LECTURE NOTES

FOR

CLINICAL VETERINARY MEDICINE

VCM – 421

PREPARED BY

DR. A.U. BHIKANE
ASSOCIATE PROFESSOR AND SECTIONAL HEAD,
DEPARTMENT OF CLINICAL MEDICINE,
ETHICS AND JURISPRUDENCE

COLLEGE OF VETERINARY AND ANIMAL SCIENCES,
UDGIR, DIST: LATUR.

CONTENTS

CHAPTER-1
GENERAL SYSTEMIC STATES

THERMOREGULATION

- Mammals maintain core body temperature within normal range despite extremes in environmental conditions.
- Core body temperature is not constant but shows diurnal variation.
- The normal range of body temperature for individuals within a species may vary by 1^0C (2^0F)

*Regulation of body temperature-

Maintenance of body temperature is under neuronal control in a negative feed back system

- Warm and cold sensitive neurons within the hypothalamus sense existing core body temperature.

Warm sensitive neurons	Cold sensitive neurons
↓	↓
• Increase respiratory rate	•Vasoconstriction
• Vasodilatation	•Piloerection
• Sweating	•Muscle activity
• Seeking cool environment	•Huddled posture
	•Group aggregation
↓	↓
Increase heat loss	Heat conservation
↓	↓
Decrease in body temperature	Increase in body temperature

- **Heat Production**
  It occurs primarily from – a) Muscle activity and
  b) Digestion of food
- **Heat conservation**
  It occurs from a) adrenergic autonomic stimuli to decreased peripheral circulation
  b) Piloerection and
  c) Behavioral means Viz.- huddled posture
  - Group aggregation and
  - Seeking sheltered environment
- **Heat loss**
  It occur from-a) conduction, convection and radiation from body surfaces
  b) Evaporation
  c) Sympathetic vosodilation of cutaneous vessels contributes to surface cooling.

1) HYPOTHERMIA

Definition: It means decrease in body temperature due to disturbances in thermoregulatory mechanism.

Etiology:

1. Exposure to extremely cold, windy / rainy (wet) weather results in heat loss from body.
2. Shock result in - peripheral vosodilation & increased heat loss
3. Hypocalcaemia, anaesthesia, sedation and acute ruminal impaction. These decreases muscle tone.
4. CNS disease resulting in damage to the thermoregulatory centers within the hypothalamus.

Predisposing factors:

Following factors increases susceptibility to hypothermia

1. **Age:** Newborn and animals.
   a) Young / new born pups, kids, calves & piglets are more susceptible as their thermoregulatory mechanism is not developed.
   b) Old animals are also highly susceptible because their vital capacity goes down.
2. **Body condition :** Poor body condition / emaciation.
3. **Nutrition** : Under nutrition or malnutrition or starvation.
4. **Hair Coat :** Thin / sparse hair / wool coat

Pathogenesis :

Exposure to Extreme cold climate

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Stimulation of cold sensitive neurons within hypothalamus

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- Peripheral vasoconstriction
- Decreased sweating
- Piloerection
- Increased muscle activity
- Huddled posture
- Group aggregation

↓

Heat conservation and decreased heat loss

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Body temperature is maintained

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This is the compensatory mechanism for maintaining body temperature.

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Continued exposure to extremely cold climate

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Failure of compensatory mechanism

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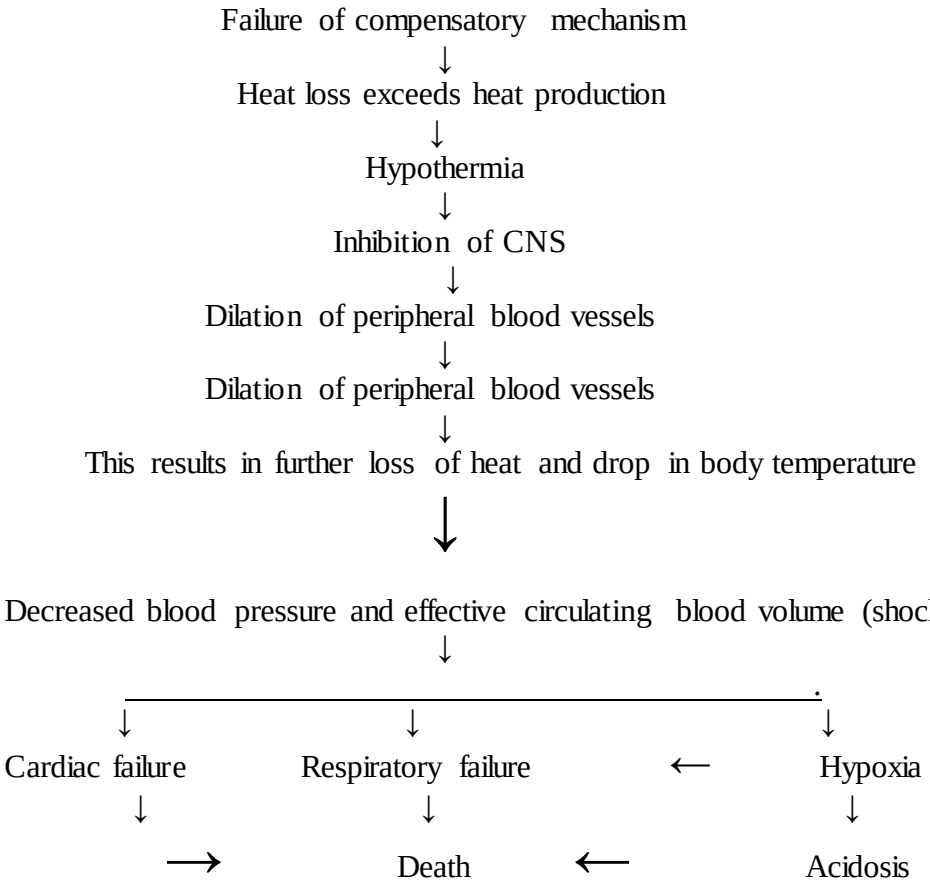


Signs :

- Subnormal temperature (temperature less than 86^0 F observed in severe cases of hypothermia)
- Tachycardia weak pulse
- Dyspnoea
- Anorexia
- Profound dullness and depression
- Cold skin and extremities
- Decreased reflexes
- Muscle weakness leading to recumbency
- Coma
- Death

Diagnosis

1. **History :** Cold environment ,age factor etc.
2. C**linical signs** – Subnormal body temperature, cold skin & extremities

Prognosis :

Concurrent signs of septic disease in hypothermic animals indicates guarded prognosis.

Treatment :

1. Provide warmth Animals with severe hypothermia by taking following measures
    a) Provide adequate covering with blankets
    b) Keeps kids, calves in closed room during late night in winter.
    c) Provide high wattage bulbs so as to maintain sufficient warmth in calf / kid sheds.
    d) Protect animal from wind / rains by covering sides and windows with gunny bags.
    e) Give gastric (rumen) or rectal lavage with warm fluids.
2. Adequate fluid therapy (Warmed to body temperature) provide energy viz. Dextrose 5 to 10% i/v
3. Corticosteroids to prevent shock e.g. Dexamethasone @ 1 mg / kg iv / im
4. Correction of acidosis by using 1.3 % sodium bicarbonate solution in.
5. Oxygen therapy for treatment of hypoxia

Prevention:

1. Animals should be kept indoor during night hours.
2. Neonates and old animals should be provided warmth during late night hours of chilling winter by using high wattage bulbs, keeping them in wing proof rooms and adequate covering with blankets and providing dry place.
3. Clipping of hairs and wool in buffaloes and sheep should be avoided during cold waves.
4. Avoid exposure of very young, senile and debilitated animals to rain.
5. Provide dry place during rainy season.

2) Hyperthermia

Hyperthermia – It is the elevation of body temperature resulting from excessive heat production or absorption or to deficient heat loss due to physical causes.

Heat stroke - It is acute hyperthermia resulting from exposure to extremely high environmental temperature.

Etiology :

A) High environmental temperature accompanied by
    1) High humidity (when humidity is 60%) of evaporative heat loss
    2) Decreased air current / inadequate ventilation
    3) Overcrowding
    4) Decreased water intake
    5) Decreased sweating - Anhydrosis
    6) Heavy coat / Fleece, thick body coat
    7) Transportation
    8) Strenuous exercise
    9) Excitement
    10) obesity

B) Other causes

a) Neurogenic hyperthermia –

It occurs due to damage to hypothalamus e.g. Haemorrhage.

b) dehydration –

Reduced heat loss by evaporation due to insufficient tissue fluids.

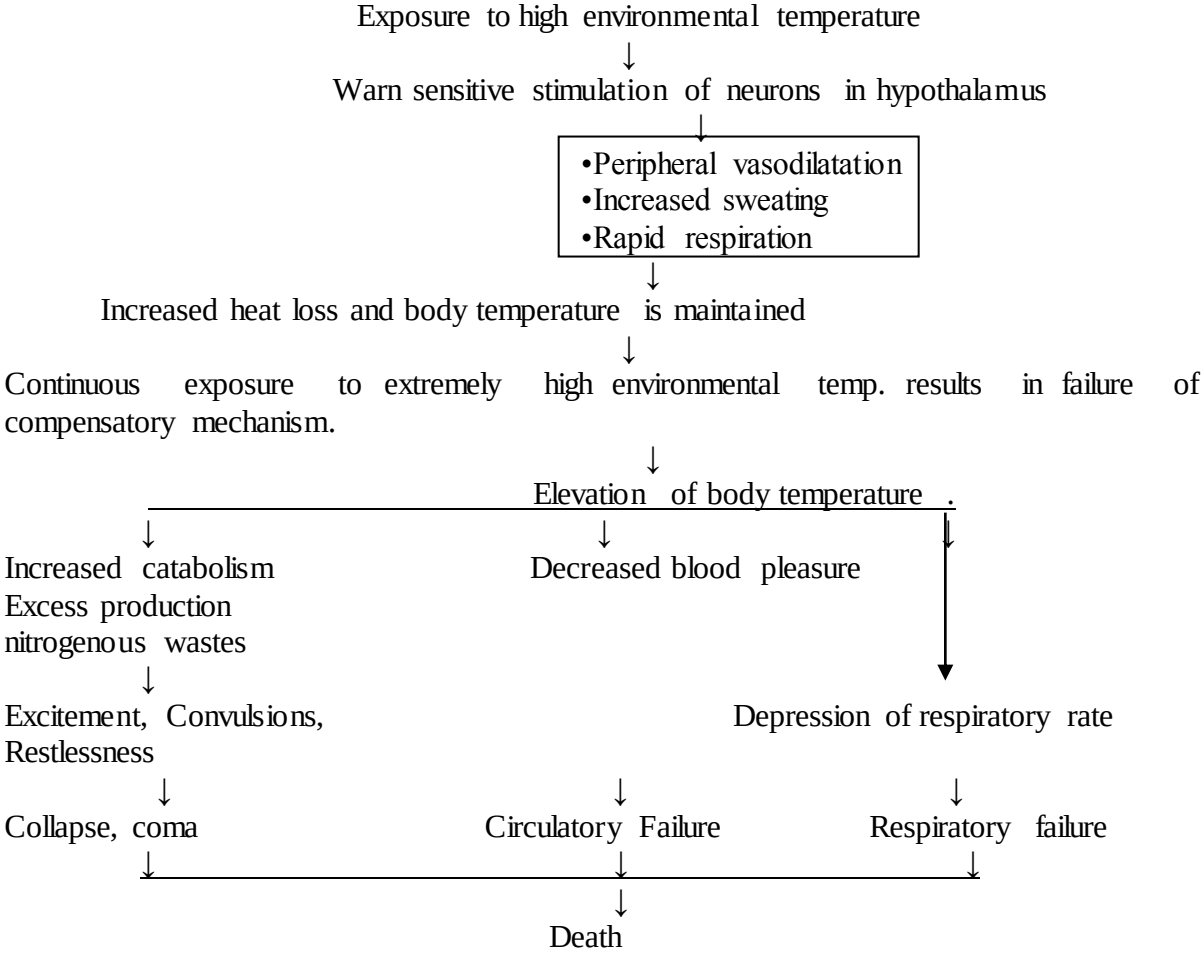
c) Excessive muscular activity -

e.g. Strychnine poisoning , chlorinated hydrocarbon poisoning

Animals susceptible

1) Heat stroke is more common in pure bred / cross bred cattle (H.F/Jersey) because of heir inability to sweat due to less number of sweat glands.
2) Buffaloes are more susceptible heatstroke due to less number of sweat glands and weak thermoregulatory center.
3) Sheep having heavy fleece are more susceptible to hyperthermia because of decreased heat loss.
4) Race horses also suffer due to their strenuous exercise.
5) Pigs are liable to suffer due to their limited capacity of lungs and obese nature.

Pathogenesis :



Clinical signs

First stage : •Temperature increased up to $106 – 110^0$ F
•Tachycardia
•Rapid and deep breathing
•Rapid & weak pulse
•Frothy discharge from nostrils
•Salivation

•Sweating occurs initially followed by absence of sweating
•Oligurea
•Dullness and depression
•Stumbling while walking, tendency to lie down
•Congestion of eye mucous membranes
•Anorexia
•Animal seeks cool place

Second stage : •Increased thirst due to dryness of mouth.
•Dry muzzle
•Excitement
•Restlessness
•Convulsions
•Dyspnoea – open mouth breathing – protrusion of tongue.

Third stage
•Collapse
•Coma
•Death occurs in most species when temperature reaches 106 108^0
•Abortions may occur if hyperthemia in prolonged.

Differential diagnosis :

1) Fever :
Body temp occurs seldom exceeds 106^0 F whereas in hyperthermia it frequently does.

2) Septicemia -
Petecheal Haemorrhage on mucosa and skin.
Blood cultures positive for bacteria.

3) Hyperthermia
Examination of environment reveals high environmental temperature.
Body temperature usually exceeds 106 F

Treatment :

a) General management :
1) Keep animal in a well ventilated shady place
2) Open all the doors and windows, if animal is indoor.
3) If possible make the arrangement of fans.
4) Give cold water both to animals preferably with ice cold water.
5) Rub ice blocks over the skin of animal particularly on forehead region.
6) Provide ample amount of cold drinking water
7) Give ice blocks to animals for licking.
8) Give ice cold rectal enema.

b) Antipyretics : They are given to reduce body temperature :
e.g. 1) Novalgin - Large animals @ 20 – 30 ml i/m
Small animals @ 1.5 ml i/m
2)Ketoprofen(ketop/vetoprofen) @ 3mg/kg i/m
3)Nimesulide (Nemovet) @ 4mg/kg

c) Tranquilizers :
They may be given to sedate the animal in case of nervous excitement
e.g. chlorpromazine HCL @ 1mg / kg body wt. i/m.

d) Corticosteroids -

They are used for prevention and management of shock e.g. dexamethasone in higher doses. ie 1mg/kg body wt. iv /im

e) Fluid therapy -

Intensive fluid therapy with cold normal saline or 5 % dextrose to correct dehydration & electrolyte losses.

f) Diet management:

Provision of adequate glucose and protein to compensate for increased utilization.

Prevention / Control

1) Animal should be provided will ventilated shady place during summer.
2) Animal should not be let loose for grazing during hot hours of the day in summer ie in between 11 A.M. to 4.00 P.M.
3) Bullocks should be used for agricultural operations in morning and evening hours.
4) Transportation of animals should be done during cool hours of the day.
5) During summer, animals should be provided ample cold drinking water.

3) FEVER

Definition: "It is the syndrome in which hyperthermia and toxemia are produced by substances circulating in the blood stream" jIt is an elevation of core body temperature above the critical level normally maintained by animal.

It is a combination of hyperthermia and toxemia. It may be septic or aseptic.

Etiology :

A) Infectious agents (Septic/Specific fever)

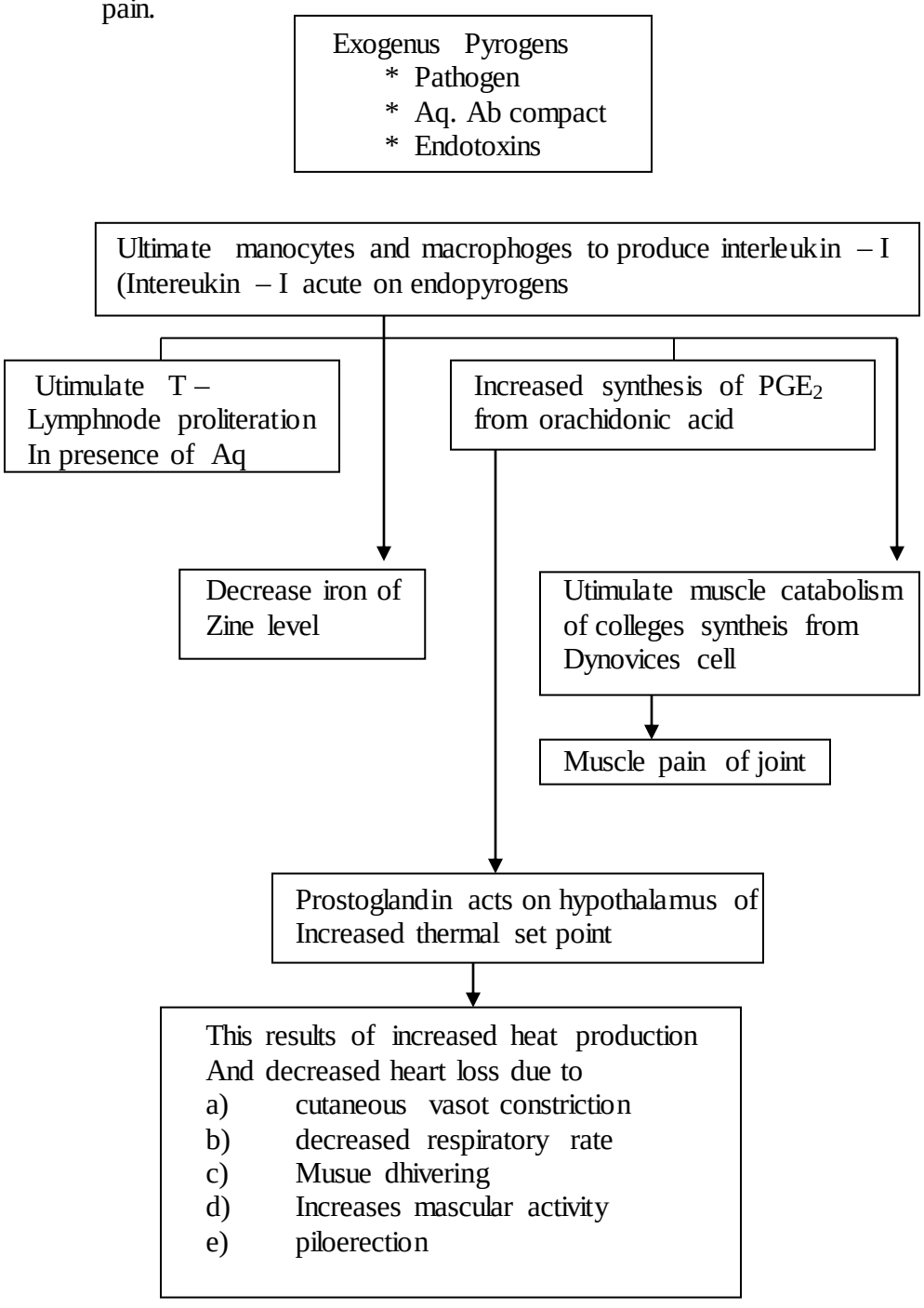
1) **Bacterial diseases** - Anthrax, H.S., B.Q., listeriosis leptospirosis.
2) **Viral diseases** - Rp, BVD, FMD, epimeral fever, PPR, BT.
3) **Protozoal diseases** – Theileriosis, Babesiosis, Anaplasmosis, Surra
4) **Systemic diseases** – pneumonia, enteritis, Metritis,
5) **Localized infections** - Abscess, cellulites, emphysema.

