis

- It can be diagnosed based on history and clinical signs that are observable on animals.
- But the diagnose gives tentative diagnostic result.

Disturbance cont'd---

➤ Confirmatory diagnose can be done by laboratory by measuring the PH of body fluids.

Principles of fluid and electrolyte therapy

➤ 1. The most important principle is to prevent or minimize dehydration and electrolyte loss through: -

- | Provision of adequate water supply
- | Continuous supply of salt and minerals

➤ 2. The next most important principle is to treat potential losses of fluid and electrolyte as quickly as possible to minimize the degree of dehydration and acid-base imbalance.

➤ There are at least four possible abnormalities which could exist at the same time and which must be corrected.

- | 1. Fluid volume deficit
- | 2. Plasma osmolar deficit (total electrolyte deficit)
- | 3. Specific electrolyte imbalances

Disturbance cont'd---

➤ The two major problems in fluid and electrolyte therapy are

1. Determination of the nature and degree of abnormalities present

2. And to decide which fluid and electrolyte replacement should be used.

➤ The ideal situation would be to make both a clinical and laboratory evaluation of the animal.

➤ The history may give indications of the possibilities of dehydration, electrolyte imbalance and acid-base

Solutions	Solutions	Electrolytes present in the fluid
0.9% sodium chloride (isotonic saline)	0.9% sodium chloride (isotonic saline)	Na⁺ and Cl⁻
1.3% sodium bicarbonate	Acidosis	Na⁺ and HCO₃⁻

Table cont'd---

1.3% sodium bicarbonate in 5% dextrose	Acidosis	Na⁺, HCO₃⁻, 5% dextrose
5% sodium bicarbonate (hypertonic)	Severe acidosis	Na⁺ and HCO₃⁻
Equal mixture of isotonic saline and isotonic sodium bicarbonate	Acidosis and dehydration	Na⁺, Cl⁻ and HCO₃⁻
Lactated Ringer's solution	Acidosis	Na⁺, K⁺, Cl⁻, Ca²⁺, lactate
High-sodium, alkalinizing solution: Lactated Ringer's solution plus Sodium bicarbonate (5 g/l)	Acidosis and hyponatremia	Na⁺, K⁺, Cl⁻, HCO₃³⁻ and lactate

Table cont'd---

High-sodium, high-potassium alkalinizing solution: Lactated Ringer's solution plus 1 g/l potassium chloride and 5 g/l sodium bicarbonate	Acidosis, hyponatremia, hypokalemia	Na⁺, K⁺, Cl⁻, HCO₃⁻ and lactate
High-potassium acidifying solution: Isotonic saline plus 2.5 g potassium chloride/l Alkalosis,	Alkalosis, hypochloremia, hypokalemia	Na⁺, K⁺ and Cl⁻
Mixture of:: 1 Ltr. Isotonic potassium chloride (1.1%) 2 Ltrs. Isotonic saline	Metabolic alkalosis in cattle with abomasal disease	K⁺, Cl⁻, Na⁺ and 5% dextrose

Quantity of fluids required and routes of administration

- The amount of fluid required will depend on : -
 - ‖ 1. The degree of dehydration already present
 - ‖ 2. The amount of the continuous losses which are occurring during treatment
 - ‖ 3. The maintenance requirements of the animal during treatment assuming its dietary intake of water and electrolytes and nutrients is minimal.
- The fluids are usually given in two stages.
 - **Hydration therapy** - given in the first 4 - 6 hours
 - **Maintenance therapy** - given in the next 20 - 24 hours depending on the severity and the course of the disease.

Notes: - Please follow this method.

- ‖ 1. The total amount of estimated hydration therapy should be given intravenously in the first 4 - 6 hours in order to increase and maintain

Disturbance cont'd---

circulating blood volume.

→ Because restoring circulating blood volume will restore renal function which will assist in acid-base and electrolyte balance.

2. If acidosis or alkalosis is present, it should be treated immediately.

→ As the immediate correction of acidosis will return the tissues to their normal physiological activity.

3. The intravenous (IV) route is preferred for treatment of dehydration (hydration therapy) and correction of severe acid-base and electrolyte imbalances.

→ However, all other routes (intrapertioneal, intramuscular, subcutaneous and oral) are not satisfactory in the presence of severe dehydration.

4. The rate of administration and the dose will depend on

→ The size of the animal

→ The severity of the illness,

→ The types of fluids being administered

Disturbance cont'd---
after administration.

5. Whenever possible, the oral route can be used to deliver the maintenance requirements provided that there are no abnormalities of the digestive tract which will interfere with the oral administration or the absorption of the fluids.

1.6. Hypothermia, hyperthermia and fever

➤ Hypothermia, hyperthermia and fever are characterized by significant changes in body temperature of animals.

Introduction

➤ Farm animals maintain a relatively constant body core temperature as they are **homoeothermic animals**, during extreme ranges of thermal environments.

➤ This homoeothermic state is achieved by **physiological** and **behavioral mechanisms** that

Hypothermia cont'd---

body energy reserves.

- For the body temperature to remain constant in changing thermal environments, the rate of heat loss must equal the rate of heat gain.
- The body temperature is a reflection of the balance between heat gain from **the environment** (radiation, conduction, convection) or due to **metabolic activity** (maintenance, exercise, growth, lactation, gestation, feeding) and heat loss to the **environment** (radiation, conduction, convection, evaporation) or due to **metabolic activity** (milk removal, fecal elimination, urinary elimination) .
- Absorption of heat from the environment occurs when the external temperature rises above that of the body.

Heat production

Hypothermia cont'd---

- Shivering is a thermo genesis which is a response to sudden exposure to cold and is a major contributor to enhance heat production.
- There is also a **non - shivering thermo genesis** which can induce heat during exposure to cold and is the mechanism in which heat is produced by the **calorigenic effect** of **epinephrine** and **norepinephrine**, which are released into the blood in increased amounts.

Heat loss

- Heat is transferred to or from an animal by the four standard physical phenomena

- 1. Convection
- 2. Conduction
- 3. Radiation
- 4. Evaporation

- **1. Convection** is a transfer of heat between two media at different temperatures, such as the coat surface and the air.

Convective heat transfer depends on

Hypothermia cont'd---

- }\ The temperature gradient between the coat surface and air
- }\ The surface area
- }\ The air speed over the surface

2. Conduction is the transfer of heat between two media that are in direct contact, such as the skin and water.

3. Radiation is the absorption or emission of electromagnetic radiation at the body surface, and depends on

- }\ The skin surface temperature
- }\ Area of contact

4. Evaporation is the heat transferring process where heat is lost by the evaporation of water.

➡ It is dependent on

- }\ The water vapor pressure gradient between the

Hypothermia cont'd---

- Evaporation occurs by

- └ Sweating

- └ Salivation

- └ Respiration, with the relative importance varying between species.

Balance between heat gain and loss

- The balance between heat gain and heat loss is controlled by the heat-regulating functions of the **hypothalamus**.

- The afferent impulses derive from peripheral hot and cold receptors and the temperature of the blood flowing through the hypothalamus.

- The efferent impulses control respiratory center activity, the caliber of skin blood vessels, sweat gland activity and muscle tone.

- Heat storage occurs and the body temperature rises when there is

- └ A decrease in rate and depth of respiration

- └ Constriction of skin blood vessels

Hypothermia cont'd---

- These physiological changes occur in, and are the basis of the increment and decrement stages of fever.
- **Hypothermia** is caused by exposure to low environmental temperatures.
- While **hyperthermia (heat stroke or heat exhaustion)** is caused by exposure to high environmental temperatures, are the major abnormalities of body temperature associated with extremes of environmental temperatures.
- There is a term called **anhidrosis** occurring primarily in horses in hot humid climates and associated with the inability to sweat.

1. Hypothermia

- Hypothermia is a lower than normal body temperature, which occurs when excess heat is lost or insufficient is produced.

Hypothermia cont'd---

Etiology

➡ There are 3 main etiological factors that play a crucial role in hypothermia.

➡ These are

1. Excessive loss of heat

2. Insufficient heat production

3. Combination of excessive heat loss and insufficient heat production

1. Excessive loss of heat

- Exposure to excessively cold air temperatures causes heat loss if increased metabolic activity, shivering and sustained muscular contraction and peripheral vasoconstriction are unable to compensate.

2. Insufficient heat production

- Insufficient body reserves of energy and insufficient feed intake result in insufficient heat production

Hypothermia cont'd---

- Hypothermia also occurs **secondary to many diseases** in which there may be a decrease in the ability to shiver and skeletal muscle contraction associated with decreased cardiac output, decreased peripheral perfusion and shock.
- Other reason for hypothermia at the terminal stage of many diseases is because of **reduction of metabolic activities**.
- So that sudden fall in body temperature in a previously febrile animal, the so- called **premortal fall**, is an unfavorable prognostic sign.
- Examples include parturient paresis, acute ruminal acidosis (grain overload), and during anesthesia and sedation.

3. Combination of excessive heat loss and insufficient heat production

Hypothermia cont'd---

➤ Insufficient energy intake or starvation of newborn farm animals in a cold environment can be a major cause of hypothermia.

Epidemiology

➤ Hypothermia is common mostly in

➤ 1. Neonates

➤ 2. Wool or hair sheared sheep

➤ 3. Hypothermia secondary to other diseases

1. Neonatal hypothermia

➤ Newborn farm animals are prone to hypothermia in cool environments.

➤ That is why hypothermia is a major cause of neonatal mortality.

➤ As the neonates cannot maintain their rectal temperatures at normal values during the first few hours(mostly the first 6 hours in calves) after birth under cold environmental conditions.

➤ This is because

↳ The coat is wet with placental fluids and energy loss is

Hypothermia cont'd---

temperature

\ Only limited tissue substrates are available as energy sources.

\ Neonates also are exposed to a variety of environmental pathogens against as they have little specific immunity.

➤ That is why the neonatal period is one of the most critical to the survival of an animal.

➤ Because during this period the morbidity and mortality can be high under adverse environmental conditions.

The response of neonates to hypothermia (cold stress)

➤ Neonates can tolerate cold stress by :-

\ 1. Cold-induced thermogenesis

\ 2. Controlling of heat loss

\ 3. Producing heat

1. Cold-induced thermogenesis

➤ This is achieved by

\ Shivering thermogenesis in skeletal muscle tissue

Hypothermia cont'd---

contractions of skeletal muscle.

- **Non-shivering thermogenesis** is the formation of heat from brown adipose tissue, which is present in neonatal lambs, kids and calves but not in piglets.
- In neonatal lambs, approximately 40% of the thermogenic response during metabolism is attributed to non-shivering thermogenesis.
- But the rest of 60% attributed to shivering thermogenesis.

2. Control of heat loss

- The insulative nature of the external hair coat and cutaneous tissues to resist non-evaporative heat loss during cold exposure is critical in maintaining homeothermia.
- The thermal insulation is the sum of
 - ↳ Tissue insulation

Hypothermia cont'd---

conductive heat loss from the body core to the skin surface.

- Tissue insulation is influenced by

- └ Sub-cutaneous fat depth, which is minimal in neonates,

- └ Vasoconstriction peripheral blood vessels

- As you know in response to cold, blood vessels constrict, peripheral blood flow diminishes, and heat transfer is limited.

- **External insulation:** - this is thermal resistance of the hair coat and air interface to radiative, convective and conductive heat losses from the skin surface to the environment.

- External insulation is a function of **length** and **type of hair coat** and **the air interface**.

3. Heat production

- Heat production by metabolism varies directly with the level of feed intake.

- The more the animal eats, the greater the heat increment of feeding.

Risk factors for neonatal hypothermia

➤ There are different factors in calves, lambs, piglets and foals which further increase their susceptibility to hypothermia.

➤ Among them the most important risk factors are

- └ Exposure to cold whether during birth
- └ The presence or absence of dystocia
- └ Colostrum intake during birth
- └ Premature birth
- └ Small birth weight
- └ Birth from mothers of poor condition (malnutrition of the dam during late gestation)
- └ Breed differences

2. Post-shearing hypothermia in sheep

➤ Sudden unpredicted summer rainfall can cause high mortality due to hypothermia in newly shorn sheep.

Hypothermia cont'd---

- A fall in body weight in the period immediately proceeding shearing is another major risk factor for post – shearing hypothermia.

3. Hypothermia secondary to other diseases

- Hypothermia also occurs **secondary to many diseases** in which there may be a decrease in the ability to shiver and skeletal muscle contraction associated with decreased cardiac output, decreased peripheral perfusion and shock.
- Other reason for hypothermia at the terminal stage of many diseases is because of **reduction of metabolic activities**.

Pathogenesis of hypothermia

- Sudden exposure of neonatal animals at birth and during the first few days of life to cold temperature results in:

Hypothermia cont'd---

- | Decreased cardiac output and heart rate

- | Decreased blood pressure

- All these results in muscular weakness, mental depression, respiratory failure, recumbency and a state of collapse and eventually coma and death.

- The entire body especially the extremities become cold and the rectal temperature is below 37°C and may drop to 30°C in neonates.

- Deaths are the result of excessive body cooling due to low temperature, driving winds and starvation.

Clinical findings

- In general the following clinical signs are common during hypothermia

- A decrease in body temperature to below 37°C represents hypothermia for most farm animal species.

- Weakness, decreased activity, cold extremities, and varying degrees of shock are common.

- Bradycardia, weak arterial pulse and collapse of the major veins are characteristics.

Hypothermia cont'd---

- The mucus membranes of the oral cavity are cool and there is a lack of saliva.
- But typical clinical signs of hypothermia for each forms of hypothermia is as follows

In neonatal hypothermia

- In the early stages of hypothermia, affected animals may be shivering and trembling to produce heat by shivering thermogenesis and / or non - shivering thermogenesis by using brown depot fat tissue.
- But if the cold condition continues, the skin of their extremities and ears feels cool to touch.
- Hypothermic piglets will attempt to huddle together, are lethargic, do not suckle and eventually become recumbent and die.
- Hypothermic calves exposed to a cold environment will assume sternal recumbency, lie quietly, will have a weak suck reflex and will die in a few hours.

In shorn sheep hypothermia

- Sheep with hypothermia associated with recent shearing and cold weather have a range of body

Hypothermia cont'd---

- They huddle in tight groups and the animals which cannot maintain sufficient heat will become weak, recumbent and die within a few hours.

Hypothermia secondary to other diseases

- The hypothermia secondary to other diseases is usually not marked and clinical findings are related to the specific underlying illness.
- Hypothermia is common in diseases such as milk fever in cattle but returns to normal within a few hours after successful treatment with calcium salts.
- Successful treatment of the primary disease will usually return the temperature to the normal range.

Clinical pathology

- Clinical pathological examinations are usually not done because the diagnosis is frequently obvious and the variability in biochemical changes make them of limited value in reaching a diagnosis of hypothermia.
- However, in starvation induced hypothermia, there is depletion of body lipid and glycogen reserves

Hypothermia cont'd---

➤ In neonatal calves and lambs with hypothermia caused by excessive heat loss during short cold exposure, the serum concentrations of glucose, non-esterified fatty acids and Immunoglobulins may be at adequate levels.

Necropsy findings

- Lesions associated with hypothermia depend on the duration and severity of the hypothermia.
- Fatal hypothermia in lambs and calves is characterized by an absence of lesions.
- A relative absence of milk in the abomasum is common.
- But a marked reductions in the amount of perirenal adipose tissue may lead to diagnose neonatal hypothermia.

Differential diagnosis

- Neonatal hypothermia is characterized by:
 - └ Low body temperature
 - └ Bradycardia

Hypothermia cont'd---

- Weakness

- Cold peripheries

- Lack of suck reflex

- Lastly circulatory collapse

➤ So in order to differentiate hypothermia from other disease conditions which have similar clinical signs, you should consider

- The history of exposure to cold environments

- Poor availability of colostrum from the dam, together with the feeding history of the newborn are critical to the diagnosis.

➤ Neonatal hypothermia must be differentiated from

- Advanced septic shock

- Septicemia

- Acid-base imbalances

Treatment

Hypothermic newborn lambs

- A system for the detection and treatment of hypothermia in newborn lambs can improve the survival rate.
- Most lambs become hypothermic within 5 hours or at more than 12 hours after birth.
- Hypothermia in the first 5 hours of life is most commonly caused by a high rate of heat loss from the wet newborn lamb.
- Whereas a depressed rate of heat production consequent to starvation is the most common cause in the older lamb.
- Twin and triplet lambs are more susceptible to hypothermia than singles.
- Because of
 - Lower body energy reserves

Hypothermia cont'd---

higher than that of a single lamb and starvation is more likely.

- Lambs of any age with mild hypothermia ($37-39^{\circ}\text{C}$) are dried off if necessary to reduce heat loss, given ewe or cow colostrum by stomach tube and placed in a sheltered pen with the ewe.
- Lambs less than 5 hours of age with severe hypothermia ($<37^{\circ}\text{C}$) are dried off and given an intraperitoneal injection of 20% glucose at a temperature of 39°C .
- A large lamb (>4.5 kg) is given 50 ml, a medium lamb (3.0-4.5 kg) 35 ml and a small lamb $\ll 3.0$ kg) 25 ml of 20% glucose.
- Hypothermic lambs are then placed in warming pens, made of horizontally laid straw bales, two bales high.

Hypothermic newborn calves

- Clinical management of hypothermic newborn calves is similar to that of lambs.
- Supplemental heat must be provided immediately.

Hypothermia cont'd---

- Colostrum or milk should be warmed to 40°C and intubated using an esophageal feeder.
- Fluids given intravenously must be warmed if you want to treat hypothermia.
- One practical method requires submersion of the intravenous line in a sustained source of warm water.
- Intravenous dextrose (1 ml of 50% dextrose/kg BW) should be routinely administered to all hypothermic calves.
- Because most have moderate to severe hypoglycemia.
- The rectal temperature should be taken every 30 minutes during treatment to assess progress.
- A more aggressive rewarming technique involves the repeated administration of warm (40°C) 0.9% NaCl

Instrument used in Enema



Hypothermia cont'd---

Hypothermic newborn piglets

- Hypothermic piglets must be placed in a warming box with a heat lamp and treated with intraperitoneal administration of glucose for the hypoglycemia.

Control

- Management strategies to prevent hypothermia from excessive heat loss are most important in the first 24 hours after birth.
- Measures to minimize excessive heat loss include providing a dry, draft-free environment for calving and lambing.
- Providing a protective shelter for cow-calf pairs for calving and during the 1st week after birth can reduce mortality from hypothermia.
- Supply of adequate quantities of colostrum, beginning from birth is important in order to provide

Hypothermia cont'd---

after birth and they should be protected from draft/winds.

➤ Frequent monitoring of both rectal and air temperature as well as energy intake will assist in the diagnosis of thermal stress.

➤ So that appropriate action can be taken.

2. Hyperthermia (heat stroke or heat exhaustion)

➤ Hyperthermia is the elevation of body temperature due to

 | Excessive heat production and/or

 | Absorption and/ or

 | Deficient heat loss

➤ Heat stroke (heat exhaustion) is the most commonly encountered clinical entity.

Etiology

➤ The major causes of hyperthermia are the physical ones that includes

 | High and prolonged environmental temperature

Hyper thermia cont'd---

transportation.

➤ There many other causes of hyperthermia.

➤ Among them the most common are

➤ **1. Neurogenic hyperthermia** : - hyperthermia due to damage of hypothalamus.

E.g. spontaneous hemorrhage, may cause hyperthermia or poikilothermia.

➤ **2. Dehydration**: - it is due to insufficient tissue fluids to accommodate heat loss by evaporation.

➤ **3. Excessive muscular activity due to poisoning** e.g. strychnine poisoning.

Pathogenesis

➤ We know that unless the body temperature reaches a critical point, a short period of hyperthermia is advantageous to over come from infectious disease because: -

└ Phagocytosis and immune body production are facilitated

└ The viability of most invading organisms is impaired.

Hyper thermia cont'd---

- However, the metabolic rate mainly catabolism may be increased to by as much as 40-50%.
- For example liver glycogen stores are rapidly depleted and extra energy is derived from increased endogenous metabolism of protein.
- If anorexia occurs because of respiratory embarrassment and dryness of the mouth,
 - ↳ There will be considerable loss of body weight
 - ↳ Lack of muscle strength accompanied by hypoglycemia and a rise in Non Protein Nitrogen(NPN) compounds.
 - ↳ There is increased thirst due in part to dryness of the mouth there is an increased salivary secretion
- Heart rate increase during hyperthermia due to
 - ↳ The direct effect of in rising of blood temperature and / or

Hyper thermia cont'd---

- Respiration increases in rate and depth due directly to the effect of the high temperature on the respiratory center
- An increased respiratory cools the body by increasing heat loss together with exhalation air.
- Because of the reduction of renal blood flow as the result of peripheral vasodilatation, urine secretion is decreased.
- When the critical temperature is exceeded, there is depression of nervous system activity and depression of the respiratory center usually causes death by respiratory failure.
- Circulatory failure also occurs, due to myocardial weakness, the heart rate becoming fast and irregular.

Clinical signs

- The first observable clinical reaction to hyperthermia

Hyper thermia cont'd---

- There is also an increased heart and respiratory rates, with a weak pulse of large amplitude.
- Sweating and salivation occur initially, followed by a marked absence of sweating.
- The animal may be restless but soon becomes dull, stumbles while walking and tends to lie down.
- In the early stages there is increased thirst and the animal seeks cool places, often lying in water or attempting to splash itself.
- When the body temperature reaches 41°C respiration is labored and general distress is evident.
- Beyond this point the respirations become shallow and irregular, the pulse becomes very rapid and weak and these signs are usually accompanied by collapse, convulsions and terminal coma.
- Death occurs in most species when the core

Hyper thermia cont'd---

- Abortion may occur if the period of hyperthermia is prolonged .
- For example a high incidence of embryonic mortality has been recorded in sheep that were 3 - 6 weeks pregnant.
- Affected horses are fatigued and have profound fluid and electrolyte losses, characterized by hypotonic dehydration due to excessive sweating.
- The resultant clinical signs include decreased performance, depression, weakness, increased heart and respiratory rates, and marked increases in rectal temperature (usually exceeding 42°C) .

Clinical pathology

- No important clinicopathological change is observed

Hypothermia cont'd---

Necropsy findings

- At necropsy there are only poorly defined gross changes.
- Peripheral vasodilatation may be evident, clotting of the blood is slow and incomplete.
- Rigor mortis and putrefaction occur earlier than other cases.
- There are no such constant or specific histopathological changes to diagnose hyperthermia in animals.

Diagnosis

- Diagnose of hyperthermia is done based on history, clinical signs.
- Since pathological and laboratory diagnose are not applicable

Hypothermia cont'd---

- Septicemia is differentiated from hyperthermia in that
 - Petechial hemorrhages may be present on the mucousa and skin but not in hyperthermia.
 - Blood culture during septicemia shows positive for bacterial growth but not in hyperthermia.
- Hyperthermia can be differentiated from fever in that
 - In fever the body temperature of mammals seldom exceeds 41°C .
 - But it exceeds 41°C in hyperthermia most frequently.
 - Additionally in most cases of hyperthermia, examination of environment reveals causative factors.

Treatment

- The presence of adequate drinking water is essential and together with shade and air movement is of considerable assistance when multiple animals are

Hyperthermia cont'd---

severity or duration of the hyperthermia.

- Affected animals should be immediately placed in the shade and housed on the midline of the back with cold water so that their coats are saturated.
- Fans should be immediately placed in front of the animal to promote evaporative cooling, and cooled water, with and without added electrolytes, should be made available for the animal to drink.
- The rectal temperature should be monitored frequently during cooling, and water application should be stopped when the rectal temperature has returned to normal.
- If affected animals may not be interested in or capable of drinking, the intravenous administration of fluids such as 0.9% NaCl is needed.
- Horses often need 20-40 liter of intravenous fluids over the first few hours of treatment.

Control measures

- Shade alone is a most important factor in maintaining

Hyperthermia cont'd---

preventing heat stress.

- Shade reduces the heat gain from solar radiation and can be provided by trees or artificially by roofs or shades made from cloth or artificial material.

- The efficiency of metal shades can be increased by painting metal shades white on the top side and black on the underside.

- In dairy and feedlot cattle, the following measures should be taken to manage heat stress:

- }\ Provide cool clean water and plenty of trough space for drinking

- }\ Enhance airflow by fans or by providing mounds for cattle to stand on it.

- }\ Adjust rations and feed a larger percentage of the ration in the evening when it is cooler.

- }\ Minimize handling during periods of greatest heat stress

Hypothermia cont'd---

- It is independent to the effects of ambient conditions on body temperature.
- It is important to realize that fever is a combination of hyperthermia and infection or inflammation that results from an elevated set point for temperature regulation.

Etiology

- Fevers can be
 - | 1. Septic fever
 - | 2. Aseptic fever

1. Septic fever

- It is the most common type of fever.
- It is caused by infectious agents like bacteria, viruses, protozoa or fungi.
- Septic fever can be due to

Fever cont'd---

- **1. Localized infection** such as abscess, cellulitis, empyema.
- **2. Intermittently systemic infections**, as in bacteremia, endocarditis.
- **3. Consistently systemic infections**, as in septicemia.

2. Aseptic fevers

- Aseptic fever is caused by non infectious agents.
- The most common non infectious agents aseptic fever are

- **1. Chemical fevers**, caused by injection of foreign protein, intake of dinitrophenols.
- **2. Surgical fever**, due to breakdown of tissue and blood during surgical operations.
- **3. Fever from tissue necrosis**, e.g. breakdown of muscle after injection of necrotizing materials.
- **4. Severe hemolytic crises** (hemoglobinemia)
- **5. Extensive infarction**
- **7. Extensive necrosis** in rapidly growing neoplasms

Pathogenesis of fever

- Most of fevers are mediated through the action of pyrogens.
- An exogenous pyrogen generates fever through its capacity to stimulate the production and release of endogenous pyrogens (EPs).
- Endogenous pyrogens are substances produced in an inflammatory response that act on receptors in the hypothalamus to cause an upward alteration of its temperature set up.
- Pyrogens are produced by granulocytes, monocytes and macrophages in response to pathogens and to their metabolic products.
- The febrile response is initiated by the introduction of an exogenous pyrogens to the body.
- Exogenous pyrogens include
 - └ Bacteria

Hypothermia cont'd---

- Antigen-antibody complexes
- Hemoglobinemia in a hemolytic crisis
- Many inorganic substances like dinitrophenols
- One of the most potent exogenous pyrogens is the **lipopolysaccharide** of Gram-negative bacteria.
- In hypersensitivity states, soluble antigen – antibody complexes may act as mediators.
- The endogenous pyrogens includes
 - Interleukin – 1
 - TNF - α (Tumor Necrosis Factor alpha)
 - IFN – α (Interferon Alpha)
- The most important and best known endogenous pyrogen is **interleukin-1**, produced by monocytes and macrophages.
- In addition to their pyrogenic activity, cytokines mediate the acute phase response, which is a term now

Due to different reasons the pyrogenic substances are metabolized and their production ceases. reaction of animals to pathogen invasion, tissue injury, immunological reactions and inflammatory processes.

Endogenous pyrogens

- Endogenous pyrogens are proteins released from monocytes and, to a lesser extent, by lymphocytes.
- These proteins were originally designated as **monokines** and **lymphokines** respectively.
- But are now more commonly referred to under the more general term of **cytokines**.
- One of the pyrogenic cytokines is **interleukin -1**, formerly known as **lymphocyte activating factor**.
- Because interleukin-1 stimulates T-lymphocyte proliferation in the presence of antigen and thereby enhances the immune response.
- The mediators between endogenous pyrogen and the hypothalamus appear to be **prostaglandins** and the level of **calcium** in the hypothalamus appears to regulate its activity.

Fever cont'd---

anterior hypothalamus.

➤ The elevated prostaglandin levels in the hypothalamus raise the **thermostatic set point** and induce the mechanisms of

↳ Heat conservation (vasoconstriction) and

↳ Heat production (shivering thermogenesis) until the blood and core temperature are elevated to match the hypothalamic set point.

➤ Prostaglandins are synthesized from arachidonic acid by the following chemical sequence.

Endogenous pyrogens cause the release of arachidonic acid, with subsequent synthesis of prostaglandins

Arachidonic acid breakdown products modulate the hypothalamic thermoregulatory mechanism, resulting in an increase in the set point value

Diagram cont'd---

Prostaglandin synthetase-inhibitor

antipyretics lower fever by blocking the synthesis of prostaglandins or prostaglandin precursors from arachidonic acid.

- The diagram can be cleared as follows
 - After their production, the EPs circulate through the blood and the the hypothalamus is exposed to these EPs.
 - As the result set point of temperature increases in the hypothalamus.
 - The animal initiates response to conserve and produce heat until the body temperature reaches to the new set point.
 - That is why shivering, vasoconstriction, peripheral piloerection, and huddling behavior are all characteristics of animals during fever.
 - As the body temperature reaches at a new set point, the animal maintains its body temperature at the new fever temperature until the pyrogen is metabolized and

Fever cont'd---

➤ When this occurs, the set point in hypothalamus decreases back to normal, and the animal initiates heat-losing mechanisms to decrease body temperature.

Effect of pyrogens on the hypothalamus

➤ The effect of bacterial and tissue pyrogens is exerted on the thermoregulatory center of the hypothalamus so that the thermostatic level of the body is raised.

➤ There are 3 periods in the development of fever

└ 1. Period of increment

└ 2. Period of fever, the fastigium (period of constant temperature)

└ 3. Stage of decrement(fever defervescence)

1. Period of increment

➤ The immediate response on the part of organs involved in heat regulation is the prevention of heat loss and the increased heat production.

➤ The period is called period of increment(chill).

➤ The period is manifested by: -

└ Cutaneous vasoconstriction resulting coldness and

Fever cont'd---

skin and an absence of sweating.

↳ Respiration is reduced and muscular shivering occurs, while urine formation is minimal.

↳ The extremities are cold to the touch and the rectal temperature is elevated the pulse rate increased.

➤ When the period of heat increment has raised the body temperature to a new thermostatic level the second period of fever, **the fastigium, or period of constant temperature**, follows.

2. The fastigium or period of constant temperature

➤ In this stage the mechanisms of heat dissipation and production return to normal.

➤ Cutaneous vasodilatation causes flushing of the skin and mucosae, sweating occurs and may be severe, and diuresis develops.

➤ Metabolism is increased considerably to maintain the body temperature, and tissue wasting may occur.

➤ There is also an inability to maintain a constant

Fever cont'd---

decrement, or fever defervescence, appears and the excess stored heat is dissipated.

3. Stage of decrement(fever defervescence)

- Vasodilatation, sweating and muscle flaccidity are marked and the body temperature falls.
- If the toxemia accompanying the hyperthermia is sufficiently severe, the ability of tissues to respond to heat production or conservation needs may be lost and as death approaches there is a precipitate fall in body temperature.
- The fall in body temperature after the initial rise is accompanied by a decline in plasma zinc and plasma total iron concentrations.

The role of fever

- Recent studies has shown that body temperature typical of fever enhances many aspect of immune

Fever cont'd---

slowly when temperature is elevated.

- In some bacteria, temperature elevation interferes with the uptake of iron that the bacteria requires for growth and multiplication.

NB. In conclusion, these advantages collectively provide a significant benefit to animals the clear the infectious agents and/or prevent further infections at the same time.

Clinical findings

- The effects of fever are the combined effects of hyperthermia and infection or inflammation.

➡ The following are the main clinical features of fever

- Elevation of body temperature

Increasing of heart rate with a diminution of amplitude and strength of the arterial pulse

Hyperpnea and wasting

Fever cont'd---

- ‖ Increasing thirst, anorexia, constipation with scant feces
- ‖ Depression and muscle weakness.
- The temperature elevation is always moderate and rarely goes above 42°C (107°F) .

Form of the fever

- There are different clinical forms of fever.
- The most common are
 - ‖ 1. Transient fever
 - ‖ 2. Sustained fever
 - ‖ 3. Remittent fever
 - ‖ 4. Intermittent fever
 - ‖ 5. Atypical fever
 - ‖ 6. Biphasic fever

Fever cont'd---

1. Transient fever

- Fever with significant diurnal variation.

2. Sustained fever

- Fever without significant diurnal variation.

3. Remittent fever

- Fever when the diurnal variation is exaggerated.

4. Intermittent fever

- Fever when fever peaks last for 2-3 days and are interspersed with normal periods.

5. Atypical fever

- Fever when body temperature variations are irregular

6. Bi phasic fever

- Fever consisting an initial rise, and fall to normal and rise secondary.

Fever cont'd---

- In farm animal practice the most common cause of a fever is the presence of an inflammatory process such as pneumonia, peritonitis, mastitis, encephalitis, septicemia, viremia and the like.
- The clinical abnormalities that are typical of the particular disease must be detected and differentiated in the process of making a diagnosis.
- In the absence of physical causes of hyperthermia, the presence of a fever indicates the presence of inflammation, which is not always readily apparent.
- A fever of unknown origin occurs commonly in farm animals and requires repeated clinical and laboratory examinations to elucidate the location and nature of the lesion.
- But mostly the reasons of fever are difficult to know them and fever may last 2 - 3 days and returns to normal without any treatment.

Fever cont'd---

Clinical pathology

- There are no clinicopathological findings that are specific for fever.
- However, the haemogram will reflect the changes associated with the cause of the fever.
- You know inflammation is characterized by marked changes in the total and differential **leukocyte count** characteristic for each disease.
- You are going to deal about this in small and large animal medicine courses.

Necropsy findings

- The necropsy findings will be characteristic of the individual disease process and are commonly characterized by
 - └ The degrees of infectious process(per acute, acute, sub acute and chronic)
 - └ The severity of the disease
 - └ The length of illness and
 - └ Whether or not treatment had been given.

Fever cont'd---

necropsy findings.

- These gives you tentative diagnose of a disease.
- Confirmatory diagnosis can be done by identifying reasons of fever
- Wide variety of tests can be performed to identify the location and nature of the lesion causing the fever.
- The most commonly used include:
 - └ Microbiologic testing of blood samples
 - └ Analysis of serous fluids from body cavities
 - └ Cerebrospinal fluid analysis
 - └ Milk sample analysis
 - └ Reproductive tract secretion analysis
 - └ Joint fluid analysis
 - └ Biopsies
 - └ Exploratory laparotomy.

Fever cont'd---

Medical imaging may be necessary to detect deep abscesses and other pathological lesions.

Differential diagnosis

➤ Fever must be differentiated from

Hyperthermia due to a physical cause such as heat stroke or exhaustion .

In fever of unknown origin.

➤ To differentiate fever from others that have similar clinical manifestation, you must consider : -

➤ The history, physical examination, laboratory findings and epidemiological setting should be reviewed.

➤ Localizing clinical findings may provide a clue to the body system or organ involved .

➤ Common inflammatory processes that cause fever in animals include:

Abscesses of the peritoneum, pleura and lungs

Septic metritis

Endocarditis

Fever cont'd---

└ Polyarthrititis

└ Pyelonephritis.

Treatment

➤ Effective methods to treat fever varies and depends on the ability of clinician to identify the cause of fever.

➤ So that we can use different methods singly or in combination to treat fever.

➤ The most common methods are

└ 1. Therapy directed to causative agent(etiological therapy)

└ 2. Surgically

└ 3. Symptomatically therapy

1. Therapy directed to causative agent

➤ It is therapy directed to microbes and their toxins that cause fever.

➤ Since the most important aspects of the clinical management of fever should be directed at its cause.

➤ The main objective is to identify and treat the primary disease

Fever cont'd---

bacterial infections.

- The selection of antimicrobial, the route of administration and the duration of treatment depend on:
 - └ The cause of the infection
 - └ Its severity
 - └ The accessibility of the lesion to the drug.
- The use of antimicrobial agents to prevent secondary bacterial infections in animals with viral diseases is controversial and of doubtful benefit.
- In animals with a fever of unknown origin, broad-spectrum antimicrobial agents seem rational.
- However, blind therapy is not recommended because it may lead to drug toxicity, super infection due to resistant bacteria
- So that isolation, identification and antibiotic

Fever cont'd---

2. Surgical intervention

➤ In some cases it may be necessary to remove surgically by drainage techniques the source of the infection located in abscesses or body cavities such as the pleural and abdominal cavities.

3. Symptomatically therapy

- It is one of the methods of treatment directed to remove undesirable symptoms of diseases.
- So that antipyretics are advisable to use during fever.
- But fever ordinarily does little harm and usually benefits the animal's defense mechanism and creates unfavorable conditions to microbes.
- That why antipyretic agents are rarely essential and may actually obscure the effect of a specific therapeutic agent or of the natural course of the disease.

Fever cont'd---

inappetance, or is so high that death due to hyperthermia it is possible to use anti pyretic.

➤ The most common anti pyretic that are used commonly are **NonSteroidal Anti-inflammatory Drugs** (NSAIDs).

➤ Because most NSAIDs are the **inhibitors of prostaglandin** synthesis and act centrally to lower the thermoregulatory set point.

➤ The most common NSAIDs that are commonly used are

1. Flunixin meglumine

➤ Route of administration are IV or oral

→ Horse : - Administration can be IV or oral in dose of 1mg/ kg BW once daily for up to five days.

→ Cattle : - Administration can be IV in dose of 2.2 mg/ kg BW once daily for up to five days.

Fever cont'd---

or under skin(subcutaneously in dose 1mg/ kg BW for up to 3 days.

2. Metamizole sodium

→It is commonly used in horses in dose of 2. 2 g/ kg BW for IV administration.

3. Paracetamol

→It is used for dogs orally in dose of 10 – 20 mg/ Kg of BW for 2 – 3 times daily

4. Acetyl salicylic acid(Aspirin)

→Horse : orally in dose of 15 – 100 mg /Kg BW twice daily

→Dogs : Orally 10 – 25 mg/ kg BW 3 times daily

→Cats: Orally 10 – 25 mg/Kg BW once daily

→Cattle : Orally 100 mg/ kg BW twice daily.

1.7. Allergy and anaphylaxis

Allergy cont'd---

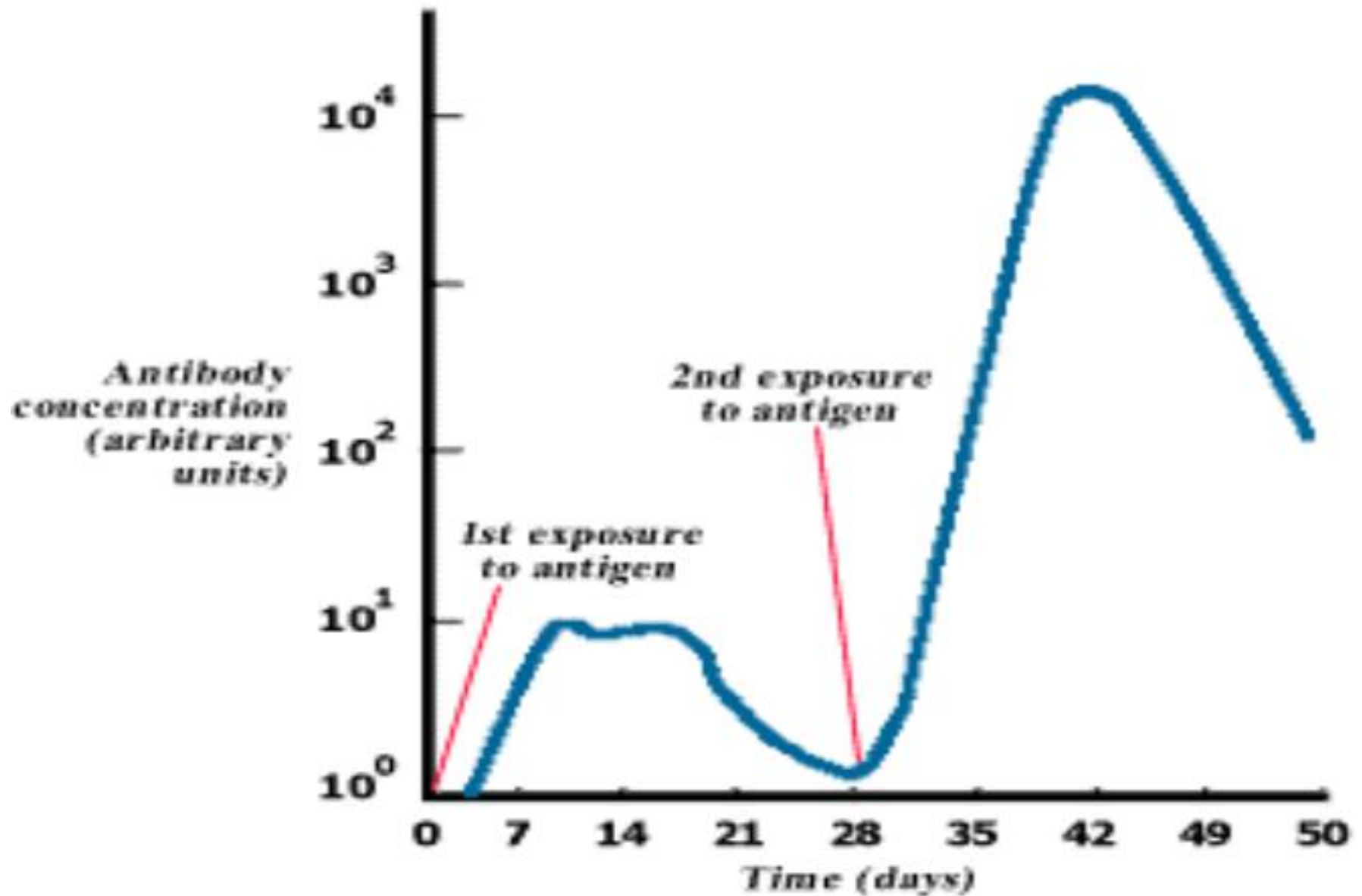
state of increased reactivity of the animal's tissues to that antigen that means a state of **specific immune responsiveness** is achieved.

- In most animals these responses are defensive and beneficial .
- But, on occasion, they can be detrimental to the host.
- In these cases inappropriate immune reaction of organism to antigen may appear .
- This inappropriate immunoreactions of organisms to antigen is called **allergy**.
- That means a state of **hypersensitivity** is said to exist, which is clinically recognizable as **allergy**.
- When the reaction is sudden and clinically severe it is called **anaphylaxis**.
- And if sufficiently severe it may result in **anaphylactic shock**.
- There are number of immune reactions that can be harmful to tissues.
- But in large animals the **immediate**

Allergy cont'd---

- ‖ Pulmonary (potentially Recurrent Airway Obstruction (RAO) in horses .
- ‖ Dermatological diseases (different allergy itches) are the most important.
- There are four major mechanisms for the induction of a hypersensitivity responses.
- They are classified as types I - IV based on the immune mechanism that elicits the disease state.
- These are
 - ‖ 1. Type I hyper sensitivity(Ig E mediated)
 - ‖ 2. Type II hyper sensitivity (Anti body mediated cytotoxic hypersensitivity)
 - ‖ 3. Type III hyper sensitivity(Immune – complex mediated)
 - ‖ 4. Type IV hyper sensitivity(Delayed type hyper sensitivity)

Diagram of normal immune response of the organism to antigen



Allergy cont'd---

1. Type I hyper sensitivity(Ig E mediated or immediate hyper sensitivity)

- Immediate hyper sensitivity response differs from normal humoral response in that plasma cells secrete IgE.
- The IgE with high affinity binds to Fc receptors on the surface of tissue mast cells and blood basophiles.
- Such Ig E coated mast cells and basophiles are said to be **sensitized**.
- The later exposure to the same allergen cross - links the membrane bound Ig E on sensitized mast cells and basophiles causing degranulation of these cells.
- Due to this allergen - antibody reaction(Ag - Ab) reaction **biological active mediators** are released.
- The mediators may act immediately on the site of antigen - antibody reaction or they may carry in the blood to produce effects in susceptible tissues at sites remote from the primary focus.
- The most common example of Type I hyper sensitivity is **anaphylaxis and anaphylactic shock**.

Allergy cont'd---

2. Type II hyper sensitivity (Anti body mediated cytotoxic hypersensitivity)

- Blood transfusion reactions in which host antibodies react with foreign antigen on the incompatible transfused blood cells.
- This mediate destruction of these cells by activating the complement system to create pores on the membrane of foreign cells.
- Antibody can also mediate cell destruction by antibody – dependent cell mediated cytotoxicity(ADCC).
- In this process cytotoxic cells bind to Fc region of antibody on target cells and promote killing of the cells.
- The most common diseases due to this type of allergy are
 - 1. Haemolytic diseases of the new born
 - 2. ABO blood group incompatibility

Allergy cont'd---

1. Haemolytic diseases of the new born

- This develops when maternal IgG antibodies specific for foetal blood group antigen, cross the placenta and destroy foetal red blood cells(RBCs).
- Sever haemolytic disease of the new born called **Erythroblastosis foetalis**.
- This mostly develops when Rh +(Rhesus factor plus) fetus expresses an Rh + antigen on its blood cells that Rh - mother does not express.

How does this disease develop?

- During pregnancy , fetal RBCs are separated from the mother's circulation by the layers of cells in the placenta called **trophoblasts**.
- During her first pregnancy with an Rh+ fetus, an Rh- woman is usually not exposed to enough fetal RBCs to activate specific B - cells against Rh + of fetus.

Allergy cont'd---

- Because during pregnancy , there is no transferring of blood from mother to fetus and vice - versa.
- At the time of delivery , however , separation of placenta from uterine wall allows large number of fetal umbilical cord blood to enter the mothers circulation.
- These fetal RBCs activate **Rh⁺ - specific B - cells** resulting in the production of **Rh⁺ - specific plasma cells** that can be differentiated in to **effector** and **memory cells**.
- The effector plasma cells secrete **Ig M antibodies** and clear the Rh + **fetal RBCs** from mother's circulation.
- But the memory cells remain and are threats to any subsequent pregnancy with an Rh⁺ fetus.
- Subsequent pregnancy of mother with Rh⁺ fetus activates the memory cells to differentiate them selves to **effector** and **memory cells**.

Allergy cont'd---

- Effector cells secrete antibody IgG which can pass through placenta and damage RBCs of fetus.
- As the result mild to severe anaemia develops with high fatality.
- In addition, conversion of haemoglobin to **bilirubin** can present additional threat to the newborn.
- Because the **lipid soluble bilirubin** may accumulate in the brain and cause brain damage.

Method of prevention of haemolytic disease of the newborn caused by Rh incompatibility

- Haemolytic disease of the newborn caused by **Rh incompatibility** in a subsequent pregnancy can be almost prevented by administering antibodies against Rh⁺ antigen to the mother within 24 – 48 hours after the first delivery.
- These antibodies against Rh⁺ antigen are called

Allergy cont'd---

at the time of delivery and facilitate their clearance before B- cells activation and ensuring memory cell production can take place.

2. ABO blood group incompatibility

- This incompatibility occurs when type A or B blood group of fetus carried out by type O blood group of mother.
- Since there is transferring of blood through uterus wall from umbilical cord of fetus during delivery, the mother develops antibody against blood group A or B of fetus.
- Subsequent pregnancy of mother with fetus which has blood group of A or B cause destruction of RBCs.
- Because there is transferring antibodies(IgG) against blood group A or B of the fetus.
- This results **anaemia, increased level of bilirubin**

Allergy cont'd---

adsorb non – specifically to protein on RBCs membranes.

- As the result they form a complex similar to **hapten – carrier complex**.
- This drug – protein complex induces formation of antibodies.
- Subsequent administration of the above drugs binds with protein of membrane of RBCs cause to induce antibodies and cause **complement mediated lysis of RBCs and develops progressive anaemia**.
- But when drug is withdrawn, **haemolytic anaemia** disappears.

3. Type III hyper sensitivity (Immune – complex mediated)

- You know the reaction of antibody with antigen generates **immune complexes**.

Allergy cont'd---

lead to **tissue damaging type III hyper sensitivity reactions.**

- The magnitude of reaction depends on
 - ↳ The quantity of immune complexes
 - ↳ Their distribution within the body
- Depending on where these complexes are carried, different tissue – damaging reactions can be observed.
- These are
 - When complexes are deposited in tissues very near the site of antigen entry , **a localized reaction develops.**
 - When complexes are formed in blood, a reaction can develop wherever the complexes are deposited.
- However the complexes are more frequently seen in
 - ↳ Blood vessels
 - ↳ Synovial membranes of joints

Allergy cont'd---

Mechanism of development of Type III hyper sensitivity

- Type III hyper sensitivity reactions develop when immune complex activate **complement system**.
- This results to array immune effector molecules like C3a, C4a and C5a that cause the local mast cell degranulation.
- Degranulation of mast cells leads secretion biological active mediators
- These biological active mediators
 - Increases the local vascular permeability
 - Have chemotactic effect to neutrophils which cause to accumulate in large numbers at the site of immune complex deposition.
 - Can induce aggregation of platelets and the release of

Allergy cont'd---

- The recruitment of neutrophils to the site develops an inflammatory lesion, with **indurations, erythema, edema, hemorrhage and necrosis**, a few hours (mostly 4 – 8 hours) after intradermal injection of antigen into a previously sensitized animal producing precipitating antibody.

Types of type III hyper sensitivity

- Depending on the location of antigen – antibody complex deposition, it can be

1. Localized type III reaction

2. Generalized type III reaction

1. Localized type III reaction

➡ When complexes are deposited in tissues very near the site of antigen entry , a localized reaction develops.

➡ Localized reaction develops when the antigen is entered **intradermally** or **sub - cutaneously** to

Allergy cont'd---

the given antigen.

- It results in the formation of localized **edema**, **erythema** at the site within 4 – 8 hours after injection.
- Severity of the reaction varies from **mild swelling** to **redness** up to **tissue necrosis**.
- This localized type III hyper sensitivity reaction called **Arthus** reaction.
- A good example of Arthus reaction is localized **allergy due to insect bite**.

2. Generalized Type III reaction

- When complexes are formed in blood, a reaction can develop wherever the complexes are deposited.
- Administering of antitoxins containing foreign serum such as anti **tetanus serum**, **diphtheritic antisera** are good examples.
- Because their administration can result within a day

Allergy cont'd---

administration the individual begins to manifest symptoms like **fever , weakness, oedema and erythema.**

➤ Generalized type III reaction that develop after administration of sera is called **serum sickness.**

➤ Another examples are **rheumatoid arthritis** and **drug reaction like allergies to penicillin and sulphonamides.**

4. TYPE IV(Cell-mediated or delayed hypersensitivity)

➤ Type IV hypersensitivity develop when antigen activates sensitized **T_{DTH}** which are sub population of T_H cells..

➤ In this reaction some population of T_C cells are also involved.

➤ When antigen enters for second time intradermally or

Allergy cont'd---

- The cytokines cause
 - The drawn of macrophages in to the area
 - Activation of macrophages to promote increased phagocytic activities.
 - Production of lytic enzymes for more effective killing
- All these lead to localized tissue destruction.
- The reactions take 48 - 72 hours to develop.
- The above mentioned time is a time required for **T_{DTH} cells activation, cytokines secretion** that are the mediators for accumulation of macrophages and subsequent release of their lytic enzymes.
- Type IV or delayed type hyper sensitivity reaction is very important in host defense against parasites and bacteria that can live with in the cells.
- Since the above pathogens are found with in the host cells

Allergy cont'd---

circulating antibodies can not reach them.

- But due to type IV hyper sensitivity reaction that cause heightened phagocytic activity and the building up of lytic enzymes from macrophages in the area of infection leads to non – specific destruction of cells and thus of the intra – cellular pathogen.
- But when this defense process is not entirely effective, the continued presence of the pathogen antigen can provoke a chronic DTH reaction.
- And it is characterized by excessive number of macrophages continued release of lytic enzymes and consequent destruction.
- The granulomatous skin lesion seen in with *Mycobacterium leprae* and lung cavitation seen with *M. tuberculosis* are examples of tissue damage that can result when chronic DTH reactions develop.

Allergy cont'd---

➤ Cell-mediated or delayed hypersensitivity is very important to diagnose many chronic infectious diseases of animals and human beings like tuberculosis, leprosy, glanders, coccidiomycosis and brucellosis by using allergen.

➤ Examples are

→ Bovine, avine and swine tuberculosis by using **tuberculin** called **PPD**.

→ Human leprosy by using allergen called **lepromin**.

→ Coccidiomycosis by using allergen called **coccidioidin**.

→ Glanders by using allergen called **mallein**

→ Brucellosis by using allergen called **brucellin**.

Anaphylaxis and anaphylactic shocks

➤ They are caused by antigen-antibody reaction which is IgE mediated.

Allergy cont'd---

- If severe it may result in anaphylactic shock.

Etiology

- Most commonly, severe anaphylactic reactions are seen in farm animals following the parenteral administration of a drug or biological product.
- Other routes of entry of the allergen, such as via the respiratory or gastrointestinal tract, may also result in **anaphylactic reactions** but not **anaphylactic shock**.
- The reaction may occur at the site of exposure or in other areas.
- In general, the reaction is due to sensitization to a protein substances entering the blood stream and second exposure to the same substances.
- Anaphylactic reactions can occur in some of the following circumstances:-
 - ↳ Repeated intravenous injection of biological

Allergy cont'd---

glandular extracts.

- └ Repeated blood transfusions from the same donor
- └ Repeated injections of vaccines, e.g. those against foot-and-mouth disease and rabies
- └ Rarely, after a first injection of a conventional drug such as penicillin, usually procaine or benzathine penicillin.
- └ In large animals this may occur after the injection of sera and bacterins.

Pathogenesis

- Anaphylactic reactions occur as the result of antigen reacting with circulating or cell-bound antibody.
- In humans and dog a specific class of antibody called IgE, has been identified and has particular affinity for fixed tissue mast cells.
- But in farm animals the classes of antibody involved

Allergy cont'd---

➤ Antigen-antibody reactions occurring in contact with, or in close proximity to fixed tissue **mast cells, basophiles and neutrophil** result in the activation of these cells to release **pharmacologically active substances** that mediate the subsequent anaphylactic reaction.

These substances include: -

‖ **Biogenic amines** such as histamine, serotonin and catecholamines

‖ **Vasoactive polypeptides** such as kinins, cationic proteins and anaphylatoxins.

‖ **Vasoactive lipids** such as prostaglandins

‖ **Slow reacting substance of anaphylaxis (SRS-A)** and others.

➤ These biological active substances acts on smooth muscles, small blood vessels, mucous glands, blood

Allergy cont'd---

- Increasing mucous secretion of mucosal glands
- Facilitate the aggregation of platelets that activates blood clotting factors.
- Decreasing of sensation of sensory nerve endings.
- All this leads in the development of **anaphylaxis** and in severe case **anaphylactic shock**.

Clinical findings

Cattle

- In cattle, initially there is a sudden onset of severe dyspnea, muscle shivering and anxiety.
- In some cases there is profuse salivation, in others moderate bloat and yet others diarrhea.
- Additional signs are urticaria, angioneurotic edema and rhinitis.
- Muscle tremor may be severe and a rise in

Allergy cont'd---

Sheep and pigs

- In sheep and pigs, acute dyspnea is common.
- Laminitis also occurs rarely in ruminants

Horses

- In the horse, naturally occurring anaphylactic shock is manifested by severe dyspnea, distress, recumbency and convulsions.
- Death may occur within less than 5 min but it usually requires about an hour.
- Laminitis and angioneurotic edema are also common signs in the horse.

Clinical pathology

- Blood histamine levels may or may not be increased.
- There is a marked increase in the packed cell volume

Allergy cont'd---

Necropsy findings

- In acute anaphylaxis in young cattle and sheep the necropsy findings are confined to the lungs.
- In the lungs you can find pulmonary edema and vascular engorgement.
- In adult cattle there is edema and emphysema without engorgement.
- In pigs and sheep pulmonary emphysema is evident and vascular engorgement of the lungs is pronounced in the latter.
- Pulmonary emphysema and widespread petechiation in the horse

Differential diagnosis

- Acute pneumonia may be confused with anaphylaxis.
- But there is usually more toxemia and the lung changes are more marked in the ventral aspects in

Treatment

General principle to treatment of allergy

- The treatment of allergic states is by the use of **functional antagonists**.
- Functional antagonists have opposing effects to those of the **allergic mediators**.
- Specific pharmacological antagonists, especially **antihistamines** and **corticosteroids** are commonly used.
- Some examples of antihistamines are acrivastine, cetirizine, desloratadine, fexofenadine, chlorphenamine, clemastine, cyproheptadine, hydroxyzine, ketotifen and promethazine that can be administered as droplet, syrups, tablets and injection.
- The functional antagonists include the **sympathomimetic drugs** like those related to **epinephrine(adrenaline) and**

Allergy cont'd---

- From sympathomimetic drugs there is a choice between those with an **alpha-response**(vasoconstriction and maintaining vascular permeability) and those with a **beta response**(bronchodilator and cardiac stimulatory).
- Antagonists, antihistamines have very limited usefulness, being effective only when the allergic mediator is only histamine.
- However the corticosteroids and NSAIDs have very wide applicability.
- The most common corticosteroids that are used for treatment of inflammation and allergy are
 - Betamethozone
 - Dexametazone
 - Prednisolone acetate

Allergy cont'd---

└ Phenylbutazone

└ Meclofenarnic acid

➤ Both the corticosteroid and NSAIDS are effective anti allergic drugs because all inhibit **prostaglandin synthesis** and thus reduce inflammation.

Treatment of anaphylaxis and anaphylactic shock

➤ Treatment should be administered immediately.

➤ Because a few minutes' dalliance may result in the death of the animal.

➤ Epinephrine (adrenaline) is the most effective for treatment of anaphylaxis and anaphylactic shock.

➤ Epinephrine administered intramuscularly or subcutaneously.