B)Non-infectious agents (Aseptic/Non-specific fever)

1) Injections of foreign protein (chemical fever)
2) Breakdown of tissues and blood in surgical operations (Surgical fever)
3) Tissue necrosis - Break down of muscles after injection of irritating material.
4) Intravascular hemolysis e.g. Post parturient haemoglobinuria.
5) Neoplastic causes - Bovine leukosis, lymphosarcoma.
6) Immunological causes – Anaphylaxis , allergy ,angioneuroticedema.
7) Drug induced fever - prolonged use of antimicrobials may cause cryptic fever.
8) Miscellaneous causes - Snake bite , burns.

Pathogenesis

1) The fever is induced by exogenous pyrogens.
2) The pathogens , Ag-Ab complex and endotoxins act as pyrogens.
3) These exogenous pyrogens stimulates monocytes or macrophages to produce a low molecular weight protein called as interleukin-I (Best known endogenous pyrogen)
4) Interleukin-I stimulates prostaglandins E_2 (PGE_2) production from arachidonic acid in the anterior hypothalamus. These prostaglandins acts on hypothalamus to raise thermal set point which in turn activates heat production and conservation (vasoconstriction) mechanism That's finally results in rise in body temperature.
5) Interleukin I is also responsible for activation or proliferation of T & B lymphocytes, there by stimulates host defense mechanism.
6) Interleukin I also stimulates the muscle catabolism of induce collagen synthesis from synovial cell. These processes contributes to muscle and joint pain.



f) Minimal urine formation

Stage of increment or chill.

When the body temp. reaches neco thermostratic level then the mechanism of heat production and loss return to normal

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Stage of constant temperature tastigium

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When the pyrogenic sunstances ceuse to act the excess tract is dissipated and temperature falls due to

a) Cutaneous vasodilation .increased sweating
b) Increase respiratory rate
c) Muscular rlacuidity, rall in body temp.

Stage of decrement / deteruescence

↓

Clinical signs :

- Rise in body temperature (rarely 103 – 105.8^{0}F)
- Increased pulse rate.
- Increased respiratory rate
- Dry muzzle
- Decreased ruminal motility.
- Dullness depression muscle weakness, reluctance to move
- Increased thirst
- Anorexia / inappetence
- Reduced milk yield in lactating animals
- Constipation (firm faeces)
- Wasting (loss of body weight)
- Oligurea (concentrated urine)

Stages of fever :

Fever develops in 3 stages

I) Stage of increment / chill / onset:

- Body temperature rises
- Pulse rate is increased
- Respiration is reduced
- Dullness
- Arched back
- Inappetence
- Skin is cold and dry (Vasoconstriction)
- Chills and feeling of cold, muscle shivering, erection of hair's due to stimulation of cutaneous nerve endings following fall in cutaneous temperature caused by vasoconstriction.

II) Stage of constant temperature / fastigium :

- Constant high body temperature.
- Profound dullness
- Congestion of eye mucus membrane
- Hot skin (cutaneous vasodilatation)
- Hot breath
- Anorexia
- Dry muzzle

III) Stage of decrement / defervescence :

- Body temperature declines
- Skin coat appears normal
- Muzzle become moist
- Appetite improves
- Urine flow increases

Differential diagnosis :

Hyperthermia

1) There is no toxemia
2) History of exposure to high environmental temperature.
3) Body temperature usually exceeds 106^0F

Prognosis :

1) Fall in body temperature coupled with decrease in frequency and improvement in quality of the pulse indicates 'recovery.'
2) Abrupt fall in body temperature, cold clammy skin, rapid and weak pulse indicates state of collapse. This occurs due to severe toxemia and ultimately results in to death. Here the body temperature falls but heat production and loss can not be controlled.

Diagnosis:

1) History: Age factor , sex factor, sudden change of climate, pica, tick infestation and vaccination.

2) Clinical signs: Degree of fever and disease specific signs

3) Blood smear examination is useful in diagnosis of HS, anthrax and haemoprotozoan infections

4) Haematology:

- Leucocytosis: bacterial fever
- Leucopenia: viral fever
- Neutrophilli: localized infection
- Anaemia: haemoprotozoan infections

5) Urine analysis:

- Proteinuria & casts in urine: Nephritis
- Proteinuria with epithelial cells: Cystitis

6) Culture of blood and other body fluids

7) Lymphnode biopsy reveals Koch blue bodies in theileriosis

8) X-ray examination reveals penetrating foreign bodies in Traumatic reticulitis and pericarditis.

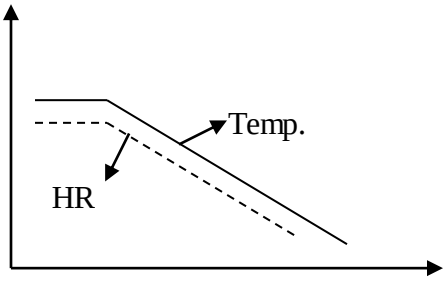
Differential diagnosis :

1) Hyperthecmia -
   * These is no toxaemia
   * H/o exposure to high environmental temp.
   * Body temp. usually exceeds 100^0F

2) Septicaemia * Petechae on mucosae

Prognosis

1) Gall in body temp. coupled with decrease in frequency and improvement in godlily of pulse indicates recovery.



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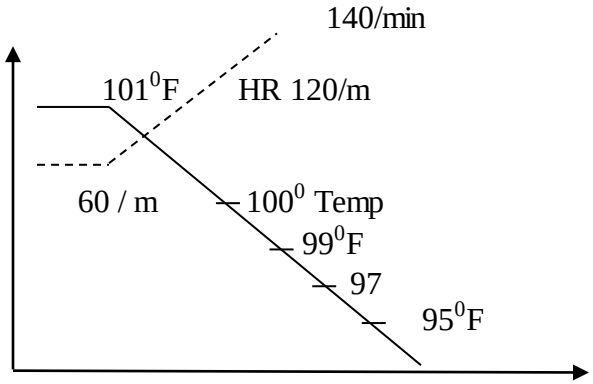


Types of fever :

1) **Transient fever :** Fever subsides within 24-28 hrs after its development e.g. Bovine ephemeral fever.
2) **Simple fever** : Body temperature rises and remains elevated with variation less than 1^0C (2^0F) for several days e.g. lobar pneumonia.
3) **Continuous fever :** Body temperature remains elevated for longer period than in simple fever e.g. Theileriosis.
4) **Remittent fever :** Body temperature rises and falls by more than 1^0C (2^0F) at short and irregular interval. e.g. Bronchopneumonia and urinary tract infections.
5) **Intermittent fever :** Regular brief attack of fever for 2-3 days with non febrile period e.g. Equine infectious anaemia, canine distemper.
6) **Recurrent fever :** Prolonged attack of fever with non febrile period of equal duration.
7) **Undulant fever :** Prolonged attacks of high and mild fever at regular intervals e.g. Brucellosis.
8) **Atypical fever :** Febrile reaction is irregular e.g. swine erysipelas, strangles.
9) **Biphasic fever :** It is characterized by initial rise followed by a fall to normal and then secondary rise e.g. canine distemper

Degree of fever :

1) **Mild fever :** Body temperature is above normal by 1-2^{0}F e.g. pulmonary T.B., surra.
2) **Moderate fever :** Body temperature is above normal by 3-4^0 F e.g. nephritis, cystitis, metritis.
3) **High fever :** Body temperature is above normal by 5-6^0 F e.g. Anthrax, HS and acute pneumonia.

Treatment :

The general principles of treatment of fever include

1) **Removal of source of toxin** :
   a) Use of antibacterial drugs / antibiotics e.g. Oxytetracycline @ 5 mg / kg im / iv
   b) Removal of necrotic material in localized infection e.g. abscess
2) **Treatment of toxemia** :
   a) Use of specific antitoxins to neutralize circulating toxins e.g. antitetanus serum.
   b) Use of corticosteroids to facilitate repair process and alleviate inflammation e.g. Dexamethasone @ 1mg / kg im or use of nonsteroidal anti-inflammatory drugs e.g. Meloxicam @ 0.5mg /kg iv / im
   c) Fluid and electrolytes therapy e.g. D- 5 / DNS
3) **Treatment of hyperthermia** :
   a) Use of antipyretics e.g. Novalgin @ Large animals – 20-30 ml, Small animals 1-2ml iv / im
   b) Cold water bath

4) TOXAEMIA

Definition: It is clinical systemic state characterized by presence of toxins in blood produced by the bacteria or injury to tissue cells.

- Toxaemia does not include diseases caused by toxic substances produced by plants and insects or inorganic and organic poisons
- A common form of toxaemia in farm animal is endotoxaemia caused by presence of lipopolysaccharide component of gram -ve bacteria.

Type of Toxins

They are of two types

1) Antigenic toxins
2) Metabolic toxins

A) Antigenic toxins : These are produced by bacteria and to a lesser extent by helminth parasites. They are of two types-

a) Exotoxins:

i) These are protein substances produced by bacteria which diffuse in surrounding medium

ii) The important bacterial exotoxins are those produced by Clostridium species. may be –
   a) ingested preformed as in botulism or
   b) produced in large quantities by heavy growth in intestine such as in enterotoxaemia or
   c) from growth in tissue as in black leg and black disease.

iii) Enterotoxin is also an exotoxin released by enterotoxigenic E.coli which act on intestinal mucosa causing fluid and electrolyte imbalance.

b) Endotoxins :

i) These are lipopolysaccharides found in outer wall of bacteria

ii) These are released in immediate surrounding when bacteria undergo rapid proliferation or destruction.

iii) The best known endotoxins are those of E.coli & Salmonella spp.

iv) Endotoxins are normally present in the intestine & are absorbed in small quantities into the portal blood. However, these toxins are detoxified by the liver & do not reach the peripheral blood. If the amount of toxin is large or if there is hepatic inefficiency, a state of endotoxaemia is produced.

v) Endotoxins are also absorbed from sites other than intestine viz. mammary gland, peritoneum, abscesses & other specific foci.

2) Metabolic toxins-

i) These toxins are produced in animal body during normal or abnormal metabolism.

ii) Normally metabolic toxins produced in the alimentary tract or tissues are excreted through urine & faeces or detoxified in the plasma & liver.

iii) These may accumulates either due to,
   a) Incomplete elimination of toxic substances normally produced by body metabolism e.g. liver/ renal diseases

b) Abnormal metabolism resulting in excess production of metabolic toxins e.g. Ketosis

Etiology-

A) Antigenic toxins

a) Exotoxins

1) Botulism
2) Block leg
3) Enterotoxaemia
4) Enterotoxigenic E.coli.

b) Endotoxins:

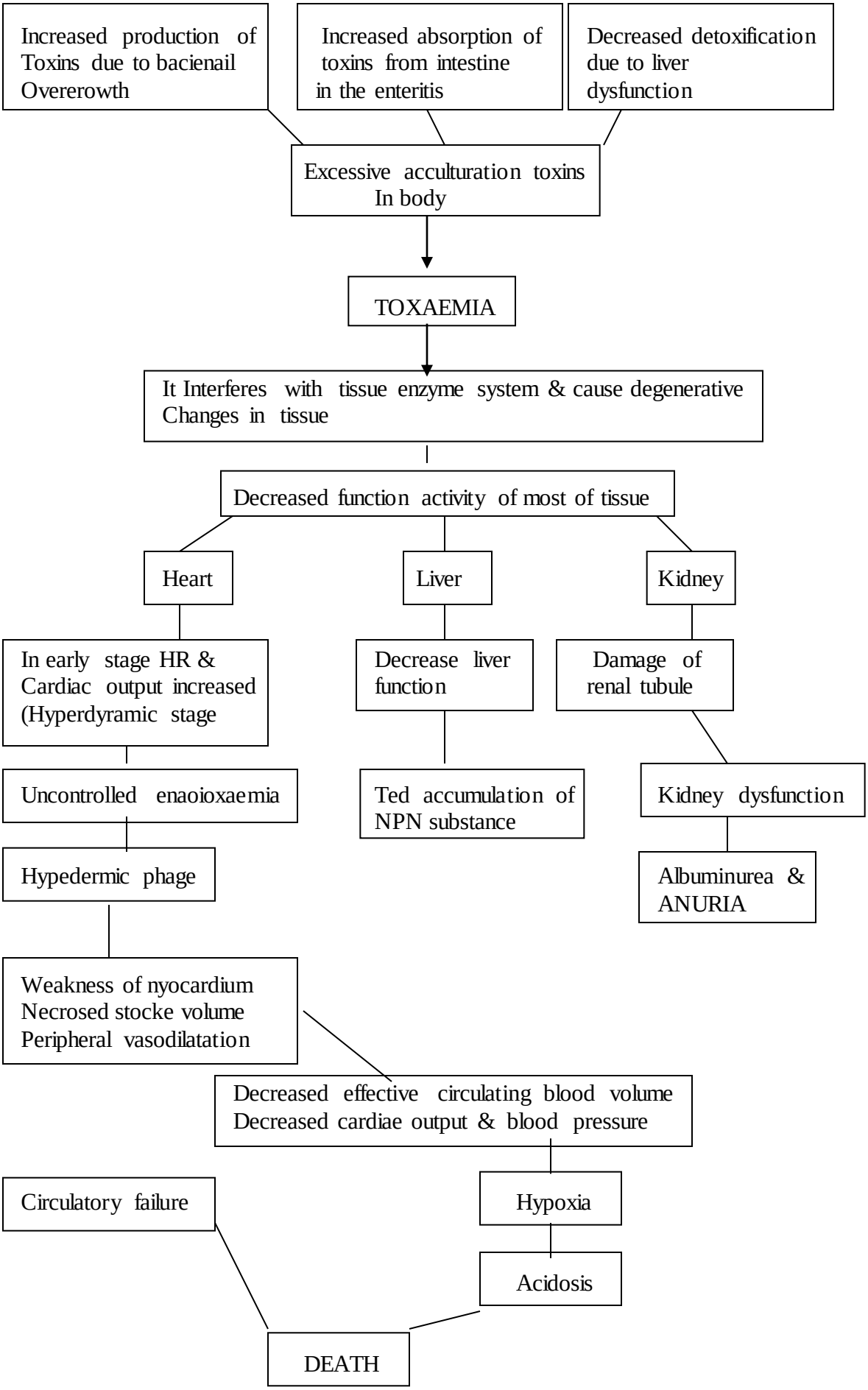
1. Coliform mastitis
2. Coliform enteritis in new born calves.
3. Peritonitis
4. Metritis
5. Abscesses and other septic foci
6. Acute intestinal obstruction
   (Gram -ve bacteria normally present in intestine / Produces endotoxins which are not normally absorbed through intestinal mucosa unless it is damaged as in enteritis)
7. Live dysfunction (absorbed endotoxins are detoxified by liver)

B) Metabolic toxins :

1) Incomplete elimination of metabolic toxins
   a) Hepatic dysfunction:- Toxic substances, (glycine, glucoronic acid , cysteines) are accumulated beyond a critical point.
   b) Obstruction of lower alimentary tract leads to regurgitation of toxins of protein putrification (Toxic phenols, cresols & amines)& their absorption from upper GI tract because of absence of protein barrier in the wall of small intestine.
   c) Renal dysfunction – uraemia

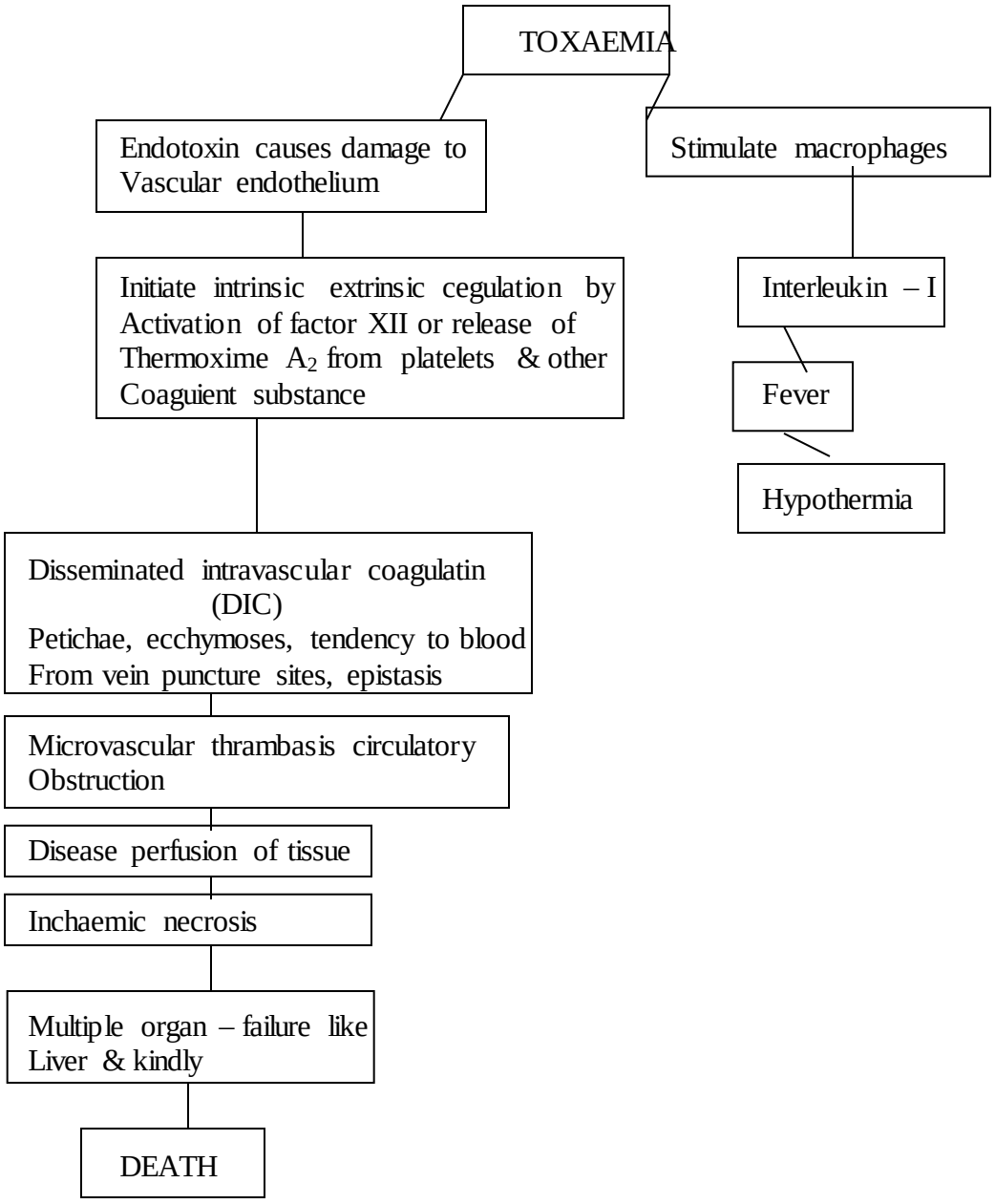
2) Production of toxins by abnormal metabolism.
   a) Release of histamine & histamine like substances from damaged tissues.
   b) ketonemia due to abnormal fat metabolism
   c) Lactic acidosis due to acute Carbohydrate engorgement

Pathogenesis :



Metabolism

Hypoglycemia sometimes hyperglycemia i.e. usually transits
Increase muscle proteolysis i.e. tissue break down & rise in NPN substance
Hypoferremia & hypozincemia with rise in blood copper & ceruloplasmin level.