➤ But in critical situation to save the life of animals it is possible to administer IV

Allergy cont'd---

given immediately following the epinephrine.

➤ Antihistamines are in common use but provide variable results due to the presence of mediators other than histamine.

Dose and route of administration

1. Epinephrine(Adrenaline)

Parenterally: Cattle, Horses, Sheep and Swine

→ 1 ml of 1:1000 solution per 45 kg of body weight injected subcutaneously or intramuscularly

Dogs And Cats

→ 0.1 to 0.5 ml of 1:1000 solution injected subcutaneously or intramuscularly.

NB. Intravenous injection is not recommended for routine clinical work, but in emergency situations, this route may be desired.

When injected intravenously, the dosage should be

2. Corticosteroids

1. Betamethozone

- **Nature:** Injectable solution of 2 mg/ ml and can be in tablet form
- Horses, cattle, sheep, goat and pigs - IV or IM route in dose of 20 – 200µg/ Kg of BW.
- Dogs and cats - IM or IV route in dose 40 – 80µg/ Kg of BW.

2. Dexametazone

- **Nature:** Injectable solution with 1 mg/ ml, 2 mg/ ml and 5 mg/ ml and can be in tablet form.
- Horses, cattle, sheep, goat and pigs: IV or IM route in dose 20 – 200µg/ Kg of BW.
- Dogs and cats- IM or IV or Sc route in dose 20 – 200µg/ Kg of BW.
- During shock use IV route in dose of 3 mg/ kg BW

3. Prednisolone acetate

➤ **Nature:** Injectable solution with 10mg/ ml and 25 mg/ ml.

└ It is used only for dogs and cats.

└ Dose 0.1 – 0.2 mg/ kg BW daily IM route.

Part 2. Practical antimicrobial therapeutics

2.1. Antimicrobial drugs and principle of treatment

Introduction

- Drugs that kill or inhibit multiplication of microbes are called **antimicrobial agents**.
- Naturally antimicrobial agents are chemical substances.
- These chemical substances which have the ability to kill or inhibit the multiplication of disease causing microorganisms are called **chemotherapeutic agents**.
- Treatment of a disease with chemical substances is known as **chemotherapy**.
- Chemotherapeutic agents can be obtained:
 - 1. Naturally from microorganisms, some plants and animals.
 - 2. Synthetically (a few antibiotics and sulfonamides etc.)
- The term chemotherapeutic has often been used to distinguish synthetic compounds from antibiotics

Antimicrobial cont'd---

- That is why the term chemotherapeutic agents is applied nowadays for both antibiotics and synthetic compounds.
- All chemotherapeutic agents are called **antimicrobial drugs**.
- But all antimicrobial drugs are not antibiotics.
- Because there are many antimicrobial drugs that are not antibiotics.
- This definition excluded substances that kill bacteria, but are not produced by microorganisms and synthetic analogs (such as gastric juices and hydrogen peroxide etc).
- It also excluded **synthetic antibacterial compounds** such as the **sulfonamides**.
- You know the word antibiotics is came from Greek word.
- Anti means "against" and bios means "life".
- That means antibiotics are chemical substances that are originated from **other microorganisms(bacteria and mold)** but are harmful to other microbes.

Antimicrobial cont'd---

➤ Example antibiotic **penicillin**, that was discovered by German scientist **Alexander Fleming** in 1928 from fungus ***Penicillium notatum*** is harm full to many gram positive bacteria.

➤ Antibiotic **streptomycin** is obtained from bacteria genus ***Streptomyces*** and is effective for both gram positive and gram negative bacteria.

➤ Antibiotic **cephalosporin** is obtained from molds of the genus *Cephalosporium*.

➤ Now a days most of antibiotics are prepared commercially from micro organisms by process is called **microbial biosynthesis**.

➤ But there are many antibiotics which are prepared synthetically.

Anti microbes and their mode of action

➤ Antibiotics have two types of actions:

- 1. Bacteriostatic action
- 2. Bactericidal action

Antimicrobial cont'd---

bacteriostatic action of antibiotics.

➤ Some common antibiotics that have bacteriostatic action are **tetracycline, spectinomycin, macrolides** and **chloramphenicol** etc.

2. Bactericidal action

➤ Action of antibiotics that cause death of bacteria is called **bactericidal action of antibiotics**.

➤ **Penicillins, cephalosporins, fluoroquinolones, vancomycin, monobactams** and **carbapenems** etc have bactericidal action.

➤ A few bactericidal antibiotics can be bacteriostatic at lower dose.

➤ To be useful chemotherapeutic agent, antibiotics must have the following qualities:

1. They should have the ability to destroy or inhibit the growth of many different species of pathogenic microorganism.

That means they must be “ **abroad spectrum**” antibiotics.

Antimicrobial cont'd---

Mode of action of antimicrobials

Bactericidal antimicrobials	Bacteriostatic antimicrobials
Beta-lactams Penicillins Cephalosporins Semisynthetic penicillins Ampicillin Amoxicillin Cloxacillin Methicillin Carbenicillin Aminoglycosides	Sulfonamides - All Trimethoprim Methotrexate Pyrimethamine Tetracyclines Macrolides Erythromycin Oleandomycin Spiramycin Tylosin

Table cont'd---

Bactericidal antimicrobials	Bacteriostatic antimicrobials
Streptomycin Neomycin Gentamicin Paromomycin Tobramycin Glycopeptides Vancomycin Rifampin Bacitracin Polymyxins Fluoroquinolones	Carbomycin Lincomycin Chloramphenicol Florphenicol

kidney and gastro – intestinal tract.

4. They should not eliminate the normal micro flora of the host.

Mechanisms of action of antimicrobials

- Antimicrobial drugs are selectively harmful to microorganisms without causing significant harm to animals.
- However, this selective toxicity depends on the concentration of the drugs in the tissue.
- Because if they are excessively administered, it may cause severe tissue damage or death of animals.
- So that the correct dose, route of administration and its frequency of administration of specific antimicrobial agents selectively act on bacterial cells through acting on specific receptors, metabolism or enzymes without affecting the metabolism of the host.
- The bacteriostatic and bactericidal action of antibiotics can be performed by the following major

Antimicrobial cont'd---

➤ Examples of this group of antibiotics are penicillins, cephalosporins, cycloserine, vancomycin and bacitracin.

↳ B. Damage to cytoplasmic membrane

➤ Amphotericin B and nystatin which are antifungal agents.

➤ Other group of antibiotics that have action on bacteria cell

membrane are polymyxin, gramicidin and tyrocidine.

↳ C. Inhibition of protein synthesis

➤ Examples are tetracyclines, streptomycin, puromycin, viomycin, kanamycin, gentamycin, and neomycin.

↳ D. Inhibition of nucleic acid synthesis

➤ Examples are mitomycin, actinomycin and griseofulvin

↳ E. Inhibition of specific enzyme systems

➤ Examples are sulphonamides and trimethoprim

Principles of anti microbial therapy

➤ The success of antimicrobial therapy depends upon

Antimicrobial cont'd---

indirectly, in the death or control of the infectious organism with minimal deleterious effect to the host.

➤ In order to achieve this aim the antimicrobial agent must have

➤ Activity against the organism at its site of infection and

➤ It must be administered in such a way as to maintain an effective inhibitory or lethal concentration to a given microbes at site of infection.

Basic requirements for successful microbial therapy

➤ For success full therapy of infected animals using antimicrobial drugs the veterinary clinician should consider the following parameters

1. Clinical diagnosis with the epidemiology of the disease

Antimicrobial cont'd---

- }\ The epidemiology of the area
- }\ The history of the animal from the owner
- }\ The result of full clinical diagnosis of the animal

➤ This helps us

- To identify the nature of infection that can be local or systemic
- To identify the severity of infection that can be per acute, acute , sub acute and chronic.

2. Microbes isolation and identification

- It starts with sample collection and followed by isolation and identification of microbes up to species level.

3. Antimicrobial sensitivity test

- Antimicrobial sensitivity testing is not required with all infections

Antimicrobial cont'd---

antimicrobial agents and in most cases these can be used for therapy.

➤ Sensitivity testing is generally reserved for members of those groups of organisms that show considerable variation in sensitivity to individual antimicrobial agents.

➤ So “*in vitro*” antibiotic sensitivity test used to determine whether the microorganism is sensitive or resistance to the antimicrobial agent at therapeutic dose.

➤ It is very important

- For selection of the best (effective) drug singly or in combination
- To reduce the emergence of drug resistance
- For cost reduction

The purpose of sensitivity testing is to determine if

Antimicrobial cont'd---

➤ In clinical terms, organisms are considered to be either sensitive or resistant to the action of an antimicrobial drugs.

Sensitivity test methods

➤ Sensitivity tests can be

 | 1. Quantitative

 | 2. Qualitative

1. Quantitative method

➤ Tube sensitivity tests, using serial dilutions of the antimicrobial drug

against a standard dose of the test organism is the quantitative method of antimicrobial sensitivity test.

➤ It provides quantitative information in terms of an exact minimum inhibition concentration (MIC) also called Minimum Bacteriostatic Concentration (MBsC).

➤ The MIC is the lowest antibiotic concentration that

Antimicrobial cont'd---

conditions of the test.

➤ Tube sensitivity testing is the gold standard because with most antibiotics, a mean plasma level 2 - 5 times the MIC needs to be sustained through the dosing interval for effective therapy.

1. The Minimum Inhibitory Concentration (MIC) of antimicrobials

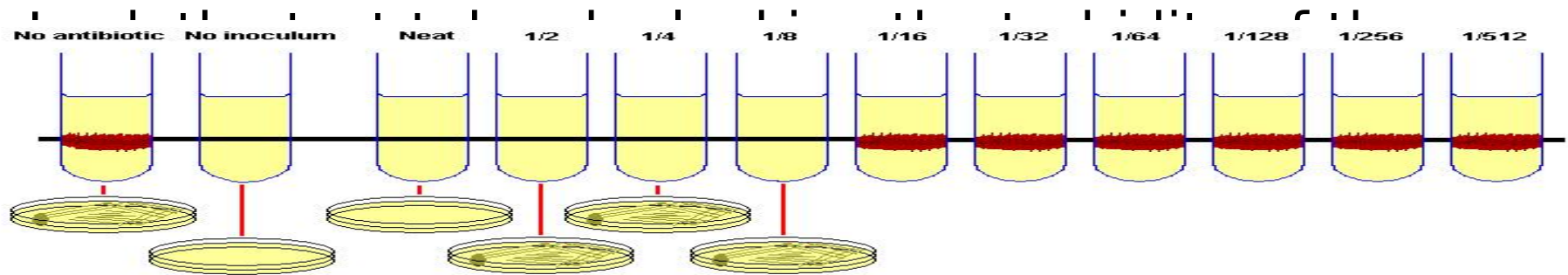
➤ **Minimum inhibitory concentrations (MICs) also called Minimum Bacteriostatic**

Concentrations (MBC) is defined as the lowest concentration of an antimicrobial agents that will inhibit the visible growth of a microorganism after overnight incubation.

➤ So the smallest amount of chemotherapeutic agent required to inhibit the growth of the bacteria "*in vitro*" is referred to as **Minimum Inhibitory**

Concentration (MIC) or also called **Minimum**

- Serially dilute the antibiotic with broth either by two or ten fold
- Add equal volume of testing bacteria in to test tubes.
 - ➡ You must have controls
 - } Inoculated broth media in test tube with out antibiotics
 - } Non - inoculated broth media with antibiotic
 - } Broth media in test tube with out bacterial culture and antibiotic
 - Mix it very well.
 - Incubate at 37 °C for 24 hours



Antimicrobial cont'd---

➤ The last vial (the higher dilution, the minimum concentration of antibiotic) in which no bacteria grow contains the antibiotic at the Minimal Inhibiting Concentration(MIC) is (1/8) in this case.

2.The Minimum Bactericidal Concentration (MBcC) of antimicrobials

➤ The Minimum Bactericidal Concentration (MBC) is the lowest concentration of antibiotic (higher dilution of antibiotics) required to kill a particular bacterium.

➤ So the smallest amount of chemotherapeutic agent required to kill the particular bacteria "*in vitro*" is referred to as Minimum Bactericidal Concentration(MBC).

Procedure

→ Prepare sterile broth media in test tubes

→ Serially dilute the antibiotic with broth either by two

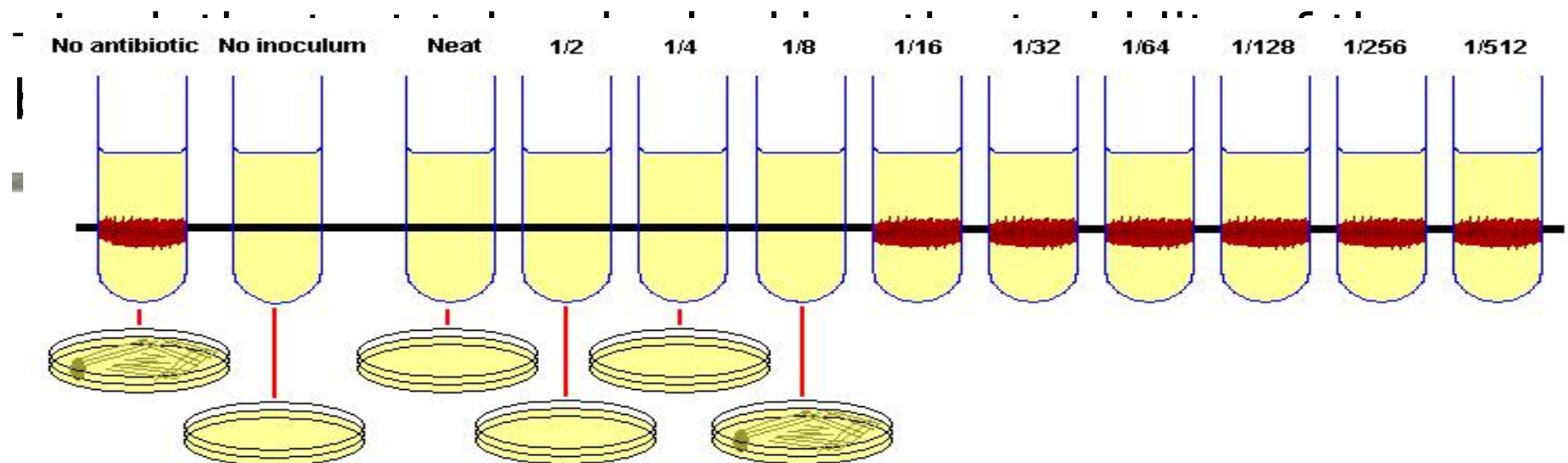
Antimicrobial cont'd---

➤ Prepare controls

- 1. Inoculated broth media in test tube with out antibiotic.
- 2. Non - inoculated broth media with antibiotic,
- 3. Broth media in test tube with out bacterial culture and antibiotic

→ Mix it very well.

→ Incubate at 37 °C for 24 hours



NB. Note that once the bacteria are removed from the drug they cannot grow on drug free medium.

2. Qualitative method

Disk diffusion or disk plate technique

- It is the most commonly used method to determine qualitative antibiotic sensitivity test “*in vitro*”.
- It is almost qualitative way of determination of antibiotic sensitivity test based on **diffusion**.
- The most common test medium is called **Mueller-Hinton Agar(MHA)** or its modification(**Isosensitest agar, Oxoid**).
- However there are exceptions of bacteria like *Streptococci* and *Actinomyces pyogenes* that do not grow on MHA.
- So use **5 - 10% sheep blood agar media** to test antibiotic sensitivity of the above bacteria.
- The principle of disk diffusion or disk plate technique is that small discs containing different antibiotics, or impregnated paper disks, are dropped in different

Antimicrobial cont'd---

bacteria can grow, the antibiotic will diffuse in the area surrounding each tablet, and a disc of bacterial lysis will become visible.

➤ The area around the disc where there is no growth of bacteria is called **zone of inhibition**.

Procedure

Preparation of MHA

→ Prepare MHA from the dehydrated medium according to the manufacturer's instructions.

NB. The media should be prepared using distilled water which is free from Ca^{2+} and Mg^{2+} ions.

→ Heat with frequent agitation and boil to dissolve the medium completely.

→ Sterilize by autoclaving at 121°C for 15 min.

→ Check the PH of each preparation after it is sterilized, which should be between 7.2 and 7.4 at room temperature.

distilled water or by allowing a little amount of medium to gel around a PH meter electrode.

PH meter



- Cool the agar medium to 45 -50°C in water bath.
- Pour the agar medium into sterile Petri dish on a flat surface by giving a uniform depth.
- Allow to solidify at room temp

MI



Mueller Hinton Agar Plate

Antimicrobial cont'd---

→Prior to use, dry plates at 30 - 37°C in an incubator, with lids partly on a gar, for not more than 30 minutes or until excess surface moisture has evaporated.

NB. Media must be moist but free of water droplets on the surface.

→Because presence of water droplets may result to swarming bacterial growth, which could give inaccurate results and they are also easily contaminated.



in the incubator

Antimicrobial cont'd---

**MHA stored in refrigerator
inside airtight plastic bags at 2-8°**



**Transferring of the test bacteria to broth or
saline solution**

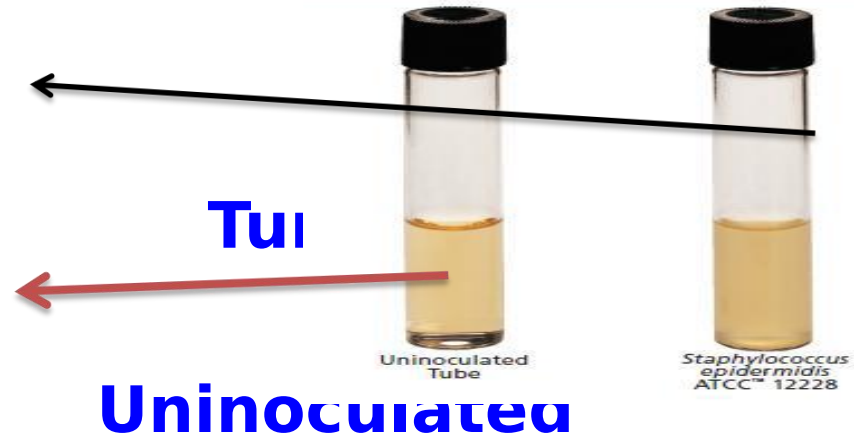
Method one

- Take 4 - 5 colonies of isolated bacteria from non - selective media.
- Transfer these colonies to a tube containing 4 - 5 ml of **soybean casein digest broth (Trypcase Soy Broth)** or to 4 - 5 ml of **tryptone soya broth**.
- Incubate the broth at 35 - 37°C or at an optimum growth temperature until it achieves or exceeds the turbidity of **0.5 McFarland turbidity standard**.
- Compare the turbidity of the test bacterial suspension with that of **0.5 McFarland** (vigorously

Antimicrobial cont'd---

background with contrasting black line under adequate light.

Soybean casein digest (Trypcase Soy Broth)



soya broth

Unino



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Antimicrobial cont'd---

Method two

✿ The colonies are selected as above and a suspension is made in buffered saline or broth with out the pre - incubation in broth.

✿ This method is suggested as the best method for testing staphylococci, especially with suspected **methicillin - resistant strains**.

NB. Before you inoculate the bacterial suspension to MHA, you must determine turbidity of the grown bacteria.

Adjusting the turbidity of bacterial suspension

✿ The turbidity of both of the pre incubated broth(method one) and the suspension of bacteria in broth or saline with out pre incubation(method two) is adjusted by comparison with a 0.5 McFarland turbidity standard.

Preparation of 0.5 McFarland turbidity standard solution

1. Preparation of solution A(0.048M BaCl_2)

Take 1.175 gm of $\text{BaCl}_2 \cdot 2\text{H}_2\text{O}$

Antimicrobial cont'd---

- Take 1 ml of H_2SO_4 with specific gravity of 1.84
- Make up to 100ml with distilled water.

Preparation of standard 0.5 McFarland turbidity solution

- Take 0.5 ml of solution A
- Then add 99.5 ml of solution B
- Shake vigorously and dispense into 4 - 6 ml sealed tubes or screw - capped vials.
- Store in the dark at room temperature.
- Compare the turbidity of the test bacterial suspension with that of **0.5 McFarland** (vigorously shaken before use) against a white background with contrasting black line under adequate light.



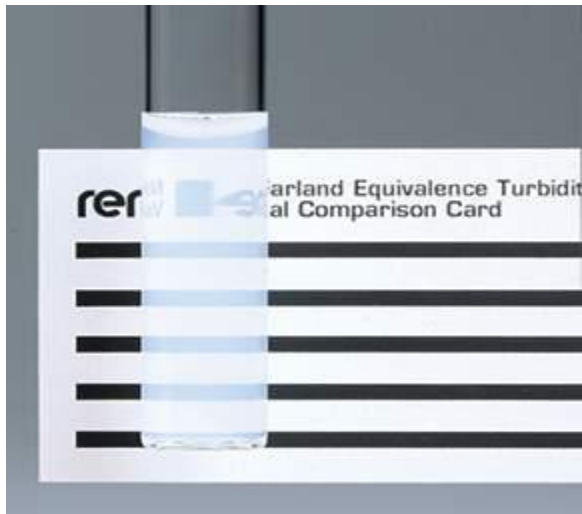
McFarland turbidity solution

Antimicrobial cont'd---

Standard 0.5 McFarland turbidity solution is also available commercially with its back ground



Comparison of the turbidity of broth culture with standard 0.5 McFarland turbidity



Antimicrobial cont'd---

Inoculation of the bacterial suspension to MHA

- Use sterile cotton swab to transfer the bacteria suspension to MHA
- Dip a sterile cotton swab into the standardized bacterial suspension.
- Remove excess inoculum by lightly pressing the swab against the tube wall at a level above that of the liquid.
- Inoculate the agar by streaking with the swab containing the inoculum.
- Rotate the plate by 60° and repeat the rubbing procedure.
- Repeat two times. This will ensure an even distribution of the inoculum
- Allow the surface of the agar to dry for 3 - 5 minutes but not longer than 5 minutes to allow for absorption of excess moisture



Mueller Hinton Agar Plate

Antimicrobial cont'd---

Sterile swab



Bunsen burner



Transferring of bacterial suspension using st



Antimicrobial cont'd---

Method of inoculation of the MHA by streaking with the swab containing the inoculum.



Dispensing of anti microbial discs on MHA

- The number of antimicrobial agents to be tested should be limited.
- To make the test practical and relevant, include only one representative of each group of related drugs those that have veterinary use to control or prevent disease.

Antimicrobial cont'd---

- Disks should be properly stored in a tightly sealed container with desiccant at 2 - 8°C.
- Expired disks should not be used

Different antibiotic discs



NB. All procedures in antibiotic sensitivity test must be done aseptically.

- Then invert the plates and placed in the incubator at 35 °C with in 15 minutes of applying the discs and incubate aerobically for 16 -

Antimicrobial cont'd---

Single antibiotic disc dispenser Multi antibiotic

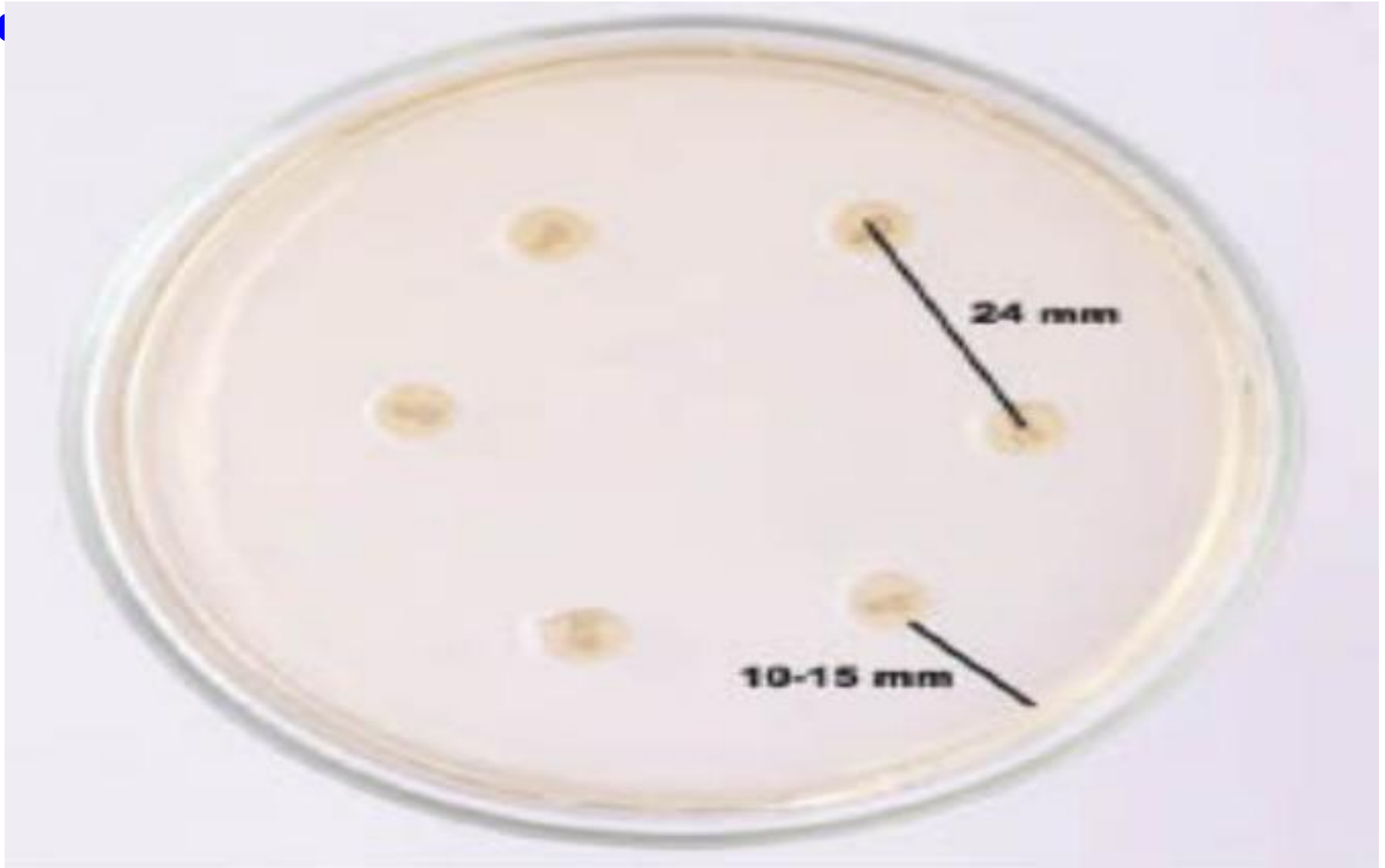


Thumb forceps



Antimicrobial cont'd---

The correct position of discs in 90 mm of Petri



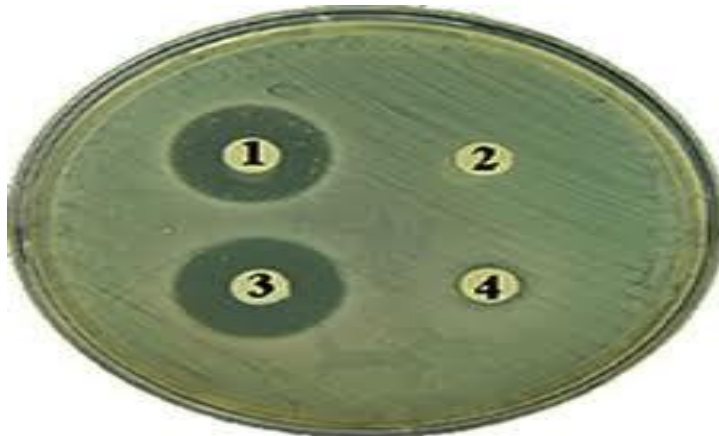
Antimicrobial cont'd---

18hours(24 hours for staphylococci).

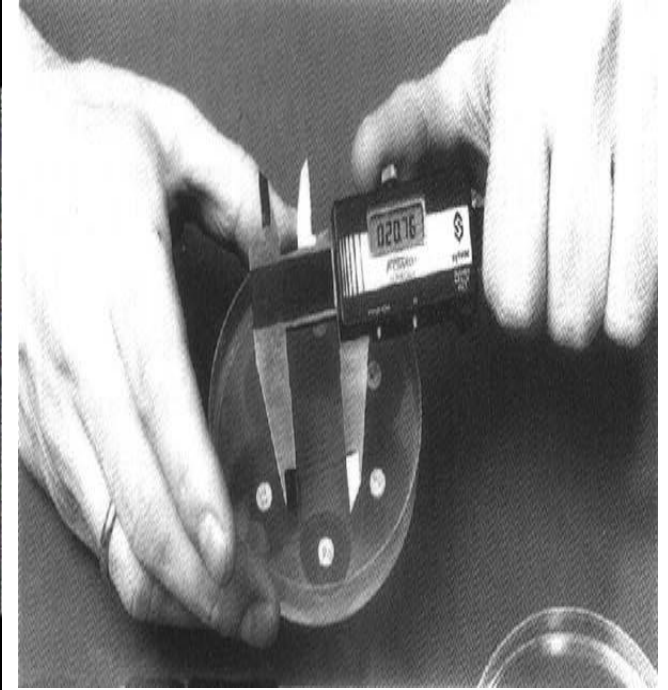
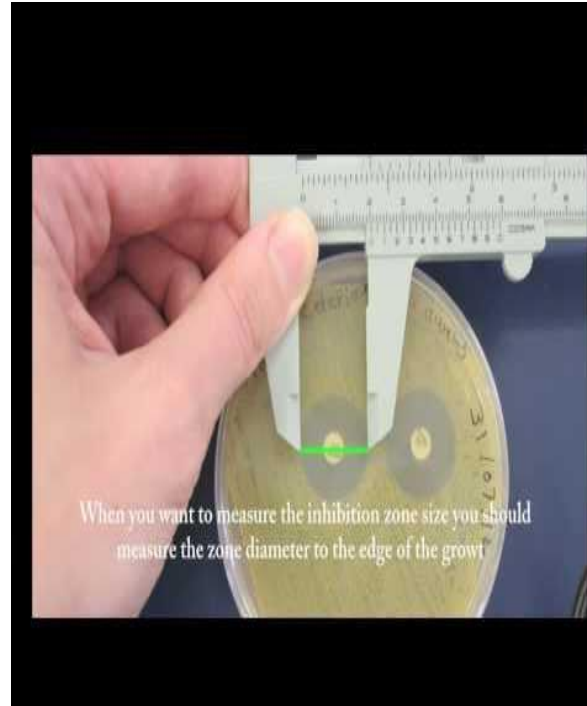
→After incubation, the diameters of the zones of inhibition are measured to the nearest mm using ruler or caliper across the center of the discs.

Result

- If the bacteria is sensitive to the given antibiotics there will be inhibition of bacterial growth around the discs with different diameter.
- This area around the disk where there is no growth of bacteria is **zone of inhibition of bacterial growth**.



Measuring zone of inhibition by ruler or caliper



Interpretation of results

- An interpretation of the size of the zones of inhibition is made by comparing the diameter of the zone of inhibition of the test isolates with those in the chart of interpretative standard for veterinary pathogens which is indicated below.
- Depending on the result, the bacterium is reported as

Antimicrobial cont'd---

- 1. Susceptible
- 2. Moderately susceptible
- 3. Intermediate
- 4. Resistant

1. Susceptible: the infection may respond to the treatment at the normal dosage.

2. Moderately susceptible: the pathogen may be inhibited by attainable concentrations of antimicrobial agent by providing a higher dosage.

3. Intermediate: the result is equivocal and if the bacterium is not fully susceptible to an alternative drug, then the test should be repeated.

4. Resistant: the bacterium is not inhibited by the usually achievable systemic concentrations of antimicrobial agent and efficacy has not been reliable in clinical studies.

Zone size interpretation chart modified by National Committee for clinical Laboratory Standards (NCCLS in 1996)

APPENDIX 2.2. Zone diameter interpretative standard for veterinary pathogens.

Antimicrobial Agent	Disk Content	Zone Diameter (mm)			
		S	I	F	R
Amikacin*	30µg	≥ 17	15-16		≤ 14
Gentamicin*	10µg	≥ 15	13-14		≤ 12
Kanamycin*	30µg	≥ 18	14-17		≤ 13
Spectinomycin	100µg	≥ 14	11-13		≤ 10
Amoxicillin-clavulanic acid*					
Staphylococci	20/10µg	≥ 20	-		≤ 19
Other organisms	20/10µg	≥ 18	14-17		≤ 13
Ticarcillin-clavulanic acid*					
<i>Pseudomonas aeruginosa</i>	75/10µg	≥ 15	-		≤ 14
Gram(-)enteric organisms	75/10µg	≥ 20	15-19		≤ 14
Ampicillin*					
Enterobacteriaceae	10µg	≥ 17	14-16		≤ 13
Staphylococci	10µg	≥ 29	-		≤ 28
Enterococci	10µg	≥ 17	-		≤ 16
Streptococci (not <i>S. pneumoniae</i>)	10µg	≥ 26	19-25		≤ 18
Oxacillin*					
Staphylococci	1 µg	≥ 13	11-12		≤ 10
Penicillin*					
Staphylococci	10 units	≥ 29	-		≤ 28
Enterococci	10 units	≥ 15	-		≤ 14
<i>S. pneumoniae</i>	1µg oxacillin	≥ 20	-		-
Streptococci (not <i>S. pneumoniae</i>)	10 units	≥ 28	20-27		≤ 19
Ticarcillin*					
<i>Pseudomonas aeruginosa</i>	75µg	≥ 15	-		≤ 14
Gram (-) enteric organisms	75µg	≥ 20	15-19		≤ 14
Penicillin-novobiocin	10 units/30 µg	≥ 18	15-17		≤ 14
Imipenem*	10µg	≥ 16	14-15		≤ 13
Cephalothin*	30µg	≥ 18	15-17		≤ 14
Cefazolin*	30µg	≥ 18	15-17		≤ 14
Ceftiofur	30µg	≥ 21	18-20		≤ 17
Enrofloxacin (canine/feline)	5µg	≥ 23	-	17-22	≤ 16
Enrofloxacin (chickens/turkeys)	5µg	≥ 23	17-22		≤ 16
Enrofloxacin (bovine)	5µg	≥ 21	17- 20		≤ 16
Difloxacin	10µg	≥ 21	18- 20		≤ 17
Orbifloxacin	10µg	≥ 28	-	18-22	≤ 17

APPENDIX 2.2. Continuation

Antimicrobial Agent	Disk Content	Zone Diameter (mm)			
		S	I	F	R
Clindamycin	2µg	≥ 21	15-20		≤ 14
Pirlimycin	2µg	≥ 13	-		≤ 12
Erythromycin*					
Streptococci	15µg	≥ 21	16-20		≤ 15
Organisms other than Streptococci	15µg	≥ 23	14-22		≤ 13
Tilmicosin (Bovine)	15µg	≥ 14	11-13		≤ 10
Tilmicosin (Swine)	15µg	≥ 11			≤ 10
Chloramphenicol*					
Streptococci (not <i>S. pneumoniae</i>)	30µg	≥ 21	18-20		≤ 17
<i>S. pneumoniae</i>	30µg	≥ 21	-		≤ 20
Organisms other than Streptococci	30µg	≥ 18	13-17		≤ 12
Florfenicol	30µg	≥ 19	15- 18		≤ 14
Tiamulin	30µg	≥ 9	-		≤ 8
Trimethoprim-sulfamethoxazole*					
<i>Streptococcus pneumoniae</i>	1.25/23.75µg	≥ 19	16-18		≤ 15
Organisms other than <i>S. pneumoniae</i>	1.25/23.75µg	≥ 16	11-15		≤ 10
Rifampin*					
<i>Streptococcus pneumoniae</i>	5	≥ 19	17-18		≤ 16
Organisms other than Streptococci	5	≥ 20	17-19		≤ 16
Sulfisoxazole*	250 or 300	≥ 17	13- 16		≤ 12
Tetracycline*					
Streptococci	30	≥ 23	19-22		≤ 18
Organisms other than Streptococci	30	≥ 19	15-18		≤ 14
Vancomycin*					
Enterococci	30	≥ 17	15-16		≤ 14
Streptococci	30	≥ 17	-		-
Other gram-positive organisms	30	≥ 12	10-11		≤ 9

* human data taken from M100-S12 supplements to M2 and M7

S Susceptible

I Intermediate

R Resistant

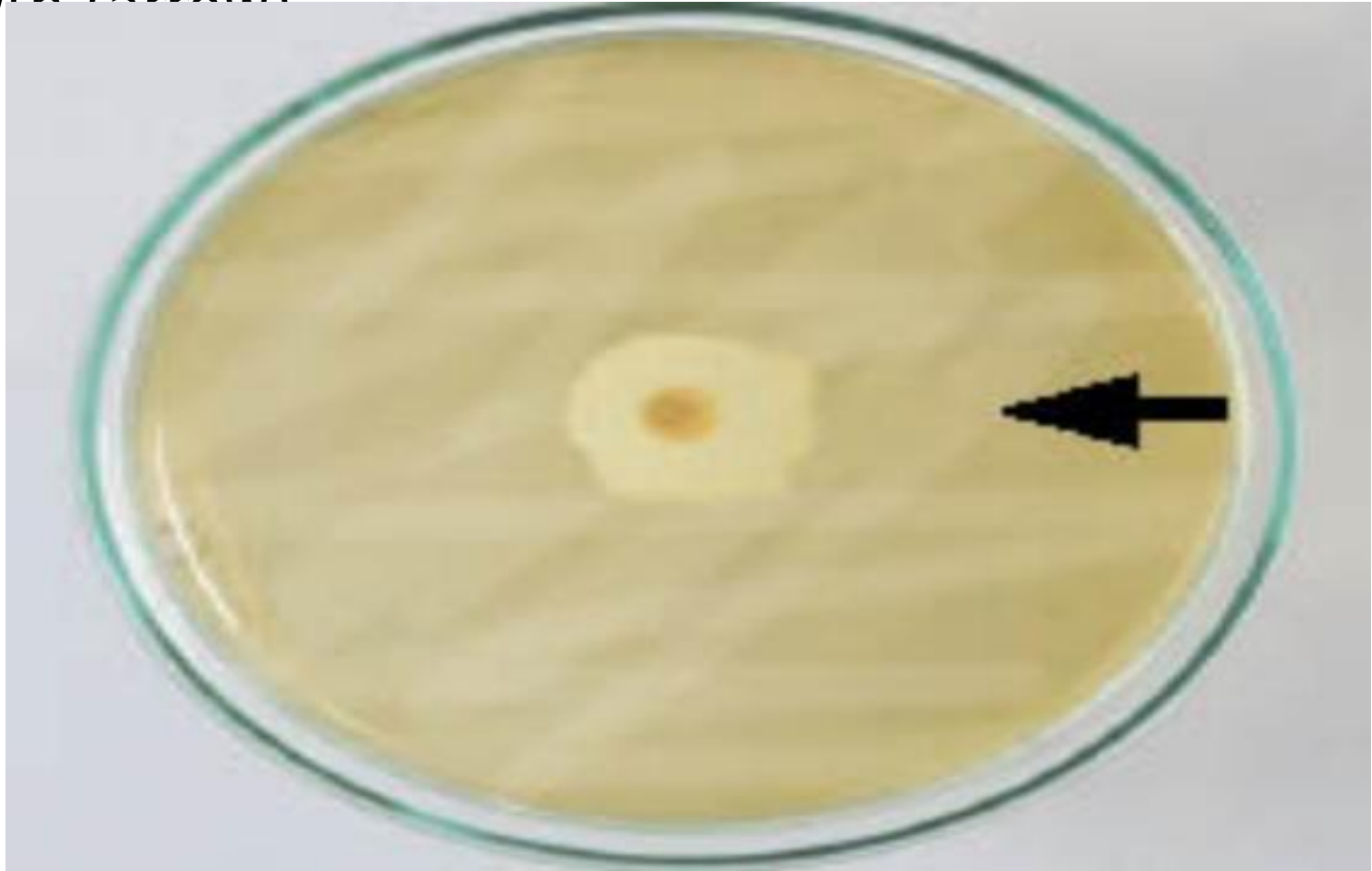
F Flexible; should be considered susceptible if appropriate dosing modifications specified in the packaging insert are applied

NOTE: Reproduced with permission; from NCCLS publication M31-A2-Performance Standards for Antimicrobial Disk and Dilution Susceptibility Test for Bacteria Isolated from Animals: Approved Standard-Second Edition (ISBN 1-56238-461-9). Copies of the current edition may be obtained from NCCLS, 940 West Valley Road, Suite 1400, Wayne, Pennsylvania 19087-1898, USA.

Antimicrobial cont'd---

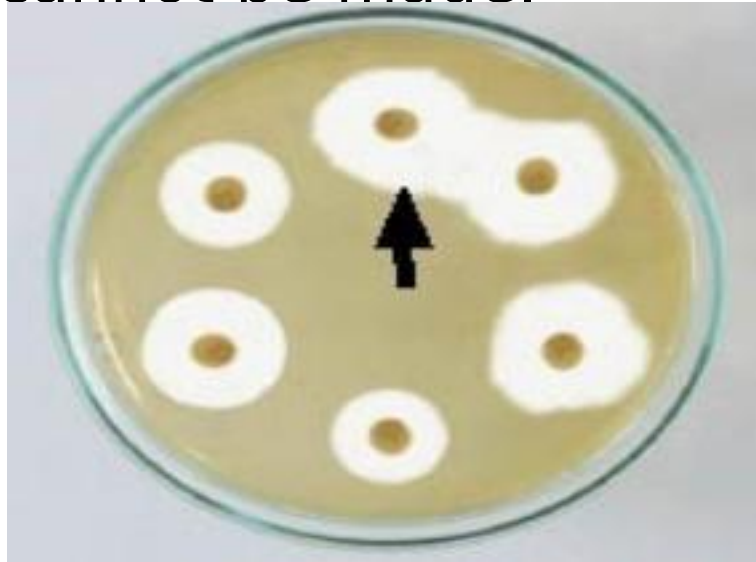
Rejection criteria

1. Do not read plates on which growth of test bacteria have isolated colonies or less than semi-confluent growth (arrow)



Antimicrobial cont'd---

2. Do not read zones of inhibition of two adjacent disks that overlap (arrow) to the extent that measurement of the zone diameter cannot be made.



3. Do not read zones showing distortion from circular (arrow).

Rational selection of antimicrobial drugs

- The success of antimicrobial therapy depends upon maintaining a drug concentration that will result, directly or indirectly in the death or control of the infecting organism with minimal side effects on the animal.
- Theoretically, the following steps should be considered before selecting antimicrobial drugs for treatment.

1. Antimicrobial effect of the drug

➤ **Bacteriostatic versus bactericidal**

- Most of the time we use **bacteriostatic drugs** as bacteriostatic drugs inhibit the multiplication of bacteria so that the host's immune system clears the microbes from the body.
- However bactericidal drugs are used when
 - ↳ The immune system of the animal is compromised

2. Spectrum of action

➤ **Narrow versus broad spectrum**

→ We use narrow spectrum drugs when the specific infectious agent is identified and its antibiotic sensitivity test is done.

→ But we must use broad spectrum antibiotics if

 | The diagnosis is doubtful

 | There is a mixed infection

3. Pharmacokinetic properties of the drug

➤ It is the possible residual effects of drugs in the milk or meat.

4. Potential toxicity of the drug

➤ Some drugs are highly toxic to some species animals.

→ Example most tetracycline groups for equines.

5. Cost

Antimicrobial cont'd---

- Cost of the drugs

- Cost of the milk that is discarded during treatment

6. Drug with drawl period

- This is the period in which antimicrobials or their residues stay at higher level in tissues. Or

- This is the time required by the animal body to clear antimicrobials and their residues to undetectable concentration.

- As the result consumption of the flesh or milk of the treated animal is not recommended for human consumption.

Practical use of antimicrobial drugs

- When you are using effective antibiotics that are selected by the above selective criteria, you must also consider

- 1. Antimicrobial dosage

1. Antimicrobial dosage

- When ever you want to treat animals with antimicrobial drugs, you must use the **recommended dose**.
- **Recommended dose** is the dose that will give the concentration of antimicrobial drugs in the blood and tissues in effective concentration to inhibit or to kill the microbes with minimal side effect to the host cells.

2. Routes of administration

- The commonly used routes of administration of antimicrobials in farm animals include:

- 1. Intravenous injection
- 2. Intramuscular injection
- 3. Subcutaneous injection
- 4. Intrapertioneal injection
- 5. Oral administration

| 6. Other routes of administration

1. Intravenous injection

➤ Intravenously administration of antibiotics is very important to attain high concentration and immediate action of antibiotics in the blood and tissue levels.

➤ This route should be used in

| The treatment of septicemia and

| Other life-threatening diseases

| In chronic infections such as *Corynebacteria pneumonia* in foals, where high diffusion concentrations are required in order to penetrate the abscess areas and the capsular material of the organism.

| It is also preferred to the intramuscular route in racehorses where

there is a need to avoid muscular soreness.

| The need to avoid muscle damage in beef cattle close to marketing may also dictate intravenous

Antimicrobial cont'd---

- The concentrations obtained are much higher than those obtained with equivalent doses of the same drug given intramuscularly or orally.
- Consequently greater diffusion concentrations are achieved at sites of infection.
- Administration by this route is not without its disadvantage.
- Because: -
 - Acute toxic reactions either to the drug or to its vehicle are more common.
 - Perivascular reactions and intravascular thrombosis are a hazard
- To reduce the above problems the following conditions must be fulfilled
 - Drugs specifically formulated for intravenous

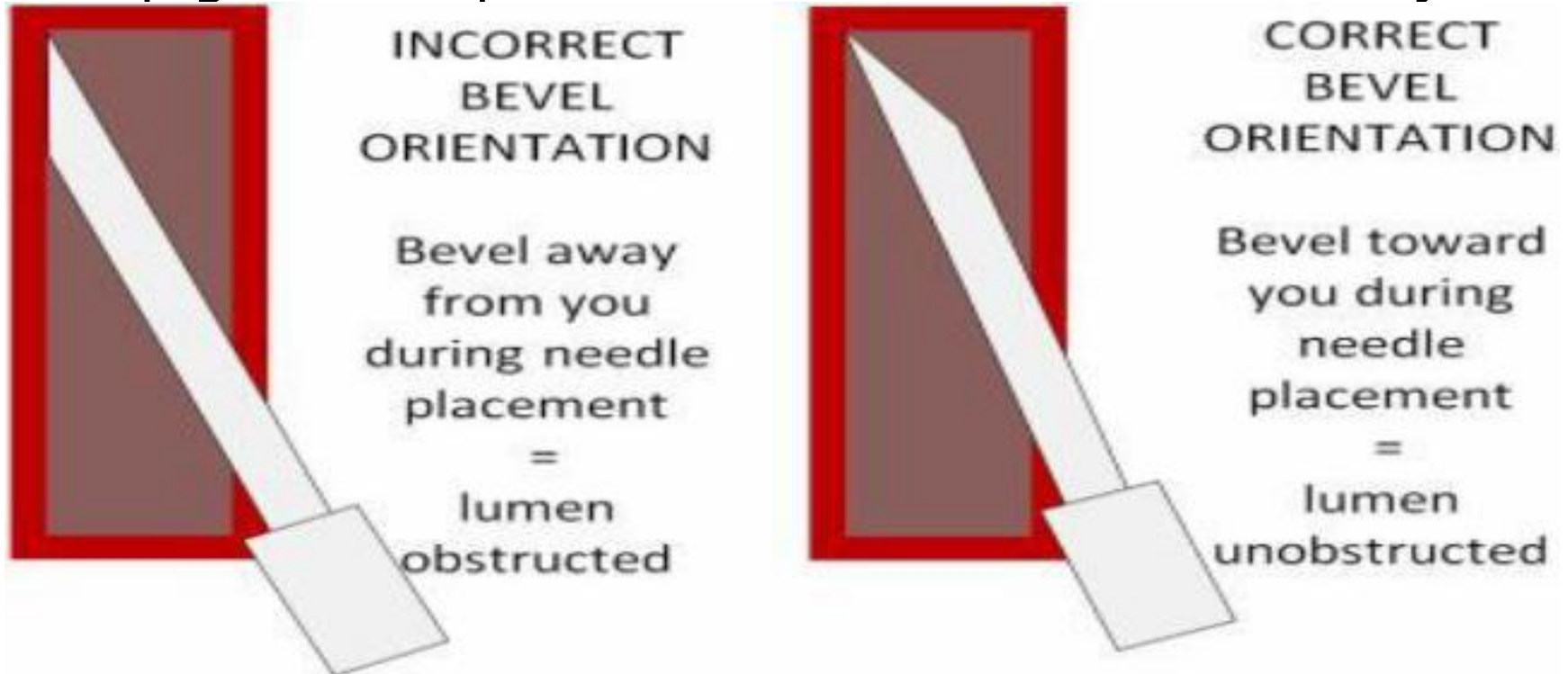
Antimicrobial cont'd---

of this route for any preparation should be followed.

NB. Injections should be given slowly and not as a bolus.

➤ The jugular vein is used in all species except the pig and dogs.

➤ In pigs the superficial veins of ear is commonly used.



Common IV Injection Sites in domestic animals

Species	Sites of injection
Sheep, goat, cow and horse	Jagular vein
Dog, cat and rabbit	Cephalic vein
Pig and rabbit	Marginal ear vein
Birds	Ulnar(wing) vein

Site of IV injection in horses



Site of IV injection in horses



Site of IV injection in cattle



Site of IV injection in cattle



Site of IV injection in goats and sheep

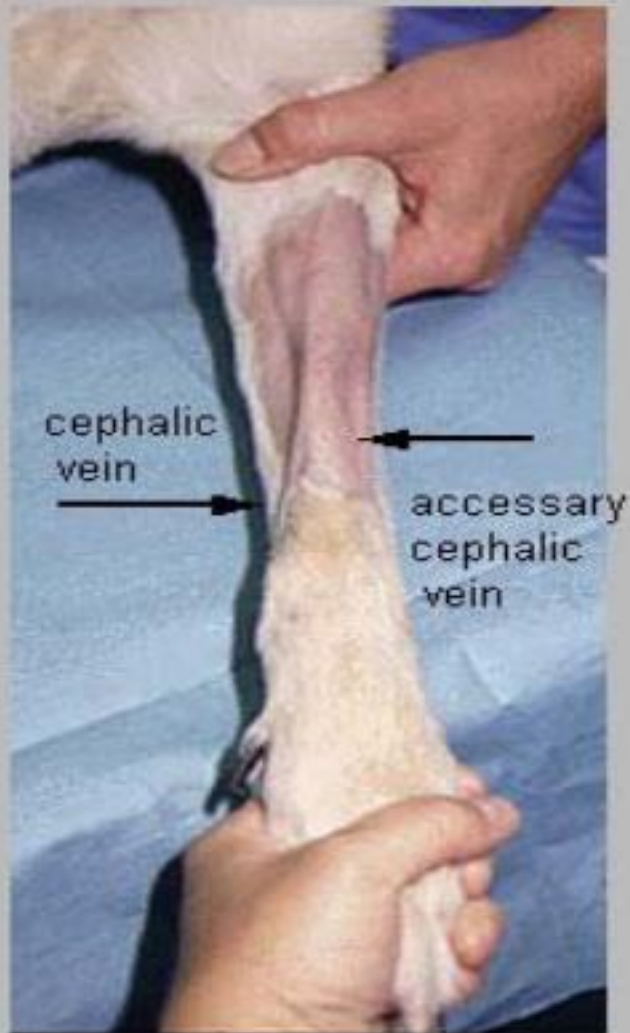




IV injection in pigs and rabbit (marginal ear vein)



IV injection in dogs(cephalic vein)



posterior-
medial

anterior-
lateral

cephalic vein

accessary
cephalic vein



IV injection in birds through ulnar(wing) vein



2. Intramuscular injection

➤ Intramuscular injection is the most commonly used method for antimicrobial administration in large animals.