Clinical Signs :

A) Acute toxemia

1) Fever (hyperthermia) in early stage followed by hypothermia.
2) Polypnoea
3) Tachycardia
4) Rapid & weak pulse

5) Depression
6) Anorexia
7) Cold skin & extremities
8) Pallor of mucosa in severe endotoxaemia. Congested mucosae due to decreased perfusion of tissues & O_2 consumption.
9) Muscular weakness leading to recumbency (loss of muscle tone)
10) Oligourea / anuria due to renal failure
11) Constipation or diarrhoea
12) Petechial or ecchymotic hemorrhages on mucous membrane & sclera with increased tendency to bleed from veine punture sites in DIC (dissiminated intravascular coagulation)
13) Collapse
14) Coma & death

B) Chronic toxemia

- Dullness, inappetence
- Failure to grow or produce & emaciation

Clinical Pathology

1) Isolation & identification of toxin is difficult.
2) Blood biochemistry

a) Hypoglycemia
b) Increased blood lactate
c) Increased NPN substances
d) Blood PH decreases

3) Urine analysis – Albuminuria
4) Hematology
a) Leucopenia and neutropaenia (in severe toxemia)
b) Leucocytosis & neutrophilia (in mild toxemia)
(Persistent neutrophilia is poor indicator)
c) Haemoconcentration
(due to movement of fluid from the vascular to extravsculor spaces).

Treatment

The principles of treatment of toxemia includes-

1) Removal of the foci of infection
2) Expansion of intravascular volume
3) Prevention of actions of endogenous mediators of stocks.

1. Fluid & electrolyte therapy :

- Large quantities of fluid & electrolytes is a high priority in the management of toxemia
- Large volume of isotonic fluid have been standard practice
- Lactated ringers solution or balanced electrolyte solution must be given over several hours.

- Give fluids equivalent to 0.5-1.0 time estimated blood volume of animal over a period of several hours.

2. Corticosteroids:

- Corticosteroids are commonly used for treatment of toxemia, especially when shock occurs.

> Role of Corticosteroids-
>
> a. Improve cellular metabolism & gluconeogenesis
> b. Improve microcirculation
> c. Decreases production of endogenous toxins

- Dexamethasone @ 1 mg/kg i/v daily is highly effective but it is cost prohibitive in farm animals.

3. Non steroidal anti-inflammatory drugs: CNSAID6J can also be used
e.g.- Phenylbutazono @2-4 mg/kg im daily. or
Ketoprofen @ 3mg/kg im daily. or
Meloxicam @ 0.5mg/kg im/iv daily

4. Antibiotics :
Broad spectrum antibiotics to remove foci of infection.
e.g. Amoxicillin-cloxacillin combination @10mg/kg iv daily.

4) Liver tonics with Vit. B complex-
e.g.- Inj. Belamyl @ 5ml i/m

5) Use of anticoagulants-
Disseminated intravascular coagulation (DIC) can be treated heparin.

5) SEPTICAEMIA

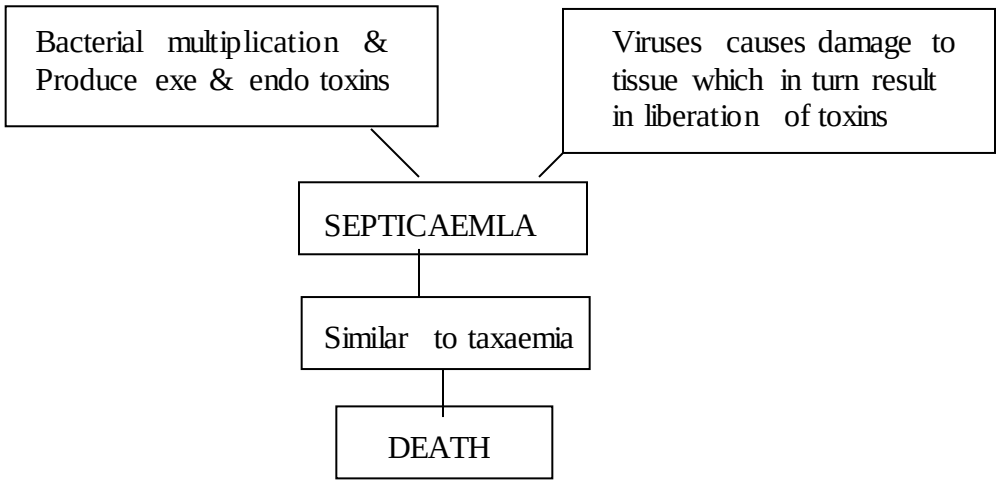
Definition:- It is disease state characterized by presence of toxins and large number of infectious micro organisms. (bacteria, viruses & protozoa) in blood stream"

Etiology :

1. **Bacterial diseases :** Anthrax, pasteurellosis, salmonellosis, leptospirosis, collibacillosis, streptococcus.

2. **Viral diseases :** Hog cholera, African swine fever, FMD, RP.

Pathogenesis:-



Clinical findings:-

The clinical signs in septicemia are those of toxemia & hyperthermia.

1. Fever, tachycardia / Polypnoea, anorexia, depression, ruminal atony.
2. Petechae an conjunctive, oral & vulval mucosae.
3. Localizing signs may occur as the result of localization of infection in joints, heart valves, meninges or other organs.

Diagnosis:-

1. Presence of Petechae on mucosa suggest septicemia.
2. Isolation of the causative agent from blood.
3. Leucopenia or Leucocytosis helps in diagnosis of the type of degree of leukocyte response is of prognostic value.

Treatment :

1. Antibiotics : e.g. – Inj. Oxytetracycline @ 5-10 mg / kg i/m or Amoxycillin-Cloxicillin@ 10-20mg/kg iv.
2. Antipyretics - e.g. Inj. Novalgin 20-30 ml 1/m in adults.
3. Corticosteroids / NSAIDS – Dexamethasone @ 1mg/g iv, Melxycam@0.5mg/kg iv
4. Fluid therapy e.g. - D5 / DNS iv

5. Anticoagulants in DIC e.g. – Heparin.

6) DEHYDRATION

Definition : Dehydration means loss of water and electrolytes from the body.

Causes : Dehydration occurs either due to failure of water intake or excessive loss of water.

a) Failure of water intake –

1. Water deprivation unavailability of water in scarcity
2. Inability to drink due to painful conditions of mouth, pharyngitis, pharyngeal paralysis, oesophageal obstruction and coma.
3. Lack of thirst due to toxemia or debility

b) Excess loss of water-

1. Gastroenteritis (vomition, diarrhoea) in dog.
2. Enteritis in ruminants.
3. Acute CHO engorgement in ruminants
4. Abomasal displacement, impaction, pyloric stenosis
5. Intestinal obstruction
6. Severe peritonitis and ascites
7. Polyuria, diabetes mellitus ,diabetes inslpidus, chronic renal failure
8. Burns and profuse sweating

Classification

There are three types of dehydration

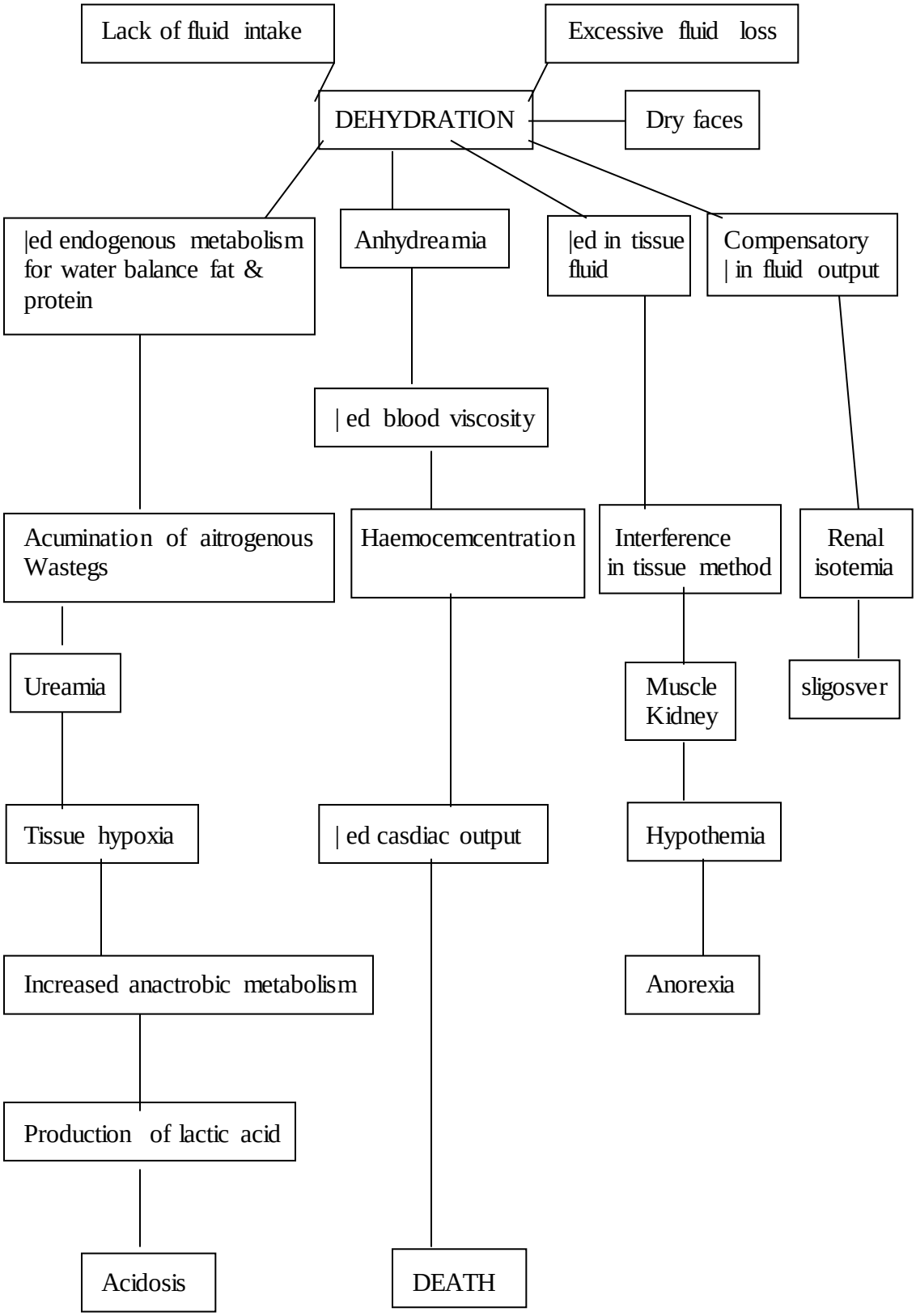
a) Isotonic dehydration :-
   i. Equal loss of water and electrolytes form the body
   ii. Dehydration is usually moderate in nature
   iii. Causes: simple enteritis, profuse sweating, nephritis
   iv. Treatment: Normal saline or Ringers solution i/v

b) Hypertonic dehydration:-

   i. It means loss or deprivation of water with minor loss or deprivation of sodium
   ii. It is uncommon and dehydration is usually mild in nature
   iii. Causes: Water deprivation, Inability to drink water, diabetes
   v. Treatment : water orally and or Dextrose 5%.I/v

c) Hypotonic dehydration :-
   i. It occurs when there is loss of electrolytes in excess
   ii. Dehydration is severe / fatal in nature.
   iii. Causes : severe diarrhoea (salmonelliosis / collibacillosis), Intestinal obstruction.
   iv. Treatment : Normal saline, Ringer's solution or 5% NaCl

Pathogenesis :-



Clinical signs:-

1. Dryness and wrinkling of skin.
2. Sunken eyes.
3. Loss of body weight
4. Muscular weakness
5. Dry mucous membrane- Dry(isotonic), perched (Hypertonic) and sticky moist (Hypotonic)
6. Muzzle dry
7. Oligurea
8. Dry faeces
9. Hypothermia- cold skin and extremities
10. Anorexia.

Clinical pathology:-

Blood : 1) Increased packed cell volume (PCV), Haemglobin (Hb) Total erythrocytes count (TEC), Total leukocytes count (TLC).
2) Increased Blood urea nitrogen (BON)
3) Increased Total serum proteins (TSP).

Assessment of degree of dehydration:-

a) History: The history pertaining to following points will be helpful in assessment of the nature of imbalance.
   1. Duration of illness
   2. Nature and volume of vomitus / faeces passed.
   3. Frequency of vomition / defaecation
   4. Amount of food and water intake

b) Clinical signs:
   1. Dryness and wrinkling of skin
   2. Sunken eye ball
   3. Loss of body weight and muscle weakness
   4. Oliguria (Normal urine output is 1.5 ml / kg/ hour. When it is < 0.5m / kg /hr then it is called as oliguria)

c) Laboratory aid :-
   They are useful in knowing severity of dehydration and confirming the nature of fluid imbalance.
   1. Increased PCV Hb & Total serum proteins
   2. Increased Blood –urea-Nitrogen and creatinine

Degree of dehydration	Sunken eyes	Retension of skin fold (Sec)	(%)PVC
04-06 %	Not sunken	Nil	40-45
06-08 %	Slight	02-04	50
08-10 %	Moderate	06-10	55
10-12 %	Deep	50-45	60

Treatment :

Objectives of fluid therapy

a) To restore blood volume & body fluids
b) To correct acid – base & electrolyte imbalance
c) To provide nutritional supplementation

I) Dose :-

The quantity of fluid required depends upon degree of dehydration, losses occurring during the treatment and maintenance requirement of animals.
The fluids are given in two stages.

a) Hydration therapy:

1. It is given in first 4-6 hrs.
2. It is given for restoring circulating blood volume and for correction of electrolyte imbalance.
3. I/V route is preferred for this therapy.
4. The dose of fluid for hydration therapy is calculated as follows.

Degree of dehydration	Approx dose required (per kg iv)
Mild – 4-6%	25 ml
Moderate – 6-8%	50 ml
Server – 8-10%	75 ml
Very severe –10-12%	100 ml

Quantity of fluid required= [body wt. x (%dehydration)] / 100

b) Maintenance therapy :-

1. It is given in next 24 hrs. following hydration therapy.
2. It is given for maintaining restored blood volume
3. It is given for meeting the normal daily requirement (40-50 ml/kg/day) and for replacing ongoing abnormal losses.
4. It can be given by both intravenous and oral routes.
5. The maintenance dose in general is as follows
   a) 50-100 ml/ kg body weight over a period of 24 hrs.
6. The maintenance dose may be calculated as follows –
   Maintenance dose = BW(kg) X 50 ml + quantity required for replacing ongoing losses.

The loss through vomition is approximately 1 ml / kg / vomitus

II) Route:

1. The route of administration of fluid depends upon
   a. Type of disease
   b. Condition of patients and
   c. Severity and type of dehydration and electrolyte acid base imbalance

2. The fluids can be given by oral route, iv, sc and intraperitoneal route.
3. The oral route is preferred for maintenance therapy, the iv route is preferred for hydration therapy and intraperitoneal route is preferred in large animal practice.

III) Rate / speed of administration :-

It depends upon following factors.

1. Size of animal : Fluids can be given at rapid rate in large sized animals.
2. Severity of dehydration: In severe cases, fluids are given rapidly
3. Type of fluid being administered : Hypertonic solution should be given slowly as compared to isotonic solutions.
4. Type of illness: In pneumonia and cardiac diseases, fluid should given slowly.

In general fluids are given at the following rates.

Hour of administration	Rate / kg / hr
Fist hour	13-14 ml
Second hour	10 ml
Third hour	4-5 ml
Fourth and subsequent hour	2 ml

Drops/min=[(drops/ml)x(ml/kg)x body wt]/60

IV) Frequency of administration :-

The frequency of fluid administration depends upon the severity of the condition.

1) Very severe causes - Continuous drip/ thrice daily
2) Moderate cases - Twice daily
3) Mild cases - Once daily

V) Response to fluid therapy :

During fluid therapy animal must be constantly monitored for clinical and laboratory evidence of improvement or deleterious effects.

A) Favorable response :

1. Urination within 30-60 min.
2. Improvement in mental attitude viz. alertness, activeness
3. Evidence of hydration on performing skin fold test and examination of eye.

B) Unfavorable response :

1. Dyspnoea due to pneumonia.
2. Pulmonary oedema – usually due to rapid administration of fluid
3. Failure to urinate- Indicate renal failure or paralysis of bladder.
4. Tetany: Result from excess administration of alkalies

C) Unusual responses :

1. Fever, shivering, trembling, champing of jaws, restlessness
2. Sometimes frequent defaecation and urination, urticaria.
3. Sweating particularly in horses.

VI) Measures to be taken to avoid unusual responses during administration of fluid

1. Selection of the fluid should be based upon the type of disease condition, state of dehydration & electrolyte imbalance.
2. The selected fluid should be clear, free from any type of contamination (dust particles, etc) There should not be any breakage to fluid bottle.
3. The I/ V set and needle chosen should clean & sterilized.
4. Warm the fluid in bottle to normal body temperature of animal in winter season or whenever required to avoid shivering and other untoward reactions.
5. Calculate the proper dose of fluid required to animal.
6. Fluid should be administered slowly in animals suffering from cardiac diseases (congestive heart failure, traumatic pericarditis) and respiratory distress (pneumonia, broncho pneumonia)
7. Hypertonic solutions (Manitol, Dextrose 20%) should be administered slowly.
8. Continuous monitoring during administration of fluid is essential.
9. If any untoward reactions like shivering, dyspnoea, nasal discharge, urticaria, trembling is seen, then stop the administration of fluid and give antihistaminic and cortico steroids.

ACID - BASE IMBALANCE

The blood pH is maintained in the range of 7-35-7.45 by buffering system. The proportion of the compounds of buffer system viz. dissolved CO_2 and bicarbonates are maintained at constant level by respiratory adjustment and urinary excretion of bicarbonate and hydrogen ions.

The acid- base imbalance may lead to either acidosis or alkalosis.

ACIDOSIS

It develops due to excess production of acid metabolites or as a result of loss of alkali (i.e. bicarbonates) from body or retention of CO_2.

Causes :-

a) Metabolic acidosis:- It is due to

* It develops due to excess

1. Excess loss of alkali i.e. bicarbonates production of acid metabolites or as a regular of loss of alkali diarrhoea.
2. Increased production of acid in starvation, fever, shock, burns, diabetes mellitus, ketosis, acute CH0 engorgement in ruminants.
3. Retension of phosphates, sulphate etc. in renal failure
4. Administration of excess quantities of acidifying solutions.

b) Respiratory acidosis:

1. It develops when there is retention of CO_2 in blood due to interference with normal respiratory exchange.
2. It is observed under following circumstances.

* Poor pulmonary ventilation, pneumonia, pulmonary emphysema, pleurisy, congestive heart failure.
* Depression of respiratory center due to diseases or drugs (anaesthesia).

Clinical Signs :
1. Depression
2. Muscular weakness
3. Increased rate and depth of respiration.

Treatment :

Ringer's lactate solution or sodium bicarbonate orally and / or I/V

ALKALOSIS

It results from excess loss of acids or increased absorption of alkali or deficit of CO_2.

a) Metabolic alkalosis: It may be due to

1. Excessive loss of acids- Gastric dilation in horse, Abdominal disorders and intestinal obstruction in ruminants.
2. Overdosing with alkali viz. Bicarbonates
3. Excess absorption of alkali – urea toxicity.

b) Respiratory alkalosis:

1. It occurs in cases of hyperventilation resulting in excessive expulsion of CO_2 and low blood carbonic acid.
2. The common causes are high fever, toxamia, encephalitis and brain tumor.

Clinical Signs:

1. Slow, shallow respiration (to preserve CO_2)
2. Muscle tremor
3. Tetany.

Treatment :

Normal saline, Ringer's solution/ potassium chloride (1.1% soln).

ELECTROLYTE – IMBALANCE

1. Electrolyte maintain osmotic pressure of body fluids, thereby maintain constant volume of different fluids.
2. The electrolytes of major importance are sodium, potassium, chioride and bicarbonates.

a) Hyponatraemia:

It means depletion of sodium ions.
Sodium is most abundant ion in extracellular fluid and is mainly responsible for maintenance of osmotic pressure of extracellular fluids.