➤ The most common sites are

→ Muscles of the neck

→ Gluteal muscles

→ Pictorial muscles

Sites of IM administration in large animals



Hypodermic needle and syringe for animals



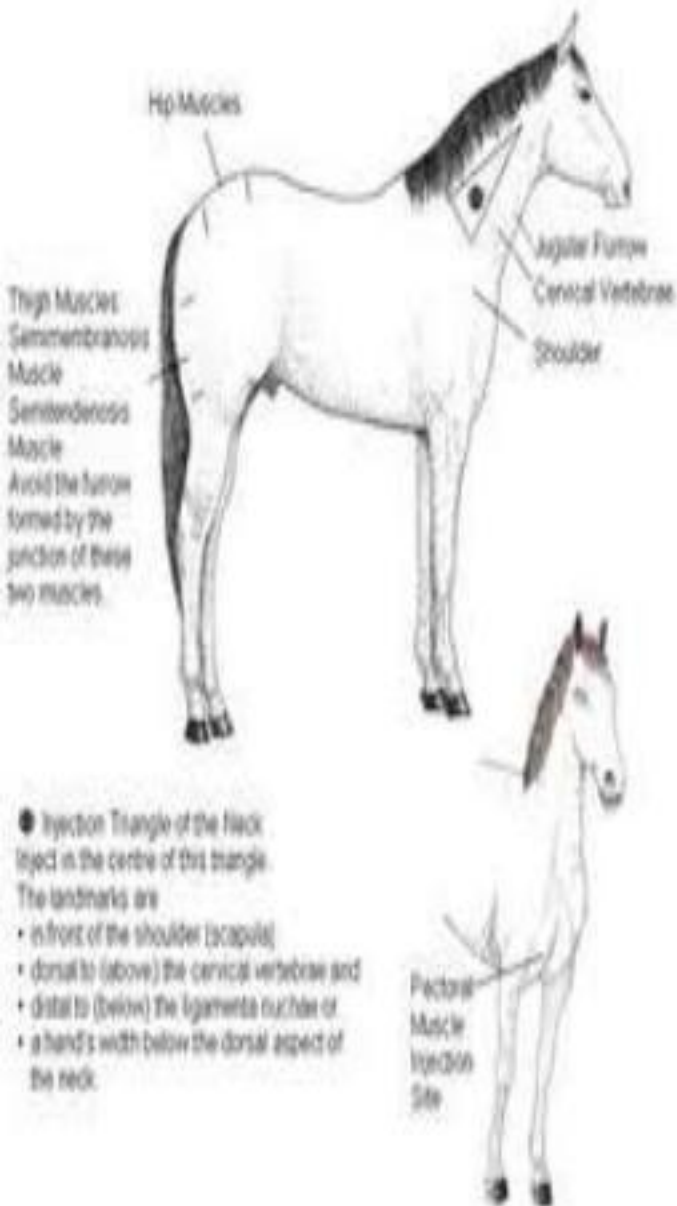
Hypodermic needle for small animals



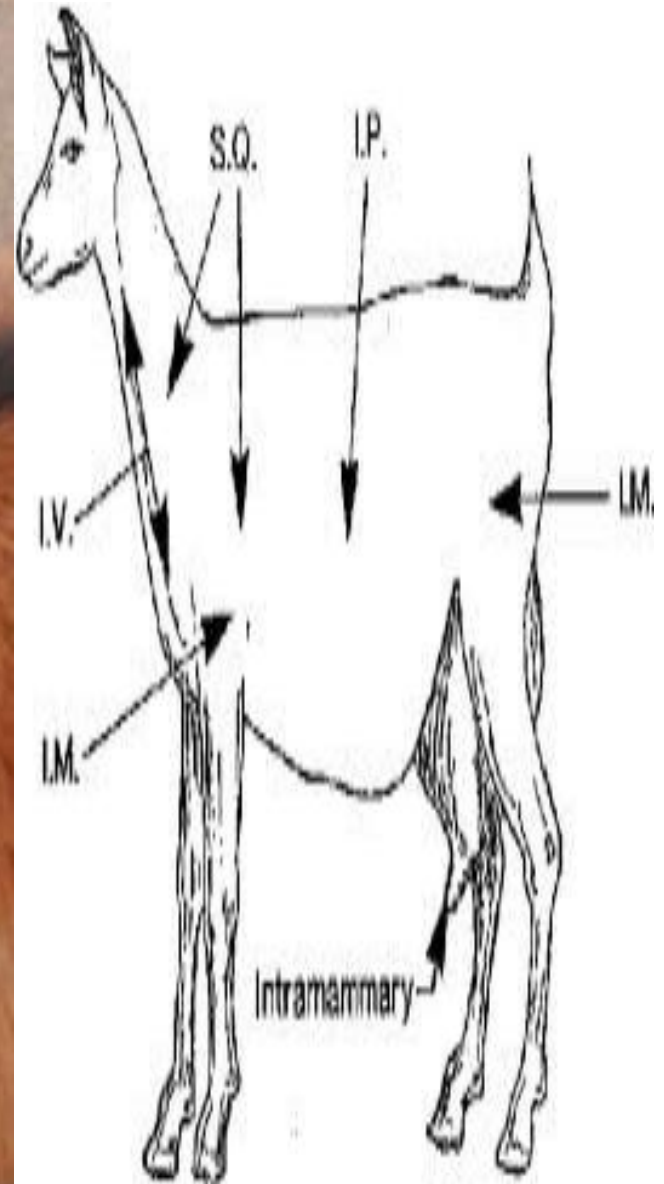
IM injection sites in equines



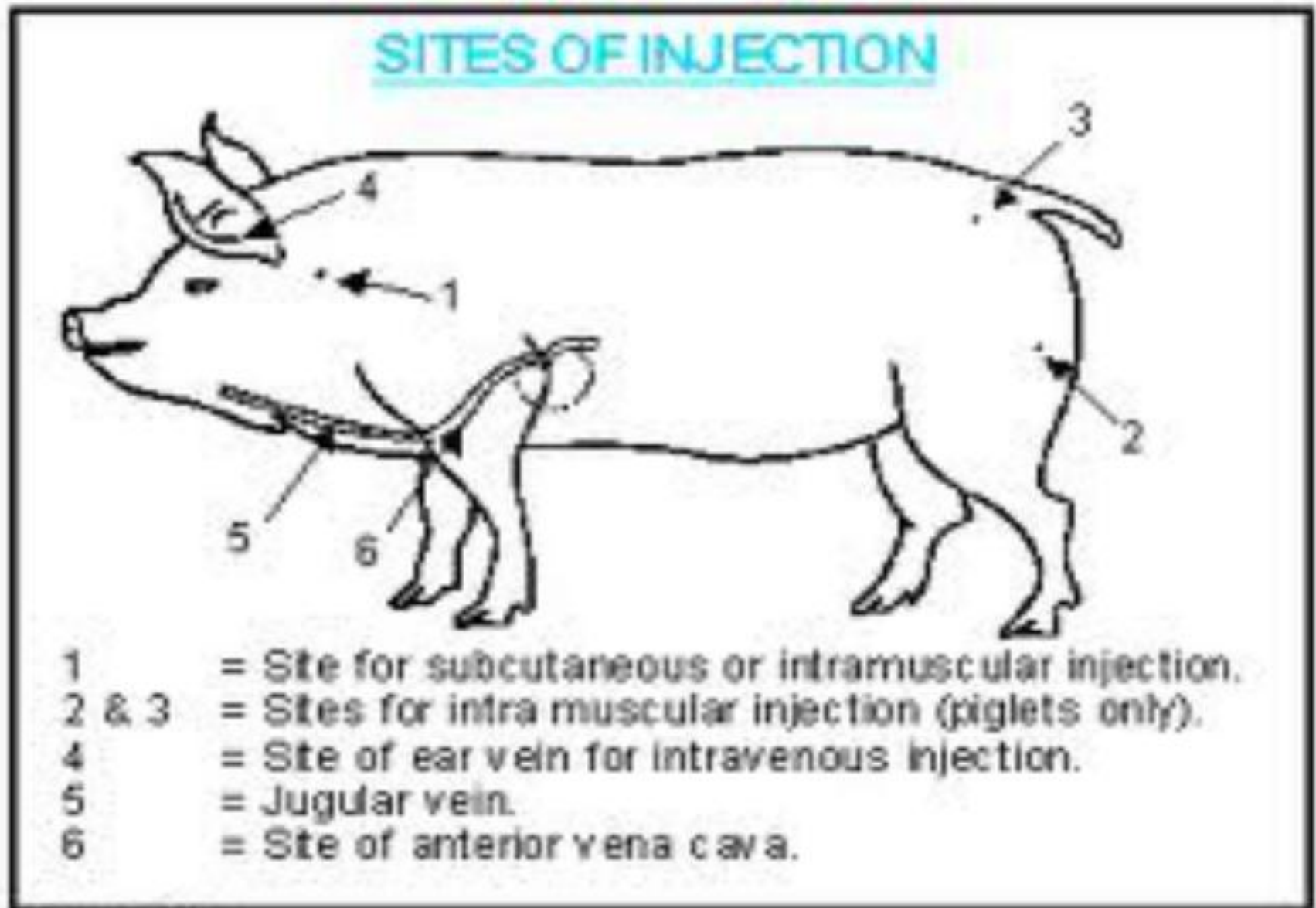
Injection cont'd---



IM injection sites in sheep and goat

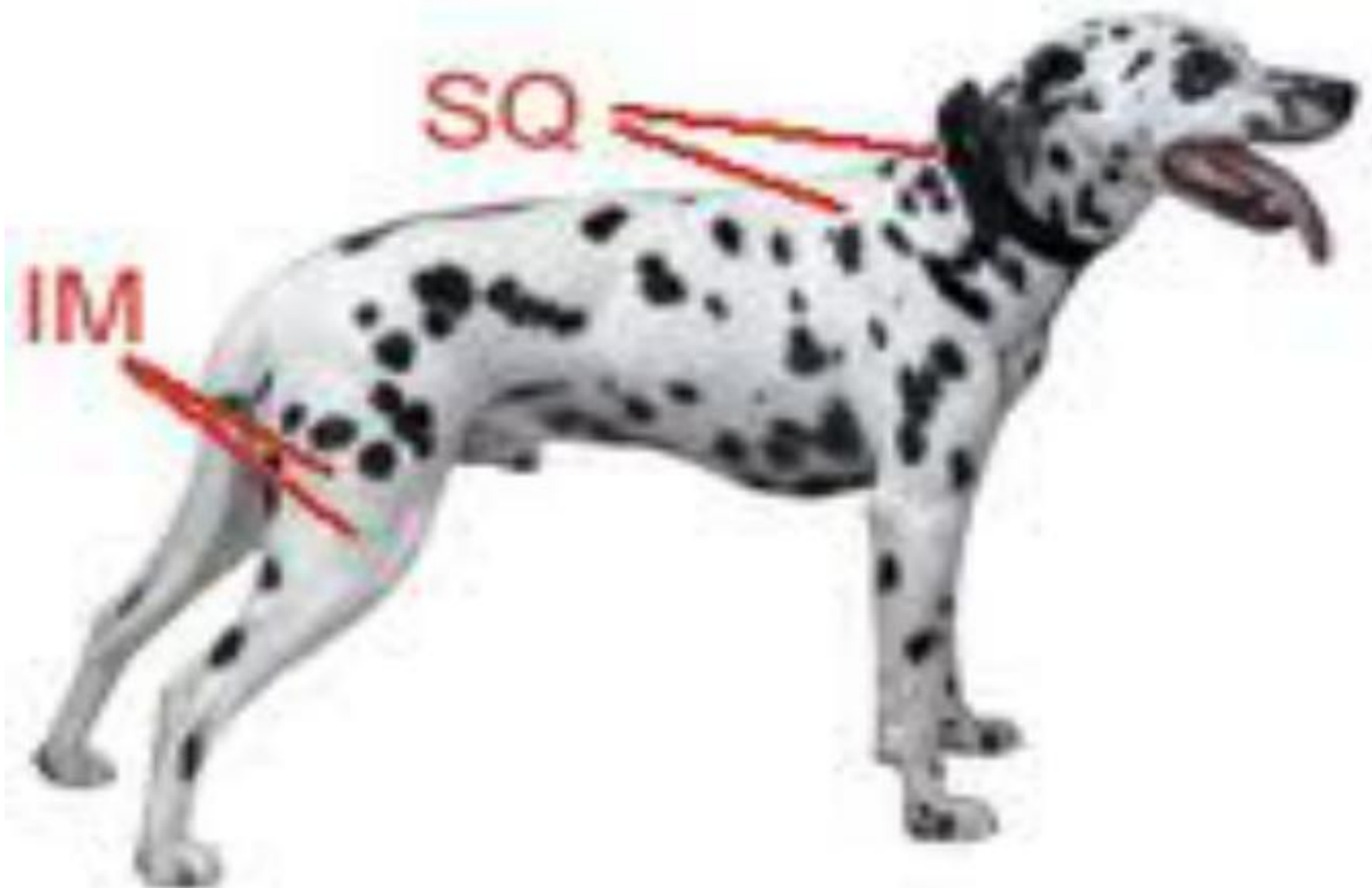


IM site of injections for pigs and dogs



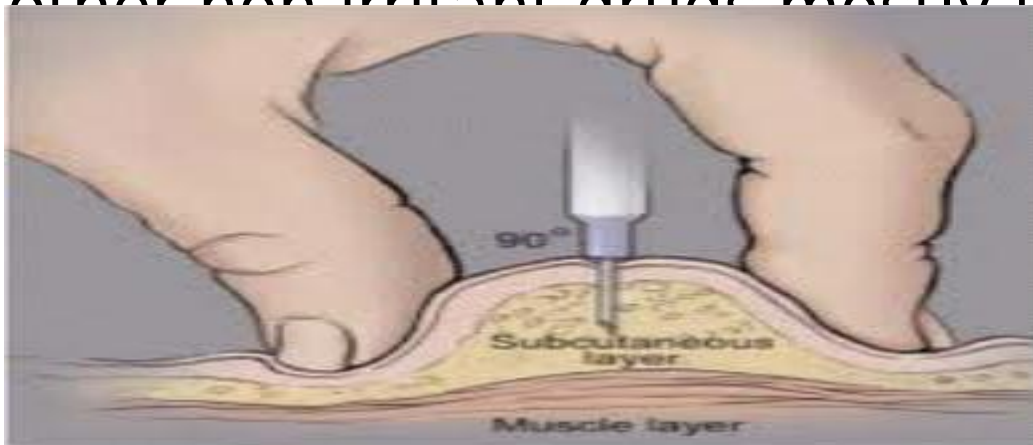
(Fig.4-4b)

Site of IM injection in dogs



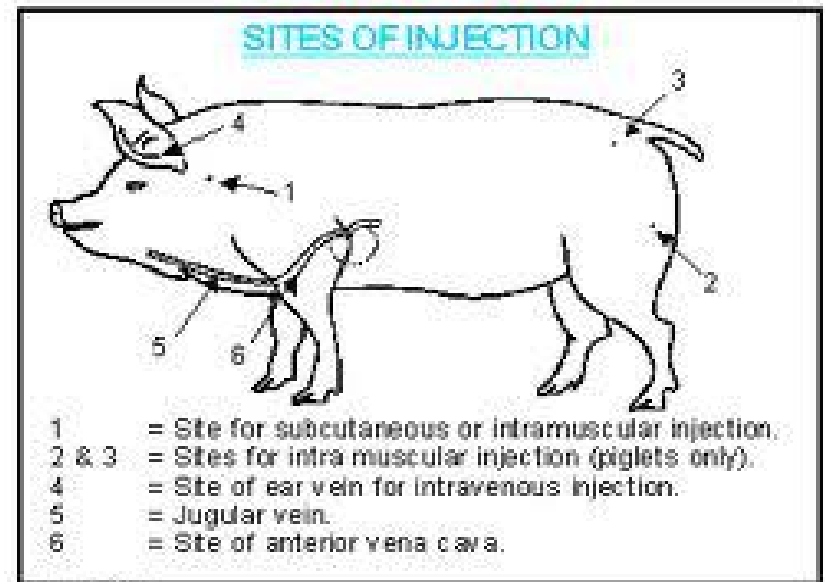
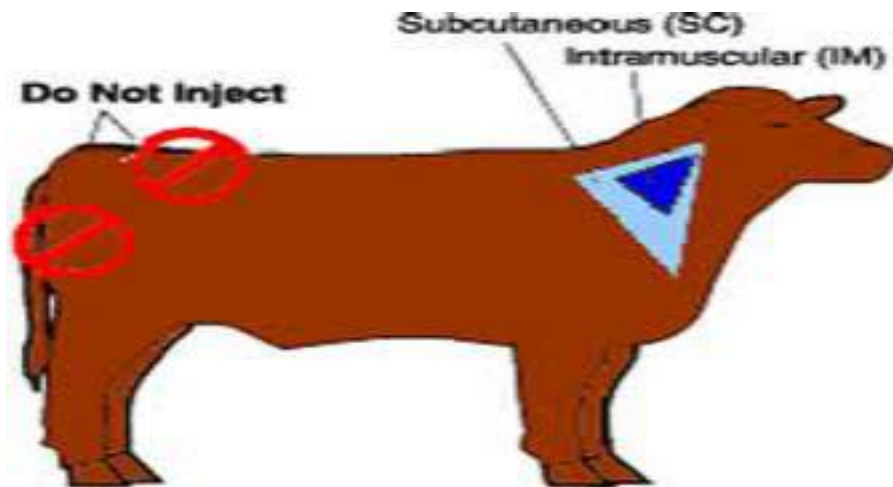
3. Subcutaneous injection

- Subcutaneous injection has not been commonly used in large -animal practice.
- Providing the drug is not deposited in a fat depot, this route provides a reasonable alternative to intramuscular injection.
- With irritant preparations there is a danger of excessive reaction and the occurrence of sterile abscesses.
- This route of administration is used to administer other non irritant drugs mostly used for vaccine

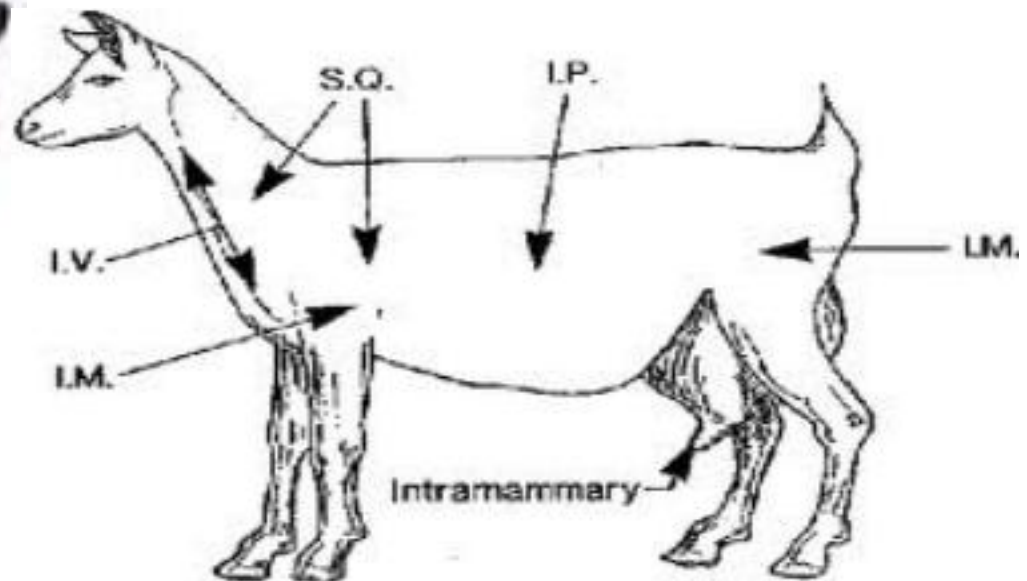
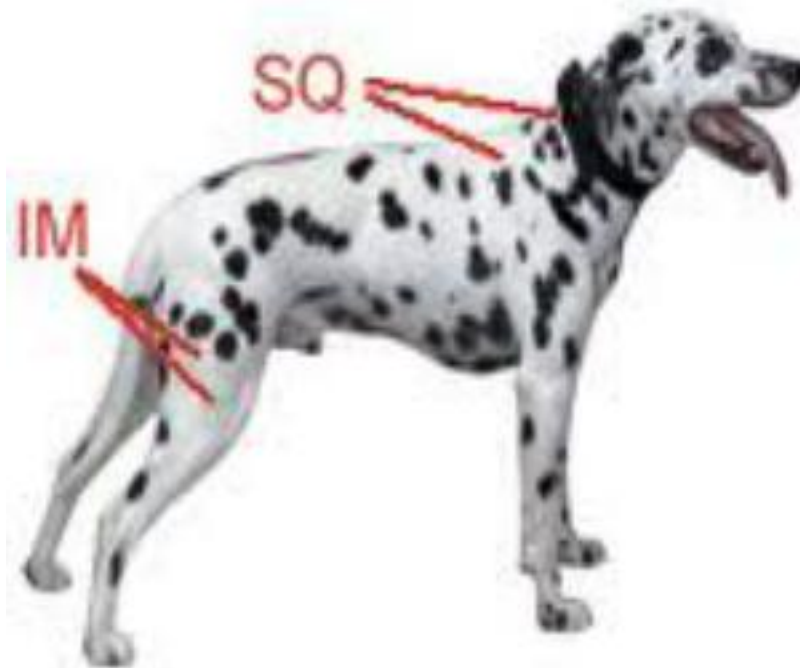


njection

Subcutaneous injection sites for different species of animals



(Fig.4-4b)



Injection cont'd---



IM injection site breast muscle and wing muscle

SC injection site dorsally at neck

4. Intrapertitoneal injection

- Intrapertitoneal injection is occasionally used for antimicrobial administration.
 - It is used due to the following reasons
 - Specially in cattle close to market , and where intravenous administration by various reasons may be impractical.
 - It is also occasionally used in pigs with diarrhea, where the antibacterial drug is combined with fluids for rehydration.
 - Animals with peritonitis are also occasionally additionally treated by this route of injection.
- NB.** An aseptic injection technique should be used during intrapertitoneal injection.

Injection sites

- In cattle the injection is given in the right flank midway between the last rib and the tuber coxae and at

Injection cont'd---

➤ In horses with peritonitis the peritoneal cavity can be drained through a canula inserted in the ventral midline as used for abdominal paracentesis.

5. Oral administration

There are two methods of oral administration of antibiotics

- └ Water medication

- └ Animal feed medication specially with concentrates

- It is less frequently used route of administration of antibiotics in pre ruminant, in young foals and pigs.

- Because

- └ There is a need of **large dose of antibiotics (2 - 5 times)** as compared the other parenteral routes of administration.

- └ The absorption of anti biotics also depends on the **function of GIT** tract like the presence or absence of

Oral administration of antibiotics



Injection cont'd---

orally administered drug.

→For example, oxytetracycline and trimethoprim have a much lower bioavailability to calves when administered in milk, rather than in

water, because of the high degree of binding to milk.

➤Generally the oral route is the easiest method for administration, and where the cost of revisits is a significant consideration.

➤ In general, however, systemic infections are better treated by **parenteral injection** and certainly treatment should be initiated by this route.

➤The oral route is the one of choice for the treatment of **enteric infections**.

➤The oral administration of antibiotics is used mostly for prophylaxis many infectious diseases in intensive farms with prophylactic dose.

Injection cont'd---

➤ Commonly a yeast, *Staphylococcus* or *Pseudomonas aeruginosa* are involved.

What is the reason?

6. Other routes of administration

➤ Other routes of administration may be used to increase the level of antibacterial drug in areas where diffusion following parenteral administration of the drug may be limited and when high local levels are required.

➤ It includes

- ↳ Intraarticular infusion
- ↳ Intrapleural infusion
- ↳ Subconjunctival infusion
- ↳ Intramammary infusion
- ↳ Intratracheal infusion

Injection cont'd---

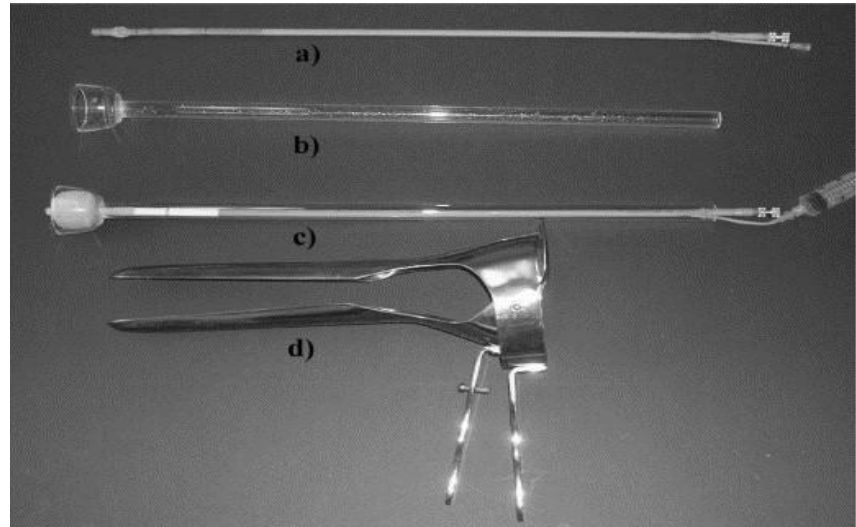
└ Intrauterine infusion

- Non-irritant preparations should be used with strict aseptic technique.
- In most cases these treatments should be supported by parenteral treatment.

Intramammary infusion antibiotic suspension



Intrauterine catheter



Part 3. Diseases of newborn animals

- This chapter considers the principles of the diseases that occur during the first month of life in animals born alive at term.
- The new born animals require special attention because
 - 1. Their immunological incompetence
 - 2. Their dependence on adequate colostrum containing adequate antibodies at the right time
 - 3. Their dependence on frequent intake of readily available carbohydrate to maintain energy
 - 4. Their relative inefficiency in maintaining normal body temperature during its upwards or downwards.
- That why in neonates there is much greater susceptibility to infectious diseases, dehydration and death.
- To solve them diagnosis and treatment must be

New born cont'd---

in order to maintain homeostasis.

➤ Generally the diseases of new born animals can be classified as

- 1. Fetal diseases
- 2. Parturient diseases
- 3. Post natal diseases

1. Fetal diseases

➤ These are diseases of the fetus during intrauterine life, e.g. congenital defects (intrauterine growth retardation and neonatal neoplasia); prolonged gestation, intrauterine infections, abortion, fetal death with resorption or mummification or maceration.

2. Parturient diseases

➤ These are diseases associated with dystocia, causing cerebral anoxia or fetal hypoxemia, and their

3. Postnatal diseases

➤ They are the diseases of newborn farm animals that occurs after birth.

➤ These are divided into

 \ 1. Early postnatal diseases

 \ 2. Delayed post natal diseases

 \ 3. Late post natal diseases

1. Early postnatal diseases

➤ Diseases of neonates that occur within 48 hours of birth

➤ Deaths that occur during this period are unlikely to be caused by an **infectious disease** unless it has been acquired congenitally.

➤ Most diseases occurring in this period are **noninfectious and metabolic** e.g. hypoglycemia and hypothermia due to poor mothering, hypothermia

New born cont'd---

- Infectious diseases are often initiated during this period.
- But most manifest clinically at a later age because of their incubation period.
- However some e.g. **navel infection, septicemic disease** and **enterotoxigenic colibacillosis**, have a short enough incubation and they may occur during this period.

2. Delayed postnatal diseases

- It is the disease of neonates at the age of 2 - 7 days.
- It may occur due to
 - Desertion by mother
 - Mammary incompetence resulting in starvation
- The above reasons increased susceptibility of neonates to different

New born cont'd---

➤ All these predispose the neonates to different infectious diseases.

➤ Examples include **colibacillosis, joint ill, lamb dysentery, septicemic disease**, most of the viral enteric infections in young animals, e.g. **rotavirus** and **corona virus**.

3. Late postnatal diseases

➤ They are the diseases of neonates that occur with **in 1- 4 weeks of age** .

➤ There is still some influence of **hypogammaglobulinemia**, with late onset **enteric diseases** and the development and severity of **respiratory disease** in this period.

➤ But other diseases not directly associated with failure of transfer of

3.1. Neonatal infections

- Infection is a common cause of morbidity and mortality in neonates.
- There are a number of specific infectious pathogens that can cause disease.
- Other infectious agents, normally considered to have low virulence, can also cause disease if the immunological status of the neonate
- is not at an optimum level.
- Maternal immunoglobulins are not transferred transplacentally in ungulates(hooved animals) and the newborns are at particular risk for infectious disease during the neonatal period.
- Because they rely on the acquisition of immunoglobulins from colostrum for passive antibody protection.

Etiology

Some of the common infectious agents which cause neonatal infection

Species of animals	Causative agents of infection
Calves	1. Bacteremia and septicemia associated with <i>Escherichia coli</i>, <i>Listeria monocytogenes</i>, <i>Pasteurella</i> spp. <i>Streptococcus</i> or <i>Salmonella</i> spp. 2. Enteritis associated with enterotoxigenic <i>E. coli</i>, <i>Salmonella</i> spp“ rotavirus and corona virus, <i>Cryptosporidium parvum</i> and <i>Clostridium perfringens</i> types A, B and C; and occasionally by the virus of infectious bovine rhinotrachitis and bovine virus diarrhea
Piglets	1. Septicemia with or without localization in joints, endocardium and meninges associated with <i>Streptococcus suis</i>, <i>Streptococcus equismilis</i>, <i>Streptococcus necridermicus</i> and <i>L.</i>

Table cont'd---

Species of animals	Causative agents of infection
Piglets	3. Enteritis associated with <i>C. perfringens</i>, <i>Campylobacter</i> spp., rotavirus and <i>Coccidia</i> spp. 4. Arthritis and septicemia associated with <i>Erysipelothrix rhusiopathiae</i>.
Foals	1. Septicemia with localization associated with <i>E. coli</i>, <i>Actinobacillus equi</i>, <i>Klebsiella pneumoniae</i>, α-hemolytic streptococci, <i>S. zooepidermicus</i>, <i>L. monocytogenes</i>, <i>Rhodococcus equi</i> and <i>Salmonella typhimurium</i> 2. Enteritis associated with <i>C. perfringens</i> types A, B, and C, <i>Clostridium difficile</i>, <i>R. equi</i>, <i>Salmonella</i> spp., <i>Strongyloides westeri</i>, <i>C. parvum</i> and rotavirus.
Lambs	1. Septicemia or bacteremia with localization in joints and/or synoviae and/or leptomeninges associated with <i>E.</i>

Table cont'd---

Species of animals	Causative agents of infection
Lambs	3. Lamb dysentery associated with <i>C. perfringens</i> type B and C 4. Gas gangrene of the navel associated with <i>Clostridium septicum</i>, <i>Clostridium novyi</i> and <i>Clostridium chauvoei</i> 5. Pyemia associated with <i>Staphylococcus aureus</i>, <i>Fusobacterium necrophorum</i> and <i>Arcanobacterium pyogenes</i> 6. Pneumonia, polyserositis and peritonitis associated with <i>Pasteurella multocida</i> and <i>Mannheimia haemolytica</i>.

New born cont'd---

Epidemiology

- The occurrence of neonatal disease is broadly influenced by two main factors:
 - They are
 - The exposure or infection pressure of the infectious agent to the neonate
 - The ability of the neonate to modulate the infection so that disease does not occur.
 - With some agents the organism is sufficiently virulent in its own right that an exposure can lead to disease.
 - With others, the majority, the defenses of the host must be compromised or the infection challenge must be very high before clinical disease occurs.
 - Management of the neonate has a great influence on both

these factors and the recognition and correction of

New born cont'd---

key to the prevention of neonatal disease in both the individual and the group.

➤ In epidemiology of neonatal infectious diseases we must consider

- ┌ 1. Source of infection
- ┌ 2. Routes of transmission
- ┌ 3. Risk factors associated with neonatal infections

1. Source of infection

Source of infection for neonates can be

- ┌ 1. Prenatal infection
- ┌ 2. Post natal infection

1. Prenatal infection

➤ Infection of fetus during its intrauterine development.

➤ Prenatal infection is mostly associated with abortion,

New born cont'd---

2. Post natal infection

- The majority of infections are acquired by the neonate after birth from
 - The enteric or respiratory tract flora of the dam and/or other animals including others infected neonates.
 - The environment
- Depending upon the specific agent, the source infection may be carrier animals or in the environment.

2. Routes of transmission

- The portal of infection is commonly by
 - ↳ Ingestion
 - ↳ Aerosol
 - ↳ Via the umbilicus
 - ↳ Trauma

New born cont'd---

- The organisms capable of to invade through respiratory and intestinal epithelium to produce a bacteremia and septicemia.

3. Risk factors associated with neonatal infections

- Occurrence of neonatal infections generally depend on the following conditions.

- | 1. Level of immunity and immune response
- | 2. Exposure pressure
- | 3. Age of exposure
- | 4. Virulence of the agent
- | 5. Animal risk factors

1. Level of immunity and immune response

- All newborn farm animals are more susceptible to infection than their adult counterparts.

New born cont'd---

of immunoglobulins and possess almost no resistance to certain diseases until after they have ingested colostrum and absorbed sufficient quantities of immunoglobulins from the colostrum.

➤ That is why failure of transfer of colostral immunoglobulins is a major determinant for occurrence of neonatal infection.

➤ All components of the immune system are present in foals and calves at birth but the immune system of the newborn animal is less mature than its adult counterpart, at least for the first 30 days of life, and does not respond as effectively to many antigens.

2. Exposure pressure

➤ The exposure pressure is a factor of the cleanliness of the environment of the neonate.

3. Age of exposure

New born cont'd---

- Examples are the importance of age with respect to susceptibility to disease associated with some enteric infections.
- Disease associated with enteropathogenic *E. coli* and with *C. perfringens* type B and C.

4. Virulence of the agent

- There are many infectious agent that are very virulent to the neonates. Example *E. coli* and *Cl. perfringens* beta toxin etc.

5. Animal risk factors

- Animal risk factors that predispose infection include those that interfere with sucking drive and colostral intake, such as cold stress and dystocia.

Pathogenesis

- After neonatal infection the pattern of disease development may take one or both of the following

New born cont'd---

- Bacteremia followed by septicemia with severe systemic signs and/ or
- Bacteremia with few or no systemic signs, followed by localization in various organs.
- If the portal of entry is the navel, local inflammation occurs -'navel ill.
- Localization of infectious agents may occur in different parts of the body depending on the affinity the animal tissues.
- The common sites of local infection includes
 - Joints(producing a suppurative or non suppurative arthritis).
 - Eye to produce a panophthalmitis
 - In the heart valves to cause valvular endocarditis
 - In the meninges to produce a meningitis

New born cont'd---

- Bladder (cystitis)
- Liver(hepatitis) and many other sites
- Dehydration, acid-base and electrolyte imbalance can occur very quickly in newborn animals, whether diarrhea and vomiting (pigs) are present or not.
- But obviously are more severe where there is fluid loss into the gastrointestinal tract

Clinical signs

- Clinical findings of neonatal infections depend on
 - The virulence of specific infectious agent
 - The age of neonates
 - The immune status of the neonates
- With organisms that have a low propensity for toxemia there is fever, depression, anorexia and signs referable to

New born cont'd---

These include

- Endocarditis with a heart murmur
- Panophthalmitis with pus in the anterior chamber of the eye
- Meningitis with rigidity, pain and convulsions and
- Polyarthrititis with lameness and swollen of joints
- With more virulent organisms there are clinical signs of toxemia as well as bacteremia, including
 - Fever
 - Severe depression
 - Prostration, coma and petechiation of mucosae
 - Dehydration, acidosis and rapid death

Diagnosis

- The principles of diagnosis of infectious disease in newborn animals are the same as for older animals.

New born cont'd---

- However, in outbreaks of a suspected infectious disease in young animals there is usually a need for more diagnostic microbiology and pathology
- The following methods are used in combination for the diagnosis of neonatal infection.
 - **1. Epidemiological**
 - Here consider
 - } Host
 - } Agent
 - } Environment
 - 2. Clinical**
 - 3. Pathological(Necropsy) findings**
 - All the above methods of diagnosis give the tentative diagnosis of the disease.

New born cont'd---

- Confirmatory diagnose is the laboratory method of diagnosis by isolating the positive agent the disease.

Treatment

- Treatment of neonatal infection should be done after isolation , identification and performing antibiotic sensitivity test.
- However it is not always practically possible.
- So that treatment of neonatal infection can be done
 - By using broad spectrum antibiotics
 - By blood or serum transfusion to affected neonate (IV in dose 2ml/ kg of body weight)
 - By using hyper immune serum
 - By using supportive treatment when ever there is dehydration, acidosis and electrolyte in balance.

3.2. Omphalitis, omphalophlebits and omphaloarteritis (Navel ill)

New born cont'd---

- Commonly in new born farm animals and appears common in calves.

- └ Anatomically the umbilical cord consists of

- └ The amniotic membrane

- └ The umbilical veins

- └ The umbilical arteries

- └ The urachus

- Normally the umbilical cord dries up with in a week after birth.

- Infection of the umbilicus occurs soon after birth.

- This may results in

- └ 1. Omphalitis

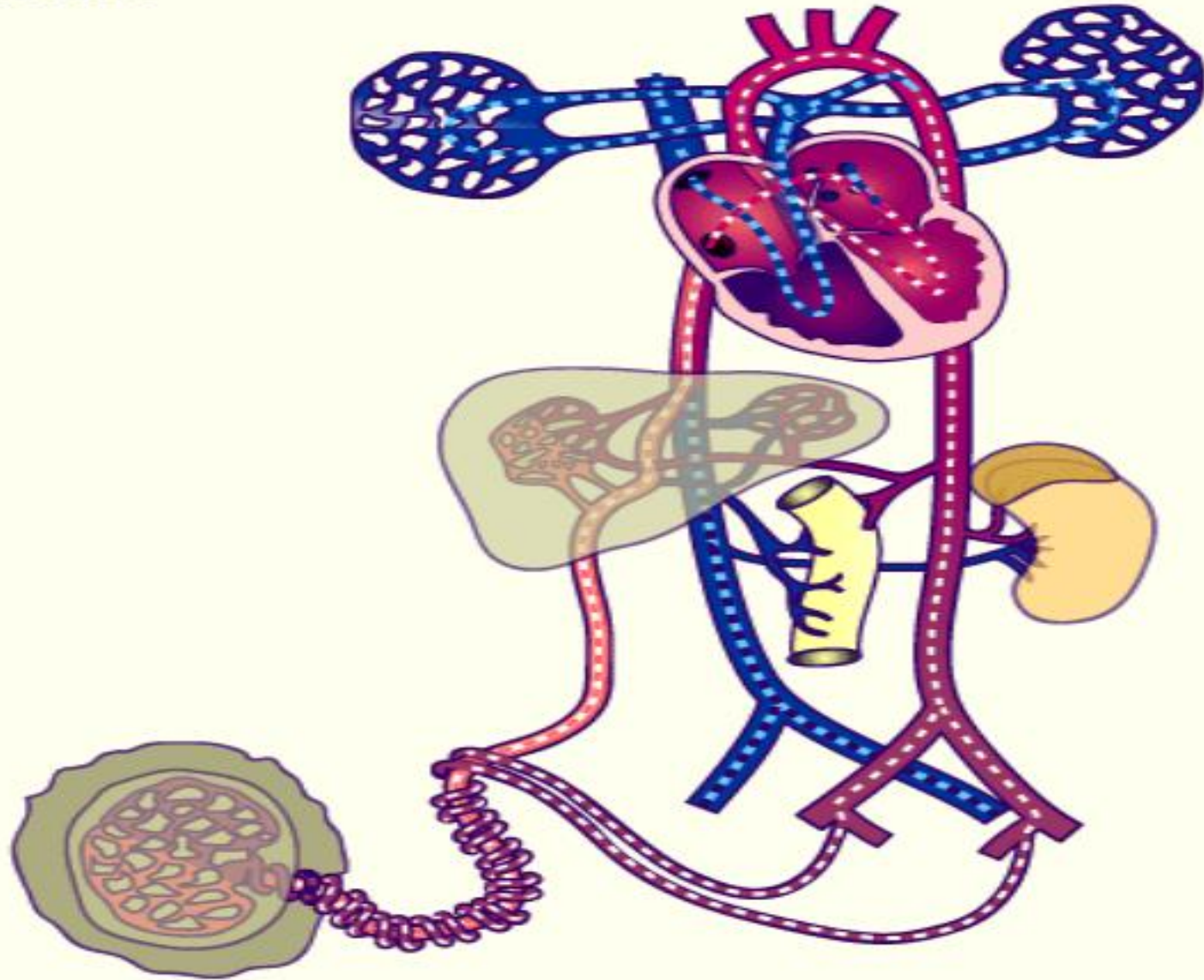
- └ 2. Omhalophlebitis

- └ 3. Omphaloarteritis

- └ 4. Infection of the urachus with possible extension of

Blood circulation in fetus

before birth



New born cont'd---

causing cystitis.

1. Omphalitis

- Infection of the umbilical cord stump in the neonatal newborn period. Or
- It is inflammation of the external aspects of the umbilicus and occurs in calves with in 2 -5 days after birth.

2. Omhalophlebitis

- It is the inflammation of the umbilical veins.
- It affects calves in 1 – 3 months of age and show unthrifty because of chronic toxemia.

3. Omphaloarteritis

- It is the inflammation of the umbilicus arteries.
- It is less common than omhalophlebitis.

Omphalitis in calves



Etiology

- The disease occurs if the umbilicus is not well disinfected after birth.
- The common mixed bacterial flora identified in navel ill includes
 - ↳ *E. coli*
 - ↳ *Proteus* species
 - ↳ *Staphylococcus* species
 - ↳ *Actinomyces pyogenes*

Pathogenesis

- If the umbilicus is not disinfected with appropriate disinfectant, the bacteria may enter to the umbilicus and may cause infection.
- Anaerobic bacteria like *C. tetani* may proliferate and cause lock jaw.

Omphalolithiasis may cause liver abscess as it has

New born cont'd---

- Complication may results in infection of urachus results in cystitis.

Clinical signs

- Clinical signs of navel – ill can be
 - ↳ Depression and anorexia
 - ↳ Unthriftness
 - ↳ The umbilicus is enlarged , pain full on palpation with purulent discharge
 - ↳ Fever in the cases of chronic toxemia

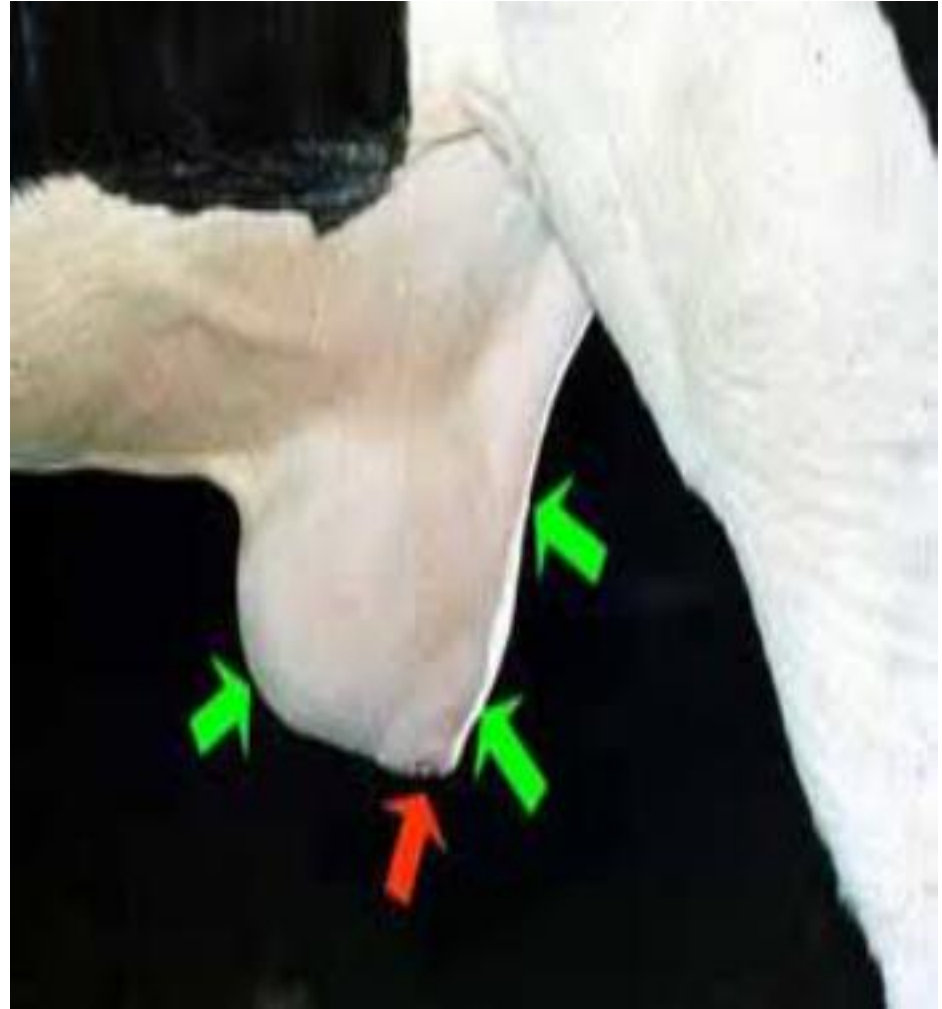
Diagnosis

- Diagnosis is based on history and clinical signs that you observe on animals.

Differential diagnosis

- You must differentiate from umbilical hernia.

Umbilicus hernia in calves



New born cont'd---

Treatment and control

- Surgical excision and removal of the abscess and dressing it with appropriate antiseptic.
- Hepatic abscess can be treated by using lipophilic antibiotics like rifampin florfenicol and drainage of abscess to exterior if it is possible.
- Use broad spectrum antibiotics parenterally after drainage of antibiotics.
- Prevention of umbilical infection can be done by improving the sanitation and hygienic condition during birth.
- Lastly umbilical infection can be controlled by disinfection of umbilical cord soon after birth with iodine texture.

THANK YOU!!!

Part 4. Diseases of digestive system

4.1. Diseases of oral cavity and esophagus

4.1.1. Stomatitis

➤ Stomatitis is inflammation of the oral mucosa.

➤ It includes

→ **Glossitis:** - It is an inflammation of the tongue

→ **Palatitis :** - It is an inflammation of the palate (hard and soft palates).

→ **Gingivitis:** - It is an inflammation of the mucosa of the gums.

➤ Clinically it is characterized by

→ Partial or complete loss of appetite

→ Smacking of the lips

→ Profuse salivation

Etiology

➤ Stomatitis may be caused by

Stomatitis cont'd---

- \ 1. Physical agents
- \ 2. Chemical agents
- \ 3. Biological(infectious agents)

➤ Biological factors being the largest group of the causes.

1. Physical agents

- They cause stomatitis by injuring mucousa of the oral cavity physically.
- Some of the examples of physical agents are
 - Trauma while administering drugs orally with a balling gun and stomach tube etc.
 - Laceration of the tongue
 - Foreign body injury
 - Malocclusion (not aligned properly) of teeth
 - Sharp awns or spines on plants

Stomatitis cont'd---

→Eating frozen feed and drinking hot water

2. Chemical agents

→Oral administration of Irritant drugs like chloral hydrate.

→Counterirritants applied to skin, left unprotected and licked by the animal like mercury and cantharides compounds.

→Irritant substances administered by mistake, including acids, alkalis and phenolic compounds.

→Some plants that produces toxic substances that have irritant effect on mucousa of the oral cavity etc.

3. Biological(infectious agents)

➤It is stomatitis caused by biological agents like some pathogenic bacteria, virus and fungi.

➤Some of the examples are

Stomatitis cont'd---

In cattle

- Oral necrobacillosis associated with *Fusobacterium necrophorum*.
- Actinobacillosis which cause wooden tongue.
- Ulcerative, granulomatous lesions may occur on the gums in cases of actinomycosis.
- Stomatitis with vesicles occurs in foot-and-mouth disease and in vesicular stomatitis.
- Erosive, with some secondary ulcerative stomatitis occurs in bovine viral diarrhoea (BVD), bovine malignant catarrh, rinderpest and bluetongue.
- Infectious Bovine Rhino Tracheitis (IBRT) in young calves may have similar lesions like that of rinder pest.

2. Sheep

Erosive lesions in bluetongue, rinderpest and Bacteraemia

Stomatitis cont'd---

- Vesicular lesions rarely in foot and mouth disease
- Granulomatous lesions due to ecthyma are not unusual in the mouth, especially in young lambs.
- Similarly, oral lesions occur in bad cases of sheep pox and mycotic dermatitis.

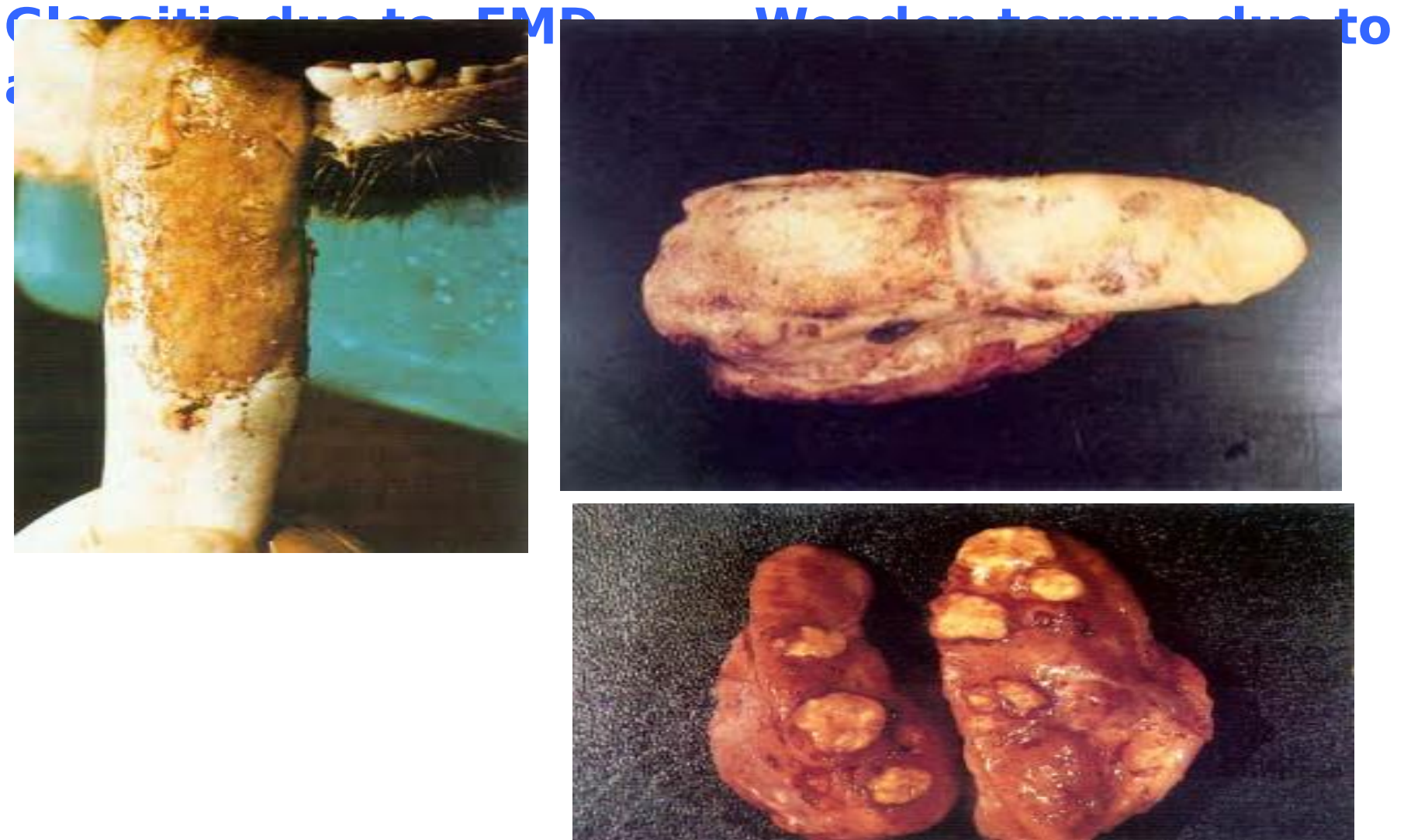
Horses

- Cheilitis and gingivitis (inflammatory nodules of the lips and gums caused by plant awns).
- Vesicular lesions in vesicular stomatitis
- Herpes virus infections are commonly accompanied by small (1 mm diameter) vesicles surrounded by a zone of hyperemia.
- Lingual abscess associated with *Actinobacillus* spp.

Pigs

Stomatitis cont'd---

vesicular exanthema of swine and swine vesicular disease.



Gingivitis due to malignant catarrhat fever



Erosion due to rinder pest



Blue tongue in cattle and sheep



Cheilitis due to mechanical damage



Ecthyma(orf) in sheep



Vesicular lesion in equines



Stomatitis cont'd---

Pathogenesis

- The lesions of stomatitis are produced
 - By the causative agents being applied directly to the mucosa and/ or
 - Gaining entrance to it by way of minor abrasions and/ or
 - By localization in the mucosa from a viremia

Clinical and pathological findings

- There is partial or complete anorexia and slow, painful mastication.
- Chewing movements and smacking of the lips are accompanied by salivation, either frothy and in small amounts, or profuse and drooling if the animal does not swallow normally.
- The saliva may contain pus or shreds of epithelial tissue.

Stomatitis cont'd---

the lesions.

→Swelling of the face is observed only in cases where a cellulitis or phlegmon has extended to involve the soft tissues.

→Toxemia may be present when the stomatitis is secondary to a systemic disease.

➤Several different lesions may be present in the oral cavity.

➤That can be

 \ Erosions

 \ Vesicles

 \ Ulcerative lesions

 \ Proliferative lesions

 \ Catarrhal stomatitis

 \ Deformity of or loss of tissue at the tip of the tongue

Stomatitis cont'd---

↓ Ulceration of the soft palate

Clinical pathology

→ Material collected from lesions of stomatitis should be examined for the presence of pathogenic bacteria, fungi and viral agent.

Diagnose

- It can be done based history and clinical signs by inspecting the mucous of oral cavity.
- To inspect the mucous of oral cavity, we must open the oral cavity by hands, rope and using mouth gags.

Mouth gags for different species of animals

Some mouth gags fo



Mouth gags for equine



Treatment

- In all forms of stomatitis, it is essential to remove the causative agent as quickly as possible.
- In stomatitis due to trauma the teeth may need attention.
- In all cases soft, appetizing food (pure water, green mass, soft hay and silage) should be offered.
- Feeding by stomach tube or intravenous alimentation maybe

Stomatitis cont'd---

resorted to in severe, prolonged cases.

➤ In easy form of stomatitis, we can treat animals by nonspecific

treatment like washing of the oral cavity with

→ 2% NaCl solution

→ 3% NaHCO₃ solution

➤ But in complicated form of stomatitis, we must wash the mucous membrane of the oral cavity for 3 - 4 times in a day with nonspecific antiseptic solutions like

↳ 2% solution of CuSO₄

↳ 2 - 3 % suspension of borax(boric acid)

↳ 1: 10000 Kmno₄ solution

➤ We can also use antimicrobial preparates like

↳ Nitrofurazone solution like 1: 5000 furacin solution

↳ 1 % suspension of a sulfonamide in glycerin.

Stomatitis cont'd---

4.1.2. Pharyngitis

➤ Pharyngitis is inflammation of the pharynx and is characterized clinically by

- ↳ Coughing
- ↳ Painful swallowing
- ↳ Variable appetite

Etiology

- Pharyngitis in farm animals is usually traumatic.
- However infectious pharyngitis is often part of a syndrome with other more obvious signs.
- The etiological agents can be
 - ↳ 1. Physical agents
 - ↳ 2. Chemical agents
 - ↳ 3. Biological agents

Stomatitis cont'd---

1. Physical and chemical agents

- Injury while giving oral treatment with balling or drenching gun or following endo tracheal intubation.
- Improper administration of a reticular magnet
- Accidental administration or ingestion of irritant or hot or cold substances.
- Foreign bodies, including grass, cereal awns, wire and bones etc.

2. Biological agents (infectious agents)

- It is caused by bacteria, virus and fungi as direct clinical signs or as symptom complex.
- Some of the examples that cause pharyngitis in cattle are
 - ↳ Necrobacillosis(*F. necrophorum*)

Stomatitis cont'd---

In equines

- As part of strangles or anthrax
- Viral infections of the upper respiratory tract, including equine herpesvirus-1, Hoppengarten cough, parainfluenza virus, adenovirus rhinovirus, viral arteritis, and influenza virus in equines.
- As part of anthrax and in some outbreaks of Aujeszky's disease in pigs.

Pathogenesis

- Physical, chemical and biological agents cause inflammation of the lymphoid tissue around the region.
- Since there is always sedimentation of microbes, feed, saliva and mucous.
- Inflammation of lymphoid tissue (tonsillitis) favor reproduction of microbes.

As the result toxin and different enzymes that are

Stomatitis cont'd---

tissue decomposition will be absorbed to blood and lymphatic system and cause fever.

- Inflammation may cause infiltration of mucosa and sub mucous and muscles of pharynx which results edema and pain around pharynx.
- All this results pain and difficulty in swallowing.

Clinical findings

- The animal may refuse to eat or drink or it may swallow reluctantly and with evident pain.
- There may be mucopurulent nasal discharge, sometimes containing blood.
- Spontaneous cough and, in severe cases, regurgitation of fluid and food through the nostrils.
- Oral medication in such cases may be impossible.
- Affected animals often stand with the head extended, drool saliva

Stomatitis cont'd---

and make frequent, tentative jaw movements.

- Severe toxemia may accompany the local lesions, especially in oral necrobacillosis and, to a less extent, in strangles.
- Empyema of the guttural pouches may occur in horses.
- If the local swelling is severe, there may be obstruction of respiration .
- and visible swelling of the throat.
- The retropharyngeal and parotid lymph nodes are commonly enlarged.
- Palpation of the pharynx may be performed in cattle with the use of a gag if a foreign body is suspected.
- Endoscopic examination through the nasal cavity is possible in the horse.

Clinical pathology

Nasal discharge or swabs taken from accompanying

Stomatitis cont'd---

assist in the identification of the causative agent.

➤ *Moraxella* spp. and *Streptococcus zooepidermicus* can be isolated in large numbers from horses with lymphoid follicular hyperplasia.

Necropsy findings

➤ Deaths are rare in primary pharyngitis and necropsy examinations are usually undertaken only in those animals dying of specific diseases.

Diagnose

➤ Diagnose is based on history, clinical signs that are observable by using external and internal palpation and in rare case by using pharyngeoscopy.

Differential diagnose

➤ You should differentiate it from paralysis of esophagus and from obstruction of esophagus due to different reasons.

Treatment

- Improve the condition in housing and feeding.
- Sick animals must be supplied with soft, palatable feeds and housed with deep litters.
- Drinking water must be warmed to room temperature.
- If the inflammation has mass character suspect infectious disease and isolate them.
- If the condition is complicated and sick animals are unable to eat and to drink,
 - Administer 25% glucose solution in dose of 300 – 400 ml IV. And
 - Administer 0.5 – 1 liter of 0.85 % NaCl IV.
 - Use antiseptics like KMnO_4 (1 :1000), Iodine glycerine (1:4) and 3% boric acid
- Pharyngitis with fever must be treated with systemic administration broad spectrum antibiotics.

4.2. Esophageal disorders

➤ Esophageal disorders are

- ↳ 1. Esophagitis
- ↳ 2. Esophageal obstruction
- ↳ 3. Stenosis of esophagus
- ↳ 4. Paralysis of esophagus

1. Esophagitis

- It is inflammation of the esophagus.
- Esophagitis is accompanied by clinical findings like
 - ↳ Spasm and obstruction of esophagus
 - ↳ Pain on swallowing and palpation
 - ↳ Regurgitation of bloodstained, slimy material

Etiology

- Esophagitis can be

Esophagus cont'd---

- 1. Primary

- 2. Secondary

- Primary esophagitis is caused by

- Feeding of animals very hot, strong, rough and pointed feed.

- Ingestion of chemical or physical irritants and is usually accompanied by stomatitis and pharyngitis.

- Laceration of the mucosa by a foreign body like bone and wire etc.

- Complications due to incorrect nasogastric intubation

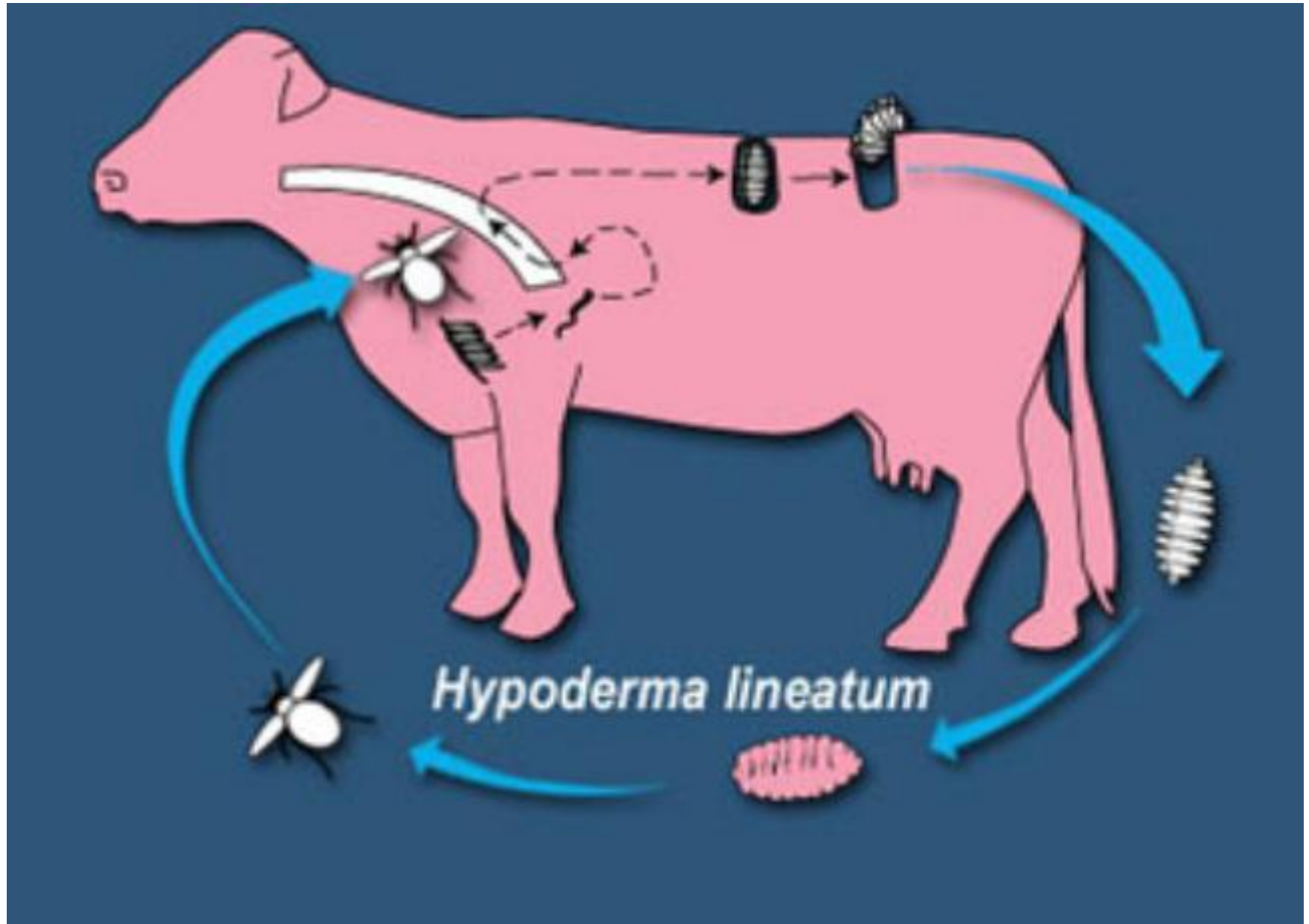
- The death of *Hypoderma lineatum* larvae in the sub mucosa of the esophagus

- External trauma

- Secondary esophagitis is developed due to

- Expansion of inflammatory process from stomach or

Esophagus cont'd---



Esophagus cont'd---

Pathogenesis

- Inflammation of the esophagus combined with local edema and swelling results in a functional obstruction and difficulty in swallowing.
- Inflammation may perforate along subcutaneous tissue and cause cellulitis.
- Perforation of the thoracic esophagus can result in severe and fatal pleuritis.
- There is extensive edema and accumulation of swallowed or regurgitated ingesta along with gas.
- The extensive cellulitis and the presence of ingesta results in severe
 - Toxemia and dysphagia
 - May cause aspiration pneumonia

Esophagus cont'd---

to swallow which cause severe pain, particularly in horses.