- **Causes :-** Collibacillosis, salmonelliosis, acute diarrhoea.
- **Signs :-** Muscular weakness, mental depression, hypothermia, dehydration.
- **Treatment:** Normal saline or hypertonic salt solution

b) Hypokalemia :

Potassium is predominant ion in intracellular fluid.
Hypokalemia means decrease in serum potassium level

- Causes :- Abomasal disorders, intestinal obstruction (High), enteritis, low dietary intake, Gastritis in dog, prolonged use of mincroalocorticoids.
- Signs:- Muscular weakness, recumbency, depression, cardiac arrhythmia, tachycardia.
- Treatment:- Ringers solution or isotonic saline, potassium chloride (2 gm/ lit)

c) Hypochloraemia:

Hypochloraemia means decrease in chloride level.

- Causes :- Abomasal disorders, intestinal obstruction (high), enteritis, acute gastric dilation in horse, gastritis in dog.
- Signs : Anorexia, weight loss, dullness, mild polydipsia and polyuria
- Treatment : similar to hypokalemia.

7) Shock

Definition : Shock can defined be as the state which is characterized by specialized acute and serious reduction in the perfusion of tissue due to severe reduction in effective circulating blood volume and blood pressure.

Etiology :

A) Hypovolemic shock

It is due to reduction in blood volume either due to loss of blood or its fluid component. The causes of this shock include.

1) Severe Haemorrhage (loss of 30% or more of total blood volume)
2) Dehydration due to diarrhoea, Vomition, intestinal obstruction, acute carbohydrate engorgement / impaction.
3) Burn – plasma is lost.
4) Adrenocortical insufficiency may also produce transient hypovolemia.

B) Distributive / vasogenic shock –

It is due to peripheral vosodilation in which large bulk of blood is pooled in one part of the vasculature. It is due to amines like histamine and serotonins.

1) Neurogenic shock – It is due to severe painful stimuli such as,
   a) Severe trauma - severe fracture, concussion of brain
   b) Severe pain – colic in horses
   c) Fear or excitement in man, wild and timid animals.
2) Severe burns
3) Extensive surgical operations
4) Rapid reduction in abdominal pressure due to rapid withdrawal of large amount of ascitic fluid.
5) Acute intestinal rupture
6) Prolapse of uterus
7) Anaphylactic shock : It occurs due to administration of antigenic substances to which the animal is previously sensitized.
   a) Drugs – e.g. Penicillin sulpha drugs
   b) Transfusion of blood, soera or plasma

C) Toxic Septic shock -

It is due to toxins (endotoxins or exotoxins) produced by gram positive or gram negative organisms (bacterial)

1) Toxemia – Black quarter, botulism, enterotoxaemia, collibacillosis of calves, coli form mastitis, acute diffuse peritonitis, acute metritis, abscess, hepatic dysfunction, intestinal obstruction , ketosis, uraemia acute CHO engorgement.
2) Septicemia - Anthrax, H.S., salmonelliosis, septicemic collibacillosis, pseudotuberculosis, leptospirosis, listeriosis Hog cholera.

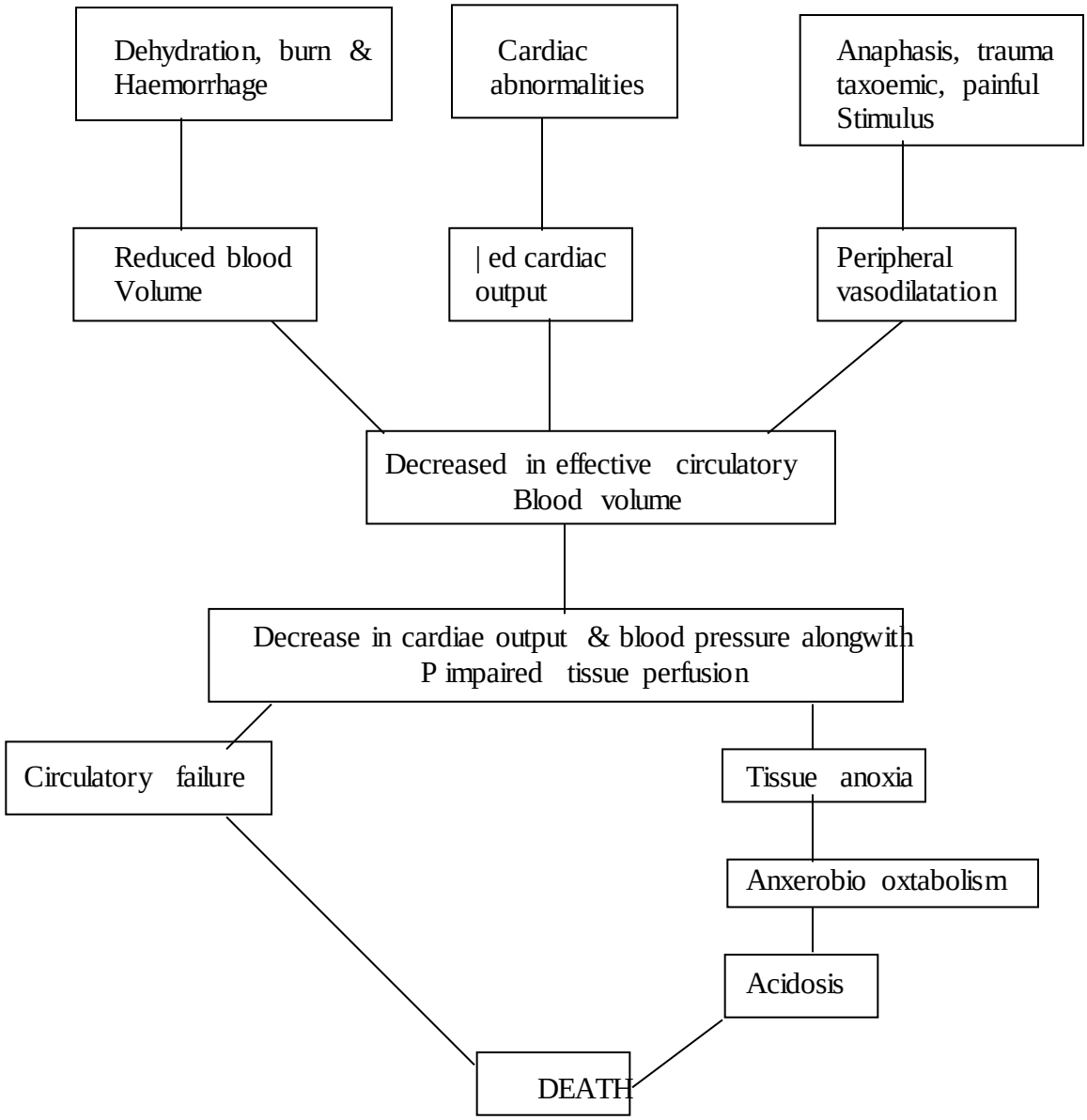
D) Cardiogenic shock –

It is due to cardiac abnormalities which results in reduction in cardiac output. It is due to

1) Myocardial disease- Myocardial infarction
   acute myocarditis
   myocardial asthenia.
2) Valvular diseases - Mitral stenosis
3) Pericardial diseases - Traumatic pericarditis,
   fibrinous pericarditis

This shock usually occurs when there is damage to 40.90% of left ventricular myocardium of is manifested by low arterial blood pressure of elevated venous pressure.

Pathogenesis :



Clinical signs :

1) Body Temperature is subnormal (hypothermia)
2) Cold skin and extremities
3) Tachycardia with diminished heart sounds
4) Rapid shallow respiration
5) Rapid thready pulse
6) Dullness and weakness
7) Mucous membranes are dry and pale
8) Oliguria / anuria
9) Muscle weakness
10) Recumbency
11) Loss of reflexes
12) Comea
13) Death

Diagnosis :

1. History - Laemorrhoge, Vomition, diarrhoea , burns, administration of some drugs severe colic severe truma etc.
2. Clinical signs : Hypothermia, loss of reflexes recumbence
3. Decreased arterial blood pressure – 60 mm Hg. (120 mm Hg)
4. Increased blood lectate > 8.3 min / L
5. Reduced venous pressure except cardiogenic shock

Clinical assessment of shock

1) History – severity of haemorrhage, vomition, diarrhoea, sweating etc.
2) Clinical findings
   i) Body temperature
   ii) Pulse rate & quality
   iii) Heart rate & intensity
   iv) Reflexes
   v)
3) Haematology –
   - Packed cell volume

Treatment

A. General principles of treatment in shock -

1) Blood volume expanders -
   - Primary objective is to restore circulatory blood volume.
   - Blood transfusion in haemorrhagic shock
   - Plasma in burns
   - Plasma expanders in severe dehydration e.g. dextran, haemacel.
   - Isotonic fluids and electrolytes in dehydration.
2) Maintenance of patency of airway and proper respiration
   - Endotracheal intubations
   - Tracheotomy
3) Use of corticosteroids :

The beneficial effects are -

- They improve microcirculation
- They stabilize endothelial function
- They Detoxify endotoxins

e. Dexamethasone @ 1/mg / kg body wt 1/v, 1/m or prednisolone Na acetate @ 5-15 mg / kg at 4-6 hrs interval i/v, i/m

4) Vasoactive drugs
These drugs are given to raise blood pressure e.g. dopamine @ 1-10 mg / kg / min

5) Use of alkalizing agents to correct metabolic acidosis
e.g. sodium bicarbonate 1.3 % or 2.5%

6) Antibiotics : Amoxicillin & cloxacillin @ 10-20 mg / kg iv

7) General care
   i) Animal should be kept in comfortable condition
   ii) Animal should be kept warm by covening blankets
   iii) Arrange adequate ventilation.

B) Treatment depending upon of type of shock

a) Hypovolemic shock : The primary objective is to restore circulating blood volume. This an be achieved by
   1) Blood Transfusion in Taemorrhage.
   2) Plasma expanders in case of severe dehydration e.g. dextran, Haemacel iv
   3) Plasma @ 20.40 ml / kg iv in case of burns
   4) Isotonic fluids and electrolytes in dehydration

b) Vosogenic shock
   1) Vosoconstrictive drugs e.g. Adrenaline @ 8-16 µg / kg v/im
   2) Corticosteroids e.g. Dexamethasone 1 mg / kg iv/im
   3) Antiprostaglandins drugs e.g. meloxicam @ 10.2–0.5mg/ kg im or iv esgipyrin @ 10-15 ml im
   4) Vosative drugs – Dopamine @ 1-10 mg / kg min iv

c) Septic Shock -
   1) Antimicrobials- e.g. amoxicillin and cloxacillin @ 5-10 mg / kg im/iv
   2) Antiprostaglandins e.g. Meloxicam @ 0.2 – 0.5 mg / kg im/iv
   3) Fluid therapy in high doses

d) Anaphylactic shock -
   1) Vosoconstrictive drugs e.g. adrenaline @ 8-10 µg / kg im/iv
   2) Corticosteroids e.g. Dexamethasone @ I mg / kg iv / im
   3) Antihistaminic drugs e.g. Avil @ 0.5-1.0 mg / kg im

e) Cardiogenic shock -
   1) Digitalization to be made to restore cardiac function
   2) Nor adrenaline to increase cardiac output.
   3) Myocardial infarction is treated with corticosteroids e.g. Dexamethasone @ 0.04 mg / kg im

f) Neurogenic shock
   1) Analgesic drugs e.g. ketoprofen 3 mg / kg im/iv
   2) Sedative drugs e.g. Diazepam @ 0.25 – 0.5 mg / im / iv.

CHAPTER – 2
Diseases of Digestive System - I
1) Stomatitis

Definition :- It is inflammation of the oral mucosa and is clinically characterized by partial or complete loss of appetite, smacking of lips and profuse salivation.
It also includes :

i)	Glossitis	:	Inflammation of tongue
ii)	Gingivitis	:	Inflammation of gums
iii)	Lampas/palatitis	:	Inflammation of palate
iv)	Cheilitis	:	Inflammation of lips
v)	Odontitis	:	Inflammation of teeth

Causes :

- It may be primary or secondary
- Primary Stomatitis is caused by physical or chemical agents
- Secondary stomatitis is commonly accompanied with systemic disease

A) Non-infectious causes:

1) Physical : Trauma or injury caused by
   a. Spines on plants
   b. Rough fodder
   c. Foreign bodies like needles, nails
   d. Eating too hot or too cold food.
   e. Dental abnormalities, sharp teeth, malposition of teeth
   f. Unskillful drenching and faulty passing of probang.

2) Chemical :

- Administration of irritant drugs like chloral hydrate and turpentine oil in higher concentration
- Accidental ingestion of acids and alkalies
- Licking of counter irritants applied over skin.

3) Nutritional deficiency :

Deficiency of vit. A, Vit B complex, Riboflavin, Niacin.
Niacin deficiency causes black togue i.e. necrotic stomatitis in dog .

4) Allergy :

Certain plant allergens, stinging plants.
[Catarrhal stomatitis usually result from direct injury by chemical/physical agents]

B) Infectious causes:

1) Bacterial :

Oral necrobacillosis (*Fusobacterium necrophorus*), *actinobacillosis, Streptococci, actinomycosis, Staphylococci.*

2) Viruses :

i)	Cattle & Buffalo	:	RP, FMD, BMC, BVD, IBR in calves
ii)	Sheep	:	RP, PPR, BT, Sheep pox, Contagious ecthyma

iii) Goat : RR, PPR, Goat pox, Contagious ecthyma.
iv) Horse : Vesicular stomatitis, herpes virus infection
v) Pig : Vesicular exanthema, FMD
vi) Dog : Canine Distemper, ICH

3) Parasites :
Plant lice, plant mites, *Entamoeba gingivalis*
4) Fungi :
Moniliasis / Candidiasis, Aspergillosis

Clinical signs :

- Partial or complete anorexia
- Increased desire for water
- Slow and painful mastication
- Difficulty in prehension of food
- Salivation either frothy or profuse of drooping
- Smacking of lips and chewing movements.
- Foetid odour from mouth in case of bacterial infection
- Oral lesions : vesicles, ulcers, erosions, necrosis, papular granulomatous.
- Enlargement of local lymph node may occur
- Body Temp may be elevated
- Traumatic lesions are usually solitary
- Mycotic stomatitis – Heavy, white velvety deposits over tongue.

Treatment :

A. Correct the primary cause :
   i. Antibiotics like Oxytetracycline, Ampicillin
   ii. Antifungal drugs like mycostatin in fungal diseases
   iii. Surgical treatment of injury
   iv. Multivitamins in Nutritional deficiency

B. Symptomatic / supportive therapy:
   i. Mouth wash (collutaria) with 0.1% potassium permangnet lotion or 2% $CuSO_4$ solution or any other antiseptic solution.
   ii. Application of boroglycerine
   iii. Cauterization of ulcers with Tincture iodine or silver nitrate sticks.
   iv. Multivitamin therapy – Inj. Multivet @ 1 ml /10 kg IV or IM
   v. Give soft liquids diet or greens
   vi. Intravenous fluid therapy in severe cases.

2) Parotitis

Definition: " It is inflammation of salivary / parotid gland"

Causes:

There are no specific causes. However following factors may be responsible for Parotitis

A. Non-Infectious causes:
   i. Trauma / injury: irritation due to grass.
   ii. Salivary calculi
   iii. Avitaminosis-A

B. Infectious Causes:
   i. Extension of infection from mouth, pharynx and retropharyngeal cellulitis

ii. Bacterial diseases:
Mycobacterium tuberculosis, Actinobacillus lignieresi, Streptococcus equi (strangles) in horse.
iii. Viral diseases: CD in dog.
iv. Lymph node abscesses.

Clinical Signs:

Acute form : One or both glands may involved.

i. Fever, anorexia, dullness & suspended rumination.
ii. Marked painful swelling of gland & adjoining areas
iii. Difficulty in mastication & swallowing due to pain
iv. Restricted movements of head & neck due to pain
v. Dyspnoea.

Chronic form :

i. Firm swelling of gland.
ii. Abscess formation .

Differential diagnosis:

a. H.S: Submandibular swelling equally progress over cheek and symmetrical
b. Snake bite: Swelling at site of bite, large asymmetrical swelling

Treatment

In acute cases :

i) Use of broad spectrum antibiotics like Oxytetracycline Streptopenicillin, enrofloxacin for 5-7 days.
ii) Use of antipyretics, anti inflammatory & analgesics e.g. meloxicam @ 1 mg / kg im for 5-7 days.

In chronic cases :

i] Hot fomentation with magnesium sulfate or massage with iodine ointment.
ii] Surgical drainage in case of abscess formation.
iii] In chronic non responsive cases inject 2.5 ml of lugol's iodine into gland to make it atrophied.

3) Pharyngitis

Definition : It denotes inflammation of the pharynx. It is mostly seen in dogs, horses but rare in ruminants.

Causes :

A) Non-infectitious:

1) Physical :

- Injury due to rough food & foreign body
- Consumption of excess not or cold food
- Faulty use of stomach tube / probang

2) Chemical :

Accidental ingestion of irritant chemicals / drugs

B) Infectitious:

1) Bacteria :

Pasteurella multocida, *Sphaerophorus necrophorus, Actinobacillus lignieresi,* Strangles in horses ,anthrax in pig., *Bordetela bronchiseptica* in dogs.

2) Viruses :

Bovine : Infectitious bovine rhinotracheitis (IBR)
Equines : Para influenza, Adeno, rhinoviruses
Dog : ICH and CD

Clinical signs :

1.Difficulty in swallowing i.e. dysphagia (extension of head & neck)
2.Salivation
3.Coughing during feeding and also on manual compression of throat / pharynx
4.Bilateral nasal discharge
5.Inspiratory dyspnoea
6.Protrusion of tongue
7.Regurgitation of food and water through nostrils in severe cases.
8.Fever in infections pharyngitis.

Differential diagnosis

a. Pharyngitis.

1.Acute onset
2.Difficulty in swallowing.
3.Coughing during feeding and drinking
4.Local pain

b. Pharyngeal paralysis

1.Caused by rabies, injury to nerve and encephalitis
2.Slow onset
3.Inability to swallow
4.Regurgitation of food and water
5.No pain reaction.

c. Pharyngeal obstruction :

1.H/o ingestion of foreign body
2.Sudden onset

3.Continuous expulsive coughing
4.Inability to swallow

Treatment

1.Parenteral broad spectrum antibiotics e.g. oxytetracycline, streptopenicillin, enrofloxacin im.
2.Antiinflammotory & analgesic drugs .e.g. meloxicam or ketofrofen im
3.Antihistaminics to reduces pharyngeal oedema e.g. pheniramine maleate i/m
4.Multivitamins for better healing e.g. Multivet im / iv
5.Give liquid soft palatable diet in mild cases
6.Fluid therapy in severe cases

4) Oesophagitis

Definition: It means inflammation of esophagus.

It is rare in animals because the esophageal mucous membrane is less sensitive to the action of irritants.

Causes

A. Physical : 1. Trauma or injury caused by foreign body, sharp object
2. Vigorous / unskilled passing of stomach tube / probang
3. Ingestion of too cold or too hot food

B. Chemical :
Irritant chemicals or drugs viz formalin, ammonia, chloral hydrate.

C. Parasites :
Horse bot fly (*Gastrophilus*) ,esophageal worm in dog (*Spirocerca lupi*)

D. Bacterial and viral infection:
Oesophagitis is always accompanied by stomatitis and pharyngitis.

Clinical signs :

- Rise of body temperature
- Profuse salivation
- Difficult painful swallowing (Dysphagia)
- Regurgitation of food and water
- Coughing and stretching movements of neck during swallowing.
- Pain on palpation in the jugular furrow in cervical Oesophagitis.

Diagnosis :

1) History : Vigorous passing of probang, Ingestion of irritant chemicals
2) Clinical signs : Dysphasia, salivation & regurgitation of food
3) Radiography : Evidence of foreign body / lesions.
4) Endoscopy : Evidence of injury, lesion or inflammation

Differential diagnosis

Pharyngitis		Oesophagitis		Oesophageal obstruction
i. Coughing is more severe	i	Comparatively less sever	i	Coughing is less prominent

ii. Pain while swallowing	ii	Severe pain while swallowing	ii	Inability to swallow food
iii Bloat is absent	iii	Bloat is absent	iii	Bloat is present
			iv	Palpable enlargement in cervical region

Treatment:

1. Give course of antibiotics to control infection e.g. Strepto-penicillin.
2. Give analgesic, antispasmodic and anti-inflammatory drugs e.g. meloxicam @ 1 mg/kg im.
3. Give soft liquid diet for few days & mild cases
4. Withheld feed and maintain animal on fluid therapy for 2-3 days in severe cases.

5) Oesophageal Obstruction or choke

Definition: "Choke is the closure or occlusion of normal oesophageal lumen by foreign body or food mass".

- Choke may acute or chronic
- If may be partial or complete
- It may be cervical or thoracic
- In horse, cattle and buffalo: obstruction in frequently observed is cervical region.
- In dog and cat, it is observed in thoracic inlet or diaphragmatic region.
- The choke in cervical region usually occurs just above the larynx or at thoracic inlet
- The obstruction in thoracic region generally occurs at the base of heart or the cardia.

Causes:

A. Intra-luminal obstruction:

a) Food mass: Mango seed, potato, turnip, beet, apple orange, water melon.

In horse: bolus of bran or hay

In dog: Fish or chicken bone large block of fish / meat

Feed stuffs are common causes of obstruction in horses allowed to eat immediately after race of work.

b) Foreign body: Stone, metal piece, plastics, polythene, leather, wood, glass etc. in dog toys.

B. Extra luminal obstruction:

a) Space occupying lesions on the oesophageal wall e.g. papilloma, neoplasm, cyst, abscesses, *Spirocerca lupi* in dog.

b) Pressure from outside on esophagus. e.g. Tuberculosis, abscesses of mediastinal lymph node

Note : A – Group : Usually causes acute obstruction
B – group : Usually causes chronic obstruction

Clinical Signs :

Acute:

- Profuse salivation in complete obstruction
- Refusal of food / inability / forceful attempts to swallow food.

- Coughing and stretching of head and neck from side to side while swallowing of food
- Continuous chewing movements.
- Regurgitation of food and water in complete obstruction and only food in partial obstruction.
- Firm cylindrical swelling along the course of neck on left side in case of cervical obstruction of esophagus.
- Nasal reflux of saliva, feed and water in affected horses.
- Horses with choke commonly regurgitate feed and water and drool saliva through nostrils due to the anatomical characteristics of the equine soft palate.

Chronic:

- The earliest sign is chronic bloat without appearance of other signs.
- Swallowing movements are usually normal until the bolus reaches the obstruction when they are replaced by more forceful attempts.
- Projectile expulsion of ingested material occurs with oesophageal diverticulum's but water is retained.

Megaoesophagus is a dilatation and along of body of esophagus usually associated with of the esophagus function and the caudal esophagus sphincters. It is sporadic and usually a congenital condition causing regurgitation and aspiration pneumonia.

Diagnosis :

1) History : Ingestion of foreign body or large food mass.
2) Clinical signs : Palpation of mass in cervical region
3) Passing of probang / stomach tube to find out nature and location of obstruction.
4) Radiography & Endoscopy.

Treatment

1) Sedate animal by using tranquilizers e.g. largactil @ 1 mg / kg im or siquil @ 0.1 mg / kg im. diazepam@ 0.25 – 1 mg / kg , im or iv
   Horses Asepromazine @ 0.05 mg / kg 1/v xylazine @ 0.5 / mg / kg 1v
2) Atropine Sulphate @ 0.03 – 0.06 mg / kg s/c for large animal to relax Oesophageal spasm
3) Solid obstruction near pharyngeal region can removed by hand
4) Solid masses can be pushed towards stomach by probang
5) Oesophagotomy

6) Gastritis in dog

Definition: Gastritis means inflammation of gastric mucosa. It is common in dogs and cats and usually accompanied by enteritis.

Etiology :

a) ***Non infections***

1) *Dietary factors:*

- Overfeeding
- Sudden change in food
- Ingestion of mouldy decomposed or highly fermented food
- Drinking of dirty / sewage / drain water

2) *Physical agents:*

- Ingestion of foreign bodies viz. sand, soil.
- Consumption of grass.
- Ingestion of too hot or too cold water / food.

3) *Chemical agents:*

- Irritant chemical e.g. As, Pb, Cu etc.
- Acids and alkalies
- Drugs like aspirin, phenylbutazone

4) *Allergens:*

- Food allergens (Specific)
- Nephritis (uraemia)
-

Infectious

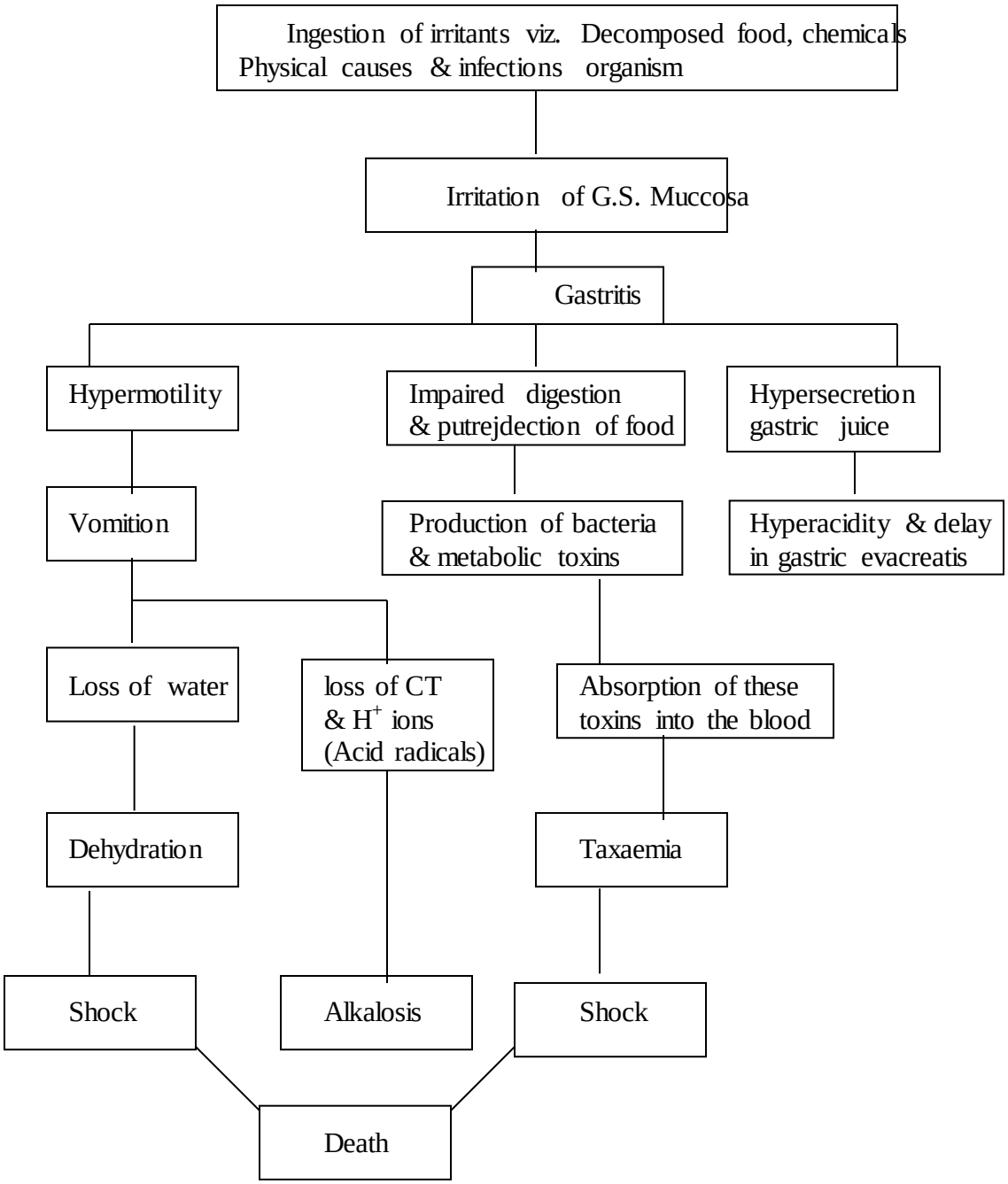
1 Bacteria: *E. coli Salmonella, Leptospirosis, Helicobacter*

2 Viruses: Canine distemper, Parvovirus infection, infectious canine Hepatitis, corona virus

3 Fungi: Aspergillus, Monila, Mucor

4 Parasites: Toxocara canis, Tapeworms

Pathogenesis :



Symptoms :

A. ***Acute :*** Sudden onset

- Loss of appetite
- Intense thirst
- Frequent vomition
- Vomitus is usually watery and may contain blood and mucus
- Salivation
- Belching of offensive smelling gases
- Abdominal pain / colic, discomfort, restlessness
- Pain on deep palpation over epigastric region
- Severe dehydration

- Death

B. ***Chronic :*** Prolonged duration of illness.

- Poor / depraved appetite i.e. pica
- Vomition usually after intake of food
- Vomitus is viscid and foetoid in odour
- Moderate abdominal pain
- Aerophagia / yawning
- Body coat is dry and rough
- Emaciation-gradual loss of body weight due to malnutrition / starvation
- Anaemia

Difference between:

Acute gastritis	Chronic gastritis
1. Sudden onset & short course	i. Prolonged course
2. Frequency of vomition is more	ii. Frequency of vomition is less
3. vomition is irrespective of feeding	iii. Usually vomition is after feeding
4. Vomitus usually watery	iv. Vomitus is viscid & thick
5. Dehydration is more marked	v. Dehydration is less marked
6. Emaciation is less marked	vi. Emaciation is more marked
7. Abdominal pain is more marked	vii. Abdominal pain is less marked
8. PCV is usually increased (haemoconcentration)	viii. PCV is usually decreased (Anaemia)

Diagnosis:

i. History: Overfeeding, change in diet, eating of spoiled food.
ii. Clinical signs: Vomition, abdominal pain, dehydration
iii. Endoscopy: Helps in detection of gastric ulcer / lesions or foreign body.
iv. Radiography: Helps in detection of foreign bodies.
v. Faecal examination: Detection of parasitic eggs.
vi. Haematology: PCV- increases in acute gastritis where as decreases in chronic gastritis
vii. Serum biochemistry: Na^+, K^+, Cl^-, HCO3 estimations.

Treatment :

Acute :

1. Antibiotics orally to check fermentation and gas production and parenterally to check bacterial infection

a) Inj. Gentamicin@2-4 mg/kg i/v and Inj. Amoxicillin + cloxacillin @20 mg IV twice daily (for 3-5 days) OR
Inj. Amikacin @10-15 mg /kg IV and Inj. Cefotaxine @20-40 mg/kg iv twice daily (for 3-5 days) OR
Inj. Chloramphenicol @20-40 mg /kg as IV twice daily for 3-5 days

b) Normet liquid 1 teaspoonful twice daily for 3 days OR
C-flox Tz tab 1 B.I.D. orally for 3 days .

2. Antiemetics to stop Vomition

Inj. Perinorm (metachlopromide 5 mg/ ml)@ 0.1-0.3mg/ /kg I/V or I/M Or
Inj. Siquil (Triflupromazine HCL 20mg/ml) @ 1-2 mg/ I/V or 2-4mg im twice daily OR Inj. Stemetil (perchlorperazine12.5 mg/ ml) @ 0.2 –0.5 mg/ kg im twice daily.

3. Fluid therapy: to correct dehydration and alkalosis a) DNS or Balanced electrolyte solution (Ringers solution) depending upon severity of dehydration b) Haemacel (polymer Na, K, Cl, Ca) @ 10-20 ml/ kg body wt in severe dehydration.
4. Antacids to check gastric hyperacidity.
   e.g. Gelusil @ 2 tea spoonful 3 times daily orally half an hour before meal and / Or:
   Histac (Ranitidine 25 mg/ ml) @ 1-2 mg / kg iv B.I.D. 10-15 min prior to meal.
5. Analgesics to relieve gastric pain e.g. Analgin @ 33-50 mg/ kg im
6. Vit – B complex : Parenterally or orally to improve appetite and G I tone.
   Inj Polybion /Neurobion /Tribivet @ 0.5-2ml im.
7. Use of coagulants / styptics in case of haemorrhagic vomition & diarrhoea.
   e.g. Inj. Stadren 1-2 ml im or
   Inj. Botropase ½ -1 ml iv
8. Diet : withheld food for first 24 hrs. Then give soft easily digestible liquid diet.

Chronic :

1. Antihelmintics: e.g. Albendazole @ 25 mg / kg po twice daily for 3 days.
2. Antacids: e.g. Gelusil @ 2 tea spoonfuls 4 times daily and / or Ranitidine @ 1-2 mg / kg orally twice daily for 2-4 weeks.
3. Antibiotics orally for 3 days
4. Bland food and milk at 4 hrs. interval
5. Haematinies: to correct anaemia

7) Abomasitis

Definition: Inflammation of mucus membrane of abomasum.

Causes:

1. Physical agents:
   i. Overfeeding
   ii. Ingestion of too hot or too cold water
   iii. Ingestion of highly fermented, mouldy decomposed food.
   iv. Feeding of inferior quality straw
   v. Drinking of dirty stagnant or sewage water
   vi. Ingestion of foreign body like sand, soil

2. Chemical agents:
   i. Irritant chemicals: Arsenic, lead, copper phenol etc.
   ii. Acids and alkalies
   iii. Drugs: Aspirin, phenylbutazone

3. Bacteria:
   E. coli, Salmonella, Clostridium perfringens, Leptospirosis, Sphaerophorus necrophorirus.

4. Viruses:
   i. Bovine: RP. BVD. BMC
   ii. Small ruminants: PPR, RP
   iii. Pig: Swine fever

5. Fungi: Aspergillus, Monilia, Mucor

6. Parasites:
i. Ruminants: *Trichostrongylus sp., Haemonchus sp. ,Ostertagi sp.*
ii. Horse: *Gastrophilus* / bot fly larvae, *Habronema sp*.

7. Allergens: Specific food.

8) ***Enteritis***

Synonyms :- Enteropathy, Diarrhoea, Malabsorption .

Definition: "It means inflammation of intestinal mucous membrane."

Etiology:

A] Noninfectious causes:

a. Dietary:
i. Overfeeding
ii. Sudden change in diet
iii. Excess consumption of green lush pasture, green grass, green leaves
iv. Feeding of moldy, decomposed spoiled rotten food
v. Drinking of dirty, sewage / waste water.

b. Physical agents:
Eating of sand, soil etc.

c. Poisoning:
i. Ingestion of irritant chemicals viz arsenic, copper , fluorine, mercury, nitrate.
ii. Ingestion of poisonous plants
iii. Molybdenosis.

d. Drugs : Like antimicrobials , anthelmintics.

e. Nutritional deficiencies : Copper (peat scours), Cobalt, vit. A deficiency

f. Psychological causes:
Fear ,excitement, tension ,stress, overwork ,transportation, work after feeding, exposure to unaccustomed environment.

g. Miscellaneous : Disaccharides deficiency in calves, foals and pups.

B] Infectious causes:

a. Bacterial diseases:
i. All species: *E. coli, Salmonella, Campylobacter jejuni, Pseudomonas, Proteus, Shigella, Pasteurella, Streptococci, Staphylococci.*
ii. Ruminants: *Clostridium perfringes*(enterotoxaemia),*Mycobacterium ,Para tuberculosis*(JD)

b. viral diseases
i. Bovines : RP, BVD, BMC, Rotavirus, Corona virus

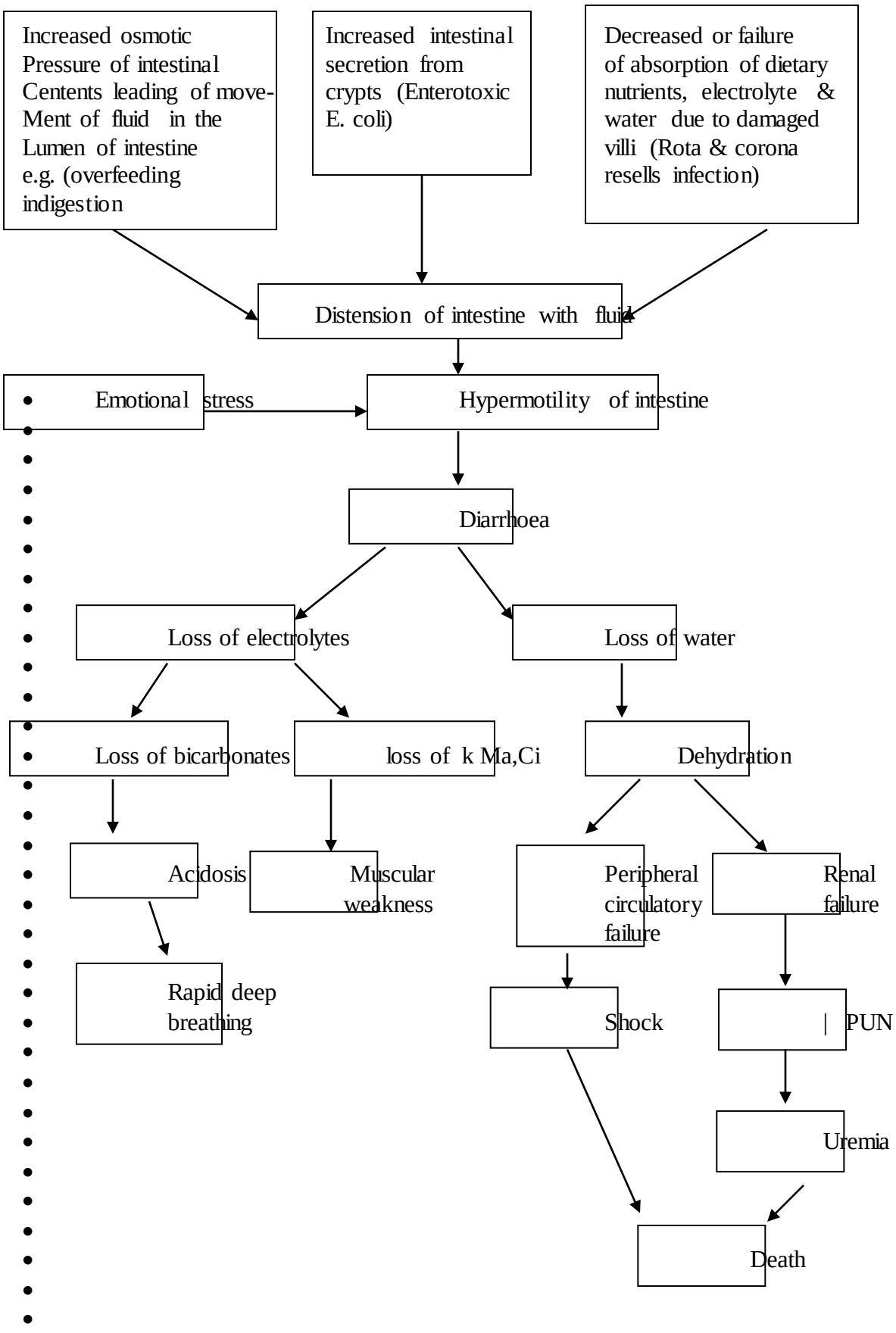
ii. Ovine & Caprine: RP, PPR, Rotavirus, Corona virus
iii. Canines : Parvo virus ,CD, ICH, Corona virus

c. Protozoan diseases
i. Ruminants : Coccidiosis, Balantidiasis, Cryptosporidiosis
ii. Canines : Giardiasis, Amoebiasis, Coccidiosis

d. Fungal diseases :
i. *Candida* sp, *Aspergillus* sp.

e. Parasitic diseases :
i. Round worms:
Ruminants : *Toxocara Trichostrongylus, Oesophagostomum, Bunostomum, Trichuris, Cooperia* etc
Canine – *Toxocara canis, Ancylostoma sp, Trichuris sp.*
ii. Tapeworms :
Ruminants : Moniezia sp.
Canine: *Taenia, Dipylidium caninum, Diphyllobothrium latem*
iii. Flukes:
Ruminants : *Fasciola* sp; *Paramphistomum* sp, *Schistosoma* sp.