➤ In some cases, attempts at swallowing are followed by

→ Regurgitation

→ Marked drooling of saliva

→ Grinding of the teeth

→ Coughing and profuse nasal discharge

➤ If the esophagitis is in the cervical region, palpation in the jugular furrow causes pain and edematous tissues around the esophagus may be palpable.

➤ Local cervical cellulitis may cause rupture to the exterior and development of an **esophageal fistula**.

➤ Perforation of the thoracic esophagus may lead to **fatal pleurisy**.

Clinical pathology

In severe esophagitis of traumatic origin a marked

Esophagus cont'd---

Necropsy findings

- Pathological findings are restricted to those pertaining to the various specific diseases in which esophagitis occurs.
- In traumatic lesions or those caused by irritant substances, there is gross edema, inflammation and, in some cases, perforation.

Diagnose

- Diagnose of esophagitis is made based on
 - History
 - Clinical signs like difficulty in swallowing and pain during palpation
 - Endoscopy and radiography

Differential diagnose

- Esophagitis must be differentiated from

Esophagus cont'd---

- Stenosis of esophagus
- Dilation of esophagus
- Obstruction of esophagus
- Pharyngitis is different from esophagitis in that pharyngitis, in which attempted swallowing is not as marked and coughing is more likely to occur.
- Palpation may also help to localize the lesion; however, pharyngitis and esophagitis commonly occur together.
- During stenosis, dilation and obstruction of esophagus there is no pain while you are doing intubation with stomach tube.
- Complete obstruction of esophagus in ruminant animals may cause bloat.

Treatment

- Feed should be withheld for 2 - 3 days and fluid and electrolyte

Esophagus cont'd---

therapy may be necessary for several days (IV 0.9% NaCl and 40% glucose).

- Parenteral antimicrobials are indicated, especially if laceration or perforation has occurred.
- Reintroduction to feed should be monitored carefully and all feed should be soft and moistened to avoid the possible accumulation of dry feed in the esophagus, which may not be fully functional.
- Administration of analgesic (pain reliever) like **50 % dipyrrone** also called **metamizole** (for dogs , cats and horses) is necessary to reduce pain.
- Administer by intramuscular or intravenous injection

Horses

→ 20 to 60 ml (average 4 ml per 45 kg of body weight)

Dogs

→ 1 to 5 ml (1 ml per 10 kg of body weight)

Esophagus cont'd---

NB. Administer 2 to 3 times daily for a maximum of 2 days.

2. Esophageal obstruction

- Esophageal obstruction is characterized clinically by
 - Inability to swallow
 - Regurgitation of feed and water
 - Continuous drooling of saliva, and bloat in ruminants.
- It can be
 - Acute
 - Chronic

Etiology

- Obstruction of esophagus may be
 - 1. Intra luminal
 - 2. Extra luminal

1. Intra luminal obstruction

Esophagus cont'd---

- Obstruction of esophagus by swallowed materials.
- Examples are solid obstructions, especially in cattle, by turnips, potatoes, peaches, apples, oranges etc.
- A trichobezoar also caused esophageal obstruction in a cow.
- The most common type of esophageal obstruction in horses is simple obstruction due to impaction of ingest.
- Feedstuffs are a common cause of obstruction in horses allowed to eat immediately after a race or workout.
- Foreign bodies in horses include pieces of wood, antimicrobial boluses and fragments of nasogastric tubes can cause esophageal obstruction.

2. Extra luminal

- It is esophageal obstruction due to pressure on the esophagus by surrounding organs or tissues.

Turnips



Esophagus cont'd---

➡ The most common causes of extra luminal esophageal obstruction are

- Tuberculous or neoplasia in lymph nodes that are found in the mediastinal or at the base of lung

- Abscess on cervical or mediastinal lymph nodes(bubo)

- Persistent right aortic arch due to congenital anomalies

- Thymoma

- Other reasons that may cause esophageal obstruction are

- Carcinoma of pyloric part of stomach

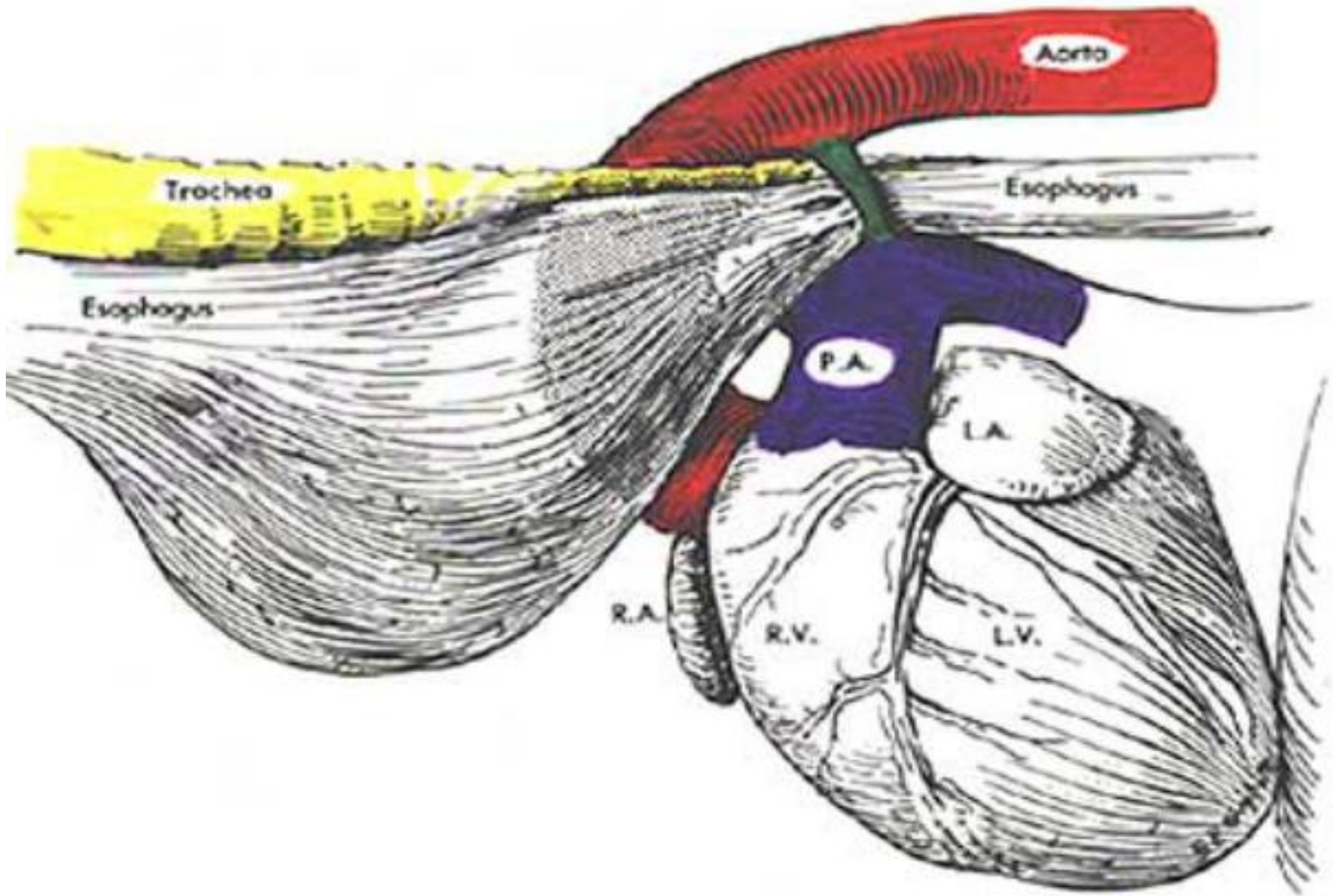
- Squamous cell carcinoma of the esophagus of a horse

- Esophageal hiatus hernia in cattle

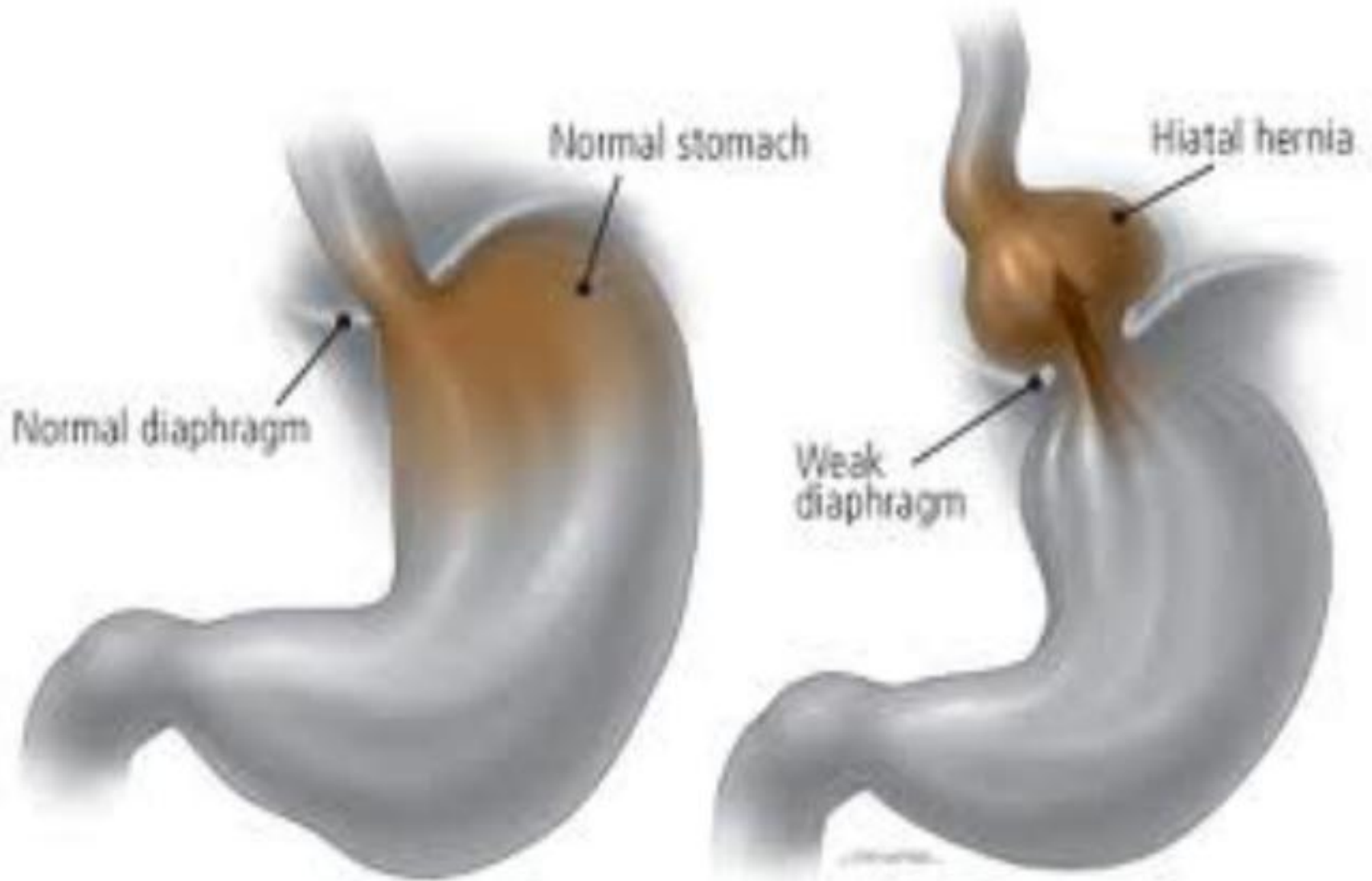
- Para esophageal cyst in a horse

Esophagus cont'd---

Persistent right aortic arch



Esophagus cont'd---



Esophagus cont'd---

Pathogenesis

- An esophageal obstruction results in a physical inability to swallow and, in cattle, inability to eructate, with resulting bloat.
- Complications of esophageal obstruction include
 - Laceration and
 - Rupture of the esophagus
 - Esophagitis
 - Stricture and stenosis
 - The development of a diverticulum

Clinical findings

- Acute obstruction in cattle has the following clinical features
 - The animal suddenly stops eating and shows anxiety and restlessness. There are forceful attempts to

Esophagus cont'd---

- If obstruction is complete, bloating occurs rapidly and adds to the animal's discomfort.
- Many obstructions pass on spontaneously but others may persist for several days and up to a week.
- In these cases there is inability to swallow, salivation and continued bloat.
- Passage of a nasogastric tube is impossible.
- Persistent obstruction causes pressure, necrosis of the mucosa and may result in perforation or subsequent stenosis due to fibrous tissue construction.
- In acute esophageal obstruction in horse, the clinical findings vary with the location, nature, extent and duration of the obstruction.
- Typically the major clinical findings are
 - Dysphagia with nasal reflux of saliva, feed and water

Esophagus cont'd---

cylindrical swelling.

Clinical pathology

➤ No typical laboratory method of diagnose.

Diagnose

➤ Diagnose of esophageal obstruction is done based on

→ History

→ Clinical signs

→ Intubation and nasopharyngeal intubation in cattle and equine respectively.

→ Special diagnostic methods like endoscopy, radiography with barium salt.

Treatment

➤ It can be done by using one of or in combination of the following methods.

Esophagus cont'd---

➤ These are

- 1. Conservative approach
- 2. By using stomach tube and endoscope
- 3. Surgical removal of foreign bodies by process called cervical esophagostomy

1. Conservative approach

➤ In acute obstruction, if there is marked anxiety and distress, the animal should be sedated before proceeding with specific treatment.

➤ Administration of a sedative may also help to relax the esophageal spasm and allow passage of the impacted material.

➤ For sedation and esophageal relaxation in the horse, one of the following is recommended:

→ Acepromazine in dose 0.05 mg/kg BW intravenously

Xylazine in dose 0.5 – 1.0 mg/kg BW intravenously

Esophagus cont'd---

- Detomidine in dose 0.01 - 0.02 mg/kg BW intravenously
- Romifidine in dose 0.04 - 0.12 mg/kg intravenously
- For esophageal relaxation, analgesia and anti-inflammatory effect we can use
 - Hyoscine: dipyrrone 0.5: 0.22 mg/kg BW intravenously.
 - Flunixin meglumine 1.1 mg/kg BW intravenously for analgesia and as anti inflammatory.
 - or phenylbutazone 2-4 mg/kg intravenously are suggested.

2. By using stomach tube and endoscope

- The passage of the nasogastric tube is always necessary to locate the obstruction.
- Gentle attempts may be made to push the obstruction caudal but care must be taken to avoid damage to the esophageal mucosa

Esophagus cont'd---

- The foreign body must be visible endoscopically and suitable snare through the scope are required.

Metallic flexible stomach tube Nasopharyngeal tube for equine



Endoscopy for animals and mono polarsnars.



Esophagus cont'd---

3. Surgical removal of foreign bodies

- It is the removal of foreign bodies by process called cervical esophagostomy.
- You will deal it in surgery course.

3. Stenosis of esophagus

- It occurs rarely in animals.

Etiology

- It can be
 - 1. Functional
 - 2. Obturation
- **Functional stenosis** is caused by repeating spasm of muscle of cardial part of esophagus.
- While **obturation esophageal stenosis** is formed as the result thickening of the mucous membrane of esophagus due to abscess,

Esophagus cont'd---

neoplasm and diverticulum in esophagus.

➤ Animals that recover from esophageal traumatic injury are commonly affected by **chronic esophageal stenosis** with distension above the stenosis.

Esophageal stenosis



Clinical signs

- Functional stenosis occurs suddenly.
- While obturation stenosis develops slowly.
- In general there is difficulty in swallowing which is manifested by formation of distension above the stenosis.
- And coming of food mass to the mouth by antiperistalsis movement.
- The animal are nervous and extends their neck while they swallow.

Diagnose

- Esophageal stenosis can be diagnosed based on history, clinical signs.
- Clinically we can know the site and degree of stenosis by intubation of esophagus with stomach tube with different diameters.
- We can also use X - rays with barium salt and

Esophagus cont'd---

- Sedatives like **acepromazine maleate** or **xylazine**.
 - For horse acepromazine maleate is administered IV or IM in dose of 30 – 100µg/ kgBW
 - For dogs 30 – 125 µg/ kg BW SC or IM or IV
 - For horses xylazine is administered IM in dose of 2 – 3 mg/ Kg BW or IV in dose of 0.6 – 1.1 mg/ Kg BW
 - For cattle xylazine is administered IM in dose of 50 – 300 µg/ kg BW
 - For dogs and cats IM in dose of 1 – 3 mg/ KG BW
- But esophageal stenosis due to obturation it is difficult to treat.
- So animals with esophageal stenosis because obturation must be culled.

4. Paralysis of esophagus

- It is characterized by the loss of esophageal muscle contraction

Esophagus cont'd---

not pass through it.

➤ Esophageal paralysis occurs very rarely in animals.

Etiology

➤ Paralysis of esophagus occurs due to

→ Trauma on esophagus

→ Esophagitis

→ Meningoencephalitis

→ Late stage of rabies

→ Paralysis of CNS because of botulin toxin

Pathogenesis

➤ If esophageal paralysis remains for long time, there will be emaciation, reduction of productivity and dehydration.

Clinical signs

➤ The animal swallows the feed easily

Esophagus cont'd---

- But never pass to stomach or rumen.
- As the result the feed is visible or palpable in esophagus.

Necropsy findings

- There will be un swallowed feed in esophagus.
- Esophageal wall will be thickened, inflamed with wounds or ulcers.

Diagnose

- Diagnose can be done based on history and clinical signs.

Treatment

- Mass of feed in esophagus must be removed by making massage around jugular sulcus after per os administration of lubricants.
- Give animals soft and liquid feed.

4.2. Diseases of the forestomachs(proventriculus) of ruminant animals

➤ Here we will see

- | 1. Hypotony and atony of fore stomachs of ruminant animals
- | 2. Simple indigestion
- | 3. Acute carbohydrate(grain)engorgement
- | 4. Ruminal tympany
- | 5. Traumatic reticulitis/ Traumatic reticulopericarditis/ Traumatic reticuloperitonitis

1. Hypotony and atony of fore stomachs of ruminant animals

- Hypotony and atony of fore stomachs are characterized by disturbance of the motility capacity of fore stomachs.
- That means hypotony is characterized by reduction of the frequency and amplitude of motility.

Fore stomachs cont'd---

- While atony means absence of motility.
- Hypotony and atony is common very often in cattle but rarely in goats.

➤ Hypotony and atony of fore stomachs can be

→ Acute

→ Chronic

Etiology

➤ It can be

→ Primary

→ Secondary

➤ Primary reasons for hypotony and atony of forestomachs are

→ Suddenly change of animal feeds from rough with high cellulose content like dry straw and hay feeding of

Fore stomachs cont'd---

→Hypotony and atony of forestomachs due to sudden change in animal feeds is common in intensive farms specially those which do not get active daily motion.

➤ It is more common in

→ Pregnant cows

→In fatten and emaciated animals

➤ Secondary hypotony and atony of fore stomachs occurs due to

→Impaction of abomasum and omasum

→Obstruction of the proximal part of small intestine

→Due to local infections like mastitis, abscess and empyema etc that can cause toxemia.

→Trauma of nerve vagus

→Any diseases that can cause fever

→Traumatic reticulitis

Fore stomachs cont'd---

Pathogenesis

- All factors that are mentioned above decrease the ability of fore stomachs to contract
- As the result
 - Rumination decreases or stops.
 - Movement and absorption functions of fore stomachs are disturbed.
 - Which results in impaction of the feed in rumen, reticulum and omasum.
 - This results changing of the micro flora and fauna of rumen and reduction of the PH of rumen to 5.8 – 6.3.
 - So that there will be production of decomposition of proteins, carbohydrates and fats etc with production of toxic products(amines, ketones, lactic acid and NH_3).
- All these inhibit

The secretory and peristaltic function of stomach and

Forestomachs cont'd---

- Reduction of the digestive function of stomach and intestine
- All this results in **auto intoxication**.

Clinical signs

- Inappetance and can be anorexic
- Rumination can be reduced or absent but eructation of gas is normal
- Bloat in rumen which hard in early stage and becomes soft as the time goes as there is high accumulation gas.
- Weak and absence of sounds during auscultation of fore stomachs and intestine.
- So there will be constipation.

Clinical pathology

- But due to intoxication the temperature may fall below norm.
- The main clinical pathology that are observed during hypotony and atony of fore stomachs is neutrophilia shifted to left.

Diagnose

- Diagnose is based on history and clinical signs.

Differential diagnose

- Hypotony and atony of rumen must be differentiated at traumatic reticulitis.

Treatment

- Primary hypotony and atony are treated
 - 1. By increasing the PH of rumen
 - By drenching using stomach tube 10% of NaHCO_3 20 – 30 liter or $\text{Mg}(\text{OH})_2$ orally 450 gm dissolved in 3.5 liter for 3 consecutive days.

Fore stomachs cont'd---

Indigestion powder



Forestomachs cont'd---

ruminatoric and carminative action without dehydration

2. By increasing the motor and secretory function of rumen using cholinergic preparates like

→ Prozerin

→ Carbocholin

Dose and route of administration

→ Carbocholin :- Cattle subcutaneously dose of 0.0025 gm

Sheep and goat subcutaneously
dose of 0.0003 gm

→ Prozerin: - Cattle subcutaneously dose of 0.2 gm

Sheep and goat subcutaneously
dose 0.02 gm

Forestomachs cont'd---

- Lubricant laxatives(liquid paraffin or white soft paraffin) or
- Bulk forming laxatives(Ispaghula husk) or
- Osmotic laxative(MgOH or MgSO_4 called Epsom salt)
- However, if bloat is not removed after all this,
- Do n't give feed for 2 -3 days.
- Administer 1 – 2 liter of fresh rumen content from other healthy animal through stomach tube.
- ➡ To stimulate the metabolism of organism which is disturbed due to auto intoxication.
- Administer 20 % glucose and 10 % NaCl IV in dose of 250 – 300 ml and 500 ml respectively.
- Sodium caffeine benzoate subcutaneously in dose of 2 – 4 gm to stimulate the function of heart.

Prevention measures

Forestomachs cont'd---

fungi

- Do n't feed animals dry feeds like wheat bran and oats etc alone for long time.
- The farm must have balanced ration to animals according to their physiological needs.
- Do not change the ration of animals from dry to wet(green mass) and vice versa radically
- Finally if the farm is intensive farm, plan for animals to have daily motion.

2. Simple indigestion

- This is a minor disturbance in ruminant gastro-intestinal digestive functions.
- Commonly encountered in indoor-fed cattle which are

Fore stomachs cont'd---

- But not commonly observed in pastured beef cattle.

Etiology

- The common causes are dietary abnormalities of minor degree including
 - Indigestible roughage like straw, bedding or scrub fed during drought periods particularly when the protein intake is low.
 - It is probable that limitation of the available drinking water may contribute to the occurrence of the disease during dry seasons.
 - Moldy, overheated and frosted feeds
 - Moderate excesses of grain and concentrate intake
 - Prolonged or heavy oral dosing with antimicrobials may cause indigestion due to inhibition of the normal

Forestomachs cont'd---

➤ But it may happen by

→ Changes the PH of rumen contents to acid called **rumen acidosis** by over eating of grain.

→ High protein diets, including the feeding of excessively large quantities of legumes or urea, also depress motility because of change of the PH of rumen to alkaline called **rumen alkalosis**.

→ Simple accumulation of indigestible food may physically impede ruminal activity.

→ Putrefaction of protein may also play a part in the production of atony.

→ Because the toxic amides and amines produces histamines which is known to cause ruminal atony.

Clinical signs

➤ A reduction in appetite is the first clinical finding, followed closely in milking cows by a slight drop in milk

Forestomachs cont'd---

- Rumination ceases and the ruminal movements are depressed in frequency and amplitude; sometimes are almost absent.
- There may be moderate tympany
- But the usual finding is a firm, doughy rumen without obvious distension.
- The feces are usually reduced in quantity and are drier than normal on the first day.
- However, 24 - 48 hours later the animal is commonly diarrheic; the feces are softer than normal, voluminous and commonly malodorous
- There is no systemic reaction and the heart rate, temperature and respirations are usually within normal ranges.

Clinical pathology

The rumen juice has pH values between 5.5 - 6.0 on

Forestomachs cont'd---

- Simple indigestion due to portentous grain, the rumen juice has alkaline PH.

Necropsy finding

- No typical necropsy findings for simple indigestion.

Diagnose

- Diagnosis of simple indigestion is based on
 - 1. History includes
 - ↳ Sudden change of feed(from green mass to roughage)
 - ↳ Access to grain
 - ↳ Long time feeding of roughage with inadequate water
 - 2. Clinical signs

Differential diagnose

- Simple indigestion must be differentiated to many diseases of forestomachs and abomasum in which ruminal atony is a common

Forestomachs cont'd---

clinical finding.

➤ And from diseases of other body systems that cause secondary

ruminal atony like

→Ketosis(acetonemia)

→Traumatic reticuloperitonitis

→Carbohydrate engorgement

→Secondary ruminal atony occurs in many diseases in which septicemia or toxemia is their clinical manifestation.

Treatment

➤ Most causes of simple indigestion recovers spontaneously with out any treatment.

➤ But if it does not recovers by it self, treatment of simple indigestion must be directed in re -

Forestomachs cont'd---

2. PH of rumen

3. Normal micro flora of rumen

1. Re - establishment of rumen motility

➤ Rumen motility can be re-established by using either

1. Ruminatoric

→ $\text{Mg}(\text{OH})_2$ or MgSO_4 called Epsom salt (0.5 - 1.0 kg per adult cow) are cheap and easy to administer.

→ Indigestion powder

2. Para sympathomimetic

→ Carbamylcholine chloride, physostigmine and neostigmine is most effective parasympathomimetic drug at a dose of 2.5 mg/45 kg body weight.

2. Re - establishment of the PH of rumen

➤ if an excessive quantity of grain is the cause of simple indigestion, use the alkalinizers like

Forestomachs cont'd---

→ Magnesium hydroxide($\text{Mg}(\text{OH})_2$) or MgO is recommended to neutralize the rumen acidity.

↳ **400 g of $\text{Mg}(\text{OH})_2$ or MgO** per adult cow (450 kg BW) is recommended.

→ **2. Acetic acid** or **vinegar** in dose of 5 – 10 liter is used when the rumen contents are alkaline due to the ingestion of high protein concentrates.

4. Reconstitution of the micro flora of rumen

➤ Reconstitution of the normal micro flora of rumen is necessary if the simple indigestion is prolonged.

➤ Because the animals stay anorexic for longer periods.

➤ Which results in depression of rumen micro flora because there have been marked changes PH of rumen content.

➤ Such cases can be treated with **cud transfer** of

Forestomachs cont'd---

Methods of obtaining rumen content

- 1. Rumen fluid can be obtained by siphoning from the rumen with stomach tube or by vacuum withdrawal with a special pump.
 - In this case best results are obtained if 20 - 30 L of water is pumped into the rumen and then allowed to Siphon by gravity flow (rumen lavages).
- 2. Rumen content can also be collected from the mouth during rumination when ingesta is regurgitated.
- 3. It can be also obtained in the abattoir

Method of transferring of rumen content

- The rumen fluid to be transferred should be strained and administered as
 - An oral drench or
 - By stomach tube

Forestomachs cont'd---

- **NB.** Commercial products comprising dried rumen solids are available and provide some bacteria and substrate for rumen activity.
- When affected animals resume eating, give them good-quality hay like alfalfa (Lucerne) or clover hay, green feed and concentrate may be added to the diet as the appetite improves.

3. Acute carbohydrate engorgement

- This is an acute disease of ruminants characterized by
 - Indigestion
 - Rumen stasis
 - Dehydration
 - Acidosis
 - Toxemia results in coordination, collapse and

Forestomachs cont'd---

- Rumen overload
- Grain overload

Etiology

- Sudden ingestion of large quantities of highly fermentable carbohydrates such as grain like wheat, barely, maize and their flours is the commonest cause.
- Less common causes include engorgement with apples, grapes, bread, baker's dough, sugar beet, mangles, sour wet brewers' grain.
- It affects both large and small ruminants.
- But it commonly occurs in feedlot cattle when they are exposed to heavy grain diets.
- The development of acute CH_2O engorgement depends up on
 - Previous diet and change of ration

Forestomachs cont'd---

Pathogenesis

- The ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2 - 6 hours by a marked change in the microbial population in the rumen.
- There is an increase in the number of *Streptococcus bovis*, which utilize the carbohydrate to produce large quantities of **lactic acid**.
- As the result the rumen PH decreases to 5 or less, which results in the destruction of the cellulolytic bacteria and protozoa.
- The low PH allows lactobacilli to grow very well in large quantities which results **in ruminal lactic acidosis**.
- The superimposition of lactic acid and its salt like **lactate**, on the existing solutes in the rumen liquid

Forestomachs cont'd---

- Which leads to the drawn of fluid into rumen.
- That results **in dehydration of the animal.**
- It can also cause **lactic acid ruminitis.**
- And its absorption to blood circulation cause toxemia called **lactic acidemia.**
- In animals that survive the low PH may facilitate fungal proliferation specially *Rhizopus* spp., *Mucor* spp. and *Absidia* spp. which in turn cause **mycotic ruminitis.**
- Proliferation of some anaerobic bacteria such as *Spherophorus necrophorus*, *Fusobacterium necrophorum* and *Arcanobacter pyogenes*.
- Which enter to the ruminal blood vessels and spread to the liver and cause **hepatic necrobacillosis.**

Clinical findings

- The severity of the disease depends on the amount

Forestomachs cont'd---

animal.

➤ The animal examined clinically within a few hours after engorgement, the only abnormalities that may be detectable are a

- Distended rumen and abdomen

- Some abdominal discomfort evidenced by kicking at the belly.

- Reduced ruminal motility(hypotony)

- Anorexia

- Animal may start eating with out specific treatment

➤ It is the easy form of CH_2O engorgement when small amount CH_2O grain are engorged.

➤ In advanced form of CH_2O engorgement

- Dullness

- Complete ruminal atony and rumen feels doughy on

Forestomachs cont'd---

- Staggering
- Heart and respiratory rates are increased
- Severe dehydration
- Subnormal body temperature
- Sunken eyes
- Profuse and watery diarrhea
- Recumbency
- Affected animals usually lie down quietly with head turned to the flank.
- Decreased response to any stimulus.

NB. Reduction of heart beat to normal, increasing of body temperature, returning of ruminal movement and passage of large, soft feces are favorable signs of recovery.

Clinical pathology

Forestomachs cont'd---

1. Reduced ruminal PH

- The PH of rumen will be reduced less than 5 suggests severe grain over load and necessity of immediate treatment

2. Disturbance of ruminal flora

- Microscopic examination of ruminal fluid on a glass slide examined at a low power reveals absence of ruminal protozoa.
- The predominantly Gram-negative bacterial flora of the rumen is replaced by Gram- positive ones.

3. Increase of haemoconcentration(hematocrit or PCV value)

- This occurs because of the increase of the drawn of fluid from extracellular space to rumen.
- As the result hematocrit(PCV - value) rises from a normal of 30 - 32 % to 50 - 60%

4. Elevation of lactic acid in the blood

- Because of an increasement of lactic acid in the rumen, the acid will be absorbed in to the blood circulation.
- Results in **acidosis** called **lactic acidemia**.

5. Urine PH

- Urine PH falls to 5 and becomes concentrated.

Diagnose

- Diagnose of CH₂O engorgement is based on
 - History - access to grain
 - Characteristic clinical signs

Differential diagnose

- Acute CH₂O engorgement must be differentiate from
 - Parturient paresis
 - Simple indigestion

Forestomachs cont'd---

- Per acute coliform mastitis
- Acute peritonitis

Treatment

➤ Principles of treatment of acute CH₂O engorgement are based on:

- 1. Correction of ruminal and systemic acidosis and prevention of further production of lactic acid
- 2. Restoring the fluid and electrolyte losses and maintaining circulating blood volumes.
- 3. Restoring fore stomach and intestinal motility to normal

1. Correction of ruminal and systemic acidosis and prevention of further production of lactic acid

➤ Ruminal acidosis can be corrected by the use of alkalizing agents as oral drench or using stomach

Forestomachs cont'd---

→ MgO – 500g/450 Kg added in warm water

2. Restoring the fluid and electrolyte losses and maintaining circulating blood volumes

→ Systemic acidosis and dehydration can be corrected by

→ Intravenous(IV) administration of **5% sodium bicarbonate (5% NaHCO₃)** at a rate of 5l ml/450 Kg for 30 minutes.

→ Followed by IV administration of isotonic sodium bicarbonate (**1.3% NaHCO₃**) for 6 -12 hours.

→ For restoration of fluid and electrolyte losses and maintenance of circulating blood volumes, we can administer **Ringer solution**.

3. Restoring fore stomach and intestinal motility to normal

→ The motility of fore stomachs and intestine can be

Forestomachs cont'd---

Ringer solution contains

→ 6.5 g NaCl,

→ 0.42 g KCl,

→ 0.25 g CaCl₂ and

→ 1 mole of sodium bicarbonate is dissolved in one liter of distilled water.

Forestomachs cont'd---

→ Neostigmine

➤ In dose of 2.5 mg/45 kg body weight by IM administration.

➤ And other Cholinergic preparates like prozerin or carbocholin

Dose and route of administration

→ Carbocholin :- Cattle subcutaneously dose of 0.0025 gm

Sheep and goat subcutaneously
dose of 0.0003 gm

→ Prozerin: - Cattle subcutaneously dose of 0.2 gm
Sheep and goat subcutaneously
dose 0.02 gm.

➤ **NB. You can also use indigestion powder that has antacid, ruminatoric and carminative action without dehydration**

Forestomachs cont'd---

stomach tube 1 to 2 times a day for 2 to 3 days.

➤ In very severe cases, if it is impossible to correct by above methods **ruminotomy** is recommended.

➤ In this case, you must evacuate all the rumen content and replace with rumen cud(10 – 20 liter) from healthy animals.

➤ **NB. Emergency slaughter may be recommended in a certain situation as in the case of CH₂O engorgement of fattening beef cattle near their finishing time.**

Prevention measures

→ Prevention of accidental access to grain and concentrates

→ Gradual adaptation of beef cattle to concentrate rations is very important

Collection of rumen content from healthy animals



Replacement of rumen



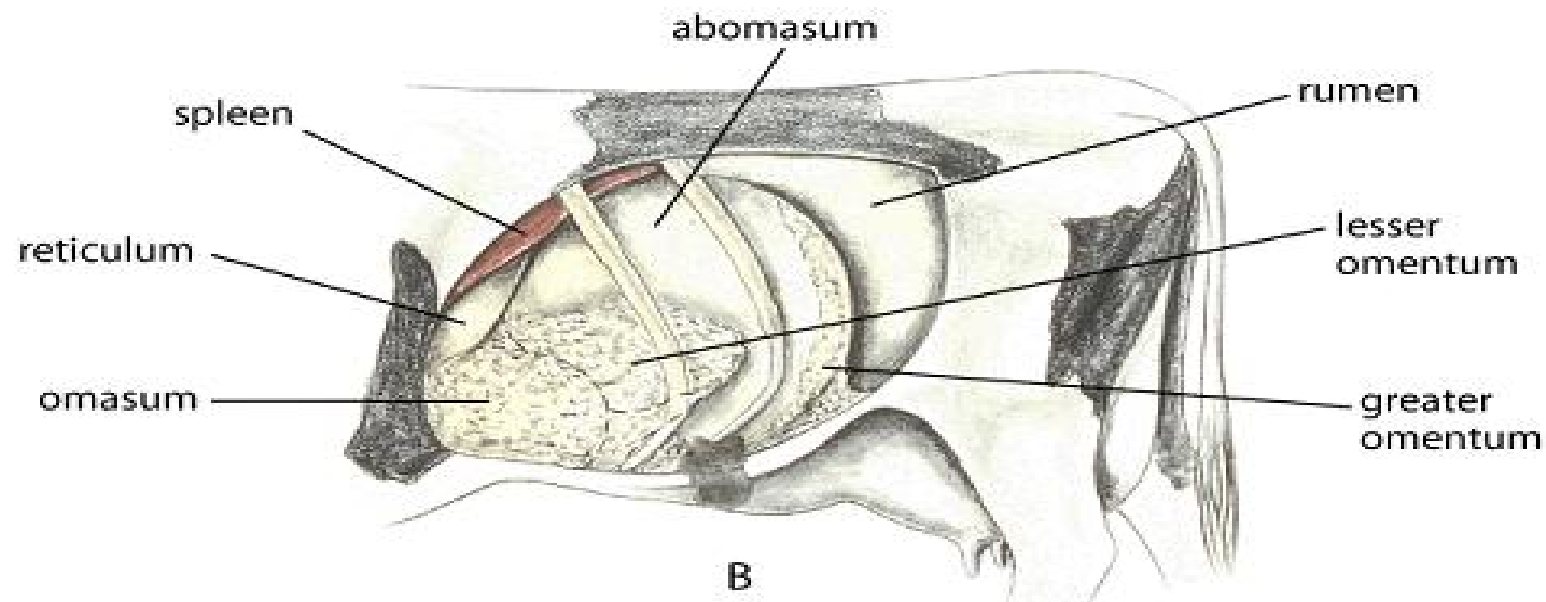
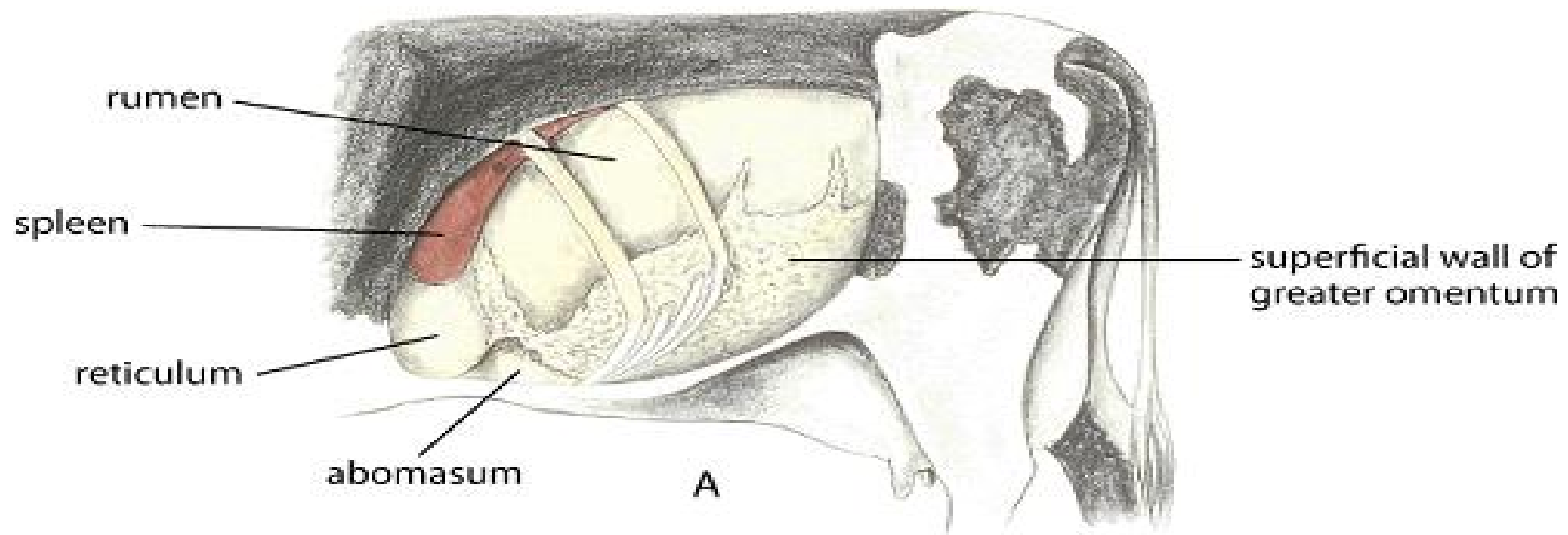
Ruminotomy

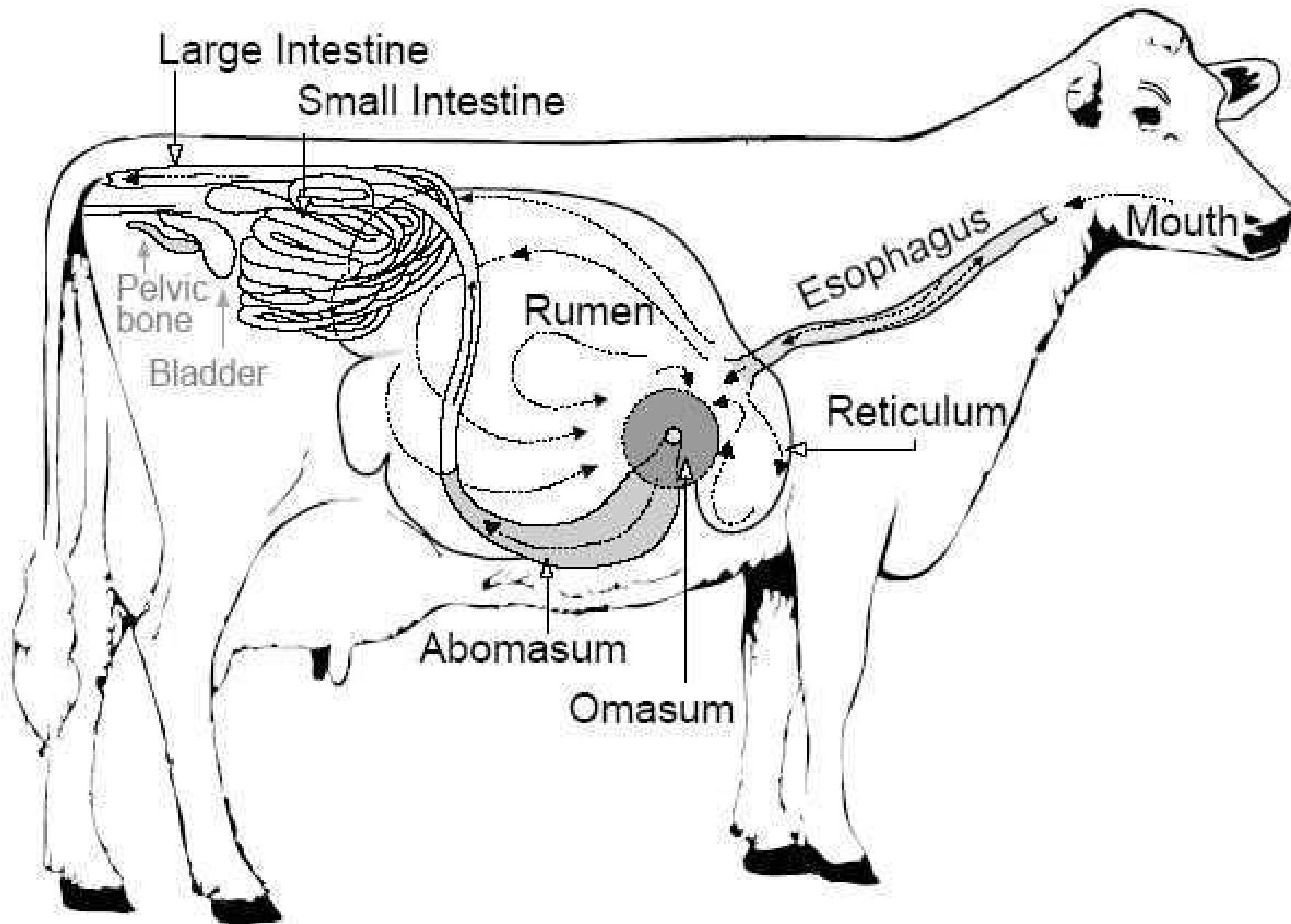


4. Ruminal tympany(bloat)

- Normally, you know in physiology that gas bubbles produced in the rumen coalesce, separate from the rumen contents to form pockets of free gas.
- These pockets of free gas are collected above the level of the contents and finally are eliminated by **eructation**.
- Disturbing of this normal physiological process may cause ruminal tympany(bloat).
- So that **Ruminal tympany** is abnormal distension of the rumen and reticulum caused by excessive retention of the gases.
- The gases can be either
 - 1. In the form of a persistent foam mixed with the rumen contents, caused **primary bloat** also called **frothy bloat/acute bloat**. or
 - 2. In the form of free gas separated from the ingesta

Topography of organs of the digestive system of cattle







Tympany cont'd ---

- **Secondary ruminal tympany(free-gas bloat)** is formed by free gas due to interference with eructation because of physical obstruction of esophagus or eructation mechanism.
- Bloat commonly affects mostly cattle , sheep and rarely goat and camel.

Etiology

- In generally the causative agents of tympany(bloat) can be either by
 - Ingestion of bloating forages or
 - Interference with eructation mechanism

1. Primary ruminal tympany (frothy bloat)

- Primary ruminal tympany or frothy bloat is caused by the production of a stable foam that traps the normal gases of fermentation in the rumen.

The essential feature is that enclosures of the small

Tympany cont'd ---

- As the result intraruminal pressure increases.
- There are two important factors for the development of frothy bloat.
- These are
 - 1. Dietary factor
 - 2. Animal factor

1. Dietary factor

- It is a factor which is related with the feed of animals.
- Primary (frothy bloat) can be
 - 1. Pasture frothy bloat
 - 2. Feedlot frothy bloat

1. Pasture(leguminous) frothy bloat

- Pasture frothy bloat occurs mainly because of
 - Grazing of very succulent green lush grasses or vegetables

Tympany cont'd---

- Grazing of immature and rapidly growing leguminous plants at prebloom stage.
- Not all pasture and leguminous plants can cause frothy tympany.
- However, the most common animal feeds that can cause frothy bloat are
 - *Trifolium* spp like that of *T. repens*(white clover) and *T. pratense*(red clover)
 - *Medicago sativa*(alfalfa)
 - Less commonly ones include cereal crops, cabbage, pea and beans, maize at their early stage of growth.
- The above pastures can be risk for frothy bloat if they are eaten by animals
 - When they are moistened with water
 - When they have dew

Some common leguminous that cause frothy bloat

Trifolium repens(white clover)



***Trifolium pratense*(red clover)**



***Medicago sativa*(alfalfa)**



Tympany cont'd---

- In general, bloat-causing legumes are susceptible to rapid digestion by rumen microflora.
- While bloat-safe legumes are digested more slowly.
- So that leguminous or pasture bloat is due to the foaming qualities of the soluble leaf proteins.

2. Feedlot bloat

- Feedlot bloat is caused by finely ground grain which promotes frothiness of rumen contents.
- Frothiness of rumen contents during feedlot is due to an increase in viscosity of ruminal fluid.
- Viscosity of ruminal fluid increases because of production of slime by encapsulated bacteria.
- As encapsulated bacteria increase their numbers on high carbohydrate diet.
- The slime may entrap the gas which is formed by fermentation so

Tympany cont'd---

that the gas has no chance to coalesce and burst.

➤ As the result the gas may mix with moisture and form foam.

➤ The foam blocks the cardiac part of rumen so that eructation is impaired.

➤ So that insoluble extracellular polysaccharides in case of feedlot bloat are important for foam formation .

Note

→1. The soluble leaf proteins in case of pasture bloat and the insoluble extracellular polysaccharides in case of feedlot bloat are important for foam formation

→2. The main cause of frothy bloat is not only excessive gas production; but it is due to trapping of produced gases of fermentation by the stable foam.

→3. Frothiness of the ruminal content causes physical obstruction of the cardiac part of rumen and this

2. Animal factors

- There are many animal factors that expose ruminant animals to bloat.
- Among them the most important are to expose a
 - **Genetics**: - Some animals are said to be genetically more susceptible to bloat than others
 - **Age** : - Young animals are said to be more susceptible to adults
 - **Body condition**: - Emaciated and fattened animals are more susceptible than animals with normal body conditions.

2. Free gas(Secondary bloat)

- It is caused by physical and/or functional defect in the eructation of the normal produced gases in the rumen.

Physical causes

- Internal obstruction(intra luminal obstruction) of

Tympany cont'd---

- Tuberculous or neoplasia in lymph nodes that are found in the mediastinal or at the base of lung
- Abscess on cervical or mediastinal lymph nodes(bubo).
- Interference in esophageal groove function as in vagus indigestion and diaphragmatic hernia.

Pathogenesis

- You know that normally, gas bubbles produced in the rumen fluid coalesce, separate from the rumen contents.
- And form pockets of free gas above the level of the contents, and are finally eliminated by eructation.
- But in the presence of large amount of easily digestible green mass that mentioned above.
- There is a formation of large quantity of gas that will be collected in the upper part of rumen during free gas

Tympany cont'd---

foam during frothy tympany.

- As the result the organism un able to evacuate the gas from rumen.
- The accumulation of this non - evacuated gas in the rumen cause bloat.
- At the beginning, the accumulated gas in the rumen irritates the receptors in the lumen of rumen and increase its motility.
- Finally the wall of rumen paralysis.
- Pressure from rumen exerts on the organs of thoracic and abdominal cavity and disturbs their functions.
- Pressure that exerts to the lung and heart through diaphragm is critical.
- Because the exerting pressure make the heart and lungs unable to contract and relax.
- As the result systolic volume of blood and gas

Tympany cont'd---

- And there will be disturbance of gas exchange.
- The pressure on abdominal cavity disturbs the function of liver and peristalsis of intestine even the movement of blood through abdominal aorta.

Clinical signs

- **In primary pasture bloat**, obvious distension of the rumen occurs quickly, sometimes as soon as 15 minutes after going on to bloat-producing pasture.
- The animal stops grazing and becomes nervous.
- There is discomfort and the animal may stand and lie down frequently.
- Frequently the animal kick at its abdomen by its hind legs.
- Temperature of animal is normal but breathing rate is increased and shallow and may reach up to 60 – 80 br. m/ minute.

Tympany cont'd---

but the entire abdomen is enlarged.

- Frequent defecation and urination are common.
- Dyspnea is marked and is accompanied by
 - Mouth breathing
 - Protrusion of the tongue
 - Salivation and extension of the head
- No rumination and eructation.
- The mucous membrane is hemorrhagic at the beginning and changed to cyanosis.
- Tenesmus and the animal expelled small amount of soft feces and urine.
- The abdominal cavity increased its volume with left asymmetry.
- If immediate help is not given the animal may die at asphaction.

Tympany cont'd---

Bloat in cattle



Bloat in cattle



Bloat in sheep and goat



Tympany cont'd---

top of the ruminal contents.

- There is usually an increase in the frequency and strength of ruminal movements in the early stages followed by atony.
- Passage of a stomach tube or trocarization results in the release of large quantities of gas and subsidence of the ruminal distension.
- If an esophageal obstruction is present it will be detected when the stomach tube is passed.

Clinical pathology

- Laboratory tests are not necessary for the diagnosis of ruminal tympany.

Necropsy findings

- In cattle that have died from bloat within an hour previously there is:-

Protrusion and congestion of the tongue

Tympany cont'd---

and neck.

- Hemorrhages of epicardium and upper respiratory tract
- The kidneys are friable
- Marked erythema is evident beneath the ruminal mucosa, especially in the ventral sacs.
- The liver is pale because of expulsion of blood from the organ.
- Occasionally, the rumen or diaphragm have ruptured.
- In animals dead for several hours because of tympany there is subcutaneous emphysema.

Diagnosis

- Diagnose of bloat depends on anamnesis and on typical clinical signs of a disease.

Differential diagnosis

Tympany cont'd---

→ 1. Free gas tympany due to different reasons like obstruction of esophagus.

↳ Free gas tympany is differentiated from frothy bloat in that percussion around left flanks produce tympanic sound, while frothy produce atympanyic (silent) sound.

↳ 2. From many febrile diseases of ruminant animals that cause hypotony and atony of rumen.

Treatment

➡ The approach to treatment bloat depends on

→ The circumstances in which bloat occurs

→ Types of bloat, whether the bloat is frothy or due to free gas

→ Whether the bloat is life threatening or not

➡ **1. If the bloat is secondary due to free gas, it is necessary**

Tympany cont'd---

- To control further gas formation in the rumen
- If the animal has difficulty in breathing due to pressure from rumen to the lung,
- Allow large ruminants to go up the mountain area slowly
- Hold the front legs of small ruminants up
- To decrease gas formation in the rumen, wash the left abdominal cavity with cold water(hold ice on it).
- The gas in free gas bloat can be removed by using ruminal intubation using stomach tube.
- The remaining gas can be adsorbed by administration of 2 – 3 liter of fresh milk or 20 gm of $\text{Mg}(\text{OH})_2$, 10 -20 ml NH_3 in 500 ml of water either in drenching or by stomach tube
- For the control of further gas formation use 50 – 150 ml of kerosene in mixture of water or with tympanol

Tympany cont'd---

➤ If the above methods does not solve the bloating problem due to free gas, use rumenocentesis.

What is rumenocentesis?

➤ It is the method of removing of gas from rumen by process called trocarization using trocar and cannula.

Site of trocarization

→ Draw the line that connect tuber coxae and the mid of the last ribs on the left abdominal cavity.

→ Find the mid point of the line

→ It is the site where trocarization must be performed.

Procedure

→ Remove the hair around the site

→ Disinfect the site with 70% alcohol or iodine texture

→ Use trocar and cannula (with diameter of 1 cm) for large animals

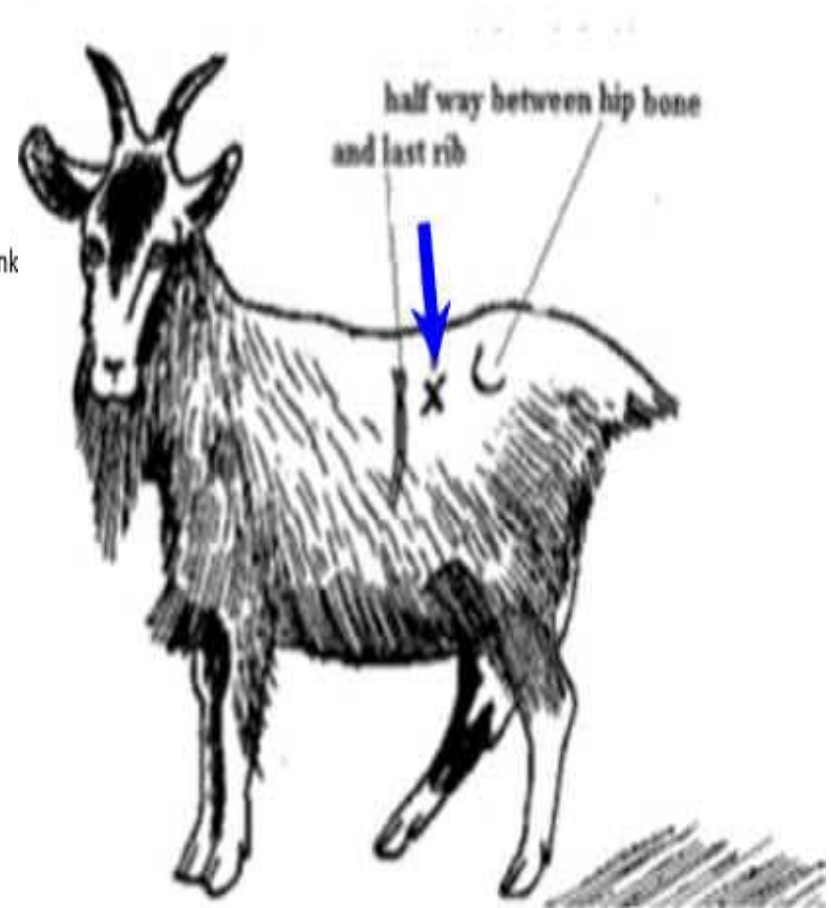
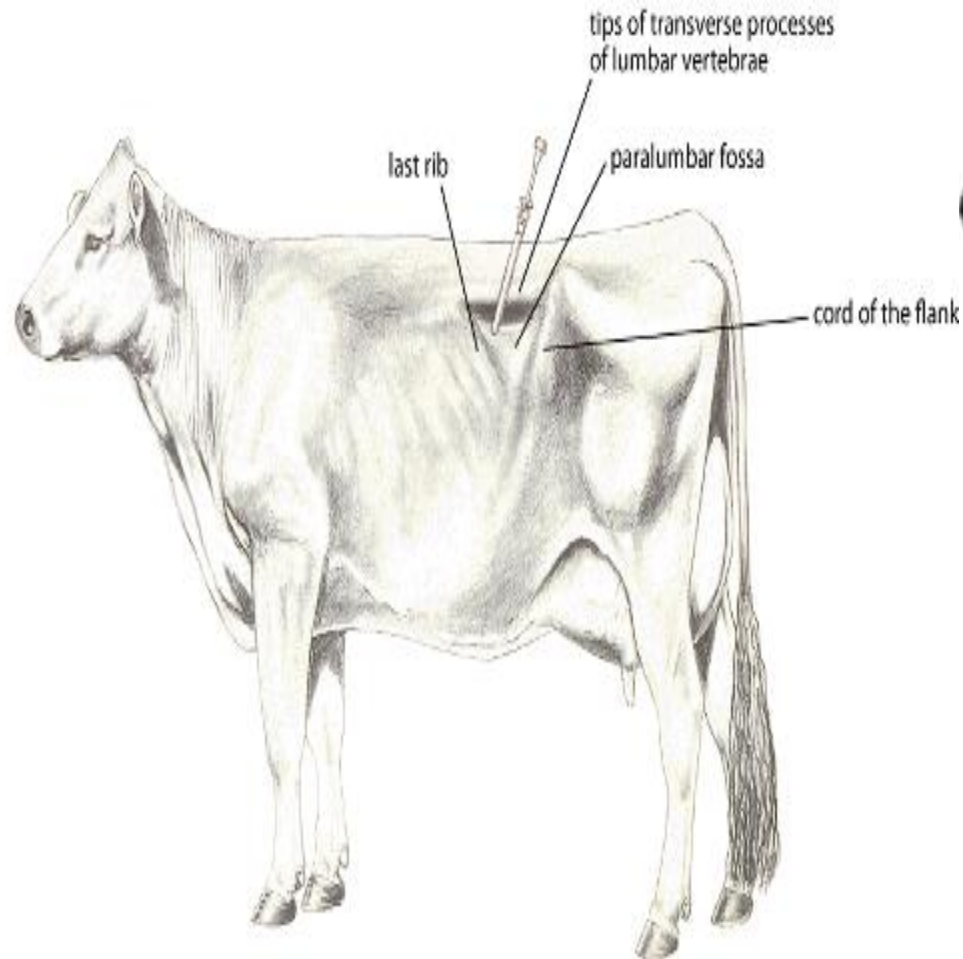
Stomach tube to evacuate gas from rumen during bloat(tympany)



Trocar and cannula for large and small ruminants



The exact site of rumenocentesis



Tympany cont'd---

and (0.4 – 0.5 cm) for small ruminants.

- Disinfect the end of cannula with iodine texture
- Then insert the trocar and cannula with strong biting on the site of trocarization with 45° in the direction of right thoracic cavity. Remove the cannula for some hours until the gas is removed
- In the case if the cannula is blocked by rumen content, remove by using cannula.
- For prevention of further production of gas, you can administer anti fermentative medicaments like tympanol and bloat remedy through trocar in dose of 300 – 500 ml for large ruminants and 50 – 100 ml for small ruminants
- Lastly remove the trocar cannula disinfect the wound with iodine texture.

2. If the bloat is frothy

Tympany cont'd---

foam, ruminal intubation and trocarization do not help the animals.

- But administration of antifoaming agents with drenching or through stomach tube or trocar cannula breaks down the stable foam.

- Results in eructation of gas through esophagus and intestine.

- The most common antifoaming or surfactants that are used to treat and prevent frothy bloat due to leguminous pasture and feed lot are

- 1. Mineral oil like **liquid paraffin oil** in dose of 250 – 500 ml.

- 2. It is also possible to use **vegetable oil and peanut oil** in dose of 200 ml of oil/ 60 kg BW.

- 3. Synthetic surfactants like **poloxalene**(oral suspension)

Tympany cont'd---

liter.

→5. Use **bloat remedy** in dose in dose of 180 ml/ 60 kg Of BW.

→6. Dimeticone (oral suspension in 1% or 2%) dose for cattle 100 ml and for sheep and goat 25 ml.

NB. If all the above methods do not help animals, you must do **ruminotomy**.

Prevention measures

→Avoid animals to feed large amount of green mass when there is a sudden change of feeding from dry mass to green mass.

→Do n't feed animals in pasture when there is dew.

→To prevent it give dry feed(straw and hay) before you allow to graze animals green mass.

→Avoid to give water before and after feeding of green mass

5. Traumatic reticulitis/ Traumatic reticuloperitonitis /Traumatic reticulopericarditis

- Perforation of the wall of the reticulum by a sharp foreign body like nail, wire and other sharp metallic materials initially produces an acute **local septic inflammation** of reticulum is called **traumatic reticulitis**.
- The disease is common in large ruminant animals.
- But rarely in sheep and goat.

Etiology

- Penetration of reticulum by metallic foreign objects such as nails and pieces of wire, including tire wire etc that were ingested by the animal and located in the reticulum.
- The metallic materials may penetrate to reticulum due to
 - The existence of different metallic perforating

TRP cont'd---

near different construction.

→Rarely licking of metallic materials, bones and rocks by milking cows and young animals due to Ca, P and microelements deficiency.

Epidemiology

Occurrence

- The disease is common in adult dairy cattle and feedlot animal.
- Because they are exposed frequently to this condition.
- However, it can also occur infrequently in yearlings , sheep and goat.

Contributory factors

➤**1. The feeding behavior of ruminants:** - The non-discriminatory feeding behavior of cattle contributes the occurrence of TR.

➤**2. Advanced pregnancy:** - Pregnancy of cows is one

TRP cont'd---

→ Because the pressing effect of gravid uterus on reticulum pushes/ forced the perforated metal to other sites.

Pathogenesis

- Lack of oral discrimination by cattle leads to the ingestion of foreign bodies that would be rejected by other species.
- Swallowed foreign bodies with feed may rarely lodge in rumen and omasum.
- But in most instances they pass the rumen and lodge in the reticulum.

➤ Why?

- This is because of the anatomical structure of reticulum mucosa which has honeycomb(net like structure).
- The honeycomb-like structure of the reticulum

The mucous structure of fore stomachs of ruminant animals



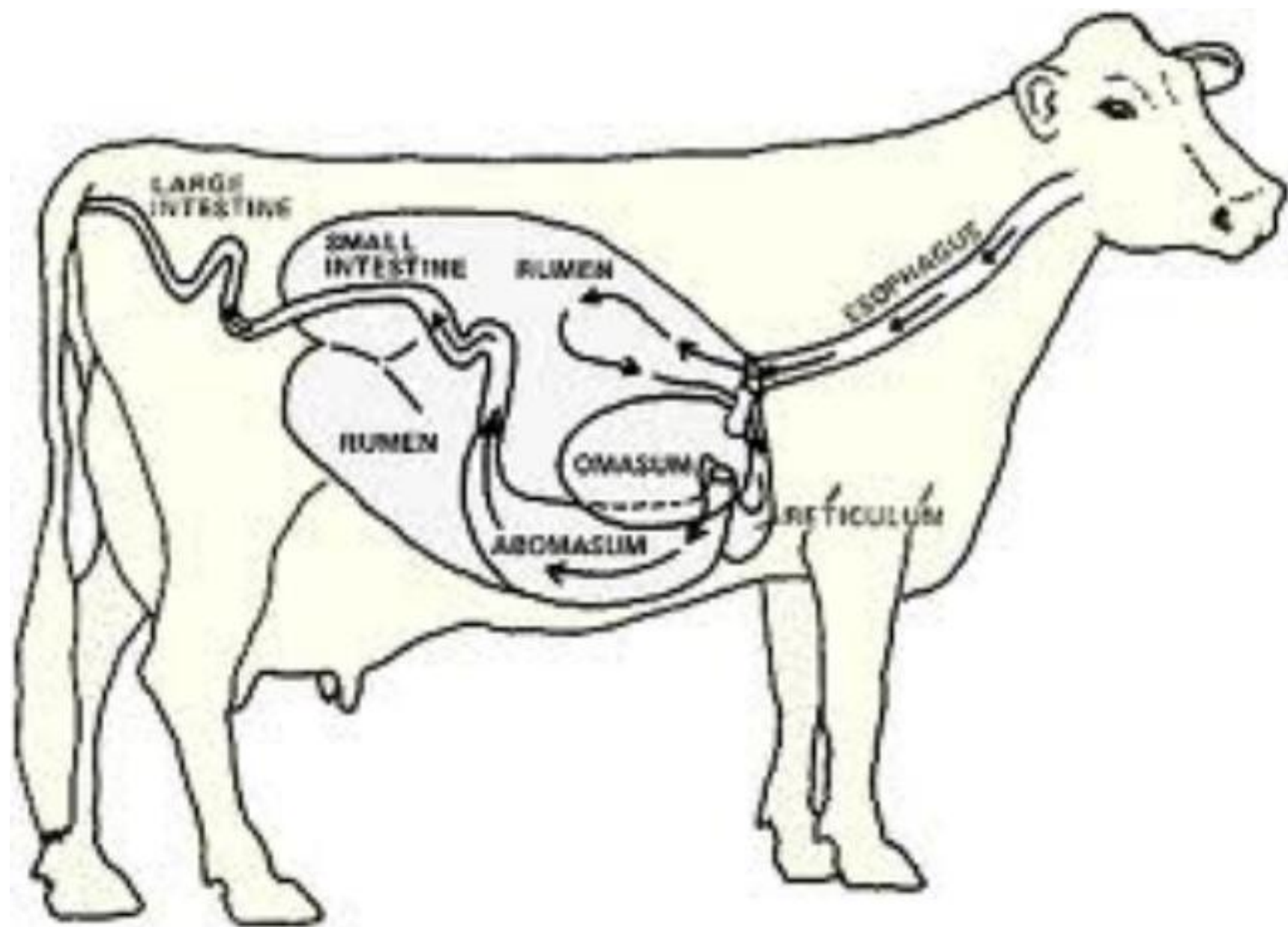
TRP cont'd---

causing harm.

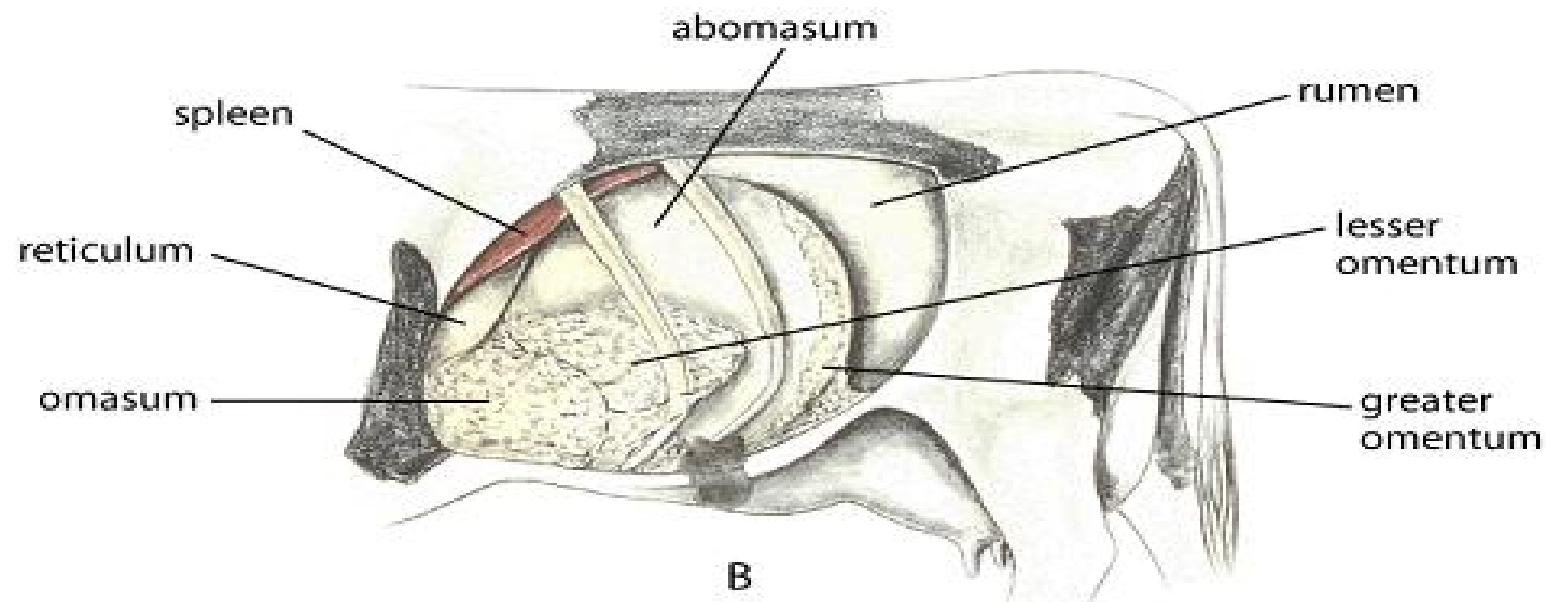
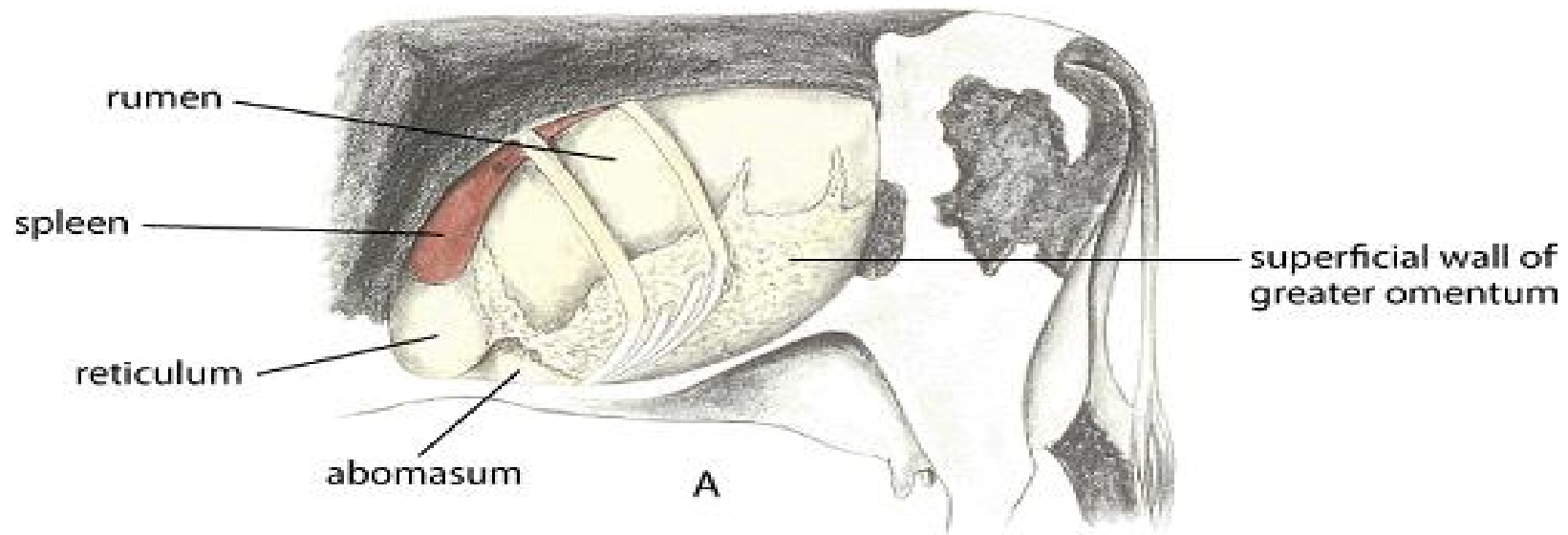
- But if a sharp - pointed objects are found in honey comb structure of the mucosa of reticulum,
- Its contraction push the metallic material to perforate the mucosa of reticulum.
- And cause **traumatic reticulitis**.
- If the reticular wall is injured without penetration to the serous surface,
 - No detectable illness occurs
 - And the foreign body may remain fixed in the site for long periods and gradually be corroded away.
- But in many causes the sharp - pointed metallic materials may perforate the myo and serous membranes of reticulum.
- And most perforations occur in the lower part of the cranial wall of the reticulum.

TRP cont'd---

- But some occur laterally in the direction of the spleen and medially towards the liver.
- Perforation may proceed beyond the reticulum to the peritoneum and cause involvement inflammation of peritoneum and cause **peritonitis(local or diffuse)**
- Further the metallic foreign body may injure different organs and membrane of the cavities.
- If the metallic material perforate the diaphragm and cause **diaphragmatic abscess or diaphragmatic hernia.**
- And further may perforate the lung and cause **pneumonia.**
- It may affect the pleura and mediastinal cause **pleurisy** and **mediastinitis** respectively.
- It may perforate pericardium of the heart and



Topography of organs of the digestive system of cattle





TRP cont'd---

- But if perforation occurs laterally in the direction of the spleen and medially towards the liver cause **spleenitis** and **hepatitis** respectively.
- NB. During perforation the metallic foreign bodies can carry microbes from one site to other and cause **septic inflammation**.
- As the result the microbes and their toxic products may be absorbed from focal foci and cause septicemia and toxemia.
- And there will be fever, neutrophilia shifting to the right and disturbance of the secretory and peristalsis of fore stomachs and intestine.

Clinical signs

- The disease appears suddenly with complete anorexia and a marked drop in milk yield in lactating cows.

TRP cont'd---

- Most animals prefer to remain standing for long periods and lie down with great care.
- Defecation and urination cause pain and the acts are performed infrequently and usually with grunting.
- This results in constipation, scant feces and in some cases retention of urine.
- Rarely, acute abdominal pain with kicking at the belly and stretching occurs.
- A moderate systemic reaction is common in acute localized peritonitis.
- And the body temperature may rise and ranges from 39.5 - 40°C.
- Rumination is absent and reticulorumen movements are markedly depressed and usually absent.

The rumen may appear to be full because of the

TRP cont'd---

➤ Traumatic pericarditis may continue to affect the myocardium and endocardium of the right part of the heart and is characterized

→ By highly pulsating jugular vein

→ As the result jugular vein is distended and visible

→ And there is brisket edema

Necropsy findings

→ Mostly in reticulum but rarely in rumen you may found metals like nails, needle and wires etc

→ In third day after perforation of reticulum, of the wall of reticulum becomes edematic and thickened.

→ In reticuloperitonitis the peritoneum is inflamed, thickened and hemorrhagic.

→ When perforated metallic material perforates diaphragm, pleura, heart and liver, there is

→ Inflammation of corresponding organs

Distended jugular vein during TR Pericarditis



TRP cont'd---

- The wall of reticulum may adhere with diaphragm, peritoneum and liver.
- Rarely on the way of perforation of metallic materials may form fistula made of connective tissue in which abscess or the metal it self is found.

Clinical pathology

- In acute local TRP, there is neutrophilia with shifting to the left called **regenerative left shift**.
- But in diffuse TRP, there is neutrophilia with **degenerative left shift**.

Diagnosis

- Diagnose of TR is made based on clinical signs like
- Sudden onset of the disease with clinical signs like
- Inappetance

TRP cont'd---

- Absence of rumination

- Hypotony of rumen

- Pain

- Further the disease can be diagnosed by

- 1. Eliciting pain

- Pain can be elicited

- Using deep palpation of the abdominal wall just caudal to the xiphisternum.

- Pinching the withers to cause depression of the back and eliciting a

- grunt is also an effective diagnostic aid.

- Allow the animal to move and walking downhill is accompanied by grunting because of pain in thoracic cavity during traumatic reticulo pericarditis.

- Other supportive diagnostic methods are

TRP cont'd---

- Rectal palpation of abdomen cause pain
- Laparoscopy during traumatic reticulo peritonitis
- Radiography of cranial abdomen and reticulum
- Ultrasonography of reticulum
- Metal detection

Differential diagnosis

- Traumatic reticulo peritonitis/ pericarditis must be differentiated at
 - Diseases of fore stomachs with clinical signs like hypotony and atony of fore stomachs (simple indigestion, acute carbohydrate engorgement and acute intestinal obstruction)
 - Pericarditis
 - Acute pleuritis
 - Postpartum septic metritis

TRP cont'd---

- Acute local peritonitis
- Pyelonephritis

Treatment

- Two methods of treatment are in general used
 - 1. Conservative treatment with or without the use of a magnet
 - 2. Ruminotomy

1. Conservative treatment with or without the use of a magnet

- ➡ Conservative treatment comprises
 - 1. Immobilization of the animal, this help
 - ↳ Facilitates for the formation of adhesion and healing
 - ↳ Reduce the pressure of abdominal organs to diaphragm
 - ↳ Lastly reduce pain

TRP cont'd---

➤ 3. Oral administration of a magnet to immobilize the foreign body

➤ Oral administration of a magnet to immobilize the foreign body is useful if

↳ The material is made up of iron

↳ It is found freely in the rumen

2. Ruminotomy

➤ Surgical removal of the foreign body through a ruminotomy incision is widely used as a primary treatment.

➤ It is used if the foreign body is found in rumen and in reticulum.

➤ **NB.** If the above measures do not help the animal, slaughter the animal.

Prevention measures

TRP cont'd---

of electromagnetic instruments

- Clean the pasture area from metallic nails, wires and needles etc as far as possible.
- To prophylaxis traumatic reticulitis, use magnetic ring.

4.3. Disease of abomasum and stomachs of monogastric animals

➤ Here we will focus on

- 4.3.1. Impaction of abomasum
- 4.3.2. Gastritis and abomasitis

4.3.1. Impaction of abomasum

➤ Impaction abomasum is the disease of ruminants caused by consumption of excessive quantities of poor-quality roughage which are low in both digestible protein and energy.

➤ The disease is common in beef cattle feed in winter

Impaction cont'd---

Etiology

- Ingestion of large quantities of low-quality roughage.
- Impaction of the abomasum with sand can also occur in cattle if they are fed hay on sandy soils or root crops that are sandy or dirty.
- The ingestion of gravel (stones) by dairy cattle kept in dry-lot facilities.
- Because the gravel, consisting of sand and small stones, may be inadvertently mixed with the feed when it is being scraped from bunker silos.
- It is also possible that some cows may ingest the gravel through pica.
- Impaction of abomasum is more prevalent in intensive farms in winter season.
- When the animals' ration contains only roughage with no or with small amount of grain

Impaction cont'd---

- To satisfy the calorie requirement of animals, the owners chopped the roughage and mixed with mill by products.
- So the animal eat large amount of chopped roughage and mill by products that contain more bran.
- Because the smaller particles pass through the forestomachs at a more rapid rate.
- As the result impaction of the abomasum occurs.

Pathogenesis

- You know chopped roughage and finely ground feeds pass through the forestomachs of ruminants more quickly than long roughage.
- However, as the feeds have low calorie value the animals may take large amount these chopped feeds.
- But they have low digestibility.
- As the result they accumulate in the fore stomachs of

Impaction cont'd---

- All this results in impaction of omasum and abomasum.
- Other reason for impaction of omasum and abomasum is ingestion of large quantities of sand.
- The sand that accumulates in the abomasum causes abomasal impaction, atony and chronic dilatation.
- The impaction of abomasum results in
 - ↳ 1. Dysfunction of the upper alimentary tract
 - ↳ 2. Irritation of the mucous membrane of abomasum that causes the continuous secretion of **hydrogen and chlorine** from the wall of abomasum.
- This results in **hypochloremia** and in **loss of hydrogen ion** from the body of the organism.
- All these result in the development of **alkalosis**.
- ↳ 3. Varying degrees of dehydration occur
- Because fluids and nutrients are not moving beyond the abomasum.

Impaction cont'd---

into the duodenum for absorption.

- Potassium ions are also sequestered in the abomasum, resulting

in a **hypokalemia**.

- ➡ Lastly the abomasal impaction can cause permanent abomasal atony.

➡ This results in

Dehydration

Alkalosis

Electrolyte imbalance and

Progressive starvation

Clinical findings

- The onset is usually slow and progressive over a period of several days.

¬Anorexia, scant feces and moderate distension of the

Impaction cont'd---

the usual presenting complaints given by the owner.

- The body temperature is usually normal
- The heart rate varies from normal to 90-100/min and may increase to 120/min.
- The respiratory rate is commonly increased and an **expiratory grunt** due to the abdominal distension may be audible.
- In advanced cases where **alkalosis, hypochloremia** and **dehydration** are marked.
- A mucoid nasal discharge usually collects on the external nares and muzzle.
- The muzzle is usually dry and cracking ,because of
 - ↳ The failure of the animal to lick its nostrils and
 - ↳ The effects of dehydration.
- The rumen and other forestomachs are usually static and full of dry rumen contents

Impaction cont'd---

Necropsy findings

- At necropsy the abomasum is commonly grossly enlarged to up to twice normal size and impacted with dry rumen-like contents.
- The omasum may be similarly enlarged and impacted with the same contents as in the abomasum.
- The rumen is usually grossly enlarged and filled with dry ruminal contents.
- The intestinal tract beyond the pylorus is characteristically empty and has a dry appearance.
- Varying degrees of dehydration and emaciation are also present.
- If rupture of the abomasum occurs, lesions of acute **diffuse peritonitis** are present.

Clinical pathology

- The most common clinical pathological changes that

Impaction cont'd---

- ‖ Metabolic alkalosis
- ‖ Hypochloremia
- ‖ Hypokalemia
- ‖ Increase in hematocrit value of blood (PCV) due to dehydration.

Diagnosis

- Diagnose can be made based on history and clinical signs
- Typical clinical signs are
 - The impacted abomasum is usually situated in the right lower quadrant of the abdomen on the floor of the abdominal wall which is usually distended.
 - Deep palpation and strong percussion of the right flank may elicit a 'grunt'.
 - This is probably due to over distension of the abomasum.

Impaction cont'd---

- Severely affected cattle will die in 3 - 6 days after the onset of signs.
- Rupture of the abomasum has occurred in some cases and death **from acute diffuse peritonitis**.
- There is a marked **hypochloremic, hypokalemic metabolic alkalosis** that can be confirmed in laboratory.

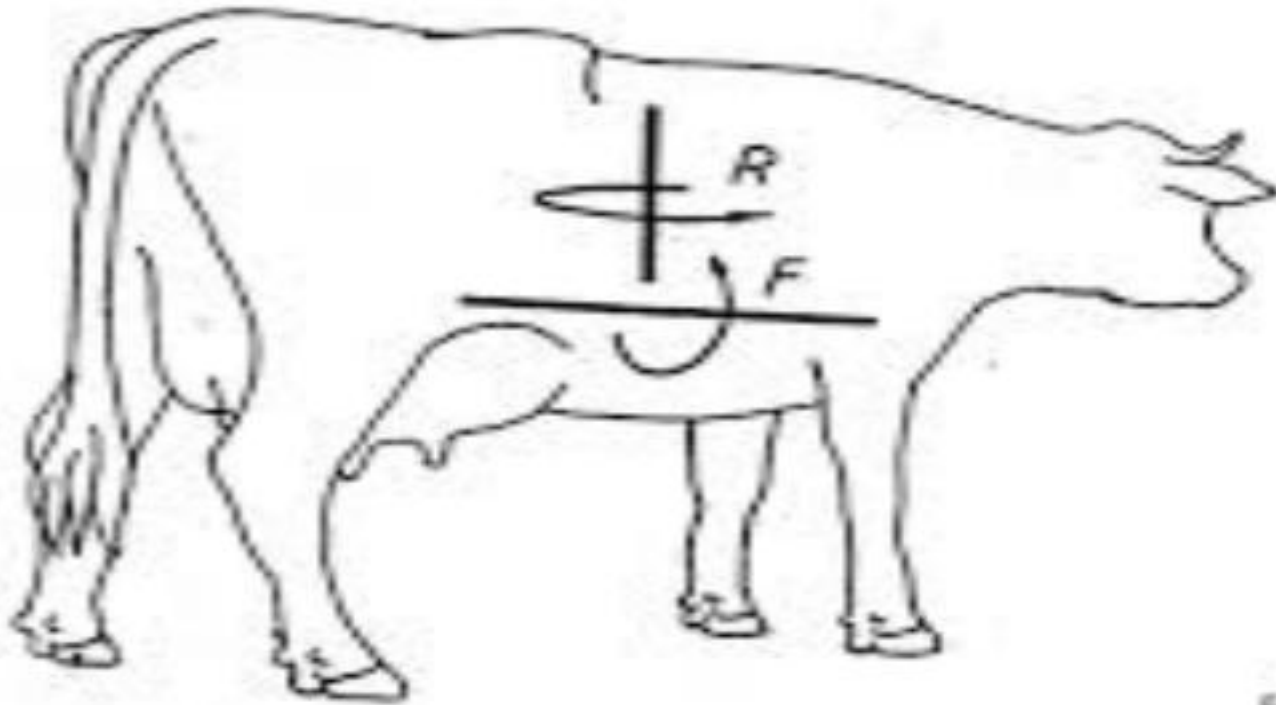
Differential diagnosis

- Impaction of abomasum must be differentiated at

1. Impaction of omasum

- It can be differentiated from impaction of abomasum in that during impaction of omasum there is
 - Normal rumen movements
 - Moderate dehydration
 - Enlarged omasum that may be palpable per rectum or

Impaction cont'd---



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Impaction cont'd---

‖ The serum electrolytes may be within normal limits if the abomasum is normal.

2. Diffuse peritonitis

‖ Diffuse peritonitis is differentiated from impaction of abomasum in that there is

‖ Grunt on deep palpation and percussion of abdominal wall.

‖ Paracentesis may yield some diagnostic peritoneal exudate during diffuse peritonitis.

3. Intestinal obstructions

‖ Abnormality may be palpable on rectal examination during intestinal obstruction.

‖ Fluid and gas accumulations in the intestines anterior to the obstruction may be detectable by rectal palpation

4. Impaction of the abomasum as a complication of traumatic reticuloperitonitis

Impaction cont'd---

↳ Cattle with abomasal impaction as a complication of traumatic reticuloperitonitis are usually a single incident and have usually been ill for several days.

↳ Whereas those with dietary impaction have usually been ill for only a few days and more than one may be affected.

Treatment

➤ Treatment of animals during impaction of abomasum depends on the actual healthy condition of the animal.

- 1. If the animal is weak and tachycardic with pulse rate of > 100 - 120/ min are poor treatment risks and should be slaughtered
- 2. If the animal is in good condition, it may be possible to treat.

➤ The rational treatment would appear to consist of

↳ A. Correcting the metabolic alkalosis

Impaction cont'd---

↳ D. Attempting to move the impacted material with lubricants and cathartics(Purgatives)

↳ E. Surgically emptying the abomasum

A. Correcting the metabolic alkalosis, hypochloremia, hypo kalemia and dehydration

➡ Metabolic alkalosis can be corrected by IV administration of sterile acidifying isotonic solutions of

↳ High - potassium acidifying solutions that contain

↳ Isotonic saline solution plus 2.5 gm of KCl/ liter of solution . Or

↳ Mixture of 1 liter of isotonic potassium chloride (1.1%) ,2 liters of isotonic saline (0.9%) and 1 liter of 5% dextrose

➡ The above electrolyte solutions are infused intravenously on a continuous basis for up to 72 hours at a rate of 1000 - 2000 ml or 500 - 1000 ml for sheep

Impaction cont'd---

that contains mixtures of

Ammonium chloride (NH_4Cl) and potassium chloride (KCl) at a rate of 20 liter per 24-hour period for a 450 kg animal.

B. Attempting to move the impacted material with lubricants and cathartics(purgative)

➤ It is method of treatment directed to move the impacted material to ward duodenum

→ By using purgative(laxatives)

→ By stimulating the tones of abomasum by using cholinergic preparates.

➤ The most common purgative that is used to treat impaction of abomasum is **25% of Dioctyl Sodium Sulfosuccinate.**

Dose and route of administration

Administered into the rumen by stomach tube at a

Impaction cont'd---

and 10 liters of mineral oil(paraffin oil)of for a 450 kg animal repeated daily for 3-5 days until recovery is apparent.

→Attempt was done to use **cholinergics** such as **neostigmine**, **physostigmine** and **carbamylcholine** have been used but appear not to alter the outcome.

C. Surgically emptying the abomasum

→Surgical correction consists of an abomasotomy through a right paramedian approach and removal of the contents of the abomasum.

→The results are often unsuccessful, probably because of abomasal atony that exists and that appears to worsen following surgery.

→An alternative approach may be to do a ruminotomy, empty the rumen and infuse **dioctyl sodium sulfosuccinate** directly into the abomasum.

Impaction cont'd---

specially during windy, cold winter season.

→ Because in a cold windy weather the calorie requirement for maintenance is increased.

→ And the animal consumes large amount of low quality roughage feeds.

→ To solve this problem the roughage should be analyzed for crude protein and digestible energy.

→ Based on the analysis, grain is usually added to the ration to meet the energy and protein requirements.

➤ Avoid to feed animals which is mixed with sand and gravel.

➤ Feed animals with the correct ration formulation based on their physiological needs.

➤ Supply adequate amounts of fresh drinking water at all times.

Because it is extremely hazardous if the animals are

Impaction cont'd---

roughage with out adequate supplying of water.

4.3.2. Gastritis and abomasitis

- It is inflammation of the mucous membrane of stomach of mono gastric animals characterized by disturbance of its secretory, musculatory and other essential function of stomachs.
- While inflammation of the mucous membrane of abomasum in ruminant animals is called **abomasitis**.
- Gastritis and abomasitis can be
 - Acute
 - Chronic
- By types of exudate gastritis and abomasitis are mostly **catarrhat** type.

Etiology

- The causative agent of gastritis in monogastric

Gastritis cont'd---

- | 1. Primary factors
- | 2. Secondary factors

1. Primary factors

- Insufficient moistening and mechanical digestion of rough feeds because of abnormality on teeth and oral cavity.
- Quickly swallowing of rough feeds that are not mechanically digested in the oral cavity
- Feeding of animals easily indigestible rough foods for long time.
- Feeding of animals which are contaminated with fungi and soil.
- Feeding of animals feeds that are very hot or cold.
- Feeding of animals with toxin plants(phytotoxin producers)
- Feeding of animals which are contaminated with

Gastritis cont'd ---

→Gastritis that occurs because of the disease of other organs(chemostasis, caprostasis, obturation, invagination, thrombosis and during some infectious diseases).

Pathogenesis

- Different materials that cause gastritis(abomasitis) enters to the lumen of stomach(abomasum) irritate the mucous membrane of stomach(abomasum).
- As the result the secretory, muscularity(peristalsis) and digestive function will be disturbed.
- Inflammation of the mucosa of stomach(abomasum) causes to accumulate exudate in the lumen of stomach(abomasum).
- All this condition creates favorable condition for growth and multiplication of bacteria.
- The bacteria may acts on undigested food and

Gastritis

➤ Experimentally it is known that there are three types of gastritis in equines.

➤ These are

 \ 1. Hyper acidic

 \ 2. Sub acidic

 \ 3. Inert type

1. Hyperacidic

➤ It is a type of gastritis characterized by increasing the total acidity

of stomach (free and conjugated HCl).

➤ It is the initial stage of gastritis characterized by retaining of feeds in the stomach and abomasum.

➤ This leads to **constipation** and an **increased bacterial decomposition** of the feed.

➤ The retained feed irritate the mucosa of stomach and

Gastritis

- Irritation stimulates vomiting center and cause **eructation** and **vomiting**.

2. Sub acidic

- Characterized by a decreased amount of the total acid in stomach.
- The content of stomach has mucous and absent free HCl content but has conjugated HCl.

3. Inert type

- Characterized by absence of secretion of HCl from the wall of stomach.
- In sub acidic and inert type gastritis, the amount of total and free HCl is decreased.
- This favors
 - For the disturbance of stomach digestion
 - Absence of bactericidal characteristics of gastric juice

Gastritis

results in bacterial decomposition of feed and production of toxins.

➤ The large amount of toxins will be absorbed in the liver and disturbs the detoxification and other functions of liver.

➤ Long time irritation of the mucosa of intestine of small intestine and large intestine may cause **enteritis** and **colitis** respectively. Or

➤ Inflammation of both small and large intestine called **enterocolitis**.

➤ So the inflammation of both stomach, small intestine and large intestine called **gastroenterocolitis**.

➤ The disturbance of stomach and intestine digestion affects the absorption of digested feed and vitamins specially retinol, ascorbic acid and vitamin B complex

Gastritis

Clinical signs

➡ Gastritis in equines and abomasitis are characterized by the following clinical signs.

→ Depression

→ Nervous

→ Inappetance up to anorexia

→ Emaciation

→ Mucous membrane of oral cavity is covered by high viscous saliva.

→ Bad smelling gas comes through oral cavity.

Gastritis

- Rarely icterus of conjugative and sclera.
- Hypoacidic type gastritis accompanied with diarrhea
- While hyperacidic is with constipation.
- As the result the faces is scanty, dry and has black colour and is covered by mucous.
- Gastritis of omnivore(pigs) and carnivores animals is characterized by
 - Eructation which is not present at norm
 - Vomiting soon after swallowing of feed and water
 - Vomited food and water is mixed with mucous that has high viscous
 - Rarely it may be mixed with blood
 - Repeated vomiting may cause mixing of vomited food with bile

Gastritis

- Haemorrhagiae of the mucosa.
- Infiltration of sub mucosa of stomach
- Histopathological there is degeneration of glands and their duct which produce pepsinogen.

Diagnosis

- Diagnose is made based on
 - Anamnesis
 - Clinical signs
 - Result of laboratory diagnose of gastric juice

Treatment

- Remove the causes of gastritis
- Wash the stomach of monogastric animals with 1 - 2 % NaHCO_3

Gastritis

➤ If the gastritis is hyperacidic type: -

→ Give the animals soft and easily digestible feeds like green mass and sup with out fiber.

➤ But if the gastritis is hypoacidic type: -

→ Give the animals feed that stimulate secretory function of stomach(like sup that has fiber).

➤ In addition to the above you can use pharmacological prepares.

➤ During constipation due to hyperacidic gastritis:-

→ You must use **osmotic purgatives** like 2% MgSO_4 or Na_2SO_4 or laxatives.

→ And you must administer broad spectrum antibiotics orally to control the growth of microbes.

➤ During hypoacidic and inert type of gastritis:-

→ You can administer natural or synthetic gastric juice

4.4. Enteritis

- You know the term enteritis is used to describe inflammation of the intestinal mucosa.
- Inflammation of intestinal mucosa results in
 - Diarrhoea and sometimes dysentery with abdominal pain.
 - Occasionally with varying degree of dehydration, electrolyte and acid – base imbalance.

Etiology

The cause of enteritis can be

- 1. Biological
- 2. Physical
- 3. Chemical
- 4. Nutritional deficiency
- 5. Dietary causes

Enteritis cont'd---

1. Biological

- Biological agents that cause enteritis are bacteria, virus, fungi and parasites of GIT
- The most common diseases that cause enteritis are

1. Bacterial

- *Enterotoxigenic E. coli (ETEC)*
- *Salmonella*
- *Clostridium perfringens*
- *Mycobacterium paratuberculosis etc*

2. Viruses

- Rotavirus infection
- Corona virus infection
- Rinder pest
- Malignant catarrhal fever etc

3. Parasites

- Emeriosis (Coccidiosis)
- Ostertagiasis
- Cryptosporidiosis(*Cr. parvum*)
- Strongylidiosis etc

2. Physical agents

- Sand soil

3. Chemical agents

- Arsenicnic,
- Fluorine,
- Copper and molybdenum
- Nitrates
- Poisonous plants
- Mycotoxicosis etc

Enteritis cont'd---

4. Nutritional deficiency

- Copper deficiency conditioned by molybdenum excess
- Iron deficiency in piglets

5. Dietary causes of enteritis

- Overfeeding
- Simple indigestion

Pathogenesis

➤ Pathogenesis of enteritis and its effect on the organism can be explained as follows

➤ A) Mechanisms of enteritis

- Under normal conditions, large quantity of fluid enters in to small intestine from the saliva, stomach, pancreas, liver and intestinal mucousa.
- The fluid including electrolytes should be absorbed mainly through small intestine.

Enteritis cont'd---

- In the horse, digestion and absorption may occur in the large intestine.
- The brush border membrane of the **villous epithelia cells** is of paramount in nutrients absorption.
- Any dysfunction of the intestines results failure of adequate **absorption** and cause **diarrhea**.

➤ **B) Location of lesions**

- The location of the lesion in the intestinal tract may also influence the severity of the enteritis.
- Lesions involving the small intestine are considered to be more acute and sever than those in large intestine.
- Because approximately 75 - 80% of the intestinal fluids are absorbed by the small intestine and much lesser quantities by the large intestine.
- Thus, in general, when lesions of large intestine is predominate, the

Enteritis cont'd---

, the fluid and electrolyte losses are not as acute nor as severe as when the lesions of the small intestine predominate.

➤ **NB.** Nevertheless the horse is an exception in that 95% of fluids and electrolyte are absorbed in the large intestine.

➤ There fore any significant dysfunction of the absorptive mechanism of the large intestine of the horse would result in large loss of fluid and electrolyte.

C) Dehydration, electrolyte and acid-base imbalances

➤ You know the net effect of an increase in the total amount of fluid in the intestinal lumen and reduction in intestinal absorption results

→ In a loss of fluids and electrolytes at the expense of body fluid and electrolyte.

Enteritis cont'd---

↓ Protein.

- Which results as follows
- Loss of protein results in **hypoproteinaemia**
- Loss of bicarbonate results in **metabolic acidosis**
- The loss of sodium, chlorine and potassium results in **serum electrolyte imbalance of the above electrolytes.**

➤ **D) Chronic enteritis**

- In chronic enteritis
 - The intestinal wall becomes thickened because of infiltration
 - The mucus secretion is stimulated and
 - The absorption of intestinal fluids is decreased.
 - Which results in negative nutrient balance because of decreased digestion of nutrients and decreased

Enteritis cont'd---

- But not of the same magnitude as in acute enteritis.
- In some cases of chronic enteritis, depending on the cause and there is: -

- A continuous loss of protein leading to clinical hypoproteinaemia.

- Good examples of chronic enteritis are **intestinal helminthiasis** of all species and **John's disease of ruminants** are classical examples.

➤ E) Replacement of villous epithelial cells

- The villous absorptive epithelial cells of the small intestine are involved in

- Almost every type of enteritis or

- Malabsorptive syndrome

- These cells which line the villi and face the lumen of the intestine contain important digestive enzymes such as **disaccharidase**.

Enteritis cont'd---

- Almost any noxious influence can increase the rate of extrusion of them and are then replaced by immature non-absorptive cells.
- The villi become shortened cells, which (villous atrophy) and chronic malabsorption occurs.

Clinical findings

- The clinical signs that are manifested during enteritis depending on either it is acute or chronic.
- However, the most common clinical signs during enteritis are
 - Diarrhea (major clinical sign)
 - Dehydration
 - Abdominal pain
 - Septicemia, toxemia and fever (especially in infectious enteritis)

Enteritis cont'd---

- May have unpleasant odor
- May contain blood (dysentery)
- May contain fibrinous casts and mucus or foreign material such as sand

➤ **2. In chronic enteritis** , there is

- Progressive weight loss and emaciation
- No systemic abnormalities

➤ **3. In parasitic enteritis and abomasitis**, there is

- Hypoproteinaemia
- Subcutaneous edema(for example bottle jaw)

Diagnosis

Diagnosis of enteritis is done based on

- History
- Clinical signs

Enteritis cont'd---

- Fecal examination

- Feces of animals must be examined during enteritis as it helps

- To determine the specific cause(infectious, parasitic and other diseases)

- To check the consistency of the fecal

- To check the presence of blood (dysentery), mucus, epithelial tissue or foreign bodies

- Other laboratory methods that must be performed are

- Hematology and serum biochemistry

- Serology and intestinal biopsy (to determine the specific cause especially in viral infections)

Treatment

- The general principles of treatment of enteritis consists of

Enteritis cont'd---

➤ It is treatment of enteritis by removing the causative agents which includes the use of

➤ **Anthelmintics** : - For the treatment of helminthiasis

➤ **Antiprotozoal drugs**: - Drugs for treatment of intestinal parasitic protozoa such as anticoccidial preparates

➤ **Antimicrobials**: - Drugs for the treatment of bacterial enteritis

| **2. Alteration of the diet**

➤ It is the method of treatment of enteritis by changing the feed of animals (for dietary enteritis)

| **3. Fluids and electrolytes**

➤ It is treatment of enteritis directs for substitution of fluid and electrolyte that are lost due to sever diarrhea.

➤ That means for the treatment of dehydration and electrolyte imbalances.

Enteritis cont'd---

- It is method of treatment of enteritis directed to coat the mucosa of intestinal wall and to reduce intestinal secretions.
- For example **kaolin and pectin** mixtures are used in animals with enteritis to coat the intestinal mucosa, inhibit secretions and decrease the bulk of the feces.

| 5. Antidiarrheal drugs

- These are drugs used to decrease diarrhea.

- The most common are

| 1. Antimotility drugs

| 2. Antisecretory drugs

1. Anticholinergic drugs

- These drugs which are administered to decrease the motility of intestinal wall.
- Because the anticholinergic drugs block the action of acetylcholine (ACh)

Enteritis cont'd---

on smooth muscle and glands.

- This results in decreased gastric secretion and emptying and reduction on both segmental and propulsive movements in the intestines.

2. Antisecretory drugs

- These are drugs which reduce the secretory function of GIT
- For example **chlorpromazine**.

4.5. Acute intestinal obstruction

- This is the occlusion(blocking) of the intestinal lumen either from
 - External pressure
 - Internal blockage or
 - Due to intestinal accidents
- It is characterized by **abdominal pain** and **shock**

Intestinal obstruction Cont'd---

Etiology

➤ The commonest causes of acute intestinal obstruction are

- | 1. Intestinal accidents
- | 2. Luminal blockages

1. Intestinal accidents

➤ The intestinal accidents are

➤ **Intussusception** – is the invagination of a portion of the intestine into an adjacent segment.

➤ **Torsion** – is obstruction of the intestine caused by the bowel twisting on its own long axis.

➤ **Volvulus** – is intestinal obstruction caused by segment of bowel twisting on its mesenteric axis.

➤ There is also a case when a loop of intestine passes through a tear in the mesentery.



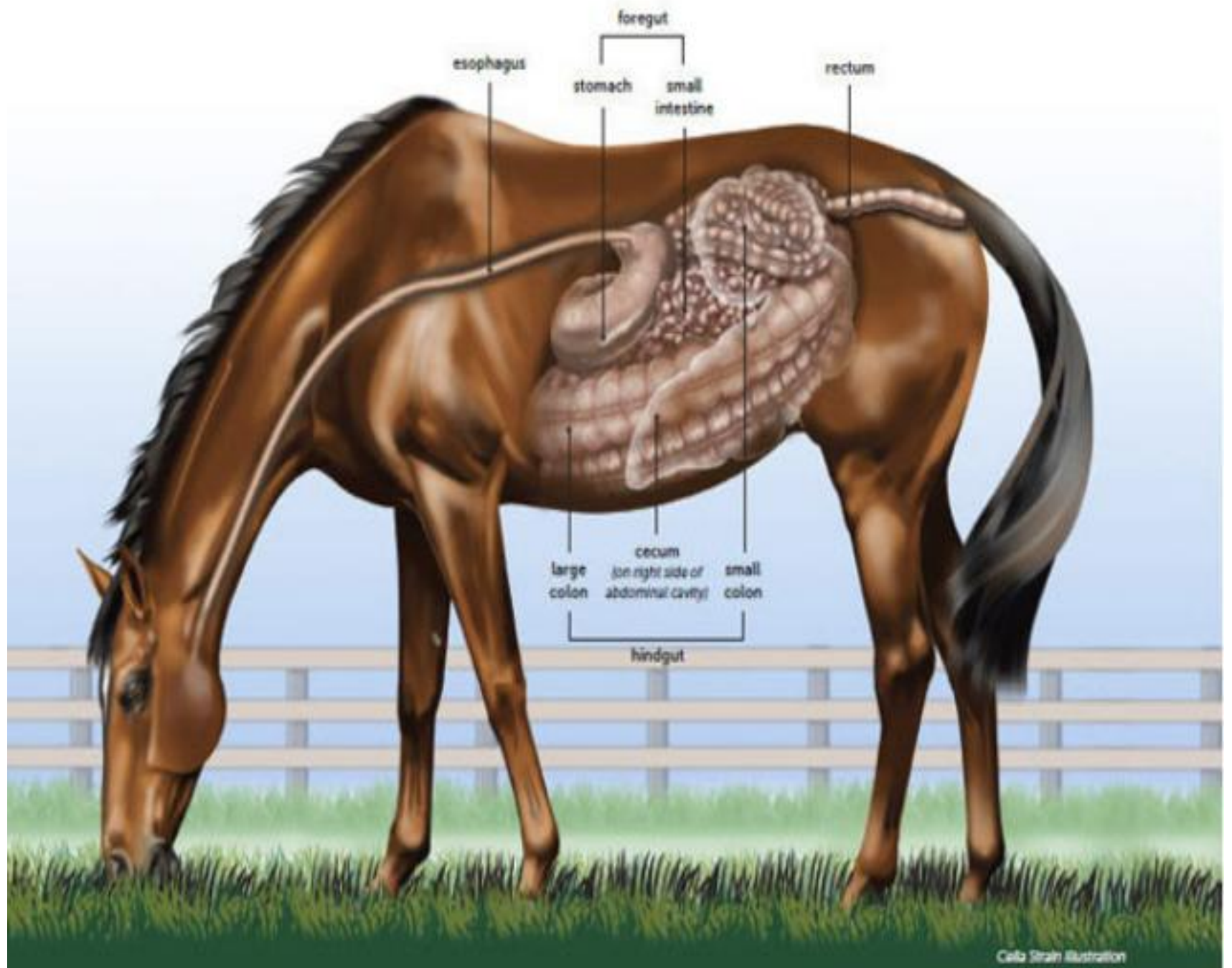


Intestinal obstruction Cont'd---

- **Incarceration** - refers to the displacement and entrapment of a segment of bowel of intestine, as **in hernia** (Eg. **umbilical, inguinal, diaphragmatic etc**).
- **Strangulation** – it is invagination of portion of intestine by ligaments.
- **Intussusception** usually results from irregular or excessive peristaltic movements, caused by such problems as enteritis, intestinal parasites, dietary disorders and bowel tumors.
- **Volvulus and torsions** occur most frequently in horses during severe exercise, jumping and rolling.
- Volvulus is common **in the small intestine**, and torsion in the large **intestine and cecum of equines**.

2. Luminal blockages

- It can be



Intestinal obstruction Cont'd---

└ Extra luminal

➤ The most common intraluminal intestinal obstructions are

➤ **1. Enteroliths** - Intra luminal blockage of intestine due to concretion of minerals such as Magnesium.

➤ **2. Phytobezoars**- Intra luminal blockage of intestine due to aggregation of plant materials in the intestine

➤ **3. Trichobezoar** - Intra luminal blockage of intestine due to hair balls.

➤ **4. Parasites** - Intra luminal blockage of intestine due to intestinal helminths. Eg *Parascaris equorum* in equines and *Ascaris gallinarium*.

➤ The common sites of intra luminal blockages/obstruction of intestine are:

→ Ileocaecal valve

→ Pelvic flexure of the large intestine

Intestinal obstruction Cont'd---

- Pyloric sphincter of stomach
- The most common cause of extra luminal obstruction in intestines are **tumors** in other organs of abdominal cavity.

Clinical findings

- The most common clinical signs that are absorbed during acute intestinal obstruction are
 - Severe abdominal pain (colic)
 - Kicking of the abdomen
 - Getting up and down
 - Rolling and flank watching
 - Abnormal postures Eg dog-sitting postures
 - Severe toxemia
 - Increased respiratory, heart rates and sweating
 - Distended loops of intestine can be felt rectally

Intestinal obstruction Cont'd---

- Absence of defecation
- Abdominal distension

Diagnosis

- Diagnosis is usually based on clinical signs like
 - Absence of defecation
 - Distended loops of intestine palpated rectally
 - Signs of pain

Treatment

- Treatment of intestinal obstruction can be made singly or in combination of using
 - Pain reliever like analgesics
 - Surgical correction (exploratory laparotomy)
 - Correction of fluid and electrolyte and acid-base imbalances
 - Using anti inflammatory drugs like that of NSAID

Colic cont'd---

→Laxatives(lubricant or bulk forming) or osmotic laxatives in cases of luminal blockages

4.6. Equine colic

- Colic is not a disease.
- But it is a sign of abdominal pain.
- Colic can be defined as any malfunction, displacement, twisting, swelling, infection or lesion of digestive system.
- Or a syndrome caused by digestive tract disorders and manifested by clinical signs of acute and sub acute abdominal pain and depression in horses is commonly referred to as colic.
- Colic is a frequent and important cause of death and is considered as the most important disease of horses for practitioners

Colic cont'd---

- Chronic (>24-36 hours)
- Recurrent (multiple episodes separated by periods of > 2 days of normality).

Etiology

➤ Colic can be classified based on its cause as:

- 1. Obstructive
- 2. Obstructive and strangulating
- 3. Infarctive
- 4. Inflammatory (peritonitis and enteritis)

1. Obstructive colic

➤ Obstructive colic is a colic in which there is obstruction to the passage of ingesta without ischemia or strangulation of the intestine.

➤ In the terminal stages there is often ischemia caused by distension of the intestine.

Colic cont'd---

- Small intestine obstructive lesions include

- Ileal hypertrophy

- Ileocaecal intussusception

- Foreign body obstruction of the lumen

- The course of the disease is about 24 - 72 hours and sometimes longer depending on the extent of obstruction.

- Large intestine obstructive lesions include impaction and simple (non-strangulating) displacements of the large colon.

- Stasis of intestinal movement can also cause obstructive colic.

- Which results in constipation due to absorption of its moisture content.

- Obstruction of small intestine by its content of feed is called **chemostasis**.

Colic cont'd---

2. Obstructive and strangulating colic

- Diseases that cause both obstructive and strangulation as an initial event, such as torsion and volvulus of the small intestine or large colon, result in severe pain.
- Obstruction causes **distension** and strangulation causes **ischemia, loss of barrier function** and **endotoxemia**.
- These diseases have a short course, usually less than 24 hours and sometimes as short as 6 hours with profound clinical signs.
- Endotoxemia and cardiovascular collapse are characteristic to these diseases.

3. Infarctive colic

- Infarctive diseases, like **thromboembolic colic**, are characterized by **ischemia** of the intestinal wall.

Colic cont'd---

- Finally it causes endotoxemia and distension of the bowel.
- The course of the disease is usually less than 48 hours and is terminated by cardiovascular collapse and death.

4. Inflammatory colic

- Inflammation of the intestine or peritoneum (enteritis or peritonitis) alters gastrointestinal: -
 - Motility and absorptive function
 - Which results in accumulation of fluid and ingesta
 - Results in abdominal distension and pain.

Clinical findings

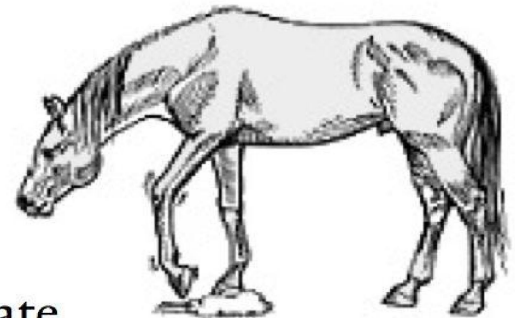
- Severe abdominal pain (colic) in equines has the following clinical signs
 - Kicking of the abdomen



HORSE HEALTH EDUCATION: COLIC

RECOGNIZING COLIC

- Leaving food; absence of appetite
- Repeatedly lying down
- Turning the head toward the flank
- Pawing
- Kicking or biting at the belly
- Repeated rolling
- Sitting in a dog-like position
- Stretching out; posturing to urinate









HORSE HEALTH EDUCATION: COLIC

TREATMENT



- Pain-relievers (analgesics or sedatives)
- Fluid therapy by intravenous or nasogastric routes
- Laxatives (mineral oil)
- Tranquilizers (acepromazine, detomidine)
- Surgery

Colic cont'd---

- Rolling and flank watching
- Abnormal postures Eg dog-sitting, “saw-horse stance”
- Severe abdominal distension
- Projectile vomiting or regurgitation of intestinal contents through the nose – this sign usually suggests terminal state.
- Increased respiratory, heart rates and sweating
- Loud **borborygmics** sounds may be present

Diagnosis

- Diagnosis of colic in equines is based on
 - Clinical signs
 - Abdominal auscultation
 - Rectal palpation
 - Paracentesis

Treatment

Principles of treatment of equine colic

1. Provision of analgesia (relief of pain)

- Analgesia is important in that it relieves
 - The horse's discomfort
 - Minimizes the pain and
 - Permits the veterinarian for easily examination.

- Common analgesics which commonly used are

- Non-steroidal anti-inflammatory drugs (NSAID)

↳ Like metamizole sodium and flunixin meglumine

2. Correction of fluid, electrolyte and acid-base imbalances

- Horses with evidence of dehydration or electrolyte imbalances should be given IV fluids preferably

isotonic poly ionic fluid such as **Ringer's solution**.

3. Gastrointestinal lubrication or administration of fecal softeners

- The commonly used intestinal lubricant is mineral oil like neutral paraffin
- But it should be given only through a nasogastric tube as its aspiration is associated with severe and fatal pneumonia.

4. Other treatments

- Antibiotics per os
- Trocarization – this may be done occasionally in cases of flatulent colic due to intestinal torsion where abdominal distension is impairing respiration.
- Surgery – the only definitive treatment for many causes of equine colic is surgical correction but it needs appropriate trained personnel and surgical facilities.

Colic cont'd---

- Causing excessive gas flatus to be created in the stomach and intestines.