Pathogenesis



Clinical Signs :

A. Acute form:

1. Diarrhoea / dysentery :
   - Faeces are soft or fluid in consistency
   - Having foul or fishy odour
   - May contain blood, mucus, epithelial shreds, fibers, casts etc.
   - Abnormal colour may seen
2. Frequency of defaecation is increased
3. Soiling of buttocks and hindquarters.
4. Straining or tenesmus may occur
5. Dehydration – sunken eyes, rough hair coat, tenting of skin
6. Abdominal pain /colic characterized by kicking at belly, rolling on ground, stretching of pain on deep palpation of abdomen
7. On auscultation fluid rushing sound or borbogymi is heard
8. Fever may be present
9. Muscular weakness
10. Loss of appetite
11. Oliguria

B. Chronic form:

1. Faeces are usually soft and homogenous in consistency
2. Faeces contain considerable mucus
3. Faeces usually do not have grossly abnormal odour.
4. Emaciation is common
5. Bottle jaw and Anaemia is observed
6. No systemic abnormalities i.e. no rise in body temperature, toxaemia, anorexia
7. Dehydration is not marked
8. Abdominal pain (colic) is not marked.

Differentiate between:

Diarrhea	**Dysentery**
1. Voluminous fluid faeces	1. Scanty sticky faeces
2. No blood , mucus or fibrin in faeces.	2. Present
3. Less / no straining	3. More straining

Acute	**Chronic**
1. Faeces are not homogenous and usually watery	1. Soft homogenous
2. Foul smelling	2. Usually do not have abnormality
3. Dehydration is marked	3. Dehydration is less marked
4. Abdominal pain is marked	4. Abdominal pain is not marked
5. Emaciation is not marked	5. Emaciation is marked
6. Systemic reaction is usually present	6. Systemic reaction is absent

Small bowel diarrhoea	**Large bowel diarrhoea**
1. Appetite is decreased	1. Unaltered
2. Loss of body wt. is marked	2. Minimum loss of body weight
3. Faeces are voluminous	3. Faeces are passed in small quantity at

	frequent interval
4. Tenesmus / straining is usually absent	4. Invariably present

Diagnosis :

- History of feeding, stress, deworming etc.
- Clinical signs : diarrhoea, dysentery ,colic, dehydration
- Faecal examination for parasitic eggs.
- Faecal culture for isolation of pathogenic microorganisms
- Haematology - Hb, PCV, TLC etc.
- Serum biochemistry – HCO_3 , Na, K, Cl, BUN and total serum proteins
- Plane or contrast radiography

Differential diagnosis

A. Acute diseases

a. Dietary diarrhoea

- History of change in diet or over feeding
- Animal looks normal
- Body temperature is normal

b. Poisoning

- History of ingestion of toxic chemicals / poisonous plants
- Severe haemorrhagic enteritis
- Subnormal body temperature.

c. .RP, BVD, PPR

- Diarrhoea & mouth lesions
- Fever may present

d. Coccidiosis

- Young animals (calves, lambs, kids) from 3 weeks to 6 months are mostly affected.
- Smudge pattern
- Soiling of buttocks with sticky faeces
- Faecal examination reveals oocysts

e. Salmonellosis

- All ages are affected
- Stress induced
- Fever, acute diarrhoea / dysentery
- Fibrinous casts / pipe like structures are voided in faeces.

f. Collibacillosis :

- Newborn animals are affected
- Acute diarrhoea
- White scour

g. Enterotoxaemia

Young calves / lambs / kids are affected
H/o overfeeding of milk / grass
Haemorrhagic entero- toxaemia, rapid death.
Pulpy kidneys

h.Winter dysentery

- Common in winter
- Mild disease

B.Chronic diarrhoea :

a. Helminthiasis / GI nematodiasis

- No systemic reaction, i.e. appetite & body temp are normal
- Progressive emaciation, anemia, pot belly, bottle jaw.
- Faecal examination reveals eggs.
- Good response to anthelmintics

b. Fascioliasis in ruminants

- History of grazing in areas near by water reservoir viz ponds, lakes, river
- Faeces may be passed like pipe water.
- Jaundice, bottle jaw ,anaemia may be present
- Faecal examination reveals *Fasciola* eggs.
- Good response to flukicides

c. J.D. in ruminants

- Mature 4-6 yrs age group of cattle are affected
- Faeces contain gas bubbles.
- No response to anthelmintics and antibiotics
- Thickening and corrugation of intestine on PM examination
- Cu deficiency or Molybdenosis
- Depigmentation of hair skin is characteristics
- Feeding of molybdenum rich / Cu deficient diet.

e. Giardiasis in dogs

- Greasy stools containing blood & mucous
- Straining during defecation
- Loss of body weight
- Faecal exam reveals cysts

f. Amoebiasis in dog

- Chronic diarrhoea / dysentery
- Abdominal pain
- Loss of body weight
- Faecal examination reveals cysts

g. Tapeworm

- Loose faeces
- Segments in faeces
- Young animals are mostly affected (lambs, kids, pups)

Treatment

1) Removal of causative agents

A) Antibacterial drugs : used in bacterial infections and as supportive in viral infection.

- It is better to give antibacterial drugs by both oral as well as parenteral routes.

i) Oral preparations:

- Tetracycline's : @ 10 mg / kg body wt. or
- Sulphadimidine @ 100-200 mg / kg or
- Sulpha-trimethoprim @ 30 mg / kg body wt. or
- Amoxicillin @ 10mg / kg or
- Norfloxacin @ 22 mg / kg
- Metronidazole + Furazolidone @ 20 mg / kg for 3-5 days
- Ciprofloxacin + Tinidazole combination @ 5 mg/kg

ii) Parenteral preparations:

- Inj. Ciprofloxacin @ 5 mg/kg im / iv
- Inj. Enrofloxacin @ 2.5-5 mg / kg once daily or
- Inj. Gentamicin @ 1-2 mg / kg in cattle & 2-4 mg / kg in dogs or
- Inj. Cefotaxime @ 20-40 mg / kg i/v in dogs twice daily

B) Anticoccidial drugs used in Coccidiosis

- Furazolidne @ 10-20 mg / kg orally for 5 days or
- Sulphadimidine @ 150-200 mg / kg orally for 5 days.

C) Anthelmintics : Used in worm infestation

- Round worm : Albendazole @ 5-10 mg / kg orally Fenbendazole @ 5-10 mg/kg orally
- Liver flukes : Oxyclozanide (Distodin) 10-15 mg / kg orally in cattle
- Tapeworms

Praziquantel : Cattle : @ 10 mg / kg PO
Dog @ 5 mg / kg PO
Niclosamide : Cattle @ 50-100 mg / kg PO
Dog @ 150 mg / kg PO
Fenbendazole 150 mg + Praziquantel 50 mg (fentas plus tab) @ 1 Tab / 10 kg body wt

2) Astringent preparation for symptomatic control of diarrhoea

- Astringent mixture (Kaolin 30 gm + Creta preparation 30 gm Pulv ginger 15 gm + Catechu 30 gm) as drench twice daily
- Neblon / kabjol powder @ 30-50 gm twice daily PO for large animals @ 10-15 gm for small animals.
- Becknoor bolus : 1 BID

3) Fluid and electrolyte therapy to combat dehydration and acid-base imbalance

- Mild dehydration: Electrolyte solution orally
- Moderate dehydration: Inj RL solution or Sodium bicarbonate 1-3 % iv
- Severe dehydration & acidosis: Sodium bicarbonate 5% @ 5-7 mg / kg iv

4) Antispasmodics to control colic and straining:

- Inj Cyclopam / Spasmovet (Dicyclomine 10mg / ml) @ 0.5 mg / kg body wt IM
- Inj. Atropine sulphate (0.6 mg / ml) @ 0.03 mg / kg IM

5)Alteration in diet:

- With held feed for first 24 hrs.
- Then provide soft liquid and easily digestible diet.

6) Use of styptics to control bleeding from GI tract.

- Inj Stadren @ 10-15 ml im daily in large ruminants and 1-2 ml im in small ruminants and dogs
- Inj Botropase 0.5-1 ml im daily in dogs.

CHAPTER – 3

DISEASES OF DIGESTIVE SYSTEM - II

General Information

- Vagus nerve regulates stomach motility.
- Hypoglycemia stimulates motility.
- The movements of the reticulorumen are of two types.
  1) Primary contractions (backward moving) that help in mixing the ruminal contents.
  2) Secondary contractions (forward moving) that help in eructation.
- The average volume of gas produced in the rumen of adult cattle may be about 2lit/min.
- Total salivary secretion can be form 90 to 190 lit/dar.
- Cattle usually spend 6-10 hrs/day in rumination.
- The gas mixture in rumen is largely composed of CO_2 and methane (30-40%).
- The gases of fermentation include CO_2, methane and H_2S.

1. RUMINAL TYMPANY (BLOAT)

Definition: "Ruminal tympany is defined as over distension of the rumen caused by excessive accumulation of gases of fermentation either due to excessive production of gas or failure of eructation of gas."

Classification :

A) On the basis of etiology :

Ruminal tympany may be classified as primary and secondary.

	Primary typmany		Secondary tympany
1	It is due to excessive production of gas	1	It is due to failure of eructation of free gas.
2	It is dietary in origin and occur in cattle fed on legume pasture and in feed lot cattle on high protein diet.	2	It is because of physical obstruction in the process of eructation of gas.
3	It is usually frothy type of bloat.	3	It is usually gaseous type of bloat.
4	It is usually peracute to acute in nature	4	It is usually subacute to chronic in nature.

B) On the basis of rumen contents :

1) Froathy bloat : Ruminal contents are froathy in nature.

2) Gaseous bloat : Excess accumulation of gas in rumen.

Etiology :

A) Primary tympany : It is mainly because of following dietary factors.

1. Excessive intake of greens (pasture / leguminous bloat)

- Grazing of green lush pasture (pasture bloat)
- Excess intake of immature leafy leguminous plants. (leguminous bloat)
- Large intake of young cereal forages and vegetables.
- Damp, wet and succulent legumes and immature grasses are bloat provocative as they fail to induce sufficient salivary secretions.
- There is decrease in incidence of bloat with increasing maturity and decreasing protein content of plants.

2. Feeding of large quantities of grain (feed lot / grain bloat)

- Excess intake of soluble carbohydrate viz. finely grounded grains.

- Excess intake of stale left over roti, chapatti or boiled rice.

3. Less intake of dry roughages. i.e. lack of fibrous feed.

4. Sudden change in feed / diet.

5. High calcium and low phosphorus in diet.

Individual difference in susceptibility of bloat may be attributed to :
a) Secretion of small quantity of saliva.
b) Difference in composition of salivary protein.
c) Difference in rumen motility and rumen structure.
d) Difference in process of cellulose digestion.

On the basis of recent research on bloat, the etiological agents are classified in to 3 groups.

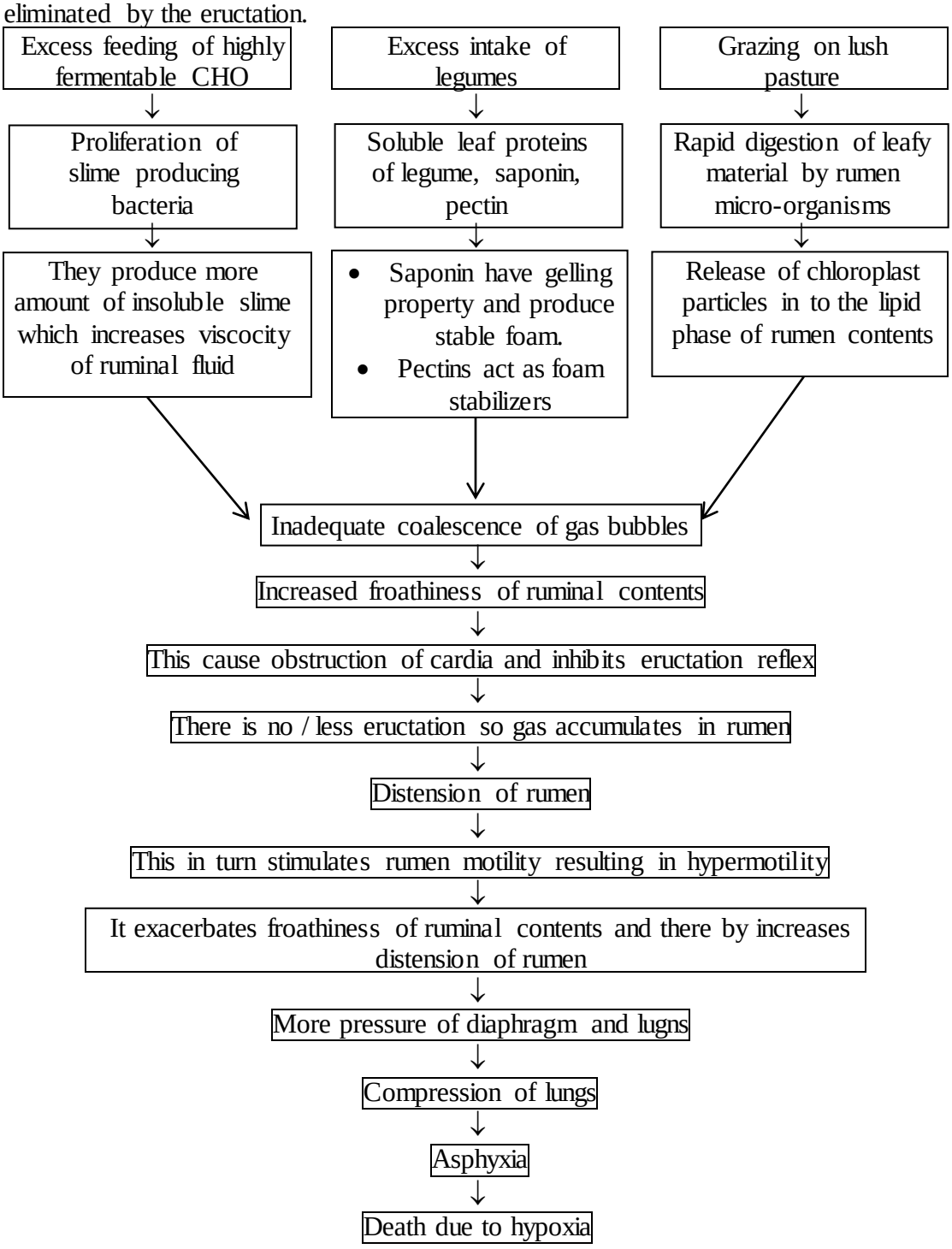
1) **Plant factor :** Plants high in nitrogen, proteins, saponins, pectins and pectin methyl esterase enzyme content.
2) **Animal factor :** Reduced secretion of saliva.
3) **Microbiological factor :** Increased mucinolytic bacteria or slime producing bacteria.

B) Secondary bloat :

1. Oesophageal obstruction
2. Vagus indigestion.
3. Diphragmatic hernia.
4. Traumatic reticuloperitonitis.
5. Reticular abscess.
6. Tetanus due to spasm of oesophagus and inability to eructation.
7. Disease responsible for decreased tone and motility of rumen and decreased rumination viz. fever, toxaemia, milk fever, allergy.
8. Unusal postures like lateral recumbency.
9. **Chemical factors :** Corrosive chemicals, large doses of acids or alkalies cause ruminal paralysis and thus interfere with eructation of gases.
10. **Drug / toxic factors :** Drug like atropine, histamine as well as endotoxins produced in rumen cause stasis of rumen musculature which leads to inhibition of eructation of gases.

Pathogenesis :

Normally, gas bubbles produced in the rumen fluid coalesce, separate from the rumen contents to form pockets of free gas above the level of the contents and are finally eliminated by the eructation.



Symptoms :

1. Enlargement of abdomen particularly in left flank.
2. Anorexia.
3. Abdominal pain – grinding of teeth, kicking at the belly, rolling on ground, restlessness.
4. Dyspnoea – Extension of head and neck, protrusion of tongue.
5. Increase in respiratory and heart rates.
6. Cyanosis of eye mucus membrane.
7. Ruminal hypermotility initially and atony in later stages.

8. Frequent urination and defaecation.
9. Frequent defaecation with passage of scanty faeces, sometimes diarrhoea.
10. Tympanic sound on percussion.
11. Crepitating sound on palpation.
12. On trocarisation, only small amount of gas will be passed in froathy bloat and huge amount of free gas in gaseous bloat.

Diagnosis :

1. History of overfeeding, sudden change in diet etc.
2. Clinical signs – distension of rumen.
3. Examination of ruminal fluid.
4. X-ray examination in non-responsive cases.

Differential diagnosis : In case of sudden death :

1. Snake bite :
a) History of snake bite.
b) Evidence of fang marks.
c) Swelling at the site of bite viz. muzzle, face, legs.

2. Lightening stroke :
a) History of lightening / thunder storm.
b) Singeing of hairs.
c) Death in group of animals.

3. Anthrax :
a) Rise of body temperature.
b) Oozing of tarry colured blood from natural orifices.
c) Blood smear positive for anthrax bacilli.

4. Black quarter :
a) Young cattle are affected.
b) Fever.
c) Crepitating swelling on shoulder / rump / thigh region.
d) Lameness in affected leg.

Treatment :

1. General care :

1. Withheld food and water.
2. Keep the animal with its anterior portion at elevated position to reduce the pressure on diaphragm.
3. Give wooden stick in mouth to promote salivation.
4. Massage of left flank.
5. Give exercise.
6. Remove gas by using trocar-canula or stomach tube or 14-16 guage needle.

2. Give antibloat drugs :

a) Carminative mixture of

- Turpentine oil – 30-60 ml
  +
- Linseed sweet oil - 400-500 ml
- Turpentine oil 5-15 ml per sheep.

} orally in cow

b) Formaldehyde 15-30 ml diluted in water orally.

c) Use of herbal antibloat drugs :

- Timpol powder @ 80 gm dissolved in lukewarm water orally or
- Tyrel liquid 100 ml orally in large animal and 50 ml orally in small animals.

d) Use of anti-foaming agents.

These agents effectively prevent froathy bloat by reducing surface tension of gas bubbles causing them to coalesce. Hence these drugs are preferred in treatment of froathy bloat.

- Silica compounds e.g. Bloatosil (Dimethicane suspension) @ 50 – 100 ml orally.

e) Poloxalene (Polyoxyethylene polyox) @ 10-20 gms/500 kg body weight orally.

3) Antibiotics : Give antibiotics to prevent bacterial fermentation.

e.g. oxytetracycline @ 15-20 ml intraruminally or

tetracycline bolus 4-8 orally.

4) Antihistaminics :

e.g. Inj.Avil/Cadistin @ 5-10ml I/m

5) Correction of rumen pH (Normal pH – 6.3 – 7.0)

Rumen pH is corrected by using acidifiers or alkaliesers.

6) Cud transplantation.

7) Rumenotomy : In severe / non-responsive cases.