Prevention measures of equine colic

- Management plays very important role in prevention of equine colic which includes
 - Establishment of regular feeding and exercise routine
 - Feed high quality forage
 - Avoid feeding excessive grain or energy rich diets
 - Divide concentrate rations into two or more feedings rather than on
 - Set up regular parasite control program
 - Provide exercise or turnout on a daily basis
 - Provide fresh, clean water (except when horse is hot from vigorous exercise)
 - Avoid medications unless they are prescribed by

Colic cont'd---

especially pain-relief drugs (analgesics).

- Because a few NSAID like metamizole may cause ulcers in GIT if they are administered for a long period of time
- Avoid feeding on ground, especially in sandy area
- Do not let horse to graze pastures which are short and are in sandy soils
- Make dietary and management changes gradually
- Reduce stress, horses experiencing changes in the environment or workloads are at a high risk of internal dysfunction.

4.7. Some of manifestations of alimentary tract dysfunction

➤ Manifestations of alimentary tract dysfunction are

↳ 4.6.1. Dysphagia

Dysfunction cont'd---

\ 4.6.3. Regurgitation

\ 4.6.4. Hypermotility and hypomotility of GIT

\ 4.6.5. Abdominal distension

\ 4.6.6. Diarrhea

4.6.1. Dysphagia

➤ You know in veterinary physiology that swallowing is a complex act governed by reflexes mediated through the **glossopharyngeal, trigeminal, hypoglossal** and **vagus nerves**.

➤ A defect in the nervous control of the reflex or a narrowing of the lumen of the pharynx or esophagus may interfere with swallowing.

➤ This interference of swallowing is called **dysphagia**.

➤ **Dysphagia (difficulty of swallowing)** is manifested by forceful attempts to swallow accompanied by extension of the head.

Disfunction cont'd---

obstruction.

- Lesions in the pharynx cause regurgitation through the nostrils or coughing up of the material.
- There is a danger that the material may be aspirated into the lungs and cause respiratory failure and cause **aspiration pneumonia**.
- When the obstruction is at a low level in the esophagus, a large amount of material may be swallowed and regurgitated.
- To differentiate regurgitate mass at vomitus mass, we must know **the PH**.
- Because the regurgitated from the esophagus is slightly **alkaline** while that of the vomitus is **acidic**.
- The main causes of dysphagia and inability to swallow include the following:
 - Obstruction by foreign body, tumor or inflammatory

Dysfunction cont'd---

- Painful condition of the pharynx or esophagus due to inflammation
- Dilation and paralysis of esophagus
- Esophageal diverticulum(sac in wall of organ)

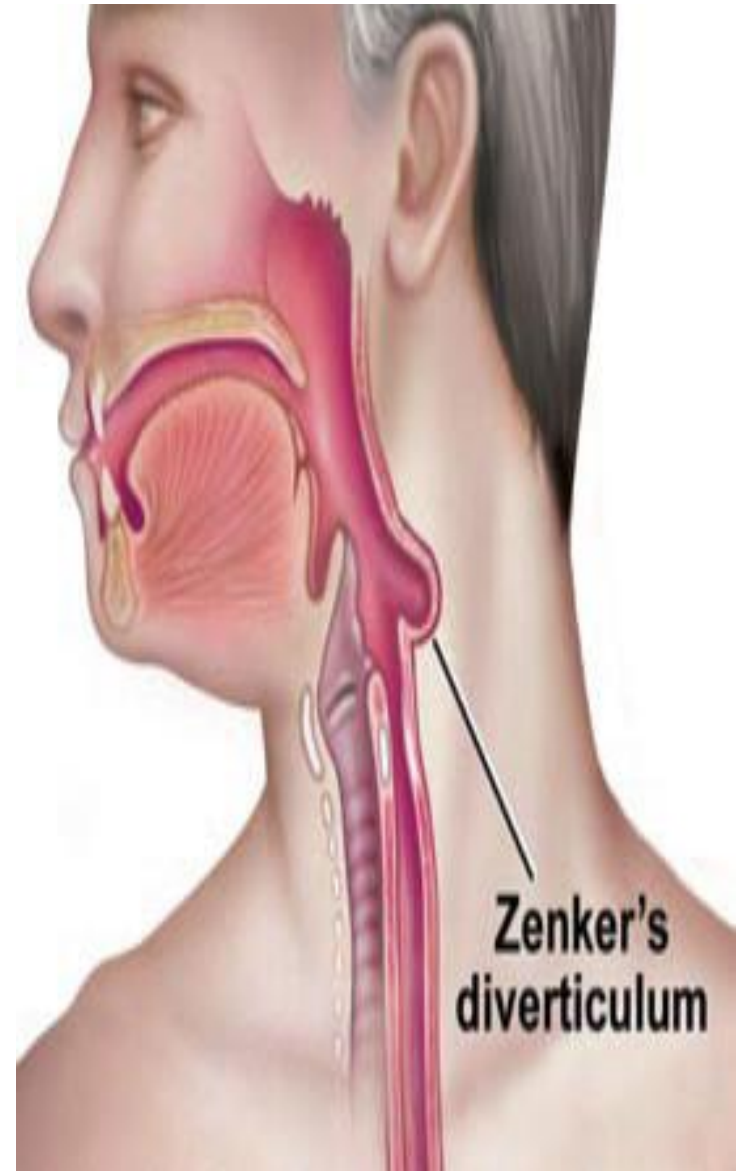
4.6.2. Vomiting

- Vomiting is the forceful ejection of the contents of the stomach and the proximal part of small intestine through the mouth.
- Vomiting happens due to complex motor disturbance of the alimentary tract.
- It is signaled by **hyper salivation, retching** and **forceful contractions** of the abdominal muscles and diaphragm.
- Vomiting is a protective mechanism with the function of removing excessive quantities of ingesta or toxic materials from the stomach (Eg. poisoning)

Esophageal diverticulum(sac in wall of organ)



Pharyngoesophageal
diverticulum (Zenker)



Dysfunction cont'd---

\ 2. True vomiting

1. Projectile vomiting

- Projectile vomiting is not accompanied by **retching movements**.
- However, large amounts of fluid material are ejected with little effort.
- It is almost always a result of **overloading of the stomach with feed or fluid**.

2. True vomiting

- **True vomiting** occurs in monogastric animals like the dog and cat and in pigs with gastroenteritis and some systemic diseases.
- It occurs very rarely in other farm animals.
- **True vomiting** is accompanied by
 - \ Prolonged and repeated retching

Dysfunction cont'd---

↳ Extension of the head.

➤ The vomitus is usually small in amount and pasty in consistency.

➤ It is commonly a result of irritation the gastric mucosa.

➤ **NB.** Among farm animals true vomiting is not a clinical feature of horses because of two reasons.

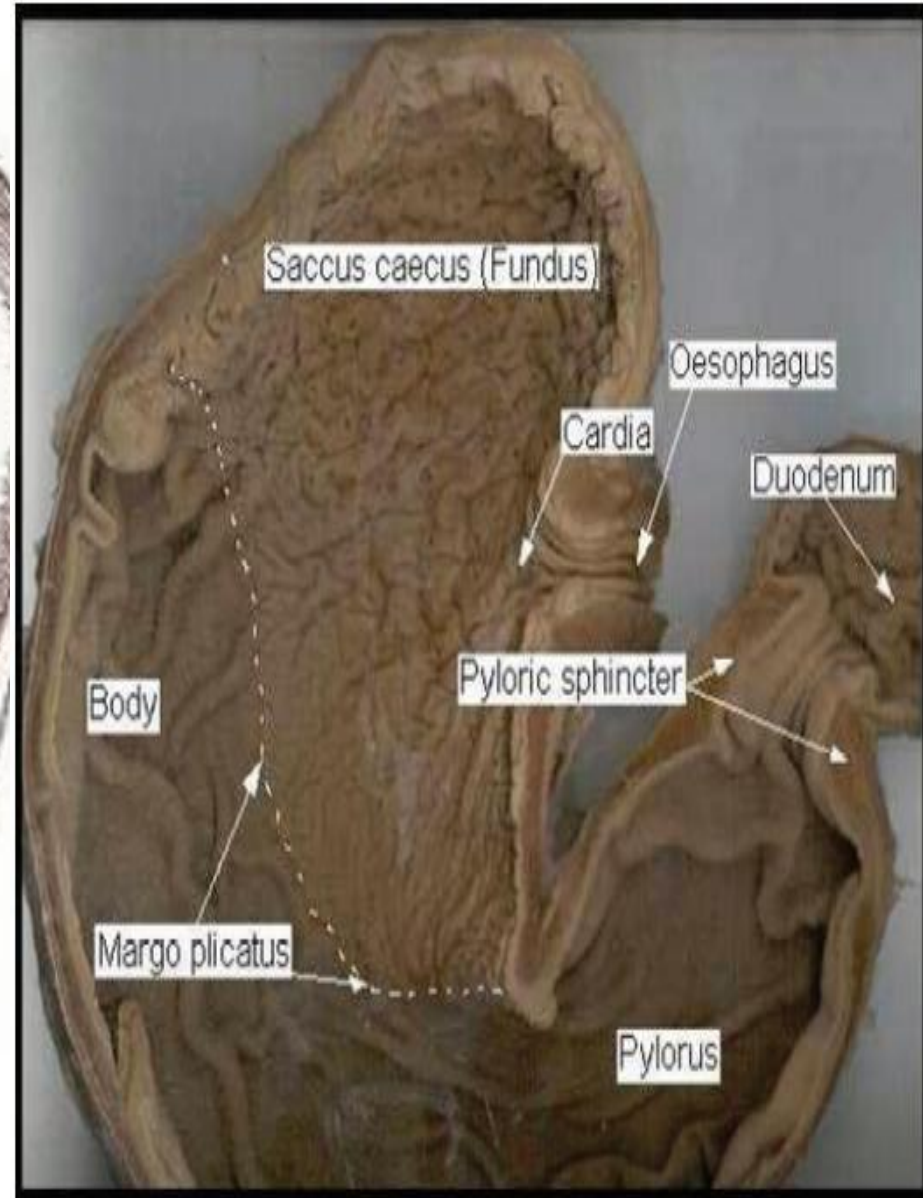
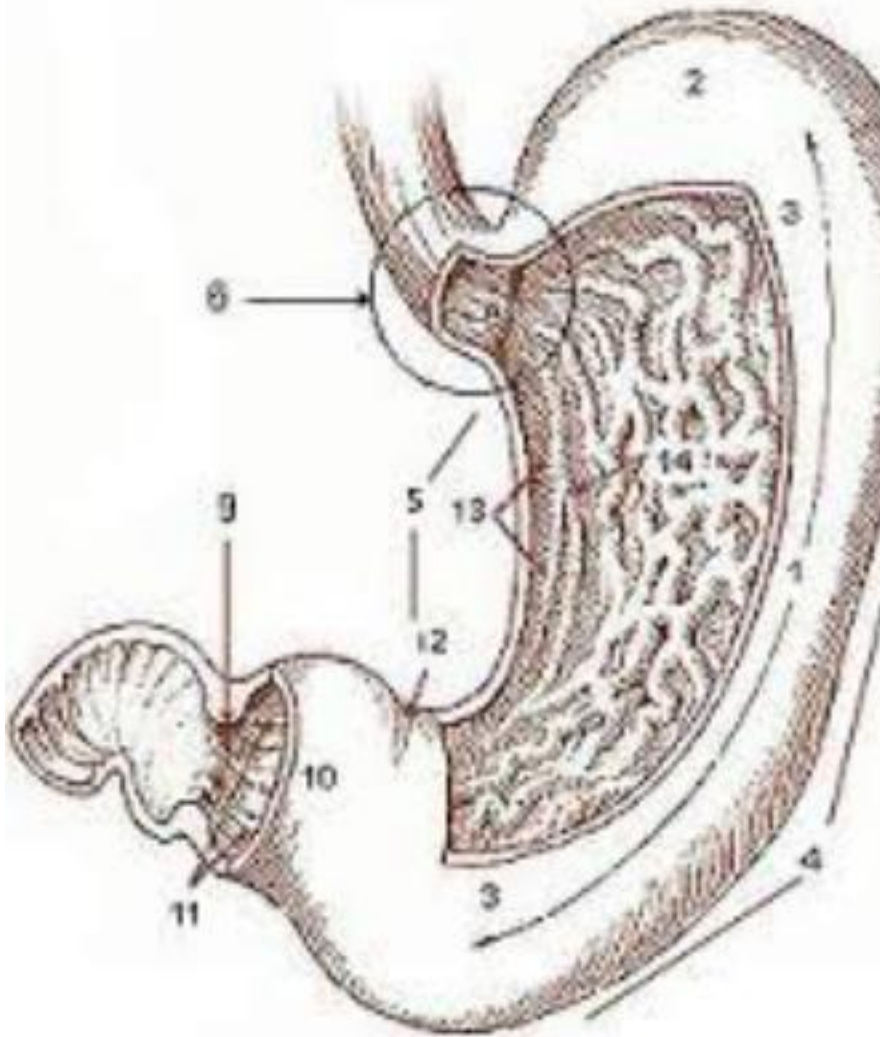
→ 1. The strong cardiac sphincter inhibits the release of stomach contents; in horses rupture of the stomach is more likely to occur before vomiting takes place.

→ 2. The soft palate and epiglottis combine to effect a seal between the oral and nasal parts of the pharynx so that any vomited stomach content is discharged through the nasal cavities and not through the mouth that may expose the horse to **aspiration pneumonia**.

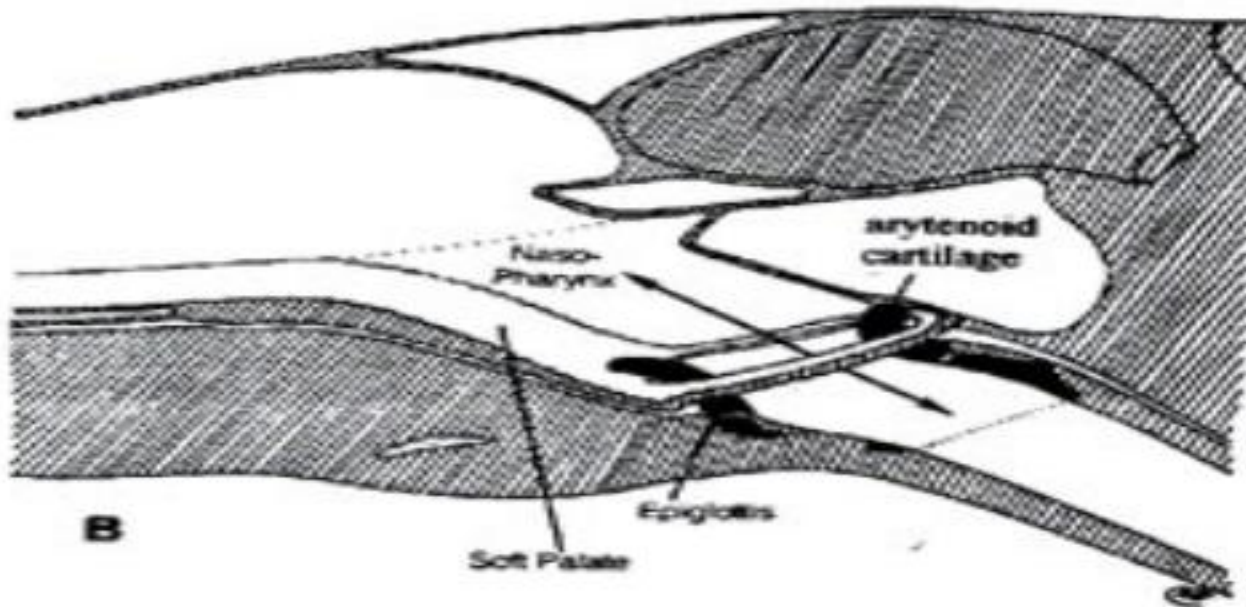
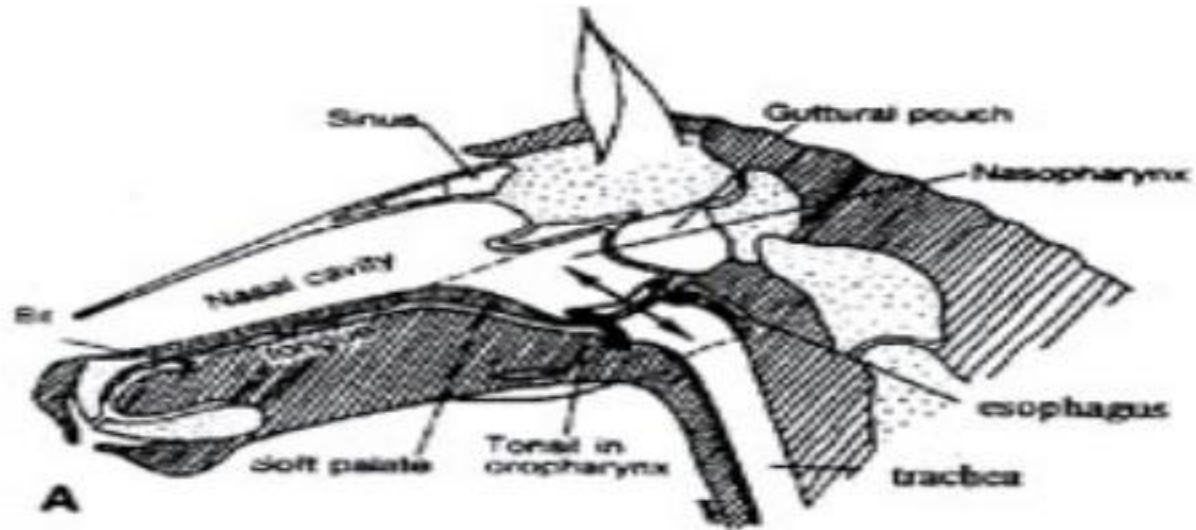
Effects of vomiting in animals

➤ 1. Fluid and electrolyte loss

Dysfunction cont'd---



Soft palate and epiglottis of horse



Dysfunction cont'd---

acidic fluid leads to alkalosis.

➤ 2. Aspiration pneumonia

➤ Aspiration of stomach contents which contain toxic substances and microorganisms into the lung alveoli cause pneumonia which is called **aspiration pneumonia**.

➤ 3. Gastric rupture

➤ Vomiting of large quantities of material in the horse is usually a terminal event and suggests **gastric rupture**.

4.6.3. Regurgitation

➤ Regurgitation is the expulsion through the mouth or nasal cavities food, saliva and other substances which have not yet reached the stomach.

➤ In most cases it is due to abnormalities of the esophagus which interfere with swallowing.

Esophagitis, esophageal obstruction, esophageal

Disfunction cont'd---

are the common causes of regurgitation.

- Ruminants regurgitate rumen contents as part of rumination but the material is not expelled from the mouth.
- Regurgitation of rumen contents through the mouth is commonly associated with loss of tone or inflammation of the cardia opening of esophagus.
- **NB.** Cardia opening of esophagus into stomach: the opening of the esophagus into the stomach, or the upper part of the stomach where it is connected to the esophagus.

4.6.4. Hypermotility and hypomotility of GIT

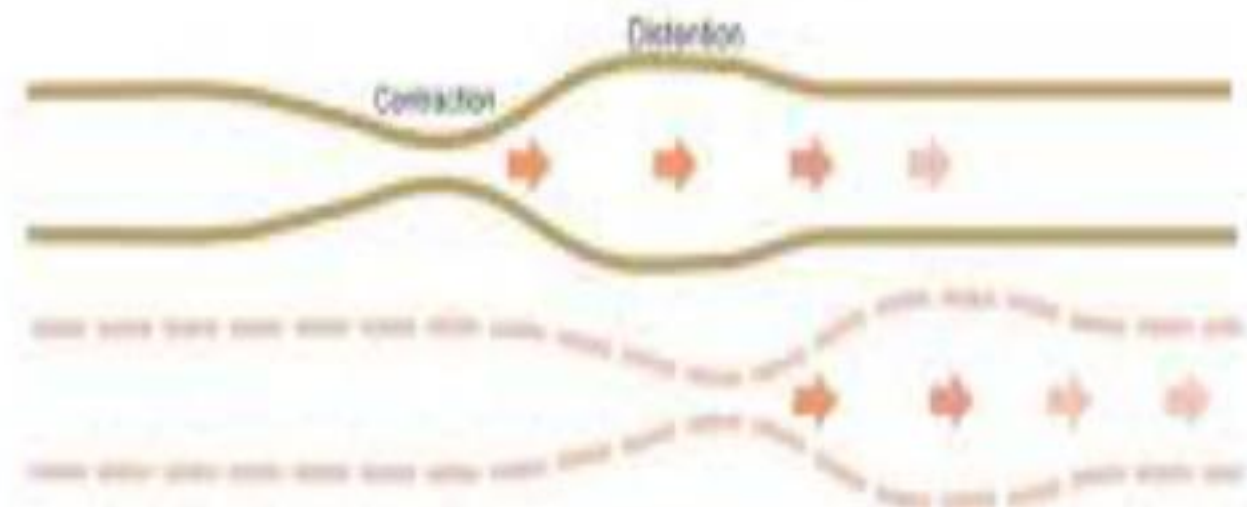
- The most important functions of alimentary tract motility are:
 - The peristaltic movements that move ingesta from the esophagus to

Segmentation Contraction



Peristaltic Wave

Health Hype
www.healthhype.com



Dysfunction cont'd---

→ The tone of the sphincters

➤ Abnormal motor function may take the form of increased or decreased motility.

➤ Abnormal motility of GIT which is manifested by increased motility of GIT above the norm is called **hypermotility**.

➤ Abnormal motility of GIT which is manifested by decreasing the motility of GIT below norm is called **hypomotility**.

➤ While absence of motility of GIT is called **atony**.

➤ You know the motility of GIT depends upon stimulation via

→ The sympathetic and parasympathetic nervous systems.

➤ This means motility of GIT dependent upon

→ The activity of the central and peripheral parts of

Dysfunction cont'd---

other system, is manifested by hypermotility or hypomotility.

➤ This means motility of GIT dependent upon

- The activity of the central and peripheral parts of these systems, and
- The intestinal musculature and its intrinsic nervous plexuses.

➤ Autonomic imbalance, resulting in a relative dominance of one or other system, is manifested by hypermotility or hypomotility.

Hypomotility and atony of GIT

➤ Hypomotility and atony of GIT is caused by

- Debility, accompanied by weakness of the musculature or
- Severe inflammation such as in acute peritonitis or

Dysfunction cont'd---

Hypermotility

- Hypermotility of intestinal wall is caused by
 - Less severe inflammation like in mild gastritis and enteritis
- Because mild gastritis and enteritis may result in an increase in muscular activity and increased propulsive activity.
- Hypermotility of intestinal wall causes **diarrhea**.
- Because of increased irritation at a particular intestinal segment increases its activity.
- And disturbs the normal down ward gradient which insures the passage of ingesta from esophagus to the rectum making it faster than the normal rate.
- However, if irritation is sever and for a long time, reverse peristalsis may occurs and make the ingesta to

Dysfunction cont'd---

➤ on digestion and absorption.

Treatment of abnormal motility of GIT

- **Hypermotility** is treated with a combination of
 - 1. Drugs that inhibits cyclic myo electric activity of intestine
 - ↳ Like **atropine sulphate**
 - 2. Drugs that are analgesic (pain reliefer) and myorelaxants like **dipyrone** or **meperidine** or **butorphanol**

Dose and route of administration

- **Atropine sulphate** can be administered IV,IM and S/C
 - Cattle - 300 mg/ 45 kg BW
 - Horses - 6.5 mg/ 45 kg BW
 - Sheep and goat - 50 mg/45 kg BW

Dysfunction cont'd---

Dose and route of administration of dipyrrone

- Dipyrrone is recommended at a dose of 5-10 grams/horse.
- It may be given subcutaneously, intravenously, or intramuscularly.
- Dosage may be repeated up to 3 times daily (every 8 hours).
- **2. Hypomotility** can be treated by administration of
 - Parasympathomimetics (cholinergic or cholinomimetic drugs) like carbocholin and prozerin
 - Purgatives (laxatives) combined with analgesics
- Purgatives that are commonly used in veterinary are
 - 1. Lubricant laxatives like **paraffin** used for dogs and cats per os in dose of 1 inch of paste 2 times daily.
 - 2. Bulk forming laxatives like that of 90% **Ispaghula**

Dysfunction cont'd---

salt.

➤ Among the osmotic laxatives the **Epsom salt** is the most commonly used in farm animals.

→ It is powder of 49% or 90%

→ Use in dose of for horses 100 -500gm/ 450 Kg BW

→ For cattle - 250 - 500 gm/ 450Kg BW

→ For pigs 25 - 125 gm

→ For dogs 5 - 25 gm

→ Cats 2 - 5 gm

4.6.5. Abdominal distension

➤ One of the major results of abnormality of motility of GIT is distension of the tract.

➤ Distension of abdomen is a common manifestation of disease of the alimentary tract.

Dysfunction cont'd---

- Generally abdominal distension associated with alimentary tract is caused by distension of viscera with gas or fluid.
- This occurs in a number of disturbance including
 - 1. Rapid accumulation of gases
 - 2. Insufficient expulsion of gases
 - 3. Complete occlusion of the lumen of GIT

4.6.6. Diarrhea

- Diarrhea is the increased frequency of defecation accompanied by
 - Feces which contain an increased amount of water and
 - Decreased dry matter content.
- The consistency of the feces may vary from soft to liquid.

Normal intestinal absorption

Dysfunction cont'd---

- This fluid including electrolytes should be absorbed mainly by the small intestines.
- In the horse, digestion and absorption may occur in the large intestine.
- The brush border membrane of the **villous epithelial cells** is of paramount importance for the absorption of water, electrolytes and nutrients.
- The common causes of diarrhea include:
 - Enteritis (infectious and non - infectious nature)
 - Malabsorption E.g. due to villous atrophy
 - Excitement
 - Lesions of the stomach and intestine (ulcers and tumors)
 - Indigestible diet E.g. lactose intolerance in calves and foals

Dysfunction cont'd---

Mechanisms of diarrhea

- Any dysfunction of the intestines will result in failure of adequate absorption and diarrhea.
- Depending on the causative agent, intestinal malabsorption may be the result of four different mechanisms.
 - \ 1. Osmotic diarrhea
 - \ 2. Exudative diarrhea
 - \ 3. Secretory diarrhea
 - \ 4. Abnormal intestinal motility

1. Osmotic diarrhea

- Absorption of water in the intestine is dependent on adequate absorption of solutes.
- If excessive amounts of solutes are retained in the intestinal lumen, water will not be absorbed and diarrhea will result.

Dysfunction cont'd---

- Some substances in the lumen of the intestine may have an osmotic effect and increase the osmotic pressure over a longer length of the intestine.
- As a result, there will be excessive osmotic movement of fluid in to the lumen of the intestine.
- The fluid is not reabsorbed & accumulates in the lumen and cause diarrhoea.
- Some the examples are
 - Saline purgatives
 - Overfeeding
 - Indigestible feeds
 - Disaccharidase deficiencies (Eg. lactose intolerance)

2. Exudative diarrhea

- Acute or chronic inflammation or necrosis of the intestinal mucosa results in

Dysfunction cont'd---

- Both in net increase in fluid production
- Inflammatory products including loss of serum proteins and
- A reduction in absorption of fluids and electrolytes
- Examples include many of the diseases caused by bacteria, viruses, fungi, protozoa, chemical agents and tumors.
- Classical examples include:
 - Enteric salmonellosis
 - Swine dysentery
 - Bovine viral diarrhea
 - Paratuberculosis etc
 - Arsenic poisoning

3. Secretory diarrhea

- A secretory-absorptive imbalance results in a large

Dysfunction cont'd---

- The enterotoxin elaborated by enterotoxigenic *E. coli* (ETEC) results in hyper secretion.
- The villi, along with their digestive and absorptive capabilities remain, intact.
- The crypts also remain intact; however, their secretion is increased beyond the absorptive capacity of the intestines, resulting in diarrhea.
- Because the mucosa remains relatively intact and retains normal absorption capacity, fluids and electrolytes.

4. 7. General principles of treatment of diseases of the alimentary tract

1. Removal of the primary cause of the disease (disorder)

➤ Examples are

➤ **Antimicrobials** – for treatment of bacterial and some

Dysfunction cont'd---

└ Eg. Anticoccidial drugs for the treatment of enteritis by coccidial parasites.

➤ **Specific antidotes** – for treatment of toxicosis caused by some bacterial exotoxins.

└ Eg. Specific antitoxins are given for the treatment of toxemia by *Clostridium perfringens* types A, B, C, D or E

➤ **Ruminotomy** for treatment of ruminal bloat or acute CH₂O engorgement

➤ Surgical correction of **abomasal displacements and torsions**

➤ Surgical correction of **intestinal accidents (torsion, volvulus or intussusceptions).**

2. Supportive treatment (replacement of fluids and electrolytes)

➤ Supportive treatment is important in cases of dehydration and loss of electrolytes due to enteritis

Dysfunction cont'd---

- **Isotonic saline** (0.9% NaCl) or
- **Ringer's solution** for the treatment of dehydration and loss of electrolytes

3. Symptomatic treatment

- Symptomatic treatment is based on the symptoms developed as a result of the underlying disorders.
- **Cathartics** – for relief of distension to remove the accumulated ingesta
- **Parasympathomimetics** – for relief of ruminal stasis or atony to increase motility
- **Ruminal acidosis** – is treated with alkalinizing agents as oral drench, through stomach tube or as intraruminal injection by
 - └ $\text{Mg}(\text{OH})_2$
 - └ MgO

Dysfunction cont'd---

- **Analgesics** – for the treatment of abdominal pain such as colic in horses
- **Purgatives and laxatives** – for the treatment of constipation
- **Systemic acidosis** – is corrected by IV administration of
 - ↳ 5% sodium bicarbonate(in sever case)
 - ↳ 1.3% sodium bicarbonate in 5% dextrose
- **Systemic alkalosis** – is corrected by IV administration of
 - ↳ High-potassium acidifying solution: Isotonic saline plus 2.5 g potassium chloride/l .
- **Rumen alkalosis** – is treated with acidifying agents
 - ↳ Acetic acid
 - ↳ Vinegar

Dysfunction cont'd---

NB. Cud is food chewed twice: partly digested food that cows and other ruminants return to the mouth, after it has passed into the first stomach, to chew again as an aid to digestion).

Part 5 - Diseases of hepatobiliary system

➡ Here we will see

5.1. Introduction to hepatobiliary system

5.2. Manifestation of liver and biliary disease(jaundice/icterus)

5.3. Hepatitis

5.4. Cirrhosis of liver

5.5. Cholelithiasis

5.1. Introduction to hepatobiliary system

➡ Hepatobiliary system comprises

→ The liver

¬The gull bladder

→ The bile duct

➤ You know in veterinary physiology in that liver is the largest gland in animals with so many functions.

The most common functions of liver are

Hepatobiliary cont'd---

- 1.Maintenance of normal blood glucose levels by providing glycogen.
- 2. Formation of some of the plasma proteins
- 3. Formation and excretion of bile salts and the excretion of bile pigments.
- 4. Formation of prothrombin
- 5. Detoxification and excretion of many toxic substances

➤What is the interesting character of liver is that it has a large reserve of function and approximately three-quarters of its parenchyma must be rendered inactive before clinical signs of hepatic dysfunction appear.

➤Morphological and functional disturbance of liver occur during many metabolic, in many infectious and parasitic diseases of liver.

➤So that the clinical signs produced by interference of

Hepatobiliary cont'd---

functions of liver are dealt with under manifestations of **hepatic dysfunction**.

➤ Hepatic dysfunction by its localization on liver can be

→ Focal

→ Diffuse

➤ Diffuse diseases of the liver can be classified as **hepatitis** and **hepatosis**.

➤ The disease of hepatobiliary system can be manifested by symptom complex called **jaundice/icterus**.

➤ Before we are going to deal about jaundice, let us see the normal physiological process of bilirubin formation and excretion from the organism.

➤ You know the **indirect bilirubin** is formed in reticuloendothelium cells.

➤ Then enters to blood system and to the liver where

Hepatobiliary cont'd---

retain by **kupffer cells** of liver.

➤ The retained bilirubin and the bilirubin that is produced by kupffer cells will be transferred to hepatocytis.

➤ Then after the mixing of bilirubin with bile pigment , it enters to intestine through bile duct.

➤ In intestine by the action of **reducing bacteria (enzyme of bilirubin)** it will be changed in to **urobilinogen**.

➤ Most of(90%) of the urobilinogen is excreted from the organism together with feces after it has changed to **biliverdin and stercobillin**.

➤ Some (10 %) urobilinogen is absorbed through small intestine and by the portal vein it enters to the liver.

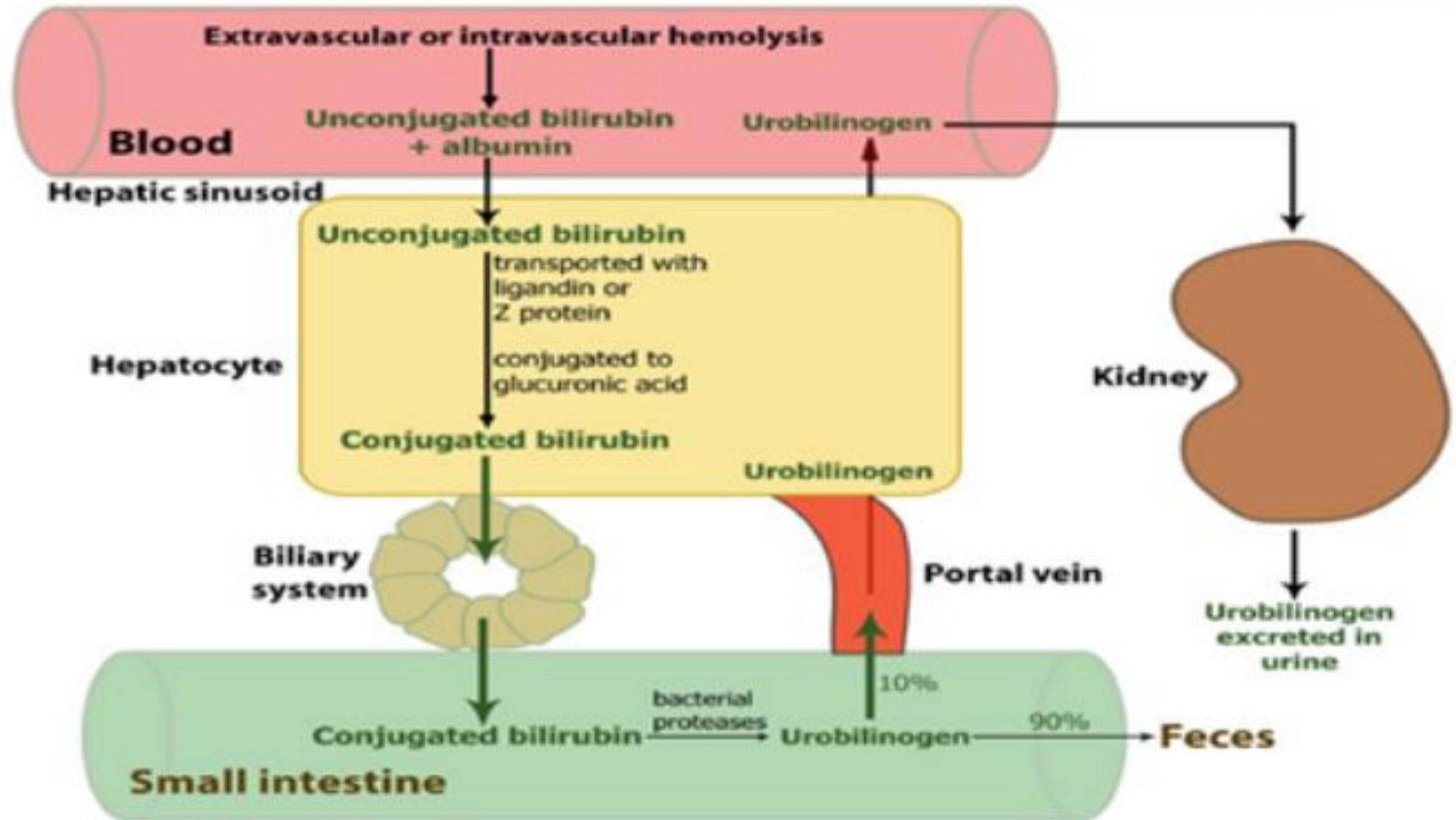
➤ Most of urobilinogen which the liver does not use enters the blood stream and will be excreted with urine

Hepatobiliary cont'd---

called **haematogenic(due to haemolysis of old RBC).**

➤ But bilirubin originated from liver called **direct bilirubin** appears in the blood of animals only if there is pathology in the liver and biliary system(gall bladder and bile duct).

Bilirubin Production & Metabolism:



5.1. Jaundice/ icterus

- **Jaundice** is a clinical sign that often arises in diseases of the liver and biliary system.
- But it also occurs in diseases in which there are no lesions of these organs.
- Although jaundice is a result of the accumulation of bilirubin, the staining is much more pronounced with conjugated (direct) bilirubin than with unconjugated (indirect) bilirubin.
- Thus the jaundice is more intense in cases of obstructive and hepatocellular jaundice than in hemolytic jaundice.
- So that we can define jaundice as clinical signs that are manifested as yellowish staining of the skin, sclera and deeper tissues with bile pigments that are

Hepatobiliary cont'd---

bilirubinaemia) and

↳ Due to other diseases that cause haemolytic crisis of RBCs(indirect or unconjugated bilirubin).

➤What is bilirubin?

- The breakdown product of haemoglobin(Hgb) from RBCs and other haem containing proteins.
- It is produced by reticuloendothelial system.
- Released to plasma and bound to albumin and transported by protein like ligandin in hepatocytes to form conjugate bilirubin and excreted through bile channels into small intestine.

➤What are the reasons to increase bilirubin in the blood?

- Overproduction by reticuloendothelial system
- Failure of hepatocytes uptake

Hepatobiliary cont'd---

→Obstruction of biliary excretion into intestine

➤**What are the reasons to increase unconjugated(indirect) bilirubin in the blood?**

→High production of bilirubin which exceeds over the ability of liver to conjugate

→Ex. During haemolytic crisis

➤**What are the reasons to increase conjugated(direct) bilirubin in the blood?**

→It is produce but not excrete

→Metabolic defect

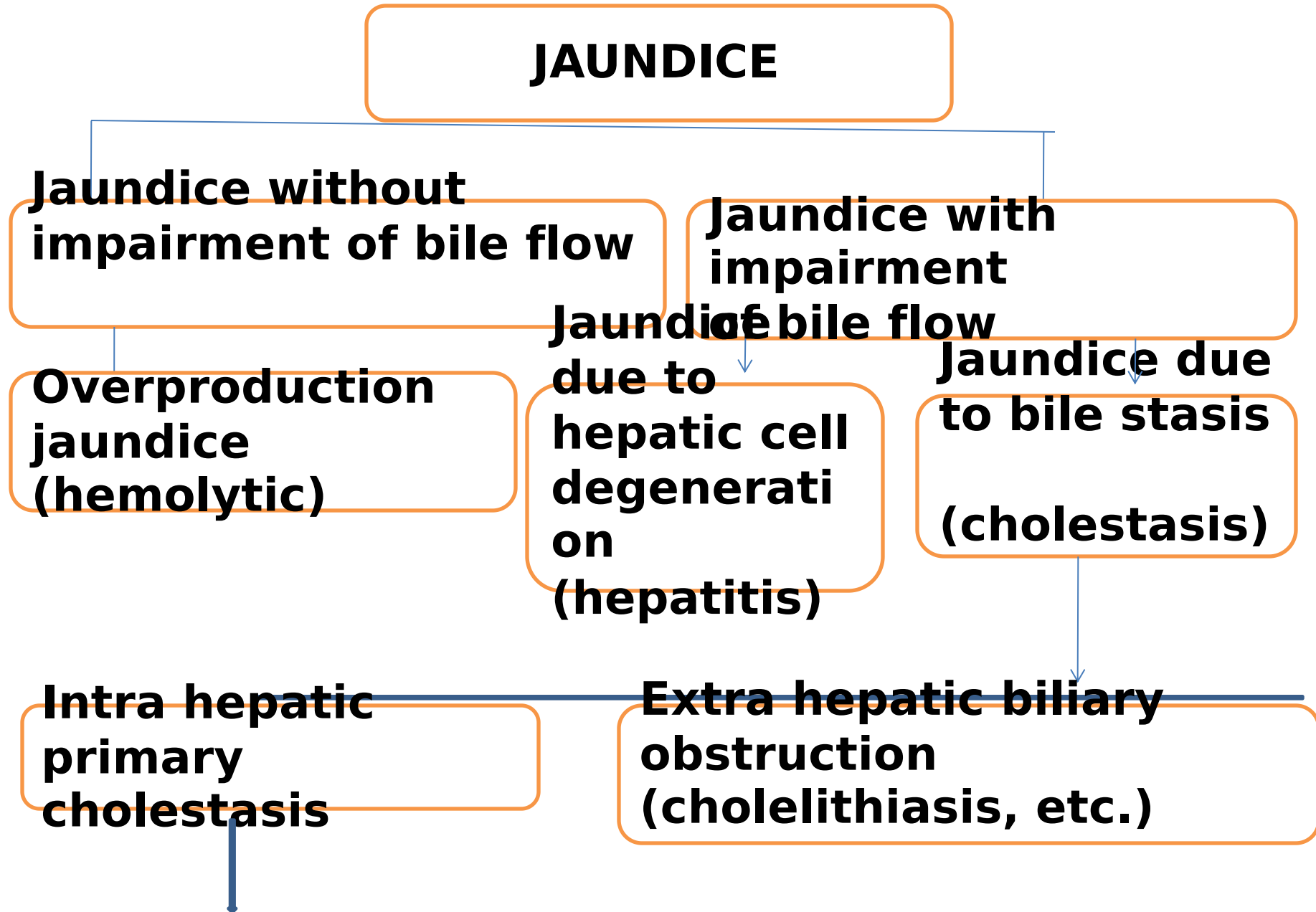
→Intra- or extra hepatic obstruction

➤By the reasons of its formation jaundice can be classified into

1. Mechanical

2. Parenchymatous

Classification of jaundice



**Mechanical
(congenital
aplasia of
ducts, fibrosis
of hepatitis)**

The diagram consists of two orange-outlined ovals side-by-side. A thin blue line connects the top of both ovals, forming a horizontal bridge. The left oval contains the text 'Mechanical (congenital aplasia of ducts, fibrosis of hepatitis)' and the right oval contains the text 'Functional (stasis of bile flow without apparent physical obstruction)'.

**Functional (stasis
of bile
flow without
apparent
physical
obstruction)**

Hepatobiliary cont'd---

\ 3. Haemolytic

1. Mechanical jaundice/ icterus

➤ It is jaundice that is formed as the result of partial or totally absence of excretion of bile from hepatocytis or bile ducts.

➤ It is caused by bile concernments, parasites and tumors etc in bile duct.

➤ It can be

\ Extra hepaticbiliary obstruction

\ Intra hepatic primary cholestiasis

Extra hepaticbiliary obstruction

➤ Obstruction of the bile ducts or common bile duct by biliary calculi or compression by tumor masses is a rare occurrence in farm animals.

➤ Commonly listed causes are obstruction of the common duct by nematodes and inflammation of

Hepatobiliary cont'd---

- A significant number of pigs die with biliary obstruction and purulent cholangitis secondary to invasion of the ducts by *Ascaris lumbricoides*.
- Parasitic cholangitis and cholecystitis also occur due to fasciolosis and infestation with *Dicrocoelium dentriticum*.
- Because of obstruction of the extra hepatobiliary duct the bile ducts will be distended and accumulate bile.
- From this the bile will be absorbed by lymphatic system of liver then it will be transported to thoracic duct and enters to blood circulation.
- Accumulation of bilirubin in the blood cause **bilirubinaemia** and icterus of sclera, mucous membrane, skin and other tissues.
- Serum levels of conjugated bilirubin rise, causing a

Hepatobiliary cont'd---

yellowish.

- Excretion of the conjugated bilirubin in urine occurs on a large scale but there is no urobilinogen because of the failure of excretion into the alimentary tract.
- Obstruction is usually complete and results in the disappearance of bile pigments in the feces.

➤ **What is its effect on the animals?**

- Bile is not toxic for the organism but its absence in duodenum disturbs the digestion and absorption of fat.
- Since there is the growth of bacteria, there will be production of bacterial and metabolic toxins.
- And the high toxin production disturbs the detoxification, metabolic function of the liver.

Intra hepatic primary cholestasis

- The mechanical stasis of biliary flow caused by fibrous tissue

Hepatobiliary cont'd---

constriction and obliteration of the small biliary canaliculi may occur after hepatitis and in many forms of fibrosis.

- Cholelithiasis, the formation of biliary calculi, is frequently reported as a cause of cholestasis in humans and has been reported in horses and cattle.
- Functional stasis is a major problem in hepatic disease in humans but has not been defined in animals.
- In both instances the defect is the same as in extra hepatic biliary obstruction and the two diseases cannot be differentiated by laboratory tests.

2. Parenchymatous icterus(jaundice)

- It is also called jaundice due to **hepatic cell degeneration**.
- The cause may be any of those diffuse diseases of the liver that cause degeneration of hepatic cells.

Hepatobiliary cont'd---

only bile metabolism but also protein, carbohydrate, fat and vitamin metabolisms.

- Because there is only partial obstruction of biliary excretion, the changes in serum and urine lie between those of hemolytic jaundice and extra hepatic biliary obstruction.
- Serum levels of total bilirubin are increased because of retention of direct bilirubin, which also passes out in the urine, causing an elevation of urine levels.
- So that the urobilinogen levels in the urine also rise.
- Parenchymatous icterus occurs during many infectious , parasitic and other diseases of animals like

➤ **1. Infectious diseases**

- Equine infectious anaemia
- CBPP and CCPP
- Leptospirosis

Hepatobiliary cont'd---

- Rift Valley fever
- Infectious necrotic hepatitis associated with *Clostridium novyi*(type B and D) etc.

➤ 3. Parasitic hepatitis

- Acute and chronic **liver fluke infestation**
- Migrating larvae of ***Ascaris* sp.**
- Fibrosing granulomas of liver in horses with **chronic shistosomiasis**
- **Hepatic sarcocystosis** in a horse etc.

➤ 2. None infectious

- Acute intoxication of liver
- Parenchymatous hepatitis
- Toxic dystrophy of liver etc

Hepatobiliary cont'd---

3. Haemolytic jaundice/ icterus

- Hemolytic jaundice is common in animals and is associated with increased process of haemolysis of RBCs in vascular and in organs that are rich in reticulo endothelial cells specially in spleen.
- As the result there is hyperplasia of spleen called **splenomegaly**.
- At the beginning of this process there is high concentration of haemoglobin in the plasma of the blood and develops **anaemia**.
- However, at the same time the **hematopoietic** function of the bone marrow has increased to composite the problem.
- As the result a lot of red blood cells which are poor in haemoglobin content will be enter to the blood circulation and cause anaemia.



Hepatobiliary cont'd---

cells is disturbed .

- That means not all haemoglobin in the plasma of blood is changed in to bilirubin.
- As the result excess haemoglobin which is not changed in to bilirubin is excreted with urine and cause **haemoglobinuria**.
- Bilirubin which is produced by reticuloendothelial cells is higher than the norm.
- And accumulates in plasma of blood.
- So that the liver does not have capacity to change all the bilirubin into conjugated bilirubin.
- Haemolytic icterus occurs in many infectious, toxic and protozoa diseases, inorganic and organic poisons and immunological reactions.
- Diseases that causes haemolysis of RBCs are

Hepatobiliary cont'd---

- Equine infectious anaemia
- Leptospirosis
- Anaplasmosis
- Piroplasmosis
- Babesiosis
- Haemolytic toxin
- Intoxication with Arsenic
- Chronic copper and selenium intoxication in sheep
- Incompatible blood infusion
- All the above problems may end with intoxication of the organism with bile which is called **cholemia**.
- Excess bile acts on nervous, cardiovascular and on blood systems.
- As the result the animals show the following clinical signs

Normal(pale - pinkish color of the mucosa of oral cavity

copyright O'Meara: Pet Informed



Anemic mucous membrane



Cyanotic mucous membrane of oral cavity



Cyanotic mucous membrane of tongue



Hemorrhagic mucous membrane



Icterus of conjunctiva, sclera and mucousa of oral cavity



Icterus on different tissues of animal carcass



- Spasm of muscles
- Dilation of pupil
- Skin itches
- Lastly somnolence (drowsiness) and, in extreme cases coma

5.3. Hepatitis

- The word hepatitis includes all diffuse, degenerative and inflammatory diseases that affect the liver.
- We can also defined as inflammation liver.

Etiology

- Although there is an extensive list of the causes of hepatitis, there are still a number of unknown factors.
- The most common causes of hepatitis are
 - 1. Hepatitis caused by toxins
 - 2. Hepatitis by poisonous plants

Hepatobiliary cont'd---

- ‖ 3. Infectious hepatitis
- ‖ 4. Parasitic hepatitis
- ‖ 5. Nutritional hepatitis

1. Hepatitis caused by toxins

➤ The common causes of toxic hepatitis in farm animals are:

- **Inorganic poisons** – Excess of(copper, phosphorus, arsenic, possibly selenium).
- **Organic poisons** – Intoxication with carbon tetrachloride, hexachloroethane.

2. Hepatitis by poisonous plants

➤ These include the following:

- "**Weeds** including *Senecio*, *Crotalaria*, *Heliotropium*, *Amsinckia* and *Tribulus* spp., *Encephalartoslanatus* and *Trachyandra* spp.

Weeds of genus *Senecio*



Senecio vulgaris

Weeds Crotalaria juncae



Weeds of genus *Trachyandra* spp.



Lupin



Hepatobiliary cont'd---

clover water-damaged alfalfa hay.

→ Trees and shrubs - lantana (*Lantana camara*); yellow wood

3. Infectious hepatitis

➤ Diffuse hepatic lesions in animals are rarely associated with infectious agents.

➤ The significant ones are:

→ The virus of Rift Valley Fever (RVF)

→ Severe cases of equine viral arteritis manifest signs of hepatitis

→ Systemic mycoses, e.g. histoplasmosis, may be accompanied by multiple granulomatous lesions of the liver

→ Other diseases in which hepatic lesions may be common at necropsy, but in which there are no overt signs of clinical disease during life.

Hepatobiliary cont'd---

- Infectious necrotic hepatitis associated with *Clostridium novyi*.
- **Fungi** – *Aspergillus flavus*, *Penicillium rubrum*, *Periconia spp.* etc

Pathogenesis

- It is not well studied.
- However, most of the scientists considered hepatitis is caused due to intoxication of the organism by different toxins.
- Which enters to liver through portal vein, blood circulation and biliary system.
- These toxins act on hepatocytes and reticuloendothelial cells and cause dystrophy and death.
- As the result the fermentative function of the liver will be disturbed.
- For example glycogenesis will be increased which lead

Hepatobiliary cont'd---

- As the result there is always **hyperglycemia**.
- Since the cell membrane of dystrophic hepatocytes becomes permeable, bilirubin which is formed from the blood will return back and accumulate in the plasma of blood.
- As the result there is parenchymatous icterus which results in disturbance of bile metabolism.
- This leads in disturbance of digestion which results in the formation of intermediate metabolic products of carbohydrate, fat and protein.
- Finally it results in intoxication of the organism.

Clinical findings

- The cardinal signs of hepatitis are
 - Anorexia
 - Mental depression

Hepatobiliary cont'd---

- Swelling of liver with abdominal pain
- Ventral body wall edema
- Parenchymatous icterus
- Yellowish urine due to the accumulation of urobilinogen
- Clotting deficiency of blood
- Muscle fasciculation

Necropsy findings

- All the mucous, serous membranes become yellowish in colour.
- Liver enlarged and the edges swollen.
- But the appearance of the hepatic surface and cross-section varies with the cause.
- In acute toxic and trophopathic hepatitis the lobulation is more

Hepatobiliary cont'd---

engorgement of the centrilobular vessels or centrilobular necrosis.

➤ There may be accompanying lesions of jaundice and edema.

➤ **In infectious hepatitis** the lesions are inclined to be patchy and even focal in their distribution.

➤ **Parasitic hepatitis** is obviously traumatic, with focal hemorrhages under the capsule and the necrosis and traumatic injury definable as tracks.

➤ **Congestive hepatitis** is marked by severe enlargement of the liver, a greatly increased content of blood and marked accentuation of the lobular pattern caused by vascular engorgement and fatty infiltration of the parenchyma.

➤ **In hepatic fibrosis** the necropsy findings vary widely depending on the causative agent, the duration

In infectious hepatitis the lesions are inclined to be patchy



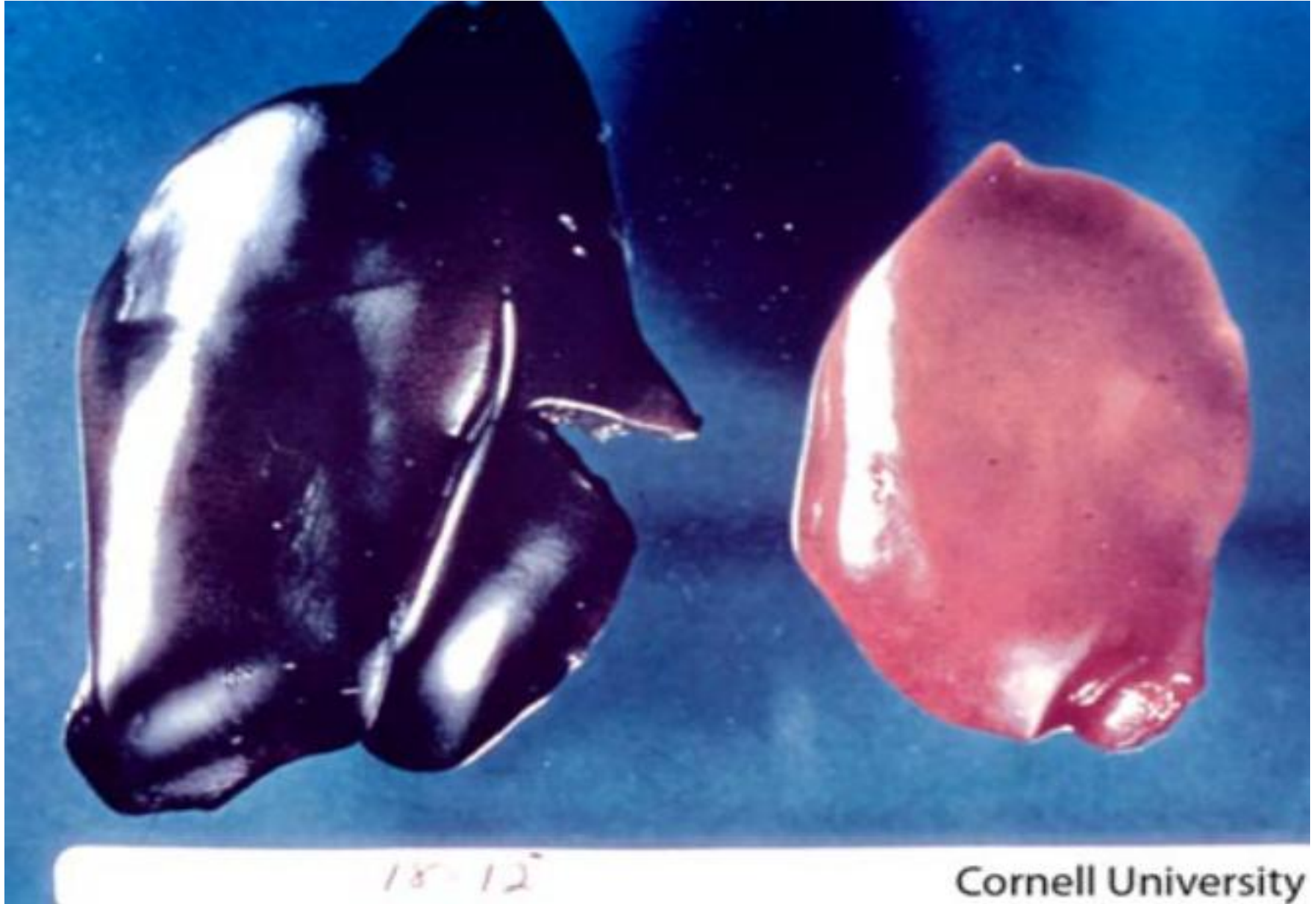
Parasitic hepatitis is obviously traumatic, with focal necrosis



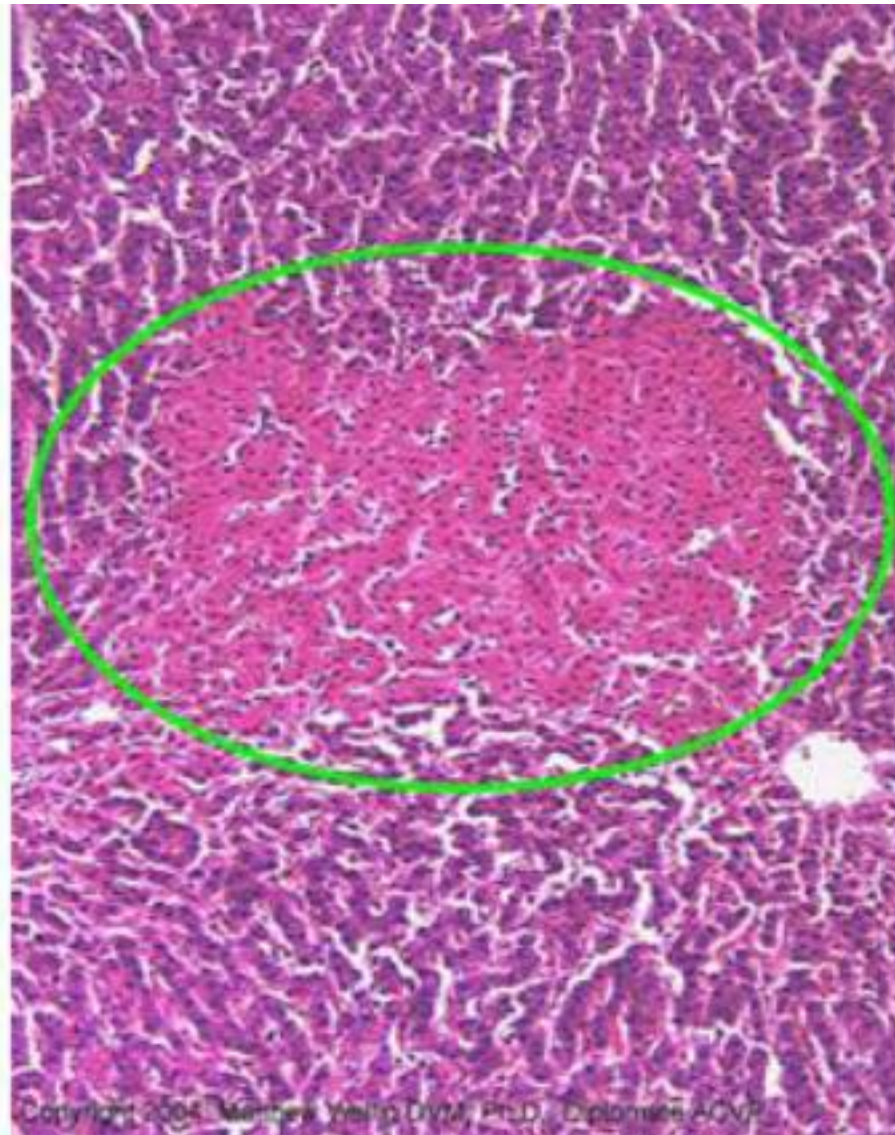
Cornell University



Congestive hepatitis



Central vein engorgement of liver

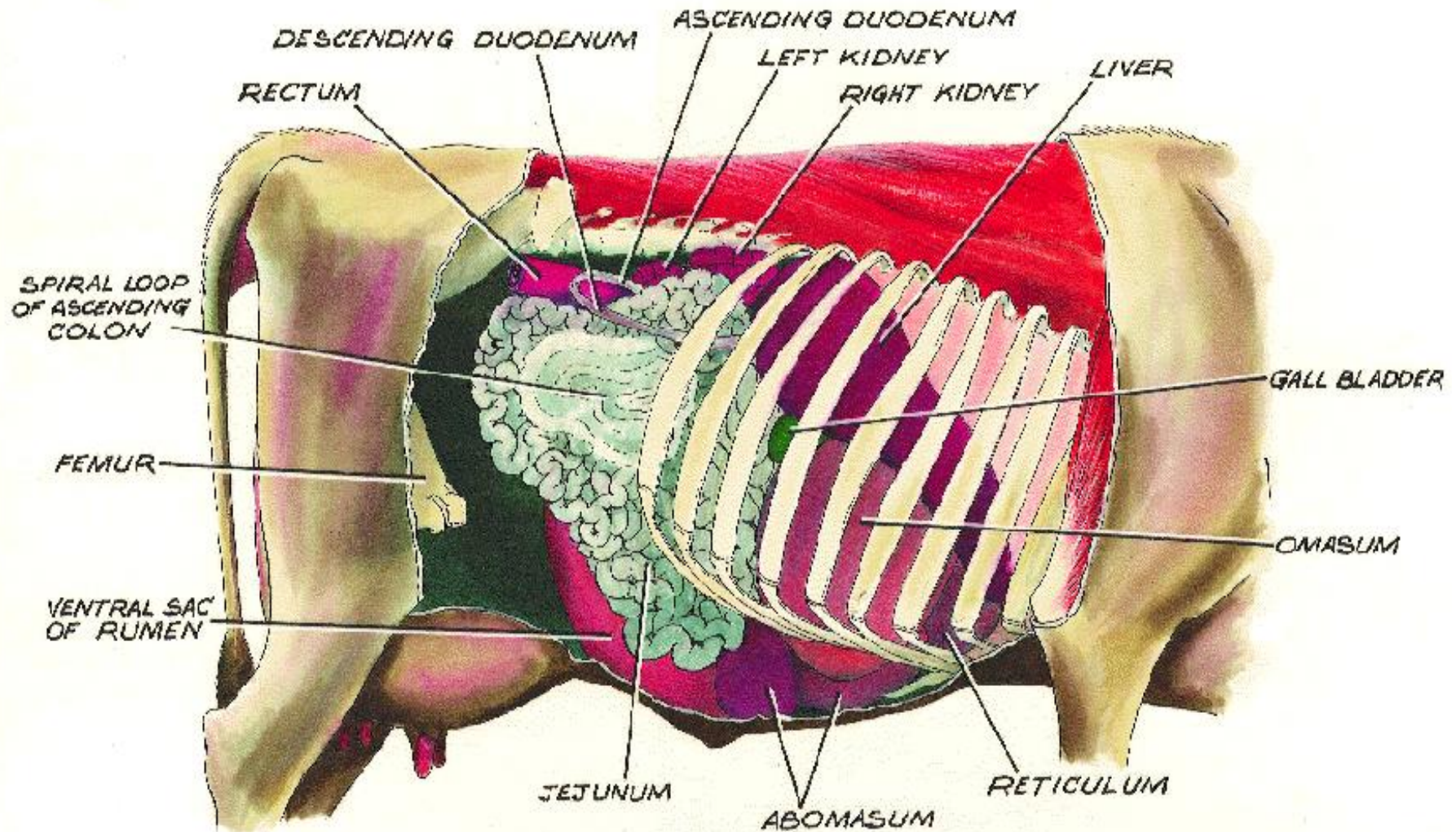


Hepatobiliary cont'd---

Topography of liver in different animals

- Liver of ruminant animals is located at the right side of abdominal cavity called doughy area of liver.
- The liver of cattle is located in the right upper part of 10 – 12 intercostals.
- Small ruminants 8 – 12 intercostals.
- The liver of equines is not accessible to diagnose in norm.
- But during the increasement of the size of liver, it is possible to

Hepatobiliary cont'd---



TOPOGRAPHY OF RIGHT SIDE OF ABDOMEN OF A FIVE-YEAR-OLD NON-PREGNANT COW

Hepatobiliary cont'd---

Diagnosis

- Diagnosis of hepatitis is done based on
 - Clinical signs
 - Laboratory diagnosis of blood and urine
 - Histopathological examination of biopsy of liver

What is biopsy?

➤ A biopsy is a sample of tissue taken from the body of live animals/ human beings in order to examine its histological structures to diagnose the disease more closely.

- As examination of liver biopsies may establish
- The presence or absence of liver disease
 - Provide a specific diagnosis
 - Guide therapeutic choice
 - And also help determine prognosis of liver diseases.

Hepatobiliary cont'd---

Biopsy technique

1. Site of biopsy

- For all animal the exact place is 11 intercostals space 2 - 3 cm below the horizontal line that can be drawn at tuber coxa or 20 - 30 cm below thoracic vertebra.
- During swallowing of liver it is possible to do on 12 intercostals space 2 - 3cm below the horizontal line drawn from tuber coxa.

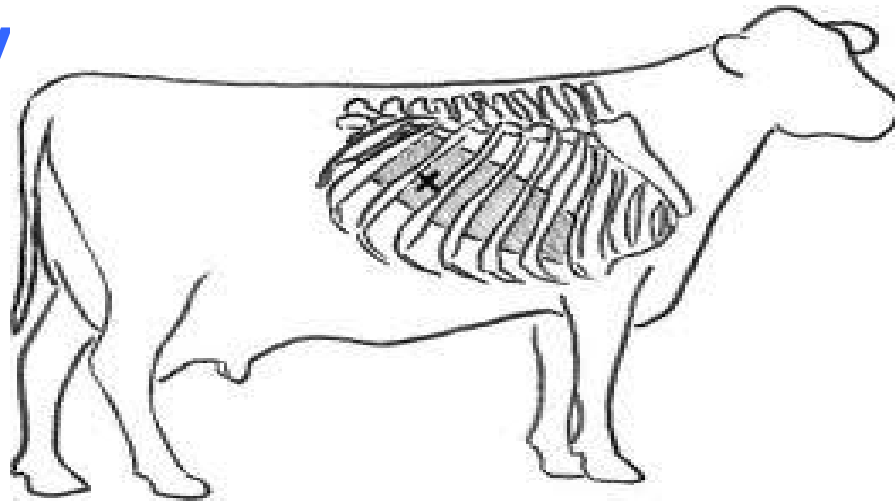
Procedure

- Wash the site
- Remove the hair
- Disinfect the site at the center to periphery
- Make small hole either by surgically pointed scalpel blade or with cannula of small ruminant
- Insert the biopsy needle which has needle wire to the direction of ulnar joint of the opposite fore leg

Hepatobiliary cont'd---

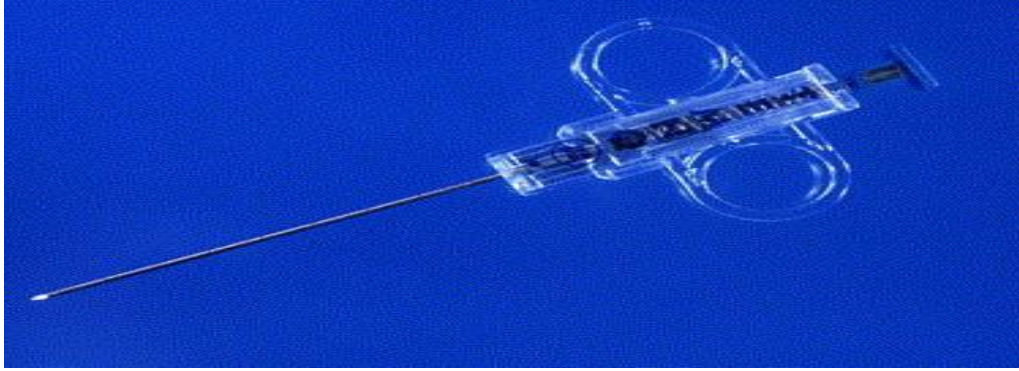
- After two strong tissue resistance from intercostals muscles and peritoneum you are in the liver.
- Then push the needle for about 3 - 4 cm.
- Then rotate the needle 180 - 360 °
- Then take the needle away and push the tissue by using the needle wire on slide.
- Examine histopathological line from tuber coxa to ribs.

Site of biopsy

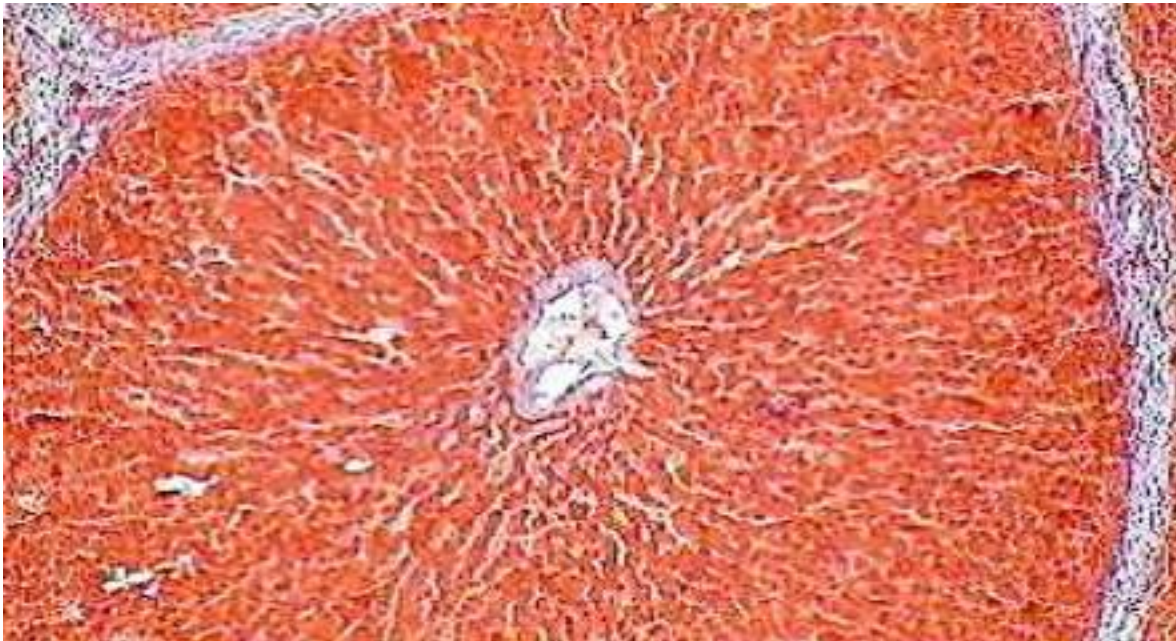


Hepatobiliary cont'd---

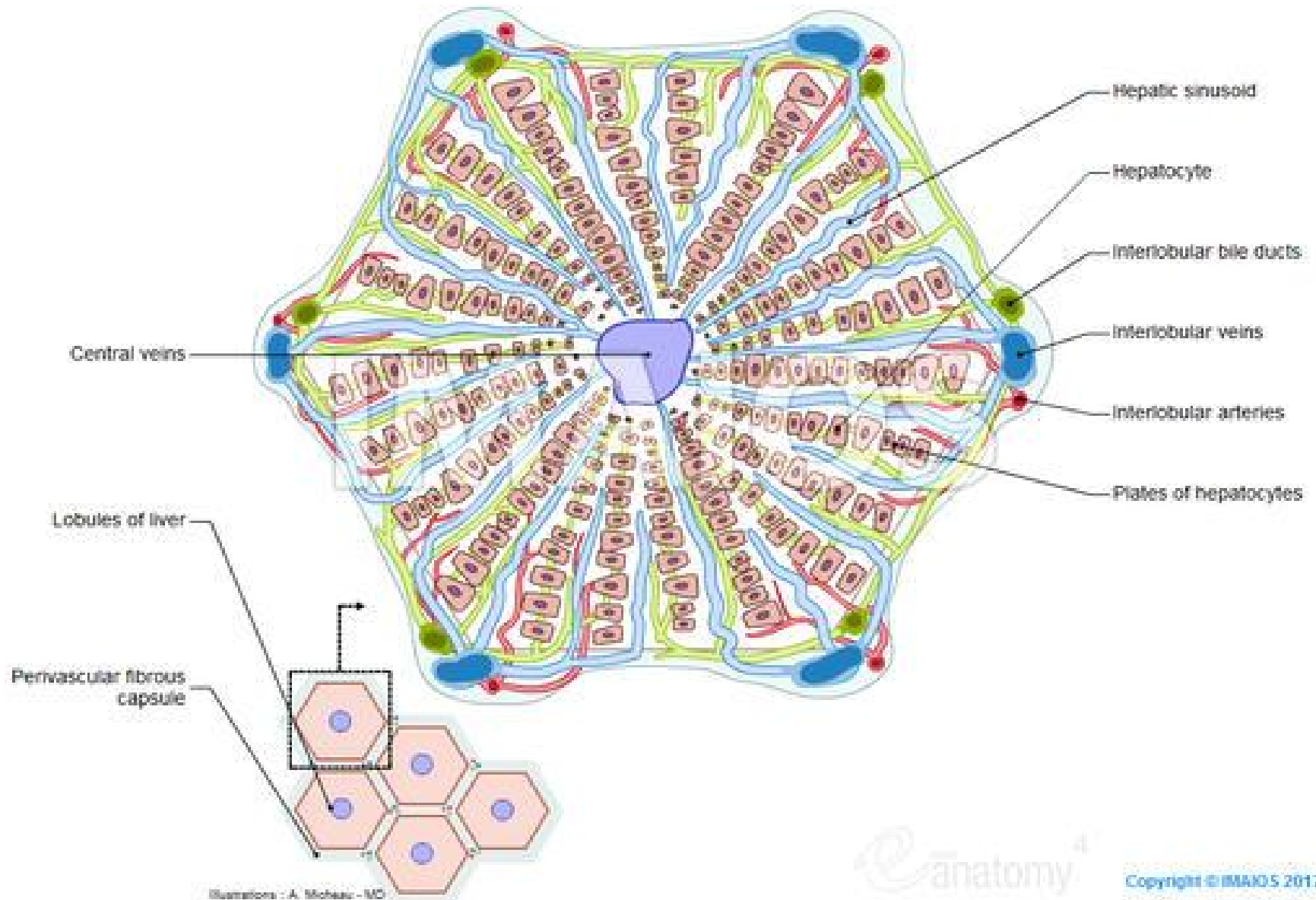
Biopsy needle



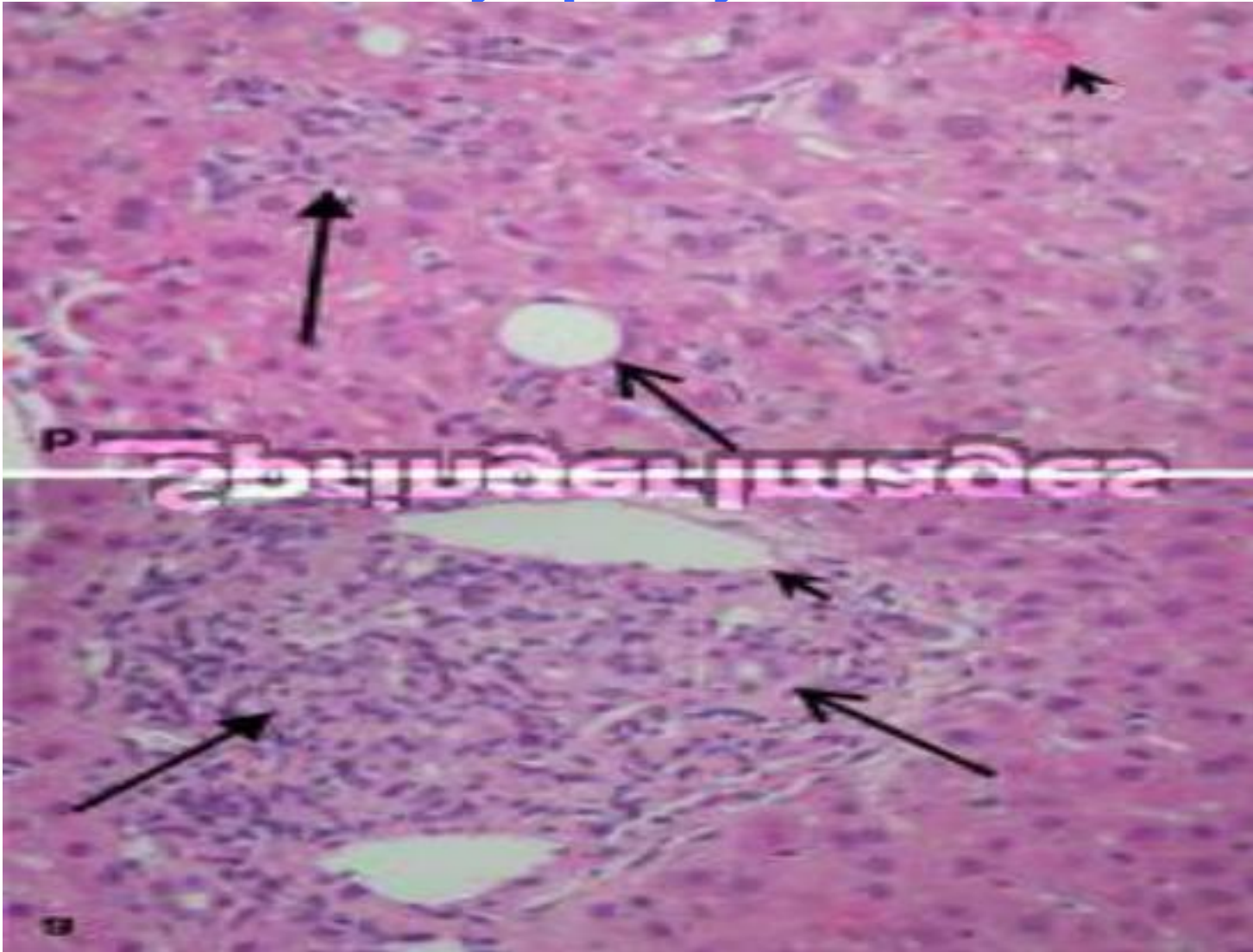
Normal histological structure of liver by Hematoxylin and Eosin (H&E) staining



Structure of lobules of liver



Liver biopsy demonstrating mixed inflammatory infiltrate in the portal area with neutrophils, plasma cells and lymphocytes



Differential diagnosis

- Hepatitis must be differentiated from cirrhosis liver and haemolytic icterus which is caused by many infectious, blood parasites and toxins.
- **Cirrhosis liver** is differentiated from hepatitis in that it has chronic character with out pain during palpation around liver.
- **While haemolytic icterus** is characterized by decreasing the **number of RBCs** and **haemoglobin** and **the presence un matured RBCs** in blood circulation which are not characteristics in hepatitis .
In serum of blood there is **high concentration of haemoglobin** and **indirect bilirubin** during haemolytic icterus that are not present during hepatitis.
- Haemolytic icterus is characterized by enlargement of spleen which is called **splenomegaly**.

Treatment

Hepatobiliary cont'd---

- The principles of treatment of hepatitis have already been outlined.
- Results are seldom good.
- Protein and protein hydrolysates are probably best avoided because of the danger of ammonia intoxication.
- The diet should be high in carbohydrate and calcium and low in protein and fat.
- So that feeds of animals must be free from protein and fat.
- Because of the failure of detoxification of ammonia and other nitrogenous substances by the damaged liver and their importance in the production may affect curtail organs like heart, brain and kidney.
- So that symptomatic therapy must be organized.

For improving the function of liver, administer

Hepatobiliary cont'd---

- Purgation and enemas have also been used in combination with oral administration of antibiotics.
- But mild purgation is recommended to avoid unnecessary fluid loss.
- Supplementation of the feed or periodic injections of the water-soluble vitamins are desirable.
- Hepatic fibrosis is considered to be a final stage in hepatitis and treatment is not usually undertaken.

5.4. Cirrhosis of liver

- Cirrhosis is a complication of many liver diseases that is characterized by abnormal structure and function of the liver.
- The diseases that lead to cirrhosis do so because they injure and kill liver cells, and the inflammation and repair that is associated with the dying liver cells causes scar tissue to form.

Hepatobiliary cont'd---

- This results in clusters of newly-formed liver cells (regenerative nodules) within the scar tissue.

Etiology

- There are many causes of cirrhosis ,they include
- Chronic intoxication of animals by bacterial, fungal toxins and other inorganic toxins.
- Many infectious disease that cause hepatitis if they are not treated on time(tuberculosis of liver, erysipelas etc).
- Many viral diseases that affects liver.
- Autoimmune liver disease in which the body's immune system attacks the liver.

Pathogenesis

- Toxic substances enter to the liver by the two ways
 - 1. Through portal vein

Hepatobiliary cont'd---

2. Through hepatic artery

➤ **When toxic substances enter to the liver through portal vein**, dystrophy and death of hepatocytes occur on periphery of liver.

➤ As the result the hepatocytes will be replaced by fibrosis and develops scar.

➤ This scar presses on blood vessels of liver and hepatocytes and cause disturbance blood supply on the rest normal cells.

➤ As the result the hepatocytes will die and replaced by connective tissue (fibrosis).

➤ And this process continues until all hepatocytes are replaced by fibrosis tissue.

➤ When large number of lobules of liver is highly affected, there is an increasement the size of liver.

➤ This cause to press the portal vein and cause the out

Hepatobiliary cont'd---

- This accumulation of plasma fluid in abdominal cavity is called **Ascites**.
- As the time going the size of the liver will be decreased(atrophy) and we call it **atrophic cirrhosis of liver**.
- Since it affects the hepatocytis, parenchymatous icterus develops

2. When the toxic substances enters through hepatic artery

- Dystrophy starts from interlobular connective tissue and diffused slowly to all lobules of liver.
- As the result the hepatocytis of lobules of liver will be replaced by connective tissue.
- In this type of cirrhosis of liver, there is
 - Enlargement of liver and called **hypertrophic cirrhosis of liver**

Clinical signs

- It is difficult to diagnose cirrhosis of liver at the beginning.
- Because the disease develops slowly as it is chronic disease.
- However, there is inappetance for along time with cathartic inflammation of stomach and intestine.
- Rarely there is colic in equines and tympany with atony of rumen in ruminant animals.
- But when cirrhosis of liver reach its maximum stage, the animal
 - Depressed and it never reacts with external environment
 - The form of abdominal cavity is changed due to accumulation of transudat in the abdominal cavity which is called ascites.
 - **During hypertrophic cirrhosis**, the size of liver has

Ascites in animals



Ascites in human beings



Hepatobiliary cont'd---

- The size of liver has decreased
- There is ascites due to accumulation of transudate in the abdominal cavity.

➤ **However, in both hypertrophic and atrophic cirrhosis**, there are both mechanical and parenchymatous icterus, as the result

- The conjunctiva, sclera and skin will have yellowish colour.
- Urine will have yellowish colour.
- Serum will give positive test result for the direct bilirubin
- Albumin and fibrinogen content serum of the blood will be reduced in both hypertrophic and atrophic cirrhosis.

Necropsy findings

➤ 1. Hypertrophic cirrhosis liver

Necropsy finding of hypertrophic and atrophic cirrhosis of liver



Hepatobiliary cont'd---

➤ The mass of liver of horse and cattle reach 20 kg and 15 kg respectively.

➤ During hypertrophic cirrhosis

→ Liver enlarged and solid in consistency

→ The external surface of liver is smooth.

→ Most of internal tissues are yellowish in colour

➤ **2. During atrophic cirrhosis liver**

→ The size of liver decreased and solid in consistence

→ Incision of the organ with knife is too difficult.

→ The external surface of the liver is rough.

Diagnosis

➤ Diagnose of cirrhosis is done clinically which includes

→ Its chronic character

→ Long time presence of icterus/ jaundice

Hepatobiliary cont'd---

→ Changing of the size of liver

→ Development of ascites

➤ **NB.** The constant clinical sign of cirrhosis of liver is **urobilinuria**.

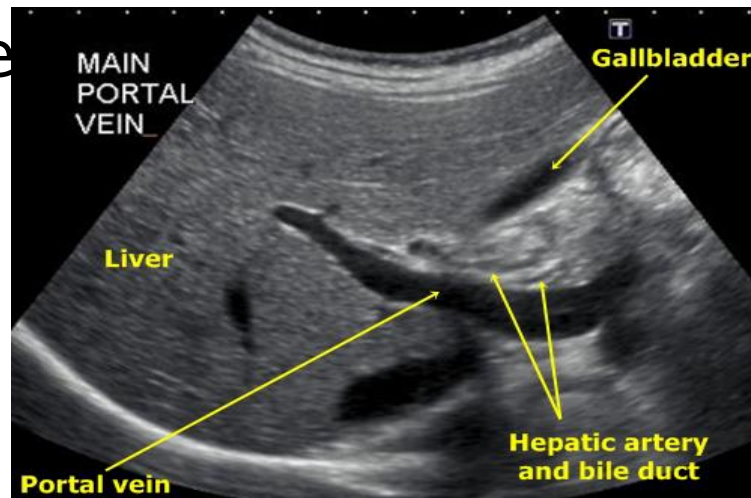
➤ Cirrhosis liver can be diagnosed in laboratory by preparing serum from animals and have

→ Positive test result for direct bilirubin

→ Albumin and fibrinogen content serum of the blood will be reduced below norm

→ Cirrhosis liver can be diagnosed by ultrasonography.

Normal liver



Ultrasonography of atrophic cirrhotic liver at the beginning stage



Treatment

- It is important to know the cause and to avoid it immediately from animals.
- Treatment of cirrhosis of liver depends on the size of affected liver.
- But in most cases give animals easily digestible feed which are rich in vitamins.
- Therapy with medicaments has no any importance mostly at the end.
- But it is important to use symptomatically therapy to remove undesired clinical signs of the disease and to extend the life of animal.
 - For this purpose use diuretics like frusemide to increase diuresis and to decrease edema.
 - Cardio tonic preparates like thiasides, chlorothiasides and hydrochlorothiazide etc.
 - Remove the accumulated transudat (ascites) from

The correct sites of paracentesis(abdomenocentensis) of domestic animals

- All animals except cattle the correct site of paracentesis is 5 – 8 cm(four fingers) back from umbilicus and at the line alba.
- But for cattle the correct site is 10 - 12 cm caudal to the xiphisternum and 10 - 15 cm lateral to the midline(line alba).

5.5. Cholelithiasis

- It is the disease of bile duct and the gall bladder characterized by the formation of **concernments in bile ducts and gall bladder.**
- Cholelithiasis is less common in animals.
- However its formation blocks the excretion of bile to the duodenum.

Etiology

- Cholestiasis occurs due to the disturbance bile

Hepatobiliary cont'd---

cholangitis and **cholecystitis** respectively.

- These predisposing factors will make the flow of the bile from gall bladder very slowly.
- As the result there will be the formation of concernments in the bile duct and gall bladders.
- Other predisposing factors for formation of cholelithiasis is over feeding of animals and absence motion of animals in intensive farms.

Pathogenesis

- Inflammation of the bile duct and gall bladder may force the bile not to evacuate from gall bladder quickly.
- As the result concernments from salts of bile may sediment to form one layer or multiple layer concernments.
- As the result depending up on the size of concernments and number concernments there will be

Clinical signs

- The disease never show typical clinical sign at begins of the disease.
- However, only after a certain time there will disturbance of fat digestion.
- That can be manifested by diarrhoea with bad smelling of faeces and mechanical icterus.
- Pain during palpation around the liver.

Necropsy findings

- In bile duct and gall bladder, we can find concrements that have different size, number and forms.
- The size of bile concrement may reach 10 cm in diameter and they may have 3 kg weight.
- They may have different shapes like round, egg and cylindrical etc.
- By their consistency they can be soft, easily breakable and hard.

Hepatobiliary cont'd---

- Surgically opening of the bile duct, gall bladder and lobules of liver shows the presence of bile that have high viscosity.

Diagnosis

- Diagnose of cholelithiasis is done based on the result of
 - Clinical signs
 - Laboratory diagnose which is based on the presence of bile pigments in the blood, urine and feces.
 - It can be diagnosed by ultra sound and radiography.

Treatment

- Treatment of animals during cholelithiasis can be done symptomatically and in rare case surgically.
- Feed animals easily digestible food like green mass for herbivores animal with parenteral administration of fat soluble vitamins.

Hepatobiliary cont'd---

- Pain due to colic must be treated by administration of analgesics/ antipyretics and sedatives.
 - Analgesics/ antipyretics like morphine chloride or pethidine hydrochloride.
 - Steroids and NSAIDs like ASA, paracetamol, phenylbutazone, metamizole, flunixin meglumine etc.
 - Sedatives like acepromazine maleate and xylazine.
- It is possible to remove the bile concretions surgically if the animal is high productive breed.
- Other wise cull the animal from production.

Part 6. Diseases of the respiratory system

- In this portion, we will see
 - 6.1. Pneumonia
 - 6.2. Bronchitis
 - 6.3. Pleuritis/Pleurisy

6.1. Pneumonia

➤ **Pneumonia** is the inflammation of the pulmonary parenchyma and usually accompanied clinically by an inflammation of the bronchioles and often by pleuritis/pleurisy.

➤ It is manifested by

- Increasing the respiratory rate
- Changing the depth and character of respiration
- Coughing, abnormal breath sounds in auscultation
- And in most bacterial pneumonias by the evidence of toxemia.

Etiology

➤ Pneumonia may be caused by

- Viruses
- Bacteria
- Fungi

Respiratory diseases cont'd ---

- 4. Contagious Bovine Pleuro-Pneumonia (CBPP) – *Mycoplasma mycoides*
- 5. Lungworm pneumonia – *Dictyocaulus viviparus*, etc
- 6. Tuberculosis – *Mycobacterium bovis*
- 7. Calf diphtheria caused by *Corynebacterium diphtheriae*

II. Pigs

- 1. Enzootic pneumonia – *Mycoplasma spp* with *Pasteurella spp* secondarily.
- 2. Pneumonic pasteurellosis – *P. multocida*
- 3. Plueropneumonia – *Actinobacillus (Haemophilus) pleuropneumoniae*
- 4. Interstitial pneumonia – septicemic salmonellosis

III. Horses

- Plueropneumonia in adult horses (*Streptococcus spp.*,

Respiratory diseases cont'd ---

spp., *E. coli*, *Bacteriodes spp*).

- 2. Strangles (*Streptococcus equi*)
- 3. Pulmonary hydatidosis

IV. Sheep

- 1. Pneumonic pasteurellosis (*Pasteurella spp*)
- 2. Lung worms (*Dictyocaulus filaria* and *M. capillaries*)
- 3. Maedivisa
- 4. Parainfluenza-3

V. Goats

- 1. Pleuro pneumonia (*Mycoplasma* strain F 38 or *M. capri*)
- 2. Retro virus infection

VI. All species

- Aspiration pneumonia

Respiratory diseases cont'd ---

Epidemiology

➤ Pneumonia is a complex disease condition which depends on different epidemiological factors:

└ Host (animal)

└ Agent (pathogen)

└ Environment and management

Pathogenesis

➤ Under normal conditions, a complex of biochemical, physiological and immunological defense mechanisms protects the respiratory tract from inhaled particles which could be injurious or infectious.

➤ The major defense mechanisms of the respiratory tract include:

→ Filtration by the nasal cavities

→ Sneezing

Respiratory diseases cont'd ---

- The cough reflex
- Muco ciliary transport mechanisms
- Alveolar macrophages
- Systemic and local antibody systems
- The path - physiology of all pneumonias, regardless of the way in which lesion develops, is based on interference with gaseous exchange between the alveolar air and the blood.
- In bacterial pneumonias, there is the added effect because of
 - Toxins produced by the bacteria and necrotic tissue
 - The accumulation of inflammatory exudates in the bronchi which is manifested by abnormal lung sounds such as **crackles**, and **wheezes** on auscultation.
- Extension of the pneumonia to the visceral surface of the pleura results in **pleuritis** **plurorppneumonia**

Respiratory diseases cont'd ---

- Restriction of gaseous exchange because of obliteration of alveolar spaces.
- In this stage blood flow through the affected part ceases.

Clinical findings

- Rapid shallow breathing is the cardinal sign of pneumonia
- Dyspnea - occurs in the later stages, when much of the lung tissue is non-functional
- Coughing in bacterial bronchopneumonia is usually accompanied by a **moist** and **painful cough**.
- But in viral interstitial pneumonia, the coughing is frequent **dry, hacking**, often in **paroxysms**.
- In acute bacterial bronchopneumonia, toxemia, anorexia, depression, tachycardia and reluctance to lie down are common.

Respiratory diseases cont'd ---

➤ **In viral interstitial pneumonias**, affected animals are usually not toxemic but they have a fever and reduced appetite or complete anorexia.

→ However some cases of **viral interstitial pneumonia** can be diffuse and severe and cause severe respiratory distress, failure to respond to therapy and death within a few days.

➤ **In chronic bronchopneumonia in cattle**, there is chronic toxemia, rough hair coat, and a gaunt appearance.

→ The respiratory and heart rates are above normal, and there is usually a moderate persistent fever.

→ A copious bilateral mucopurulent nasal discharge and a chronic moist productive cough are common.

Clinical pathology

➤ **Examination of respiratory secretions -**

Respiratory diseases cont'd ---

common diagnostic procedure performed in cases of pneumonia.

➤ So that the most common sample are

→ Nasal swabs

→ Tracheobronchial aspirates

→ Bronchoalveolar lavages samples

➤ The above samples can be collected and submitted for isolation of viruses, bacteria, fungi and determination of antimicrobial sensitivity(bacteria and fungi).

➤ Some times additional special diagnosis can be done like

→ **Thoracentesis** can be done when pleural effusion is suspected, and can be used to obtain pleural fluid for analysis.

→ **Hematology** examination can indicate the nature of

Respiratory diseases cont'd ---

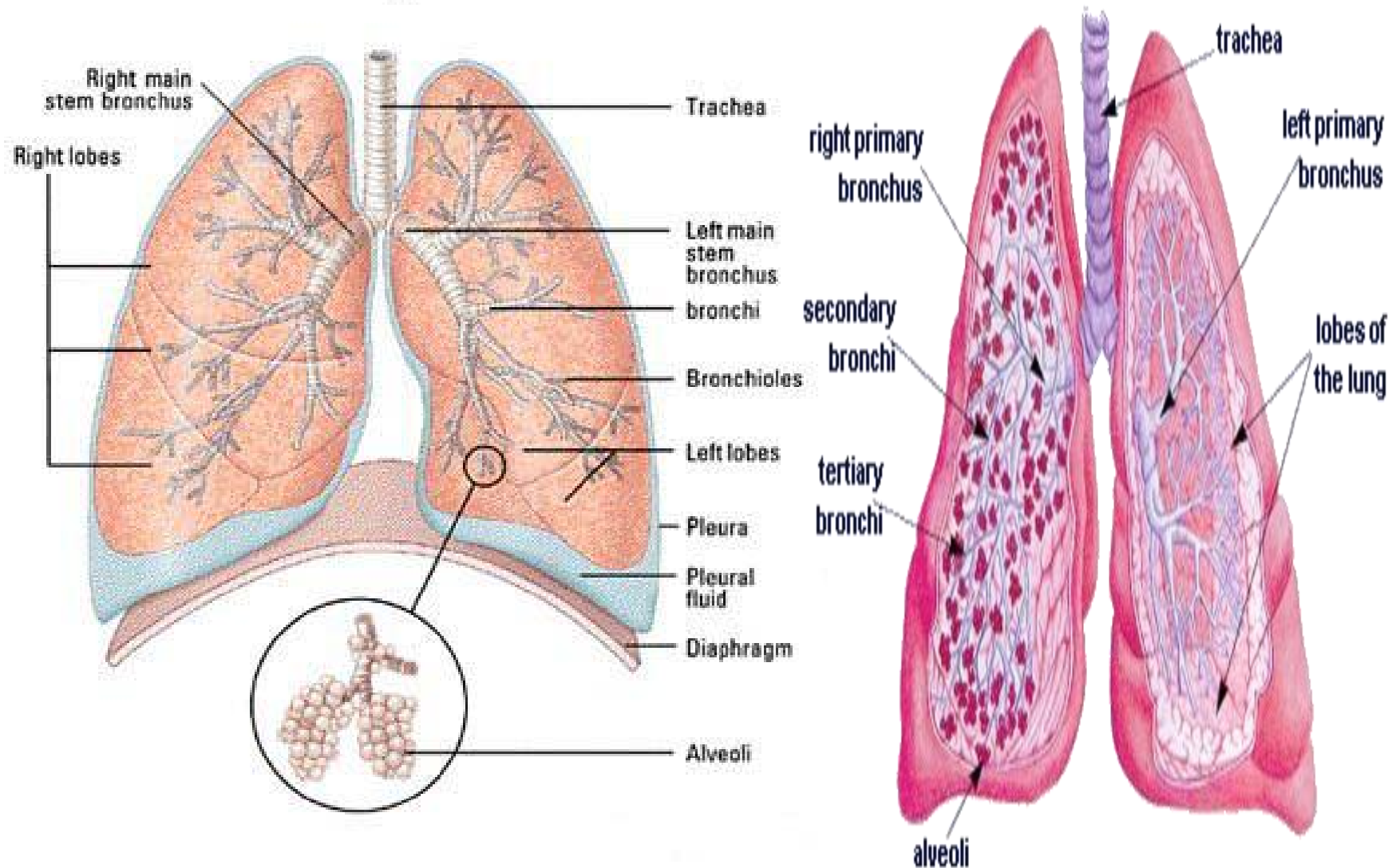
- **Fecal samples** is used when lung worm pneumonia is suspected, fecal samples can be submitted for detection of larvae.
- **Medical imaging** including thoracic radiography and ultrasonography.
- **Necropsy** is done in outbreaks of respiratory disease where diagnosis is uncertain, necropsy of selected early cases will often assist in making a diagnosis.

Necropsy findings

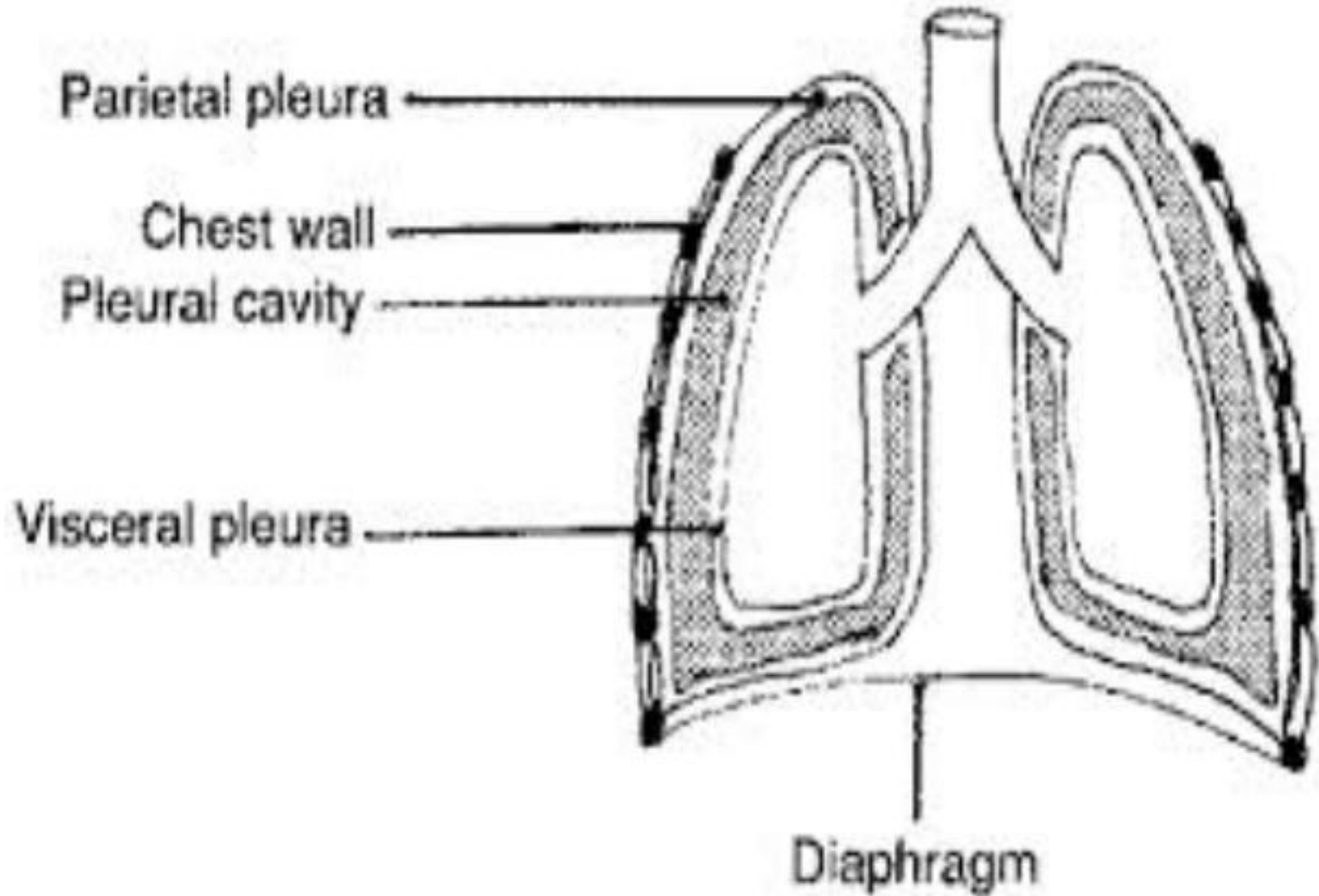
- Gross lesions are usually observed in the anterior and dependent parts of the lobes.
- The gross lesions vary a great deal depending upon the type of pneumonia present.
- **Bronchopneumonia** is characterized by
 - The presence of sero fibrinous or purulent exudates in the bronchioles and

Anatomical structure of lung

Lungs



Pleura of the thoracic cavity and lung



Blood congested lung



Red hepatization of lung



Gray hepatization of lung



Respiratory diseases cont'd ---

→ Lobular congestion or hepatization.

➤ **In severe fibrinous pneumonia**

→ There is gelatinous exudation in the interlobular septa and

→ An acute, pleurisy with threads of fibrin present between the lobes.

➤ **In interstitial pneumonia**

→ The bronchioles are clean and the affected lung is sunken, dark-red in color

→ And has a granular appearance under the pleura and on the cut surface.

→ There is firm thickening of the interlobular septa.

➤ **In chronic bronchopneumonia of cattle**

→ There is consolidation, fibrosis, fibrinous pleuritis and interstitial emphysema

Respiratory diseases cont'd ---

→ Abscesses are common.

Diagnosis

➤ The diagnosis of pneumonia requires consideration of the following factors

→ Epidemiological factors (host, agent and environment)

→ Clinical findings

→ Clinical pathology

Differential diagnosis

➤ Pneumonia must be differentiated from

➤ **1. Diseases of other body systems (which may cause polypnea and dyspnea)**

→ Congestive heart failure

→ Terminal stages of anemia

→ Poisoning by histotoxic agents

Respiratory diseases cont'd ---

➤ These diseases are accompanied by respiratory embarrassment but not by the abnormal sounds typical of pulmonary involvement.

➤ **2. Pleuritis**

- Pleuritis is different from pneumonia in that
 - It is characterized by shallow abdominal-type respiration
 - By its pleuritis friction sounds when effusion is minimal
 - By its muffling of lung sounds on auscultation.
 - Thoracentesis reveals the presence of fluid.

➤ **3. Pulmonary edema and congestion (embolism of pulmonary artery and emphysema)**

- In such cases there is no toxemia and fever.

➤ **4. Pneumothorax**

Respiratory diseases cont'd ---

- Increased intensity of the heart sound
- Increased resonance on percussion

➤ **5. Diseases of the upper respiratory tract (laryngitis, tracheitis)**

➤ **Inspiratory dyspnea**

- Reveal moist wheezing sounds on auscultation
- More frequent cough than pneumonia
- Cough can be induced by squeezing the larynx or trachea

Treatment

1. Antimicrobial therapy

- In specific bacterial infections, isolation of the affected animals and careful surveillance of the remainder of the group should accompany administration of antimicrobials to affected animals.

Respiratory diseases cont'd ---

preparations.

- However, the blood levels from the long-acting preparations are not as high as the short-acting preparations and may not be effective in severely common causes of failure to respond to treatment for bacterial affected animals.

2. Supportive therapy

- Affected animals should be housed in warm, well ventilated, draft free accommodation, provided with fresh water and feed.
- During recovery, premature return to work or exposure to inclement weather should be avoided.

6.2. Bronchitis

- It is an inflammation of bronchus.
- Bronchitis affect all species animals of different age and sex groups

Bronchitis cont'd---

- Bronchitis can be

- Acute

- Chronic

- By the origin of etiological agent, it can be

- Primary

- Secondary

- By the nature of inflammatory exudate it can be

- Catarract

- Pus

- Hemorrhagic

- Fibrous

- You know the size of bronchus is different.

- So the inflammation of large bronchus(bronchus of first, second and third orders) is called **macro bronchitis**.

Bronchitis cont'd---

- Bronchitis can be

- Acute

- Chronic

- By the origin of etiological agent, it can be

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- Secondary

- By the nature of inflammatory exudate it can be

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- Hemorrhagic

- Fibrous

- You know the size of bronchus is different.

- So the inflammation of large bronchus(bronchus of first, second and third orders) is called **macro bronchitis**, while inflammation of

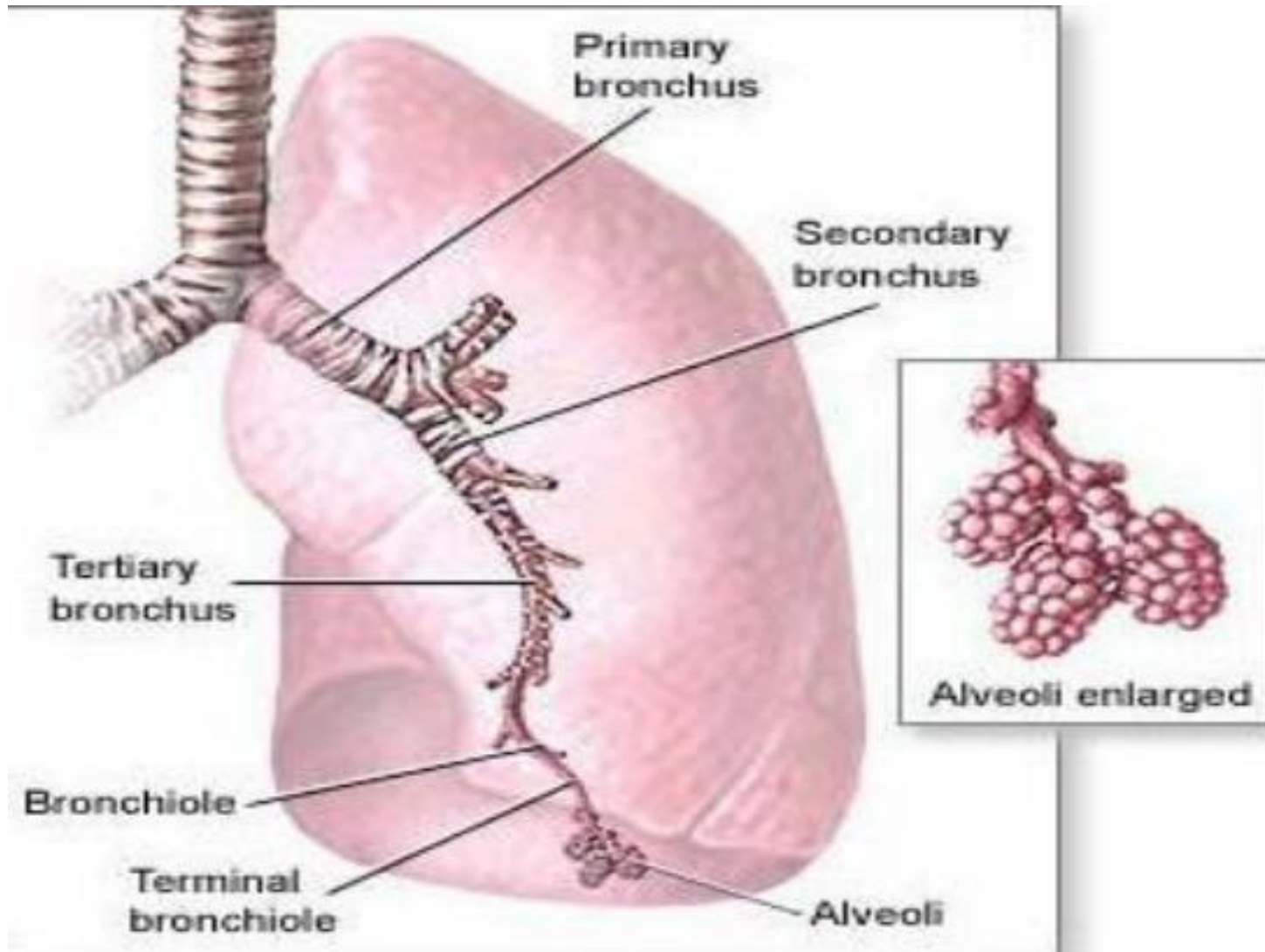
Bronchitis cont'd---

small bronchus is called **micro bronchitis**.

Etiology

- ‖ Very cold microclimatic condition
- ‖ High humid air
- ‖ High concentration of irritant gases like NH_3 , CH_4 , H_2S
- ‖ Very often changing of the temperature of animal house
- Bronchitis may have mass character in sheep after shoring their hair(wool) in cold environmental condition.
- Bronchitis may occur if foreign materials(feed, medicaments) are enter in to bronchus.
- **NB.** Many foreign materials may enters to bronchus during: -
 - Oral administration drugs

Structure of bronchus



Bronchitis cont'd---

→Bronchitis may also occur if the animals inhales dusts for along time.

Predisposing factors for bronchitis

- High stocking density of animals
- High bacterial contamination of the animal house
- Absence of UV light and canalization
- Absence of retinol in animals ration

Secondary bronchitis

➤Bronchitis which is developed as complication from upper respiratory tracts and sinuses.

Pathogenesis

➤By the action of etiological factors like irritant gases and dusts etc on the mucousa of bronchus disturbs **the neuro - humoral regulation of the function of bronchus.**

Further the capillaries dilate which results in

exudation.

➤ This creates favorable environment for reproduction of microbes.

➤ The microbes reproduce in bronchus and produce toxin which results in death of epithelial cells of bronchus.

➤ Exudate will be accumulated in large bronchus, in bronchioles and in bronchus of small calipers that have mucous, epithelial cells of bronchus, leucocytes, erythrocytes and microbes.

➤ Inflammatory product will be absorbed in to blood and lymphatic system.

➤ This all results in **intoxication of the organism** which is called **toxemia.**

➤ **Hyperemia, edema** and **accumulation of exudate** in bronchus disturbs gas exchange.

➤ If the further action of the etiological factor is removed

Bronchitis cont'd---

immunity.

- But if the etiological factor/s continues its action and if the animal is not treated appropriately, the inflammatory process will continue to parenchyma of the lung and cause **bronchopneumonia**.
- Lastly if the etiological factor of bronchitis acts on the organism for long time or if it acts repeatedly with periodical action the **acute bronchitis** will be changed into **chronic bronchitis**.
- In chronic bronchitis
 - The exudation decrease but has high viscosity.
 - There is atrophy and death of the epithelial cells of bronchus.
 - In rare case there is infiltration and death the cells of under serosal and muscle tissue.

Which results replacement of **epithelial and soft**

Bronchitis cont'd---

- Slowly the bronchus loses its defensive, elasticity and develops **functional stenosis**.
- This results in disturbance of gas exchange and develops **hypoxia**.
- In chronic bronchitis and peribronchitis there may be stenosis of bronchus because of the growth of connective tissue.
- And this blockage results in **atelectasis, focal pneumonia, dilation of bronchus or focal emphysema** of lung.

Clinical signs

- ➡ The main clinical signs in acute bronchitis in the first day are
 - Inappetance
 - Body temperature of animals is found in higher limited range of body temperature or increased by 0.5 –

Bronchitis cont'd---

- Auscultation of lung shows the presence of **hard vesicular breathing, dry chirp** that can be heard at the distance.
- In acute bronchitis if the affected animals are treated on time, they will recover quickly after 7 – 10 days.
- But complication of bronchitis because of delay in treatment result in chronic form of bronchitis.
- The chronic form of bronchitis called **bronchopneumonia.**
- Chronic bronchitis is characterized by
 - Depression
 - Fever
 - Periodic dry cough
 - Cyanosis of the mucousa and skin
 - Force full breathing

Bronchitis cont'd---

➤ Auscultation of lung during chronic bronchitis(bronchopneumonia) characterized by **dry chirp breathing sound.**

Necropsy findings

➤ **1. In acute form of bronchitis** the lesions are found in the mucousa of bronchus.

→ The mucous membrane of bronchus is hyperemic, edematic and has cataract exudate.

→ In histopathological examination of the mucousa of bronchi or bronchial tubes, there is death of epithelial cells with losing of their cilia.

→ And in exudate there are large number of dead epithelial cells, leukocytes, erythrocytes and microbes.

➤ **2. In chronic form of bronchitis also called bronchopneumonia** is characterized by the following necropsy findings

Bronchitis cont'd---

- Loss of elasticity of bronchi
- In some portion of lungs, there are also **bronchi stenosis or dilation of bronchi**(bronchiectasis) and/or peribronchitis.
- In bronchial tubes there is some **high viscous serous exudate**.
- In lung there is focus of **emphysema**.
- Rarely there is enlargement of **mediastinal lymph nodes**.

Diagnosis

- Diagnose of bronchitis is done based on
 - Anamnesis
 - Clinical findings
 - Radiography in chronic bronchitis

Differential diagnose

Bronchitis cont'd---

- For this it needs special laboratory diagnostic methods.

Treatment

- First identify the etiological factor and avoid it.
- Treatment of acute and chronic bronchitis must be done by using complex therapeutic methods.
- They are
 - ↳ Pathogenetic therapy
 - ↳ Symptomatically therapy
- Treatment of bronchitis must be directed for liquidification of the mucous that are found in bronchi.
- Liquidification of inflammatory mucous can be done by using drugs that have **expectorating** and **disinfection effect on mucous that are in** bronchi.
- The most common **expectorants** that are used in

Bronchitis cont'd---

practice are

- Ammonium chloride(NH_4Cl)
- Terpin hydrate
- Sodium bicarbonate(NaHCO_3)

Dose and route of administration

1. Ammonium chloride(NH_4Cl)

- Horse, cattle, sheep and pigs dose 0.02 – 0.03 gm/ Kg BW together with liquid feed for 5 – 7 days 2- 3 times in a day.

2. Terpin hydrate

- For the above species of animals in dose of 0.01 – 0.03 gm/ Kg BW together with liquid feed for 5 – 7 days 2 -3 times in a day.

3. Sodium hydro carbonate (NaHCO_3)

- For the above species of animals in dose of 0.1 – 0.2 gm/ Kg BW together with liquid feed for 5 – 7 days 2 – 3

Bronchitis cont'd---

animals with steam of **NaHCO₃**, **menthol** and **eucalyptus tree leaf** extraction.

➤ In chronic bronchitis and peribronchitis specially those in the way of stenosis and complete obstruction, it is very important to use complex method of treatment.