2. SIMPLE INDIGESTION

Defination : "It is simple digestive disturbance clinically characterized by inappetance to anorexia, ruminal atony and abnormal faeces. (Scanty / voluminous)

Etiology :

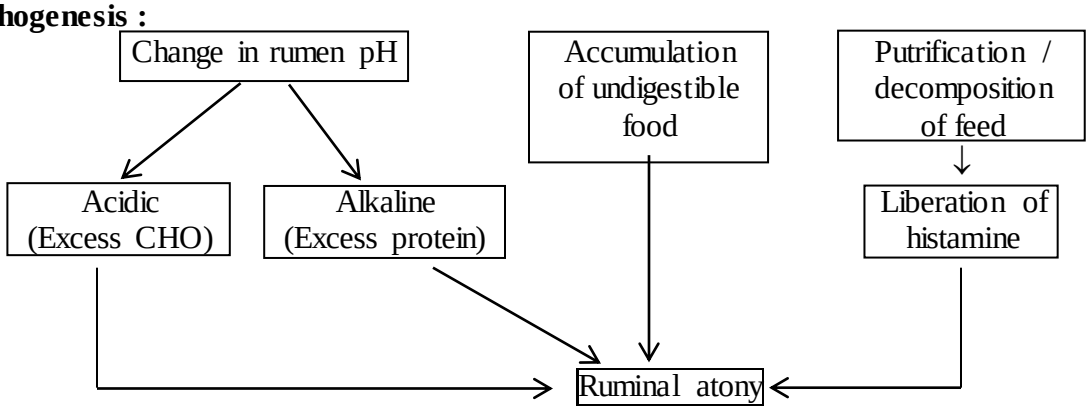
A) Dietary causes :

1. Overfeeding of grains, legumes, protein rich diets, silage.
2. Sudden change in quantity and quality of feed.
3. Ingestion of large amount of indigestible coarse roughages.
4. Feeding of moldy or decomposed feed.
5. Consumption of overheated or frosted feeds.
6. Drinking of drainage or sewage water.
7. Non – or – less availability of drinking water.
8. Prolonged or heavy oral dosing with sulfonamides or antibiotics cause inhibition of normal ruminal microflora.

B) Animal factors :

1. Greedy feeding.
2. Depraved appetite i.e. pica.
3. Improper / incomplete mastication.
4. Old age, dibility and weakness.
5. Transportation, heavy work and exertion.

Pathogenesis :



Clinical signs :

- Partial or complete anorexia.
- Fall in milk yield.
- Mild depression and dullness.
- Rumination is suspended.
- On palpation rumen is usually firm and doughy sometimes distended.
- Rumen motility is reduced in strength and frequency.
- Faeces initially dry and scanty, lateron voluminous and soft.
- Sometimes mild abdominal pain.
- Temperature, respiration and pulse normal.

Diagnosis :

1. History of over feeding, change in diet.
2. Clinical Sign : Ruminal atony with normal body temperature.
3. Rumen function tests – pH in between 5-7.5 (normal 6.5 - 7)
    1. Cellulose digestion time is more than 30 hrs.

Treatment :

1. Correction of ruminal pH to reactivate ruminal microflora.
    a) If pH is acidic, give alkalizer like sodium bicarbonate.
    b) If pH is alkaline give acidifier like vinegar 5-10%. 200-500 ml orally.
2. Use of parasympathomimetic drugs to induce peristalsis.
    e.g. Neostigmine @ 0.02 mg/kg body weight. Im/Sc.
    Physostigmine 30-45 mg/ animal SC
    Carbachol 1-3 mg/ animal SC
3. Use of rumenotoric durgs to improve ruminal motility.
    e.g. Nux-vomica, Ammonium chloride.
    Antimony potassium tartrate 8-10 gm/cow orally.
    Bolus Rumentas / Rumenton / Bovirum. @ 2-3 orally daily for 2-3 days.
4. A course of B complex with liver extract parenterally.
5. Fluid therapy to combat dehydration.
6. Rumen cud transplantation.
7. Rumenotomy if there is no improvement.
8. Massage of left flank from downward to upward.
9. Give exercise.
10. Withheld feed for 24 hrs. and then give laxative diet.

3. ACIDIC INDIGESTION

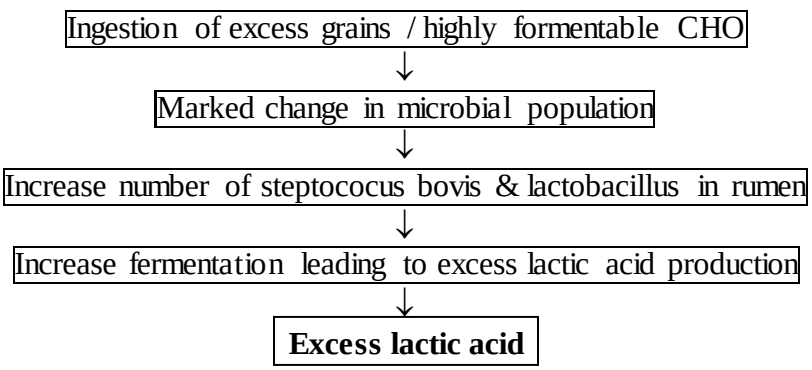
(Rumen overload, Acute carbohydrate engorgement Grain overload, Ruminal acidosis, Founders)

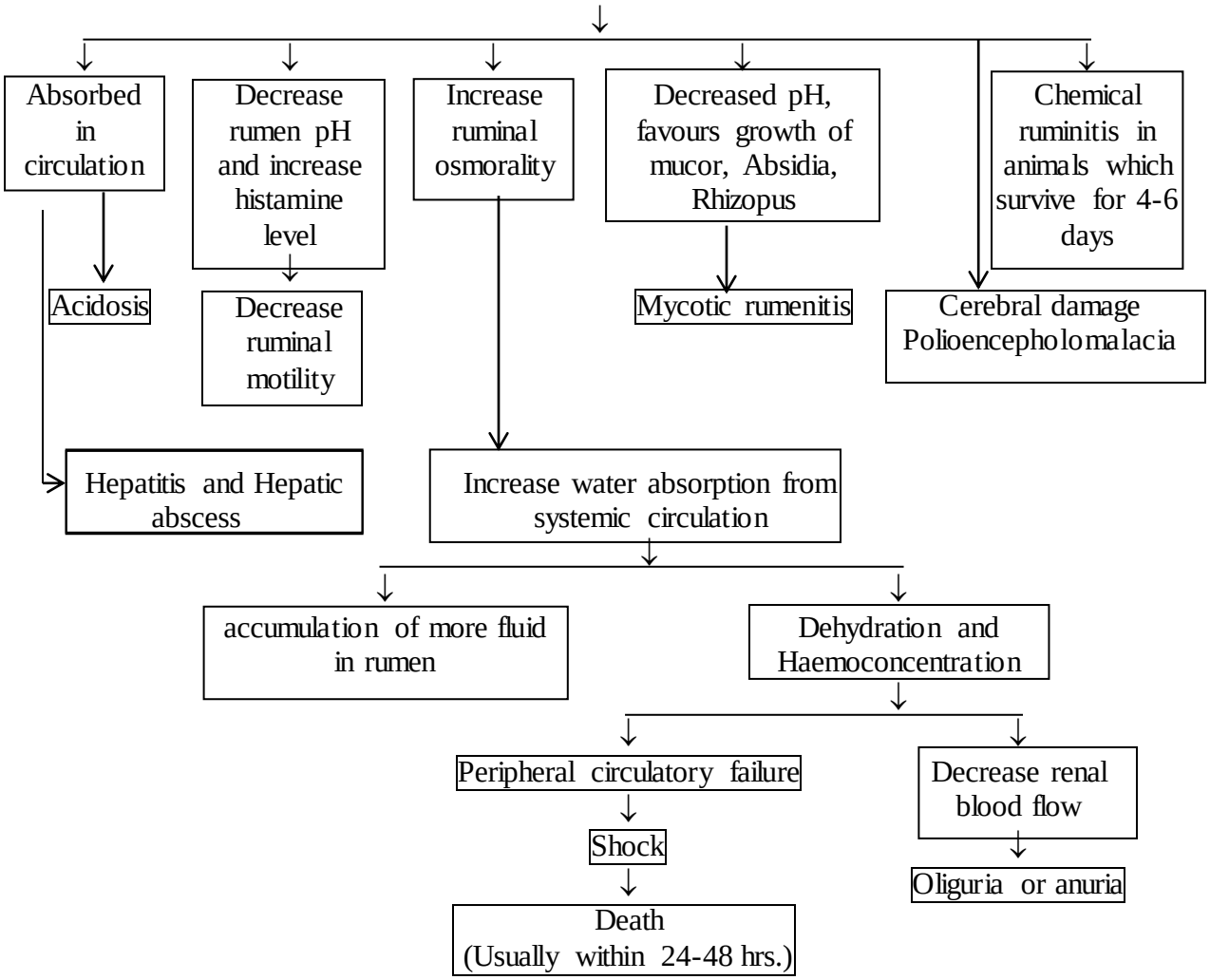
Definition : "It is an acute ruminal dysfunction caused by ingestion of large amount of carbohydrate rich feeds and clinically characterized by severe toxaemia, dehydration, ruminal stasis, weakness, recumbency and high mortality."

Etiology :

1. Accidental ingestion of excess amount of cereal grains.
    viz. jowar, rice, wheat, oat, maize.
2. Overfeeding of whole or crushed grains.
3. Sudden chage in nature of grains.
4. Feeding of left over roti, chapatti or rice.
5. Ingestion of apples, grapes, sugar beet, carrot, potato.
6. Excess feeding of sugar molasses.
7. Ground powdered grain produce more acid than whole or partially crushed grains.
8. Feeding of cereal grains @ 25-62 g/kg produces severe acidosis.

Pathogenosis :





Clinical signs :

- The speed of onset of signs is faster with ground feed than whole grains.
- The severity of signs increases with the amount of feed eaten.
- Anorexia.
- Suspended rumineation.
- Decrease amplitude and frequency of ruminal motility and later on complete atony.
- Rumen is distended with fluid in mild to moderate acidosis however, it is firm and doughy in cattle which have consumed large amount of grains.
- Gurgling fluid sound from rumen.
- Abdominal pain
  - locking at flank
  - kicking at belly
  - grinding of teeth.
- Faeces – hard, dark, mucus, coated, scanty in early stages usually loose and voluminous (diarrhoeic) in later stages.
  - They are light coloured with sweet sour odour.
  - They may contain coat or kernels of grain.
- Dehydration – Sunken eyes and supraorbital fossa.
  - Dry muzzle
  - Increase skin fold test.
- Oliguria and anuria.
- Subnormal body temperature (98^0F – 101^0F)
- Tachycardia (70-130 / min)

- Respiration usually shallow and rapid. (60-90 / min)
- Severe depression, staggering, recumbency (milk fever like posture) in severe cases.

Diagnosis :

1. History of access to grains.
2. Clinical Signs : Anorexia, depression, ruminal stasis, abdominal pain, dehydration nad staggering gait.
3. Clinical pathology.
   a) Ruminal fluid : i) pH – 5 or below 5
   mild – 5.5 to 6.5
   moderate – 4.5 to 5.5
   severe - 4.0 to 4.5
   ii) Ruminal fluid : colour - milky grey
   odour – sour.
   iii) Absence of ruminal protozoa.
   iv) Bacteria : Gram –ve replace by Gram +ve bacteria (Normally Gram –ve are predominant)
   b) Urine : pH – acidic / lowred (5.0)
   proteinuria.
   c) Haematology – High PCV increase from 30% to 50-60%.
   d) Serum biochemistry : Increase BUN and serum phosphate – due to renal failure
   - Increase blood lactate.
   - Increase bicarbonate.
   - mild hypocalcaemia – due to temporary malabsorption.

Differential diagnosis :

1. TRP
   a) Long course
   b) Fever
   c) Tucked up abdomen
   d) X-ray positive for foreign body.
2. Milk fever
   a) History of parturition.
   b) Hypocalcemia
   c) Response to calcium therapy
3. Nervous form of acetonemia / ketosis :
   a) History of lactation.
   b) Sweetish smell to breath, milk, urine.
   c) Hypoglycemia
   d) Rothera's test of urine positive.
   e) Response to glucose therapy.
4. Hepatic insufficiency
   a) Jaundice
   b) Liver function test positive

Treatment :

1. Correction of ruminal pH by giving alkalizer e.g. sodium bicarbonate, magnesium hydroxide and magnesium oxide orally.
2. Correction of fluid and electrolyte imbalance by giving isotonic sodium bicarbonate (1.3%) and balanced electrolyte solutions (Ringer's solution).
   Severe acidosis – Sodium bicarbonate @ 5 liter I/V in 30 min. followed by isotonic balanced fluids @ 150 ml/kg for 6-12 hrs.
3. Use of oral antibiotics to control growth of bacteria which lactic acid e.g. tetracycline tablet 4-8 orally.
4. Calcium borogluconate @ 200-300ml i/v for 2-3 days to combat mild hypocalcemia.
5. Liver extract with B-complex vitamins @ 5ml I/M daily for 3-5 days.
6. Oral ruminotorics to restore motility of rumen. e.g. Bovirum bolus 2-3 orally daily for 2-3 days.
7. Parasympathomimetic to stimulate gut motility e.g. Neostigmin @ 0.02 mg/kg I/M.
8. To promote the metabolism of lactic acid give e.g. Thiamine HCl @ 2-4 g I/V.
9. Antihistaminics and corticosteroids to prevent shock. e.g. Anistamin @ 0.25 – 0.5 mg/kg I/M, Dexona @ 0.04 mg/kg I/M
10. Cud transplantation @ 10-20 lit to replenish ruminal microflora.
11. Give exercise every hour for 12 to 24 hrs to encourage movement of ingesta through digestive tract.
12. Ruminal massage from downward to upward.
13. Withheld food and water for 12-24 hrs followed by laxative diet.
14. In severe case rumenotomy is done.

Prevention :

1. Small amounts of grain (8-10g/kg) which is increased every 2-4 days by increments of 10-12%.
2. Monensin, losalocid and salinomysin may be used in feed to prevent acidosis.
3. A new approach is to inoculate unadopted cattle with ruminal fluid from adopted animals which contains a large population of lactic acid utilizing bacteria.

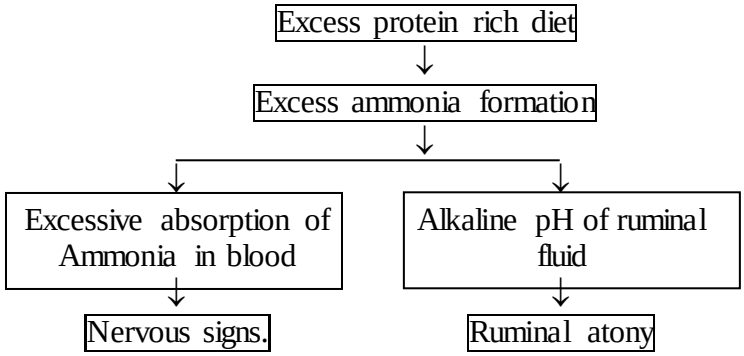
4. ALKALINE INDIGESTION

Defination: "It is caused by ingestion of protein rich feeds and characterized by anorexia, depression, alkaline rumen pH and atony of rumen."

Causes :

1. Ingestion of protein rich concentrates – GNC, soybean.
2. Ingestion of leguminous forages like Lucerne, berseem.
3. Sudden change from carbohydrates to proteins.
4. Exclusive feeding of paddy straw for long period.
5. Feeding of urea.

Pathogenesis :



Clinical signs :

- Inappetance to anorexia.
- Ruminal motility decrease in frequency of amplitude.
- Rumination suspended.
- Passage of semisolid faeces.
- Increase pulse and respiratory rate.
- Abdominal pain.
- Tremor, ataxia and convulsion.

Diagnosis :

1. History
2. Clinical signs.
3. Rumen fluid
   - pH - Increase
   - Colour – Dark brown
   - Odour - Ammonical
4. Blood – Increase BUN 15 mg% to 70 mg%.

Treatment :

1. Correction of ruminal pH. e.g. Venegar (5% acetic acid) @ 5-10ml/kg orally.
2. Oral antibiotics e.g. Oxytetracycline tab @ 4-8 orally for 2-3 days.
3. Rumenotorics. e.g. Bovirum bolus @ 2-3 orally for 2-3 days.
4. Fluid therapy. e.g. DNS/NS
5. Cud transplantation (Fresh ruminal ingesta)
6. Liver extract with B- complex vitamin. e.g. Belamyl @ 5ml I/M

5. TRAUMATIC RETICULO – PERITONITIS

Definition : "It is caused by sharp pointed foreign bodies and characterized by anorexia, mild fever, ruminal stasis and subacute abdominal pain."

Incidence :

1. Species : Disease incidence is high in cattle and buffalo because of lack of oral discrimination between food and other substances due to their indiscriminate feeding habbit.
   - Buffaloes are more susceptible than cattle.
   - This disease has also been reported in sheep and goats.
2. Age : Adults animals are more susceptible than young animals.
3. Sex : Disease is more common in females than males.
4. Milk yield : High yielding animals are more susceptible than low yielders.

Etiology :

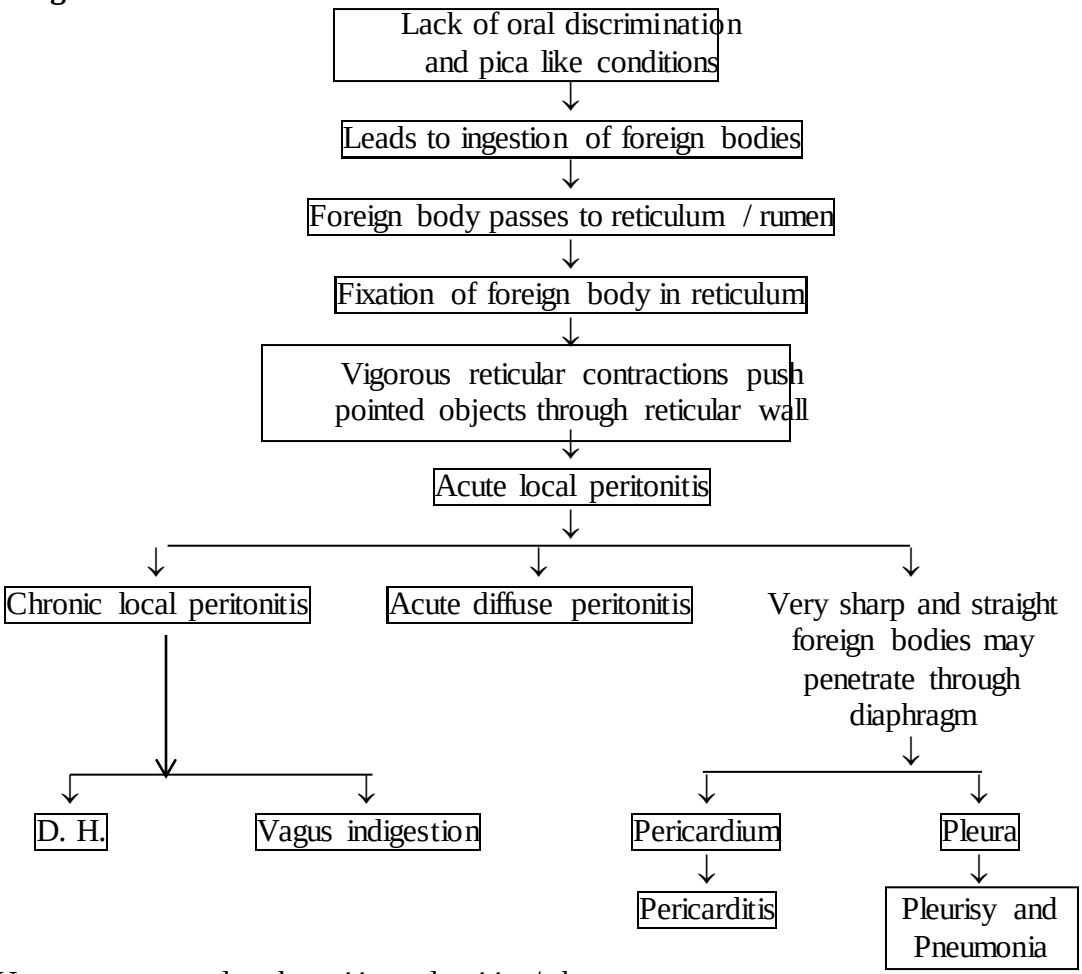
A) Exciting causes :

Accidental ingestion of foreign bodies viz. metallic wires, nails, sewing needles through feed or while grazing in pasture.