➤ For this we must use medicaments that have the following properties

- | 1. Bronchi dilators
- | 2. Proteolytic enzymes

1. Bronchi dilators

➤ For dilation of bronchi we use bronchi dilators like 5% ephedrine , euphyllin (aminophylline or theophylline).

Dose and route of administration

- Horse and cattle 7 - 10 ml subcutaneously 2 times a day.
- Small ruminants and pigs 1 - 1.5ml subcutaneously

Bronchitis cont'd---

2. Proteolytic enzymes

- They are enzymes like pepsin and trypsin that can liquidify the mucous exudate in bronchi, bronchioles and bronchi of small calipers.
- **Example** good result is obtained by intratracheal administration of **1 - 2 mg/ kg BW pepsin or trypsin** and subcutaneous administration of 5 - 8 mg/Kg BW **euphyllin** for 5 continuous days.

6.3. Pleuritis/ pleurisy

- Inflammation of pleura is known as **pleuritis** or by the older name called **pleurisy**.
- The ordinary forms of pleuritis belong to the acute exudative inflammations, usually being either **serous, fibrinous, or purulent**.
- If the pleuritis accompanies pneumonia, the pleuritic area over lies the hepatized portions of lung parenchyma, and the condition is called **plueropneumonia**.

Etiology

- The causes of pleuritis are usually infectious, and in general, they are the same kinds of organisms which cause pneumonia.
- Infection of the pleura may result by direct extension from the lung.
- But in many cases of pleuritis arise by the

Pleuritis cont'd---

the following:

- *Pasteurella multocida*
- *Mannheimia haemolytica*
- *Mycoplasma* spp. which cause
 - ↳ Contagious Bovine Pleuropneumonia (CBPP)
 - ↳ Contagious Caprine Pleuropneumonia (CCPP)
 - ↳ Pleuropneumonia in pigs
- *Actinobacillus (Haemophilus) pleuropneumoniae*
- Influenza suis
- Tuberculosis (*Mycobacterium bovis* in cattle)

Pathogenesis

- Pathogenesis of pleuritis has the following stages:-
 - **1. The early stage of pleurisy** begins with acute hyperemia and swelling of the thin covering membrane.

Pleuritis cont'd---

➡ During this stage:-

- The friction of the visceral and parietal surfaces at each respiratory movement is very painful.
- And causing a typical kind of breathing.

➡ **2. The second stage develops** after about two days and follows :-

- With appearing of **serous exudate** which lubricates and separates the two surfaces.
- The pain is assuaged.
- The exudate may remain **serous**, filling the pleural cavity and compressing the lungs or it may become **fibrinous or purulent**.
- But very often all forms co-exist, resulting in a **sero-fibrino-purulent pleuritis**.

3. In the third stage of development of pleuritis

Pleuritis cont'd---

- As the result there is restricting movement of the lungs and chest wall.
- But the interference with respiratory exchange is usually minor and disappears gradually as the adhesion stretch with continuous movement.

➤ **NB.** In all bacterial pleuritis, toxemia is common and usually severe.

➤ The toxemia may be severe when large amount of pus accumulate.

Clinical findings

➤ Clinical findings of pleuritis vary from mild to severe depending on

- The species

- The nature and severity of the inflammation

➤ **In the early stages of pleuritis**, the main clinical features are

Pleuritis cont'd---

- The animal stands with its **elbows abducted** and is dis-inclined to move (evidence of pleural pain).
- In bacterial causes of pleuritis, **toxemia** may be present and hence anorexia and depression may be observed.
- Pleural friction sounds on auscultation – common in the early stages of pleuritis.
- Subcutaneous edema
- Dullness on percussion over the fluid filled area of the thorax is characteristic of pleuritis in which there is a significant amount of pleural effusion.
- In the recovery stage** – animals with pleuritis characteristically
 - Recover slowly over a period of several days even weeks.
 - The toxemia usually resolves first but abnormalities in



Clinical pathology

1. Thoracocentesis (pleurocentesis) is paracentesis of the thoracic cavity.

- ➡ Collection of fluid from thoracic cavity by thoracocentesis (**pleurocentesis**) helps to conduct
 - Odor, color and viscosity differentiation of the fluid (exudate, transudate or water)
 - Protein concentration determination
 - To know the presence of blood, or tumor cells and
 - To collect samples for culturing to identify the etiological agents(bacterial or viral or fungal).

2. Medical imaging

- Radiographic examination
- Ultrasonography
- Pleuroscopy

3. Hematology

Necropsy findings

- **In early acute pleuritis**, there is marked edema, thickening and hyperemia of pleura, and presence of shreds of fibrin.
- **In the exudative stage**, the pleural cavity contains an excessive quantity of turbid fluid containing flakes and clots of fibrin.
- **In the later healing stages**, adhesions connect the parietal and visceral pleura.

Diagnosis

- Diagnose of pleura is done based on
 - Major clinical signs – thoracic pain, fever and toxemia
 - Result of medical imaging
 - Result examination of fluid that is collected by pleurocentesis

Pleuritis cont'd---

Differential diagnosis

➤Pleuritis must be differentiated from

➤**1. Pneumonia** – pneumonia usually occurs in conjunction with pleuritis and differentiation is difficult

➤**2. Pulmonary emphysema** – is characterized by loud crackles, expiratory dyspnea and lack of toxemia

➤**3. Hydrothorax and hemothorax**

➤**4. Pulmonary congestion and edema**

Treatment

➤Antimicrobial therapy – the primary aim of treatment is to control the infection in the pleural cavities.

➤It can be done by using systemic administration of antimicrobials.

➤We can use the following antibiotics

→Broad-spectrum antimicrobials

Pleuritis cont'd---

→ Combination of antimicrobials

- Narrow spectrum antibiotics based the result of antibiotic sensitivity test after isolation and identification of the agent.
- We can also drain the pleural fluid to allow the lung to re-expand.
- We can also use analgesics like phenylbutazone in horses to relieve pain.

Part 7: Disease of the cardiovascular system

➡ Here we will focus on

➤ I. Disease of heart

└ 7.1. Pericarditis

└ 7.2. Myocarditis

└ 7.3. Endocarditis and valvular heart disease

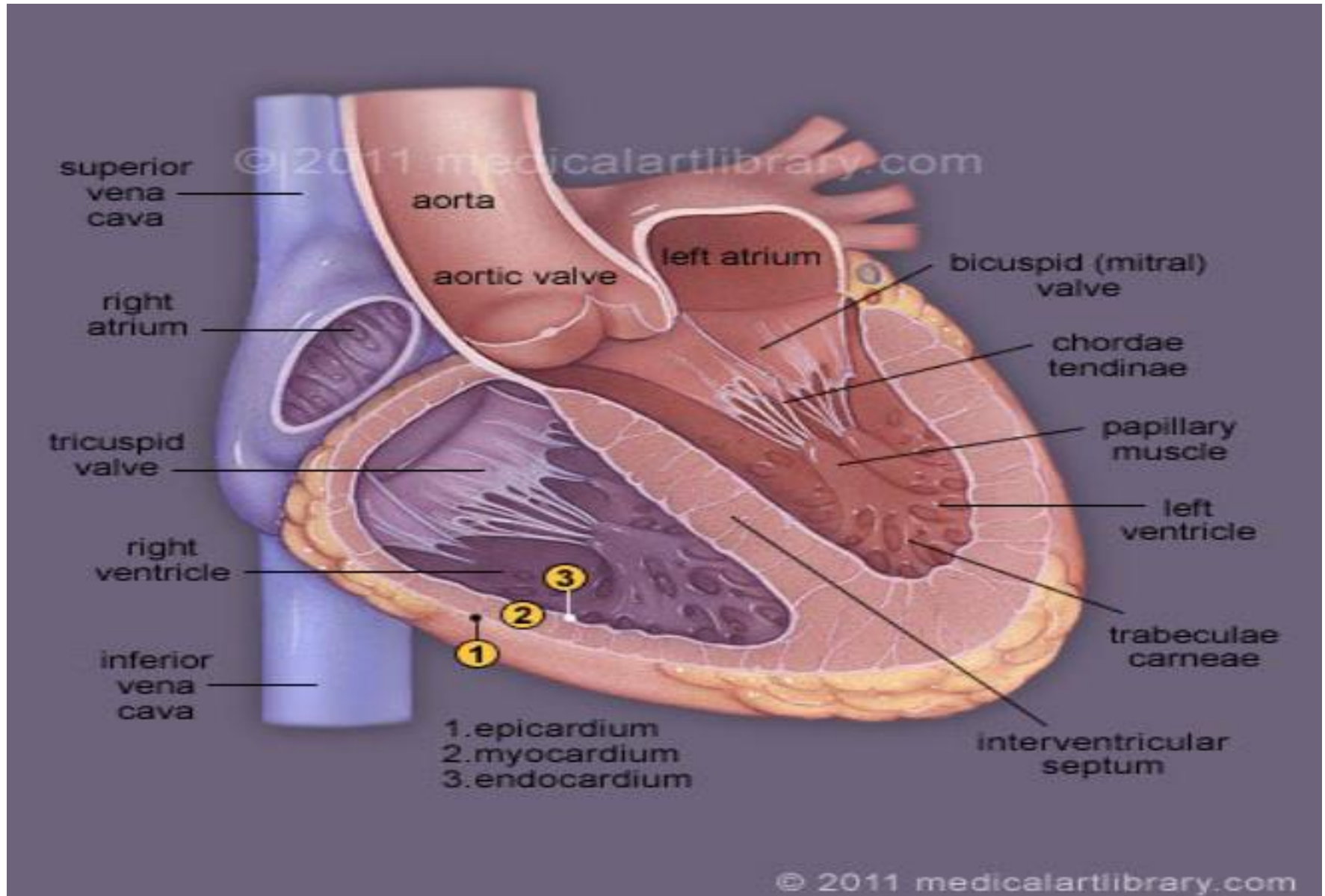
➤ II. Diseases of the blood vessels

└ 7.4. Arterial thrombosis

└ 7.5. Acute and peripheral circulatory failure

└ 7.6. Thrombosis and embolism

Anatomical structure of heart

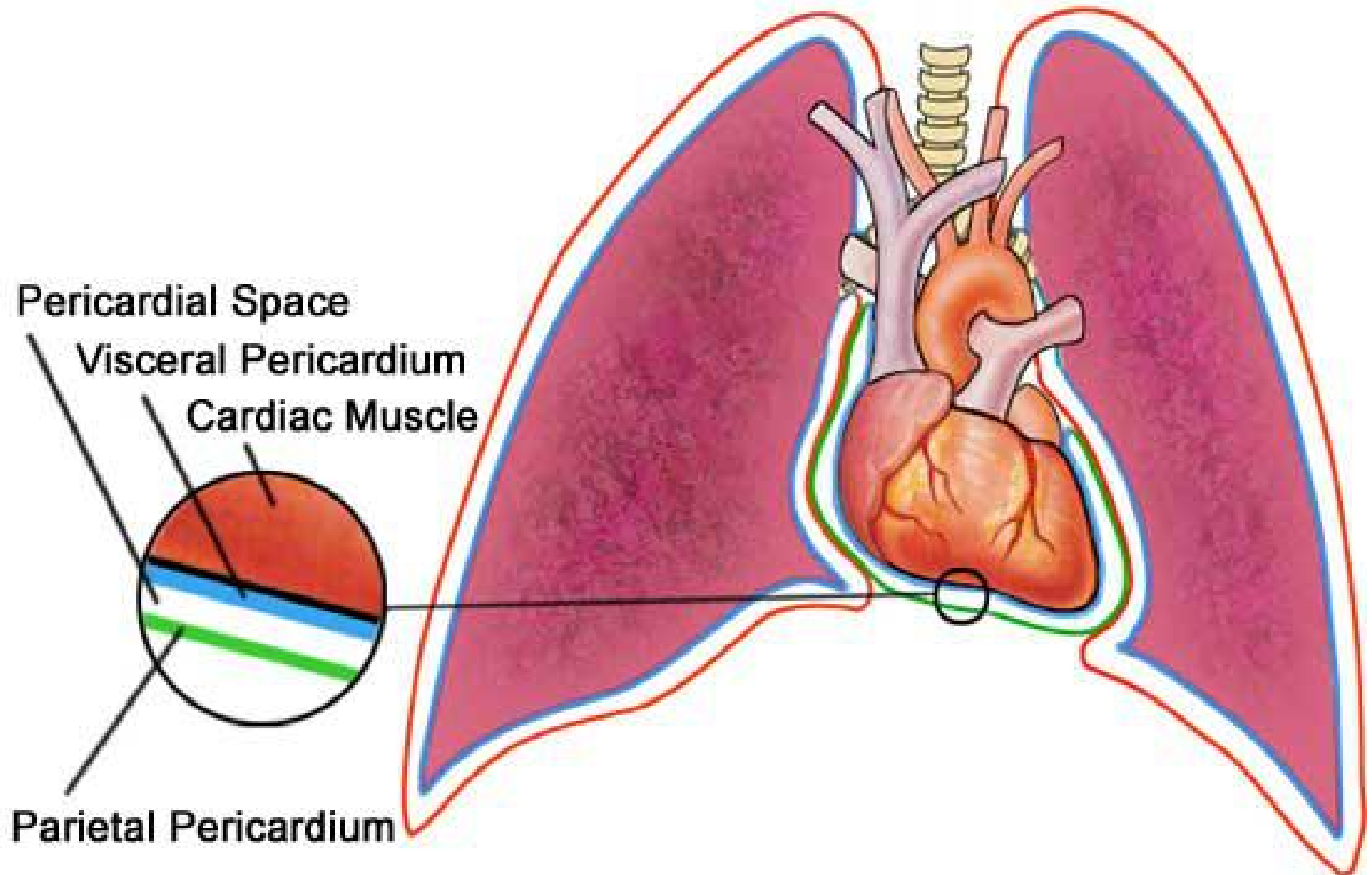


I. Diseases of heart

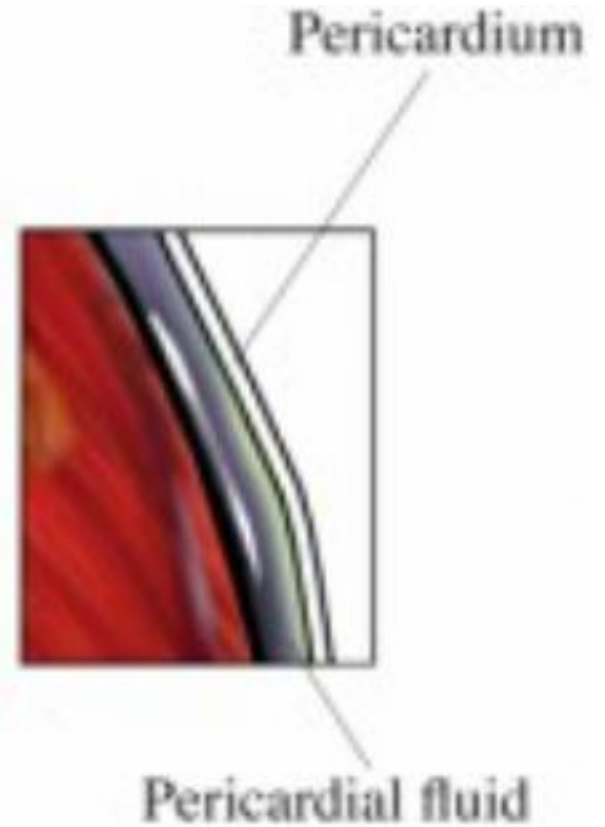
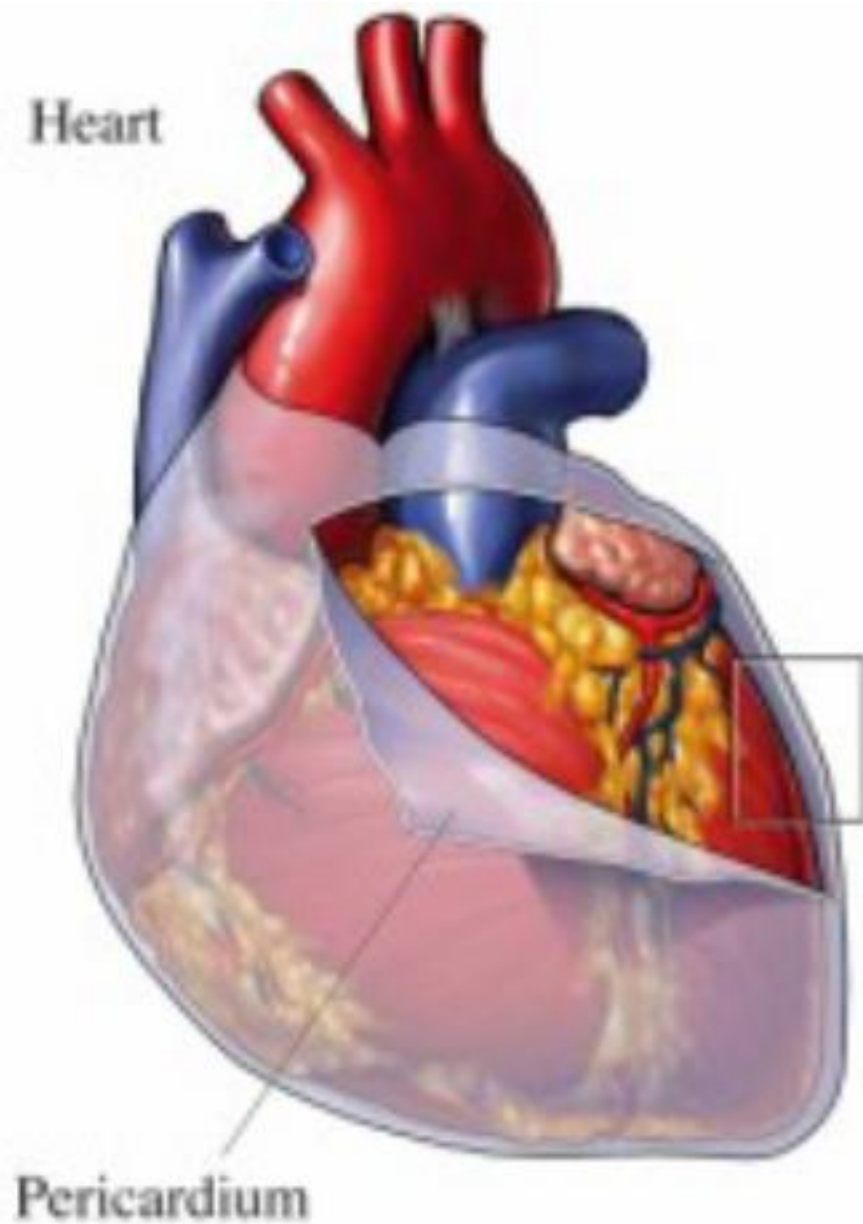
7.1. Pericarditis

- This is inflammation of the pericardial sac characterized by
 - Audible friction sound and
 - Muffling of the heart sound which leads to congestive heart failure.
- Pericarditis is not common but presents in three general forms:-
 - ↳ 1. Effusive
 - ↳ 2. Fibrinous
 - ↳ 3. Constrictive
 - ↳ 4. Traumatic
 - ↳ 5. Although combinations of one or more of the three forms can occur.

Pericardium of heart and lung



Pericardium of heart



Pleuritis cont'd---

(transudat) within the pericardial sac.

2. Fibrinous pericarditis

- It is the subsequent of effusive pericarditis characterized by fibrin deposition which can lead to fibrinous pericarditis.

3. Constrictive pericarditis

- It is the subsequent of fibrinous pericarditis formed as the result of maturation of fibrin within the pericardial sac and it may reach up to epicardium.

4. Traumatic pericarditis

- Pericarditis which is caused by perforation of the pericardial sac by an infected foreign body, occurs commonly only in cattle.
- It is the extension from traumatic reticulitis or TRP.

Etiology

- Traumatic pericarditis (extension from traumatic

Heart cont'd---

- Extension from pleurisy or myocarditis
- ➡ There are many cases of pericarditis with unknown reasons and are called **idiopathic pericarditis**.

Some common bacteria and virus that cause pericarditis in domestic animals

Species of animals	Causative agent and disease of animals
Cattle	<i>Mannheimia haemolytica</i>, Black disease - if patients survive more than 24 hours
	Pasteurellosis , <i>Staphylococcus aureus</i>, <i>Mycoplasma</i> spp.

Heart cont'd---

Species	Causative agent and disease of animals
	Sporadic bovine encephalomyelitis, <i>Haemophilus</i> spp., including <i>Histophilus somni</i>, Tuberculosis, <i>Pseudomonas aeruginosa</i>, <i>Mycoplasma</i> spp., <i>Klebsiella pneumoniae</i> <i>Actinobacillus suis</i>, Idiopathic effusive (non septic) pericarditis.
Horses	<i>Streptococcus</i> spp., including <i>S. equi</i>, <i>S. zooepidermicus</i> and <i>S. faecalis</i>, Tuberculosis, <i>Corynebacterium pseudotuberculosis</i>, <i>Actinobacillus equuli</i> in association with EHV-1 infection Idiopathic effusive (non septic) pericarditis

Pathogenesis

- In the early stages, of inflammation of the pericardium is accompanied by
 - Hyperemia and
 - Deposition of fibrinous exudates which produces a friction sound
- Because the pericardium and epicardium rub together during cardiac movement.
- As effusion develops the inflamed surfaces are separated, the friction sound is replaced by muffling of the heart sounds,
- As the result the accumulated fluid compresses the atria and right ventricle, preventing their complete filling and **congestive heart failure follows.**
- A severe toxemia is usually present in suppurative pericarditis
- Because of toxins produced by the bacteria in the

Heart cont'd---

epicardium to cause an adhesive pericarditis.

Clinical findings

➤ Initial stage of pericarditis is characterized by

- Pain

- Avoidance of movement

- Abduction of the elbows

- Arching of the back

- Shallow abdominal respiration

- Pain can be induced through palpation over the cardiac area of the chest

- Pericardial friction sound detectable on auscultation

- Elevated temperature (39 °C - 41°C)

- Increased pulse rate

- Associated signs of pleurisy, pneumonia and peritonitis

Arching of back and abduction of elbow



Heart cont'd---

- Advanced stage of pericarditis is characterized by
 - Muffling of the heart sounds
 - Signs of congestive heart failure (edema and exudates)
 - Toxemia in pyogenic cases
 - Distension of the jugular veins

Necropsy finding

- In the early stages there is
 - Hyperemia of the pericardial lining and
 - A deposit of fibrin
- When effusion occurs there is an accumulation of turbid fluid and tags of fibrin are present on the greatly thickened epicardium and pericardium.
- Gas may also be present and the fluid may have a putrid odor.

Heart cont'd---

- When the pericarditis has reached a chronic stage, the pericardium is adherent to the epicardium over a greater or lesser part of the cardiac surface.
- Loculi containing serous fluid often remain.
- Embolic abscesses may be present in other organs.
- Lesions typical of the specific causative diseases listed above are described under their specific headings.

Diagnosis

- Diagnosis of pericarditis is done based on
 - History – common in feedlot cattle occurring as TRP
 - Clinical findings like
 - ↳ Characteristic signs of thoracic pain
 - ↳ Reluctance to move
 - ↳ Depression

Heart cont'd---

- ‖ Arching of the back and grunting
- ‖ Pericardial friction sound
- Other special method of diagnose of pericarditis are
 - Electrocardiography
 - Radiography

Differential diagnosis

- Pericarditis must be differentiated from
 - Pleuritis
 - Cardiac valvular disease
 - Mediastinal abscess
 - Hydro pericardium occurs in congestive heart failure

Treatment

- Antimicrobial treatment based on sensitivity test (if possible)
- Broad-spectrum antimicrobials

Heart cont'd---

→Combination of antimicrobials (Eg, penicillin + gentamycin)

7.2. Myocarditis

➤It is an inflammation cardiac muscle called myocardium.

➤Myocardium can be

→Acute

→Chronic

➤By localization of pathological lesions myocarditis can be

→Focal

→Diffuse

➤By the origin of pathological agent myocarditis can be

→Primary

→Secondary

Heart cont'd---

Etiology

➤ Myocarditis very often occurs as secondary complication disease during many infectious, parasites and metabolic diseases of animals.

– Myocarditis may occur due to mycotic and

	Viral	Parasitic	Metabolic (nutrition)	Toxin
→ Following bacteremia, as in strangles or from navel-ill → Tuberculosis - especially horses → Tick pyemia in lambs	→ Foot-and-mouth disease - especially young animals → African horse sickness → Equine viral arteritis → Equine infectious anemia	Bacterial	→ Vitamin E/selenium deficiency in all large animal species → Some forms of chronic copper deficiency	→ Inorganic poisons - selenium, arsenic, → Mercury, phosphorus, thallium → Gossypol from cotton seed cake

Heart cont'd---

Bacterial	Viral	Parasitic	Metabolism	Toxin
→ Tick pyemia in lambs → Black quarter (black leg)	→ Equine herpesvirus-1 in fetus → Swine vesicular disease → Parvovirus in piglets → Encephalomyocarditis virus infection in pigs → PRRS virus in piglets → Bluetongue		→ Iron deficiency in piglets and calves → Copper/cobalt deficiency in lambs	The mycotoxin fumonisin when ingested by pigs and horses

Pathogenesis

- Inflammation of myocardium develops by the action of different toxin of bacterial and fungus and diseases of viral origin.
- The process often starts from coronary artery.
- At the beginning there is exudation and edema of cardiac muscle.
- There are two periods of development of myocarditis.
- These are
 - 1. Primary stage of development myocarditis
 - 2. Secondary stage of development myocarditis

1. Primary stage development myocarditis

- In this period still structural change of myocardium is not begin.
- But there irritation of the receptor of myocardium with product of inflammation and toxin of bacteria and their metabolic product.

Heart cont'd---

- Results in the increasement of blood pressure.

2. Secondary stage of development myocarditis

In this stage there is dystrophic and degenerative change in myocardium.

This leads to

- In decreasing the contraction of myocardium and blood pressure.
- Development of arrhythmia
- Development of dyspnea, cyanosis and edema

Clinical signs

- Clinical signs during myocarditis depends on its stage of development.
- However there is always fever, depression and inappetance or anorexia.
- Productivity and ability of the animal to perform work

Heart cont'd---

decreased.

➤ **During the first stage of myocarditis there is**

- Tachycardia
- Extra systole
- High amplitude and strength pulse with well filled and distended arterial wall
- Pain around the cardiac region of thoracic cavity.
- An increasement of cardiac tone specially the systole tone.
- An increasement of blood pressure

➤ **During the second stage of myocarditis there is**

- Bradycardia
- Additional sound between systole and diastole tones
- Low amplitude and strength pulse with not well filled and distended arterial wall.

Heart cont'd---

- All this leads to decrease of tones of heart
- Consequently arterial blood pressure decreased however, vein pressure is increased.
- All these leads to the disturbance of many systems organism(Breathing, liver, kidney, CNS, digestive system etc.)
- That results in the development of cyanosis, edema in different organs.

Necropsy findings

➤ Necropsy finding on myocardium depends on the stage of myocarditis

➤ In the first stage of myocarditis: -

- Infiltration and thicken myocardium
- Hemorrhagic of myocardium rarely with petechial or ecchymotic character)

➤ In the second/ dystrophic and degenerative

Heart cont'd---

myocardium): -

- Myocardium is pale in colour.
- Cardiac muscle fibers lose their structure due to the development of scar.
- In deep degenerative myocarditis, myocardium has fried meat colour.

Diagnosis

- Diagnose of myocarditis is done based on
 - Clinical signs
 - Myocardial functional test on animals by giving an exercise load.
 - By using electrocardiogram(electrocardiography).

Differential diagnosis

- Myocarditis must be differentiated at pericarditis and endocarditis.

Treatment

- Treatment of myocarditis must be started by removing of the primary cause of myocarditis(bacteria, virus, fungi and their toxins etc).
- In the primary stage of myocarditis do not administer cardiovascular drugs as the systole of the heart is increased as this stage.
- Administration of these drugs may cause paralyses of the heart muscle.
- Instead at this stage put the ice in the cardiac region to decrease the expansion of inflammatory process.
- At this stage it is possible to administer anti allergic drugs mostly corticosteroids and antimicrobials like broad spectrum antibiotics and sulfa drugs.
- In the second stage of myocarditis since there is reduction of the systole of heart, it is possible to administer cardiovascular preparates that stimulates

Heart cont'd---

- It is possible to administer sodium caffeine benzoate together with 30 – 40 % glucose IV to stimulate the function of myocardium.
- In the case if the arterial blood pressure drops, administer 1: 1000 adrenalin by 2 – 3ml, 0.5 – 1 ml for large and small animals respectively.

Prophylaxis and prevention measures

- Treat animals from primary causes of a diseases.
- Prevent animals or treat animals on time from toxico - infections.

7.3. Endocarditis and valvular heart disease

7.3.1. Endocarditis

- Endocarditis is the inflammation of endocardium.

Heart cont'd---

- By localization of pathological process it can be
- On the wall of endocardium
- Valvular

Etiology

- Endocarditis occurs as complication during many infectious and toxic diseases of animals and during different traumatic factors.
- It can also occurs during myocarditis as the result of extension of inflammatory process from

Species of animal	Infectious diseases agents that cause endocarditic
Cattle	→ Alpha-hemolytic streptococci → <i>Actinomyces</i> → <i>Corynebacterium pyogenes</i> → <i>Micrococcus and Staphylococcus spp.</i> → <i>Pseudomonas spp.</i> → <i>Clostridium chauvoei (blackleg)</i> → <i>Mycoplasma mycoides</i>

Heart cont'd---

Species of animals	Infectious disease agents that cause endocarditis
Horses	→ <i>Actinobacillus equuli</i> → <i>Streptococcus</i> spp., including <i>Streptococcus equi</i> and <i>Streptococcus zooepidermicus</i> → <i>Pasteurella/Actinobacillus</i> spp. → <i>Pseudomonas</i> spp. → Migrating <i>Strongylus</i> spp. larvae.
Pigs and sheep	→ <i>Erysipelothrix rhusiopathiae</i> (→ <i>Streptococcus</i> spp. including → <i>Streptococcus equisimilis</i>, <i>Streptococcus dysgalactiae</i>, <i>Streptococcus suis</i> → <i>Escherichia coli</i> → <i>A. pyogenes</i>.

Pathogenesis

- In endocarditis in addition of inflammatory process there is degenerative and necrotic process.
- Very often the inflammatory process starts from valves of the heart.
- From example the seminal valves of aorta and pulmonary artery are infested from the side of ventricles.
- While the arterioventricular valves (bicuspid and tricuspid valves) are infested on the side of auricles.
- These results show that endocarditis develops from valves of the heart as the result of action pathogenic microorganisms and their toxins on the valves.
- From the valves(cartilage fibers and papillary muscles) the pathologic lesions may extend to the endocardium.
- So that depends on the virulence of pathogenic

Heart cont'd---

2. Ulcerative endocarditis

➤ **1. In valvular endocarditis** the inflammatory process has superficial character with hyperplasia or necrosis of endocardium.

➤ As the result on the surface of valves accumulate fibrin, thrombocytes, leucocytes that can be changed to **thrombosis** .

➤ All these lead to in deformation of valves of heart with disturbance of blood circulation.

➤ Very rarely this disease may lead to acquired cardiac defect on animals.

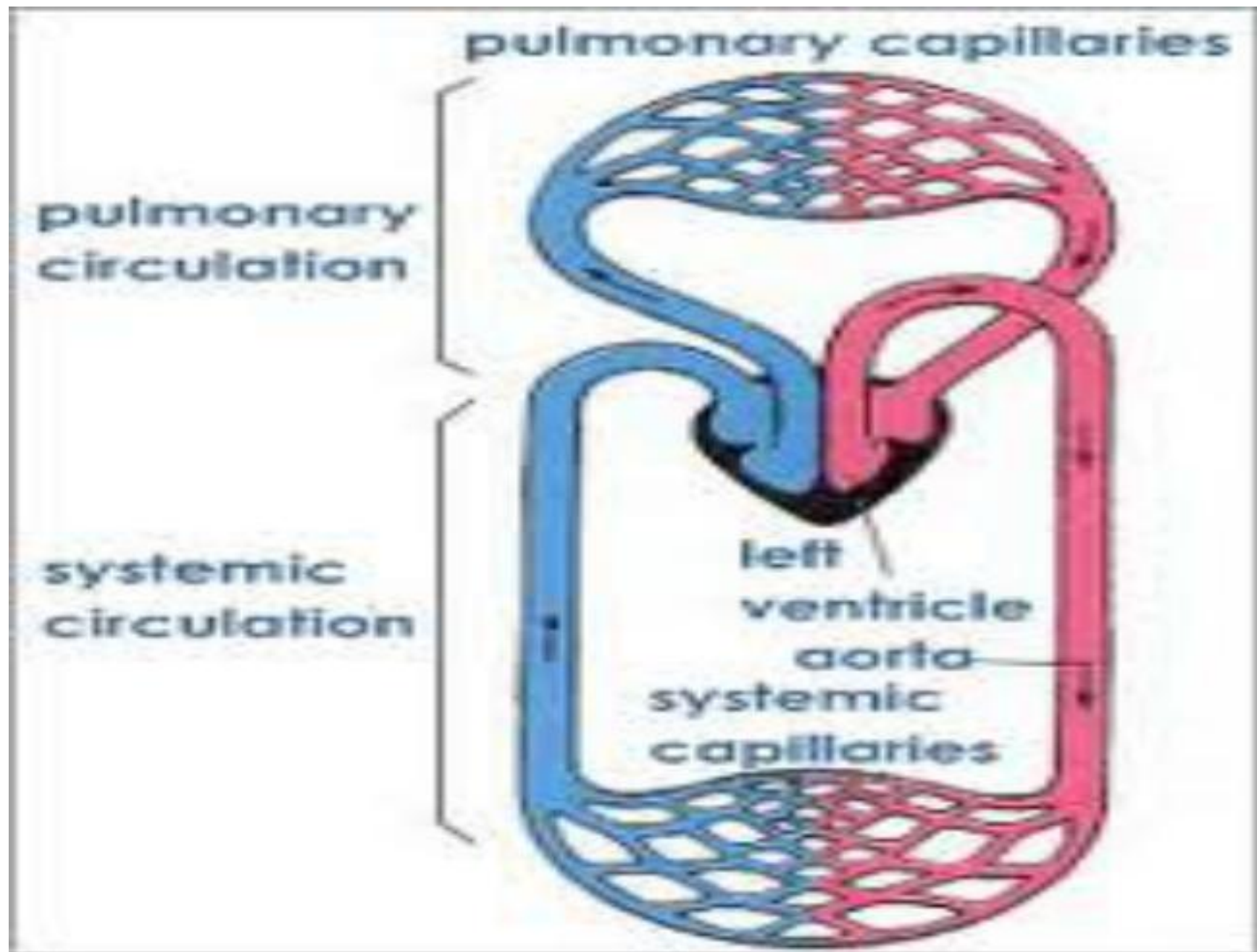
➤ **2. In ulcerative endocarditis** the inflammatory process has a deep and diffuse character with necrosis of the valves and other part of endocardium.

➤ Ulcerative form of endocarditis has malignant character in that it causes emboli of blood vessels and

Heart cont'd---

death of animals.

- There is also metastatic of pathogenic organism from endocardium.
- The ulcerative process may extend from endocardium to myocardium and pericardium.
- If the pathological process has chronic character on valves of heart, it leads to the changing of the valvular tissue to connective tissue(fibrosis).
- Which results in adhesion of fibroses on valvular parts of heart that can be manifested either in the form of
 - Deformation
 - Stenosis
 - Adhesion of valves on the endocardium
- All these lead to in the development of acquired defect of valves.
- Which results in absence of closing of the valves and



Clinical signs

Cardiac signs

- The important finding is a murmur on auscultation or a thrill on palpation of the cardiac area.
- Persistent tachycardia should be regarded as the most consistent clinical sign in endocarditis.

Embolism

- Chronic bacteremia and embolic showering of microorganisms results in signs referable to infection and infarction at other sites in the body.
- There is a constant moderate, fluctuating fever and secondary involvement of other organs may cause the appearance of signs of peripheral lymphadenitis, embolic pneumonia, nephritis, arthritis, synovitis or myocarditis.
- There is usually much loss of condition, pallor of

Diagnosis

➡ Diagnose of endocarditis is done based on

¬ Clinical signs

➡ In acute endocarditis there is

Depression

1 Anorexia

Body temperature is above the norm

Tachycardic

3 Pulse with high amplitude and fully distended.

] Cardiac ton is increased specially the first ton.

➡ In ulcerative type endocarditis

It has mostly malignant character with metastatic and embolism.

→ Cardiology

] Electrocardiographic findings suggestive of endocarditis are sinus tachycardia and decreased QRS

Differential diagnosis

- Ulcerative endocarditis must be differentiated from
 - Congenital defect of valves
 - Myocarditis
- It is difficult to differentiate congenital defect of valves of heart from ulcerative endocarditis.
- Myocarditis is different from ulcerative endocarditis in that endo cardial sound of heart is more frequently in endocarditis than myocarditis
- Other methods of differentiation is by cardiogram(Cardiography).

Treatment

- Treatment should be directed to remove the positive agent of the diseases that cause endocarditis.
- Traumatic endocarditis must be treated by administration of anti-inflammatory drugs (SAID or NSAID) with broad spectrum antibiotics or sulfonamide

Heart cont'd---

- While septic endometritis must be treated after isolation, identification of the causative agent from the blood and doing antibiotic sensitivity test.
- For this reason, it requires repeated attempt to isolate, identify the agent from the blood and doing antibiotic sensitivity test.
- However, treatment is not highly successful.
- The choice of antimicrobial agent should be one that allows
 - High concentrations in serum relative to the minimal bactericidal
 - concentration
 - That has minimal side effects over a prolonged period of administration and has a prolonged half-life.
 - In the absence of a positive culture the types of organism commonly isolated in cattle and small

Heart cont'd---

➡ The variety of causative organisms in horses, dogs and cats recommends the use of broad-spectrum antibacterial treatment.

➤ **NB.** Most of animals with endocarditis, has no good treatment response and it is better to cull the animals out of production this is because

→ 1. Duration of treatment needs to be prolonged (a period of continual

→ treatment for 4 months or with periodic treatment continuing for 14 months in a cow has been recorded

→ 2. Relapse is common

→ 3. The sequel of embolic lesions in other organs and permanent distortion of valves resulting in valvular insufficiency brings an unsatisfactory outcome of treatment.

Prevention measures

Heart cont'd---

→ Use specific and non-specific measures that help to increase the resistance of animals.

7.3.2. Valvular heart disease

➤ It is also called valvular heart defect.

➤ It is defects on the valves of heart characterized by absence of intra auricle and/ or intra ventricle septum, stenosis and/ or insufficient function valves of heart.

➤ Valvular diseases of heart can be

 | 1. Congenital

 | 2. Acquired

1. Congenital valvular defect of heart

➤ It is valvular defect of heart because of disturbance in organogenesis during intrauterine fetus development which is called anomaly.

Heart cont'd---

- Congenital valvular defect can be
 - Absence of intra auricle and/ or intra ventricle septum
 - Stenosis of tricuspid, bicuspid and semi lunar valves of pulmonary artery and aorta.
 - Opened foramen oval of the intra auricle septum after birth.

2. Acquired valvular defect of heart

- Valvular defects of heart which are characterized by stenosis and/ or dysfunction of the valves of heart and acquired after birth is called acquired valvular defect heart.
- It occurs mostly as complication after endocarditis.
- Acquired valvular defects are common in dogs, horses, pigs and hens.
- But very rarely in cattle, sheep and goats

Heart cont'd---

- | 1. Simple
- | 2. Complex
- | 3. Combined

1. Simple valvular defect of heart

- It is valvular defect characterized by either stenosis of valves or disturbance of the function of valves to close their holes.
- Therefore there are eight simple vascular defects of heart.

2. Complex valvular defect of heart

- It is vascular defect of heart characterized by both stenosis and disturbance of the function of valves to close their wholes.

3. Combined valvular defect of heart

- It is a vascular defect of heart characterized by

Heart cont'd---

- Both stenosis and disturbance of the function of valve to close its hole in one valve is the most common valvular defect in domestic animals.
- Valvular defect/s are considered as composited valvular defect if there is no disturbance in hemodynamic in the organism.
- This can be checked by absence of the basic clinical signs of dysfunction of cardiovascular system which are
 - Cyanosis
 - Dyspnea
 - Edema around legs, abdominal cavity and eye
 - Arrhythmia
- Let us see each valvular defects of heart
- The defects are eight which is 2 in each valves of heart.

Heart cont'd---

- ┌ I. Stenosis of the hole of valve/valves
- ┌ II. Insufficient function of valve/valves

I. Stenosis of the hole of valve/valves

1. Stenosis the hole of bicuspid(mitral) valve

- Blood transfer from the left auricle to left ventricle at the end of diastole of left ventricle through small hole(stenosis) of bicuspid
- Which results in the formation of pre systolic murmur sound.
- This pre systolic murmur sound is clearly audible in parts of optimum of bicuspid valves during auscultation.
- Horses and dogs in 5th intercostal space 2 - 3 fingers below the line drawn horizontally from articularis humeri on the left side of thoracic cavity
- Ruminant animals and pigs in 4th intercostal space 2 fingers below the above line on the left side of thoracic

Heart cont'd---

- hyperemia in pulmonary blood circulation.
- Which results in hypertrophy of left auricle and right ventricle.
- Hyperemia in pulmonary blood circulation results in
 - Dyspnea
 - Cyanosis
 - Pneumonia and bronchitis due to hyperemia and edema of lung.
- Since there is reduction of blood that passes through the valve there will be
- Tachycardia
- Weak pulse with amplitude and force

2. Stenosis of the hole of tricuspid valve

- Blood transfer from the right auricle to right ventricle

Heart cont'd---

- Which results in the formation of pre systolic murmur sound.
- This pre systolic murmur sound is clearly audible in parts of optimum of tricuspid valves during auscultation.
- Horses and dogs it is on 4th intercostal space 3 - 4 fingers below the line drawn horizontally from articularis humeri on the right side of thoracic cavity. .
- While ruminants and pigs 3 - 4 fingers below the line on the 3rd intercostal space of the right thoracic cavity.
- Because of the stenosis of holes of tricuspid valve, there will be hyperemia in systemic blood circulation.
- Which results in hypertrophy of right auricle and left ventricle.
- Hyperemia in systemic blood circulation results in
 - Distended vein vessels

Heart cont'd---

- Cyanosis
- Hyperemia of liver

3. Stenosis of the hole of semi lunar valve of aorta

- During the transferring of blood from left ventricle to aorta, there is systolic murmur sound due to stenosis of semi lunar valve of aorta.
- This systolic murmur sound is clearly audible by using stethoscope on the line drawn from art. humeri on 4th intercostal space for ruminants, horse and dogs on the left side of thoracic cavity.
- On the 3rd intercostal space on the line drawn from art. humeri for pigs on the left side of thoracic cavity.
- The accumulation of blood in the left ventricle cause its hypertrophy.
- As the result the top of the heart increased with low

Heart cont'd---

➤ circulation cause s hypoxia and deficient in nutrient etc in many tissues of organism mostly in nerve cells(brain) that are the most sensitive.

4. Stenosis of the hole of semi lunar valve of pulmonary artery

➤ During contraction(systole) of right ventricle, the blood transfers through small hole of the valve form systolic murmur sound.

➤ This systolic sound is clearly audible with stethoscope on the optimum sight of semi lunar valve of pulmonary artery.

➤ The sights for horse, ruminants and dog are on the 3rd intercostal space by 3 - 4 fingers below the line drawn horizontally from art. humeri on the left thoracic cavity.

➤ But for pigs all are the same but in 2nd intercostal space.

Heart cont'd---

➤ There is hyperemia in systemic blood circulation and consequently results in

→Dyspnea

→Cyanosis

→Edema

II. Insufficient function of valve/valves

1. Insufficient function of bicuspid(mitral) valve

- If there is insufficient function of bicuspid valve, blood will return back to left auricle when the left ventricle contracts.
- There fore there will be systolic murmur sound that can be audible using stethoscope on the exact sight of bicuspid valve.
- During this type of defect of valves, blood enters in to left auricle not only from pulmonary vein but also from left ventricle

Heart cont'd---

- Since the amount of blood which enters in to left ventricle increases, there will be also dilation then hypertrophy of the left ventricle.
- If this process continues for a long time, the left auricle never compensate the problem.
- Results in hyperemia in pulmonary blood circulation and hypertrophy in right ventricle.
- If the defect reach in decompensate stage, there will be
 - Hypertrophy of right auricle
 - Hyperemia in pulmonary blood circulation
- Which can be clinically manifested by
 - } Dyspnea
 - } Cyanosis
 - } Cataract bronchitis

Heart cont'd---

- \ Hyperemia and edema of lung
- \ Tachycardia
- \ Weak pulse with low amplitude and force

2. Insufficient function of tricuspid valve

- If there is insufficient function of tricuspid valve, blood will return back to right auricle when the right ventricle contracts.
- Therefore there will be systolic murmur sound that can be audible using stethoscope on the exact site of tricuspid valve (Refer back).
- During this type of defect of valves, blood enters into right auricle not only from superior and inferior vena cava but also from right ventricle.
- This results in dilation and hypertrophy of the right auricle.

Since the amount of blood which enters into the

Heart cont'd---

ventricle.

- If this process continues for a long time, the right auricle never compensate the problem.
- As the result there will be hyperemia in systemic blood circulation in vein vessels.
- Which can be manifested clinically by
 - Dilation of vein vessels
 - Edema
 - Cyanosis
 - Hyperemia in liver, kidney
 - Hypertrophy of left ventricle
- **NB.** Typical clinical sign for insufficient function of tricuspid valve is **positive vein pulse.**

3. Insufficient function of semi-lunar valve of aorta

- In this type of defect of valve blood returns back to left ventricle from aorta when it relaxes (diastole).
- Consequently there is murmur diastole sound on the sight of the valve. (Ref. back).
- Since there is back flow of blood from aorta, the left ventricle dilates and then hypertrophy occurs to compensate the problem.
- There will be increased ton of heart on the left side thoracic cavity
- When the left ventricle stops to compensate the defect.
- There will be
 - Hyperemia in pulmonary blood circulation which can be manifested clinically by
 - } Dyspnea
 - } Hyperemia and edema of lungs

\ Arterial pulse becomes past and weak.

4. Insufficient function of semi lunar valve of pulmonary artery

- In this type of defect of valve the blood will be returned back in to the right ventricle from pulmonary artery when it relaxes(diastole).
- As the result diastole murmur sound will be audible on the exact sight of semi lunar valve of pulmonary artery using stethoscope(Ref. back).
- There will be hypertrophy of the right ventricle to composite the defect.
- When it stops to compensate the defect, it dilates and cause
 - Dyspnea
 - Cyanosis
- Which are clearly visible during exercise.

Heart cont'd---

Treatment

- Veterinary doctor must be focused not to disturb the mechanism of compensation of defects.
- For this it is important to reduce rate of exploitation of animals.
- Feeding of animals with balanced and easily digestible feed.
- The animal must have systematic motion in fresh air.
- However treatment of animals must be started only when the defect is not compensated
 - For this use symptomatic treatment
 - For example low tone of heart use cardiac preparates
 - Edema anti edematous or diuretics etc.

II. Diseases of the blood vessels

‖ 7.4. Arterial thrombosis and embolism

‖ 7.5. Acute and peripheral circulatory failure

7.4. Arterial thrombosis and embolism

➤ A thrombus is a mass of platelets, red and white blood cells, and fibrin.

➤ It develops from and maintains a point of attachment to the blood vessel's wall and it never forms outside blood vessels.

Predisposing factors of arterial thrombosis

➤ Three factors predispose to thrombus formation.

➤ These include:

‖ 1. Endothelial injury/damage

‖ 2. Altered blood flow

‖ 3. Blood hyper coagulation

Vessel cont'd---

1. Endothelial injury/damage

- This is the dominant factor and by itself can lead to thrombosis.
- When a vessel's lining is damaged, endothelial cells may be stripped away or draw away from adjacent cells and expose the sub-endothelial collagen.
- This condition favors platelet adherence, activation, aggregation and initiation of thrombosis.
- Endothelial injury is particularly important in thrombus formation in the heart and arterial circulation (Eg. myocardial infarction or valvulitis).
- There are two principal mechanisms underlying arterial thrombosis.

➤ **A. Hemodynamic stress** (wear and tear of the endothelium) : – factors that contribute to hypertension will increase endothelial damage and

Vessel cont'd---

➤ **B. Arteriosclerosis** – hardening of the arterial wall by deposition of plasma proteins and calcium which affects small arterioles.

2. Flow abnormalities

➤ Change from normal pattern of blood flow is the second factor that increases platelet contact with the endothelium to favor thrombosis.

➤ The two most common hemodynamic changes are:

- 1. Reduction/stasis of the rate of blood flow
- 2. Turbulence in the blood

A. Reduction/stasis of the rate of blood flow

➤ It is blood flow reduction in the arterial system causes a decrease in the axial flow of blood.

➤ As a result, platelets in the central column move nearer to the vessel wall.

Vessel cont'd---

thrombosis.

➤ There are two principal causes of reduced flow rates in the arterial system.

➤ These are

→ **Cardiac damage:** - which reduces the heart's pumping capacity and

→ **Increased blood viscosity:** - when blood viscosity is increased, there is more resistance to flow and hence the blood moves slowly.

→ For example dehydration causes increased viscosity of blood.

B. Turbulence in the blood flow

➤ It is increased blood flow.

➤ Turbulent, irregular blood flow increases platelet contact with cardiac and vascular lining.

3. Blood hyper-coagulation

Vessel cont'd---

by various diseases and other conditions, such as

- Extensive burns
- Some kidney diseases
- Heart failure and
- Widespread metastatic tumors.

Sequelae of thrombosis

➤ There are several possible pathological consequences of thrombosis which include the following.

- ┌ A) Resolution
- ┌ B) Organization
- ┌ C) Propagation
- ┌ D) Infarction
- ┌ E) Embolism
- ┌ F) Disseminated intravascular coagulation

Vessel cont'd---

A)Resolution

- In this case, the anticoagulation system overcomes the factors that favor thrombus formation.
- Thrombi that start to form do not all continue to develop.
- Of thrombi that do form, many are completely broken down or substantially reduced in size so as to lack any significant effects.

B) Organization

- In this case, there will be phagocytic digestion of the thrombus within 2 - 3 days after its formation.
- Platelets and fibrin are then replaced by fibrous connective tissue
- Endothelium forms over the organizing tissue, with the result that the thrombus becomes incorporated into the wall.

Vessel cont'd---

C) Propagation

- The thrombus may accumulate more platelets and fibrin eventually leading to vessel obstruction.

D) Infarction

- This is a major sequelae of thrombosis
- An infarct, a region of necrosis caused by oxygen deficiency is formed
- Hypoxia is the result of ischemia, caused by thrombus that narrows or completely occludes the lumen of the vessel.
- Infarction is most common and serious in arteries because they directly supply oxygen and nutrients.
- When arterial stenosis (narrowing) occurs, it also reduces waste removal, with toxic effects adding to the tissue damage as levels of waste products rise.
- The two most damaging types of infarction are

Vessel cont'd---

and myocardial infarction.

E) Embolism

- It is another important sequelae of thrombosis.
- That can be defined as anything in the vessel other than the normally fluid blood constitutes.
- This is the sudden occlusion of a blood vessel by an embolus which is an abnormal mass moving with the blood stream.
- Interruption of blood flow by embolism is a common cause of infarction
- Thromboembolism is the most common type of embolus formed when a thrombus or part of it breaks away from the vascular wall and is carried off by the moving blood
- Emboli originating at arterial or cardiac surfaces consist mainly of fused platelets and fibrin, and are

Vessel cont'd---

F) Disseminated intravascular coagulation

- DIC is one of the sequelae of thrombosis and is characterized by the appearance of fibrin thrombi within the small blood vessels including capillaries.
- It is seen in: -
 - Severe systemic infections
 - Septic (endotoxic) shock
 - Neoplastic diseases
 - Extensive trauma or burns and following surgery.
- Some of viral diseases associated with DIC include: -
 - Hog cholera
 - Blue tongue etc.

Some specific causes of arterial thrombosis and embolism

- Some of specific causes of arterial thrombosis and

Vessel cont'd---

- ‖ A) Parasitic arteritis
- ‖ B) Viral arteritis
- ‖ C) Bacterial arteritis
- ‖ D) Vasoactive agents

A) Parasitic arteritis

- *Strongylus vulgaris* in horses – migrating larvae cause arteritis of the anterior mesenteric artery.
- Inflammation and thickening of the arterial wall results in the formation of thrombi which may partially or completely occlude the artery.
- Recurrent colic and fatal ischemic necrosis of segment of the intestine occurs.

B) Viral arteritis

- Viral arteritis is important in the pathogenesis of several viral diseases and involves several organ systems.

Vessel cont'd---

➤ Some viral diseases causing arteritis include the following.

- Malignant catarrhal fever
- Equine viral arteritis
- African swine fever
- Hog cholera
- Blue tongue
- African horse sickness

C) Bacterial arteritis

➤ Bacterial arteritis involves in different arteries of several organs and the pathogenesis depends upon the specific etiology.

➤ Some examples of bacterial infections causing arteritis leading to thrombosis include the following.

- Septicemic salmonellosis

Vessel cont'd---

- Pasteurellosis
- Salmonella dublin

D) Vasoactive agents

- *Clavisceps purpurea* (causes ergot poisoning) is a fungus which under natural conditions infests cereal rye and less commonly cereals and grasses.
- The pharmacologically active compounds (ergotoxins) include:
 - Ergotamine
 - Ergotoxin
 - Ergometrine
- These compounds stimulate smooth muscles of arterioles, intestines and the uterus.
- **Ergotamine** especially causes arteriolar spasm and capillary endothelial damage, with restriction of the circulation and gangrene of the extremities when small

Vessel cont'd---

Clinical findings

Clinical findings of arterial thrombosis and embolism depends on its location.

1. Aortic- iliac thrombosis in the horse

- Great pain and anxiety
- Increased pulse and RR rates
- Profuse sweating
- The affected limb is dry and cooler than the rest of the body
- The pain is sufficiently severe to cause the animal to lie down and refuse to get up
- Lameness on exercise

2. Pulmonary embolism

- Severe dyspnea develops suddenly and is accompanied by profuse sweating and anxiety.

Vessel cont'd---

- The temperature and pulse rate are elevated
- Pulmonary arteritis and aneurism may be followed by rupture and pulmonary hemorrhage and hemoptysis

3. Classical ergotism

- The extremities particularly the lower part of the hind limbs, tail and ears are affected.
- There is reddening, swelling, coldness, loss of hair or wool and lack of sensation of the parts initially
- Which is followed by the development of blue-black color, dryness of the skin and its separation from its normal tissues.
- The gangrene usually affects all local tissues and, after some days the affected part obviously separates and eventually sloughs.
- Lesions are not painful but some lameness is evident even in the early stages and the animal may remain

Vessel cont'd---

4. Parasitic arteritis

- Persistent low grade fever and poor appetite
- Intermittent colic and poor weight gain

Diagnosis

- ➡ Diagnose can be done based on
 - Clinical signs
 - Signs of colic in parasitic arteritis
 - Gangrene (in Clavisceps purpurea and Salmonella dublin)
 - Ultrasonography

Therapy in intravascular thrombosis and thromboembolism

- ➡ There are different approaches in the treatment of intravascular thrombosis and thromboembolism.

1. Anticoagulants

Vessel cont'd---

formation.

- Heparin can administered parenteral or orally
- The most common injectable solution of heparin is Heparin sodium solution in 10 unit/ ml, 1000 unit/ ml, 5000 unit/ ml.

Dose and route of administration

- S/C - 40 – 80 units/ kg BW 3 times daily up to 3 days.
- IV - 8 units/ kg BW in every 3 hours for 1 day.
- Orally you can administer **Warfarin** in dose of 10 mg/ kg of BW for 3 days.

2. Antiplatelet therapy

- It limits platelet adherence. Example ASA(Aspirin)

3. Promotion of the fibrinolytic system

- The agents employed are enzymes that activate plasminogen to form plasmin.

Vessel cont'd---

4. Administration of anti pains

- It is important to administer anti pains like dipyrone to relieve pain (colic due to parasitic arteritis)

5. Anthelmintics

- It is good to administer broad spectrum anthelmintics to treat parasitic arteritis.

Part 7:Disease of blood and blood forming organs

- **Here we will focus on**

| 7.1. Shock/ circulatory collapse

| 7.2. Edema

| 7.3. Anemia

7.1. Shock/ circulatory collapse

- Shock is defined as failure of peripheral circulation. Or
- It is a decreased perfusion of the tissues such that the tissues are

Blood cont'd---

- unable to meet metabolic demands.
- The fundamental disturbance is that the volume of blood is too small to fill the vascular system and the accompanying cell damage is due to inadequate perfusion of the tissues (hypoxia).

Mechanisms of circulatory shock

- To maintain normal homeostatic balance, tissues depend on an adequate blood flow reaching the microcirculation.
- The microcirculation describes the extensive networks of capillaries that pass among the cells of almost all tissues, including the arterioles which supply them and the venules that drain from them.
- Systemic blood pressure is determined by the relationship of the three key elements:
 - The heart's pumping

Blood cont'd---

→ Blood volume

➤ In circulatory shock, this normally balanced relationship is abruptly thrown out of equilibrium.

➤ Although many etiologies are involved, three basic patterns of pathogenesis contribute to the development of shock:

- | 1. Cardiogenic shock
- | 2. Hypovolemic shock
- | 3. Vascular shock