B) Predisposing factors :

1. Pica.
2. Tendency to lick metallic objects (Iron managing)
3. Greedy feeding.

Pathogenesis :



- Uncommon sequelae, hepatitis, spleenitis / abscess.

Clinical signs :

- Inappetence to Anorexia.
- Fall in milk yield in lactating animals.

- Suspended rumination.
- Rise in body temperature (usually mild to moderate)
- Tachycardia.
- Shallow respiration accompanied by expiratory grunt.
- Ruminal motility is weak and reduced / absent.
- Recurrent typany or mild to moderate chronic tympany or sometimes ruminal impaction.
- Subacute abdominal pain – characterized by
    - Immobility (prominent sign of disease)
    - Animals walk slowly particularly downhill and walking is accompanied by grunting.
    - Most animals prefer to remain in standing for long period and lie down with great care.
    - Arching of back and abducted elbows.
    - Animal appears gaunt or lucked up due to rigidity of back and abducted elbows.
    - Animal appears gaunt or tucked up due to rigidity of back and abdominal muscles.
- Constipation or scanty faeces is common.
- Pain on deep palpation of the abdominal wall just behind xiphoid process.

Diagnosis :

1. History of pica
2. Clinical signs
    - Recurrent / chronic tympany
    - Mild fever
    - Subacute abdominal pain
3. Slight, transient or no response to antibiotic therapy.
4. Clinical pathology – Leucocytosis with neutrophilia.
5. Radiological examination : Evidence of foreign body.
6. Right flank laproscopy.
7. Back pinch test.
8. Peritoneal fluid analysis.

Differential Diagnosis :

1. Simple indigestion :

- Acute onset
- No fever
- History of overfeeding or change in diet
- Good response to treatment with rumenotorics.

2. Primary tympany :

- Acute in nature.
- History of feeding of greens and leguminous plant.
- No fever.
- Good response to treatment with antibloat drugs.

3. Ketosis :

- Occurs during early lactation in high yielding females.
- Absence of fever and pain
- Ketonemia / ketonuria
- Good response to dextrose therapy.

4. Pyelonephritis :

- History of uterine infection in females.
- Urine is abnormal and may contain pus, flakes, etc.

5. Traumatic pericarditis :

- Abnormal sound i.e splashing / dull sound on auscultation of cardiac area.
- Brisket oedema, jugular pulsation.

6. Pneumonia :

- Coughing, nasal discharge
- Abnormal lung sounds on auscultation.

7. DH :

- Chronic moderate frothy bloat
- Reticular sound just behind cardiac area.
- Evidence of herniation on the Radiography.

Treatment :

A) Conservative treatment :

This is of little value

i) Immobilization or confinement of animal for several days to facilitate adhesion formation.
ii) Administration of antibiotics to control the infection.
iii) Oral administration of a magnet to immobilize the foreign body.

B) Rumenotomy :

This is the best and only way to treat cases of TRP.

Peritoneal fluid analysis

Parameters	**Healthy**	**TRP**
pH	7.55	8.42 ± 0.68
TLC	3225.00 ± 322.77	4333.33± 569
DLC – Neutrophil	2303.90 ± 304.55	29991.67 ± 367
Lymphocyte	907.90± 069.41	1341.67± 210
Eosinophils	9.30 ± 9.30	Nil
Monocytes	4.40± 4.40	Nil
Total proteins (g/dl)	1.96± 0.19	3.47± 0.22

6. DIAPHRAGMATIC HERNIA

Definition : "Herniation or protrusion of a portion of reticulum through diaphragmatic rupture."

- DH may be right or left sided.
- Right sided DH is more common than left sided DH.

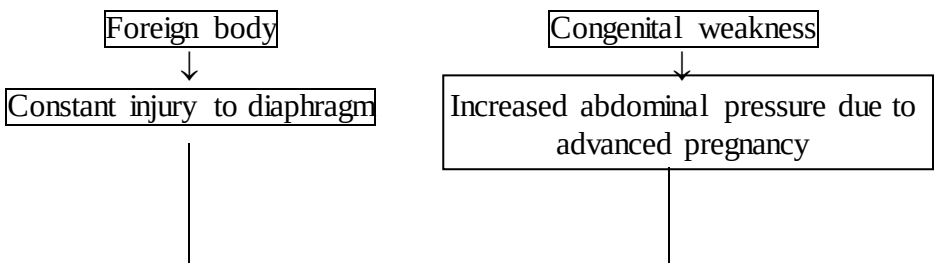
Etiology :

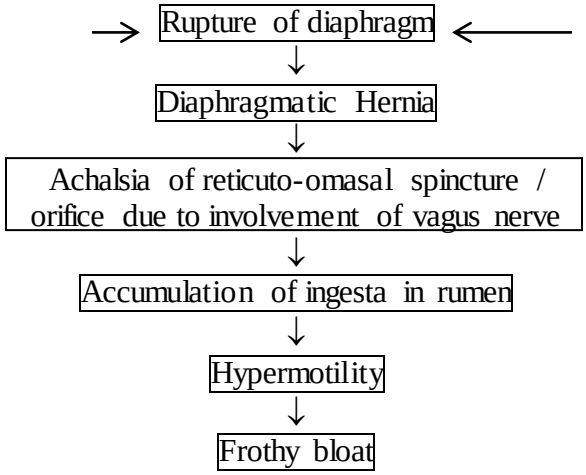
1. Weakening of diaphragm by foreign body.
2. Congenital weakness of diaphragm.

Incidence :

1. **Species :** Disease incidence is higher in buffaloes as compared to cattle due to high incidence of TRP and congenital weakness of diaphragm in buffaloes.
2. **Age :** Adult animals are mostly affected.
3. **Sex :** Females are more susceptible than males.
4. **Milk yield :** High yielding buffaloes either during advanced stage of pregnancy or immediately after parturition are more affected.

Pathogenesis :





Note : Syndrome produced is identical with that caused by vagus indigestion.

Clinical Sings :

1. Persistent moderate frothy bloat.
2. Rumen hypermotility.
3. Progressive reduction in appetite.
4. Progressive loss of body weight.
5. Faeces are scanty.
6. Temperature and respiration usually normal.
7. Heart rate is decreased (brady cardia)
8. Reticular sounds are audible just posterior to cardiac area in right sided DH.

Diagnosis :

1. History of advance pregnancy, recent calving, pica etc.
2. Clinical signs :
    - Persistent moderate frothy bloat
    - Rumen hypermotility
    - Reticular sounds
    - Loss of body weight etc.
3. No/slight response to conservative treatment.
4. Radiographic examination.
5. Exploratory laprotomy.

Prognosis : It is always poor.

Treatment :

Surgical correction of herniation.

a) Herniorraphy OR
b) Hernioplasty

by

a) Transabdominal approach OR
b) Thoracic approach.

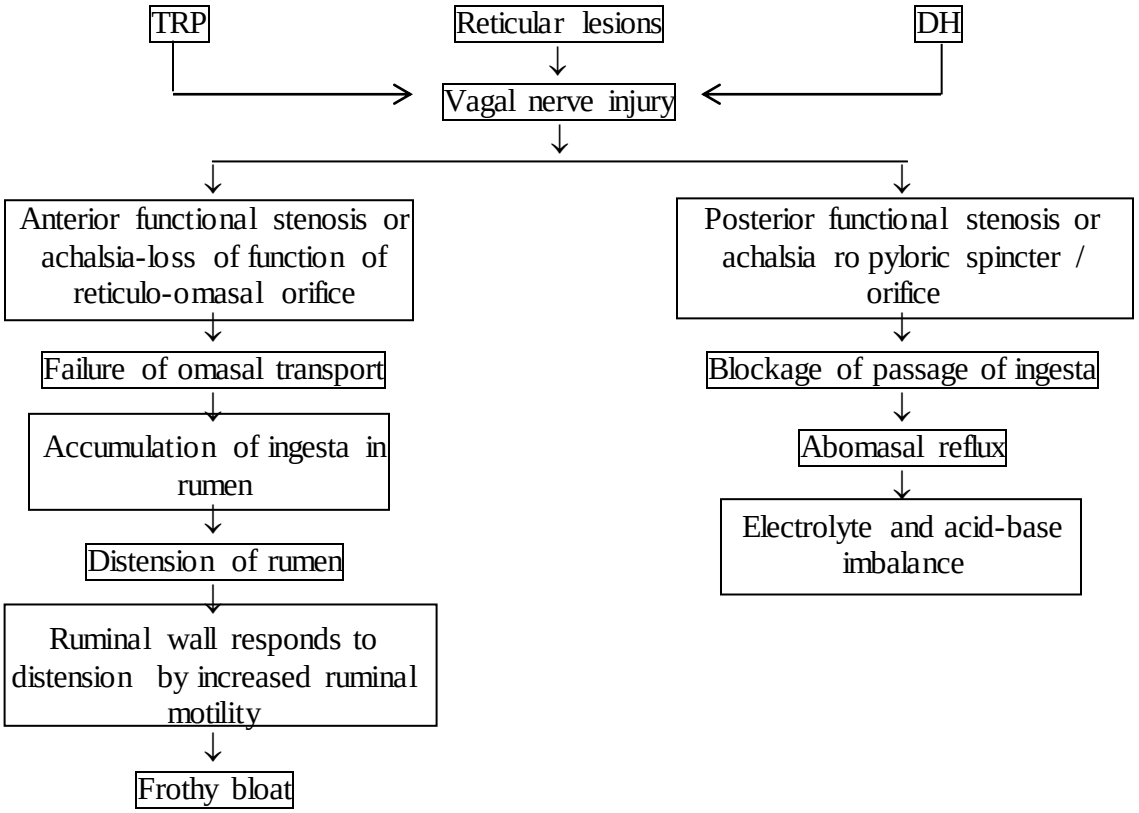
7. VAGUS INDIGESTION
(Hoflund syndrome)

Definition : "Vagus indigestion is a chronic disease of the forestomach and abomasum characterized by chronic frothy bloat, papple shaped abdomen, decreased appetite, scanty faeces and weight loss."

Incidence : It is most common in buffaloes and cattle and has been recorded in sheep.
Etiology : Vagal nerve injury or dysfunction may occur due to –

1. Traumatic reticulo-peritonitis.
2. Diaphragmatic hernia and adhesions of reticulum to diaphragm.
3. Reticular lesions – abscess, cyst, tumor, actinobacillosis
4. Tubercular abscess, lymphomatosis, enlargement of mediastinal lymphnodes exerting pressure on vagus nerve.

Pathogenesis :



Clinical signs:

- Reduction in appetite.
- Progressive loss of body weight
- scanty, pasty faeces.
- Chronic moderate to severe frothy bloat.
- Papple shaped distension of abdomen i.e. apple shaped on left and pear shaped on right. The upper left abdomen is bilaterally distended.
- Ruminal motility is increased (3-6 /min) – Hypermotility.
- Ruminal contractions are visible and palpable as waves of undulation of the left flank.
- Ruminal contractions may not be audible because of the ruminal contents are homogenous and porridge like due to prolonged maceration in the rumen.
- Large L shaped rumen on rectal palpation.
- Temperature is usually normal.
- Bradycardia may be present – Ruminal distension
  - Compression of heart
  - Parasympathomimetic effect.
- Weakness
- Dehydration

- Recumbency

Diagnosis :

1. Chronic frothy bloat, hypermotility
2. X-ray examination.
3. No response to conservative treatment.
4. Laparotomy.

Prognosis of vagus indigestion is always poor.

Differential diagnosis :

1. **Left abomasal displacement :**
   - i) History of calving.
   - ii) Contracted abdomen.
2. **Intestinal obstruction :** Usually no passage of faeces.
3. **Diaphragmatic hernia :** Splashing reticular sound on ventral thorax.

Treatment :

There is no effective treatment, however the following line of treatment may be used.

1. Evacuation of rumen contents by
   - a) Using stomach tube
   - b) Administration of mineral oil-liquid paraffin 2.5 lit orally for 2-3 days.
   - c) Rumenotomy.
2. Nervine tonics Inj B1+B6+B12 daily to stimulate rumen musculature.
3. Use of parasympathomimetics, Neostigmin @ 0.02 mg/kg body wt I/m
4. Fluid therapy and electrolyte therapy – DNS 2.5 lit. i/v daily till recovery.
5. Use of calcium preparation to increase muscle tone.
6. Removal of cause viz. foreign body, abscess, tumor.

❍ ◆ ❍

8. ABOMASAL DISPLACEMENT

- Abomasum is movable organ because of this the organ is easily displaced from its original position.
- The displacement may be on left or right side.
- Left sided displacement (LDA) is more common (80%) than right sided dipsacement (RDA) (15%).

Incidence :

1. It usually occurs in large sized, high producing dairy cows immediately after parturition.
2. It is more common in exotic cattle. (HF cattle)
3. It is less frequent in indigenous cattle.
4. Lactation : 3-4 th
5. Age : 4-5 years.

Causes :

a. Hypomotility of abomasums in cattle fed on more concentrate and less fibre.

- Parturition is an important predisposing factor.
- Hypocalcaemia is also important contributing factor.

Pathogenesis :

Heavy feeding of grains / concentrates

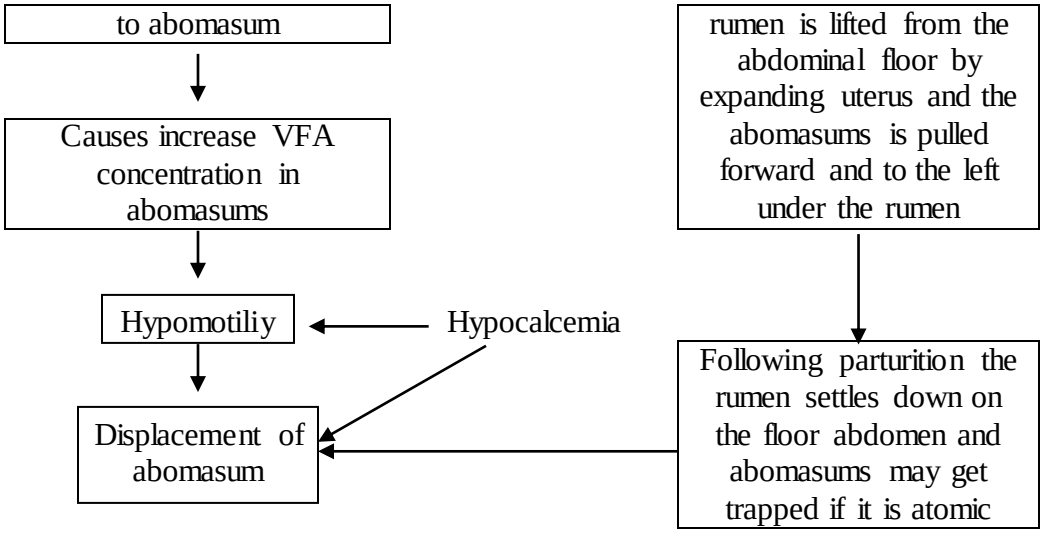
↓

Increased flow of ingesta

Parturition is important predisposing factor.

↓

During late pregnancy,



Hypocalcemia is also important contributing factor in left abomasal displacement.

Clinical Sings :

- Inappetence to complete loss of appetite within few days or weeks after parturition.
- Marked drop in milk production.
- Scanty, pasty, loose faeces usually but sometimes diarrhoea.
- Left abdomen is usually slab sided because rumen is smaller than normal and displaced medially.
- Ruminal movements decrease in intensity and frequency.
- Temperature, respiration and pulse – within normal range.
- Auscultation of an area below a line from centre of the left paralumbar fossa, to just behind the left elbow (9^{th} – 12^{th} intercostal space) reveals the presence of abomasal sound – splashing / tinkling sounds.

Diagnosis :

1. History of parturition, high milk yield, Age 4-5 years, 3^{rd} – 4^{th} lactation.
2. Clinical signs
3. Clinical pathology : Ketonuria, hypocalcaemia.

Differential diagnosis :

a) Intestinal obstruction : No defaecation

b) Vagus indigestion : Chronic froathy bloat.

Treatment :

- Conservative treatment is not effective.
- Rolling the animal on its back and massage of anterior part of abdomen form left to right.
- Symptomatic treatment with dextrose and antibiotics.
- Surgical correction (abomasoplexy i.e. fixing of abomasum to the wall) will prevent the recurrence of the condition.

9. COLIC

Definition : "Colic involves all types of acute abdominal pain."

It occurs in all animal species but it is most common in horses.

A) Classification on the basis of origin of pain :

1) True colic : Pain in abdomen originating from GI tract is known as true colic.

2) False colic : Abdominal pain originating from organs other than GI tract is known as false colic e.g. liver, kidney, uterus, peritoneum.

B) Aetiological classification :

1) Physical colic : Pain due to presence of physical agents like stones, sand, feed mass / bolus, enteroliths etc in GI tract.

2) Functional colic : Pain because of altered function of GI tract due to some infections (virus, bacteria, parasites) irritant chemicals and plant poisonings.

C) Clinical classification :

1) Tympanic / Flatulent colic : Pain due to excessive accumulation or over production of gases in stomach. (gastric / ruminal tympany) or intestine (intestinal tympany) It results from feedings of easily fermentable feed stuffs.

2) Spasmodic or Idiopathic Colic : Pain due to spasmodic contraction of GI tract. It usually occurs due to irritation caused by unsuitable feed stuffs, greedy feeding, drinking of cold water after heavy work, excitement due to thunder storm or lightening. This is frequently observed in nervous temperament group of horses.

3) Impactive Colic : It is because of impaction of GI tract due to impacted food mass, stones, sand and other foreign bodies. Impaction may be observed in stomach or large intestine.

4) Extraluminal Colic : It is because of obstruction of lumen from outside of bowel due to intussuception, volvulus / torsion, strangulation and hernia.

5) Duration of disease :

1) **Acute :** Above 24 – 36 hrs.
2) **Chronic :** Below 24 – 36 hrs.
3) **Recurrent :** Multiple episodes separated by periods > 2 days of normality.

Etiology :

1) Dietary causes :

i) Overfeeding.
ii) Feeding of poor quality roughages / grains.
iii) Sudden change in feed.
iv) Excessive feeding of bran in horses.
v) Feeding of mouldy straw or rotten food material.
vi) Ingestion of irritant chemicals / poisonous plants.
vii) Ingestion of sand, stones and other foreign bodies.
viii) Insufficient drinking water.
ix) Drinking of excess quantity and cool water after heavy work.

2) Animal factors :

i) Greedy feeding.
ii) Bad teeth – improper mastication.
iii) Nervous temperament of animals.
iv) Exhaustion.
v) Old age and debility.
vi) Feeding after race in horses.
vii) Autonomic imbalance.

3) Environmental factors :

i) Excitement by lightning / thunderstorm.
ii) Placing in unaccustomed environment.
iii) Weather change – exposure to cold.

4) Infective factors :

i) Bacteria – Salmonellosis, colibacillosis.
ii) Viruses – Equine viral arteritis.
iii) Fungus – Feed infested with aspergillus and condida.
iv) Parasites – Strongylosis, Ascariasis, Gastrophilus larve (bots).
v) Protozoa – Cocidiosis.

5) Mechanical factors :

i) Volvulus / Torsion – Twisting of intestine.

ii) Intussuception – Telescoping of intestine, invagination of one part of intestine into other part.

iii) Hernia.

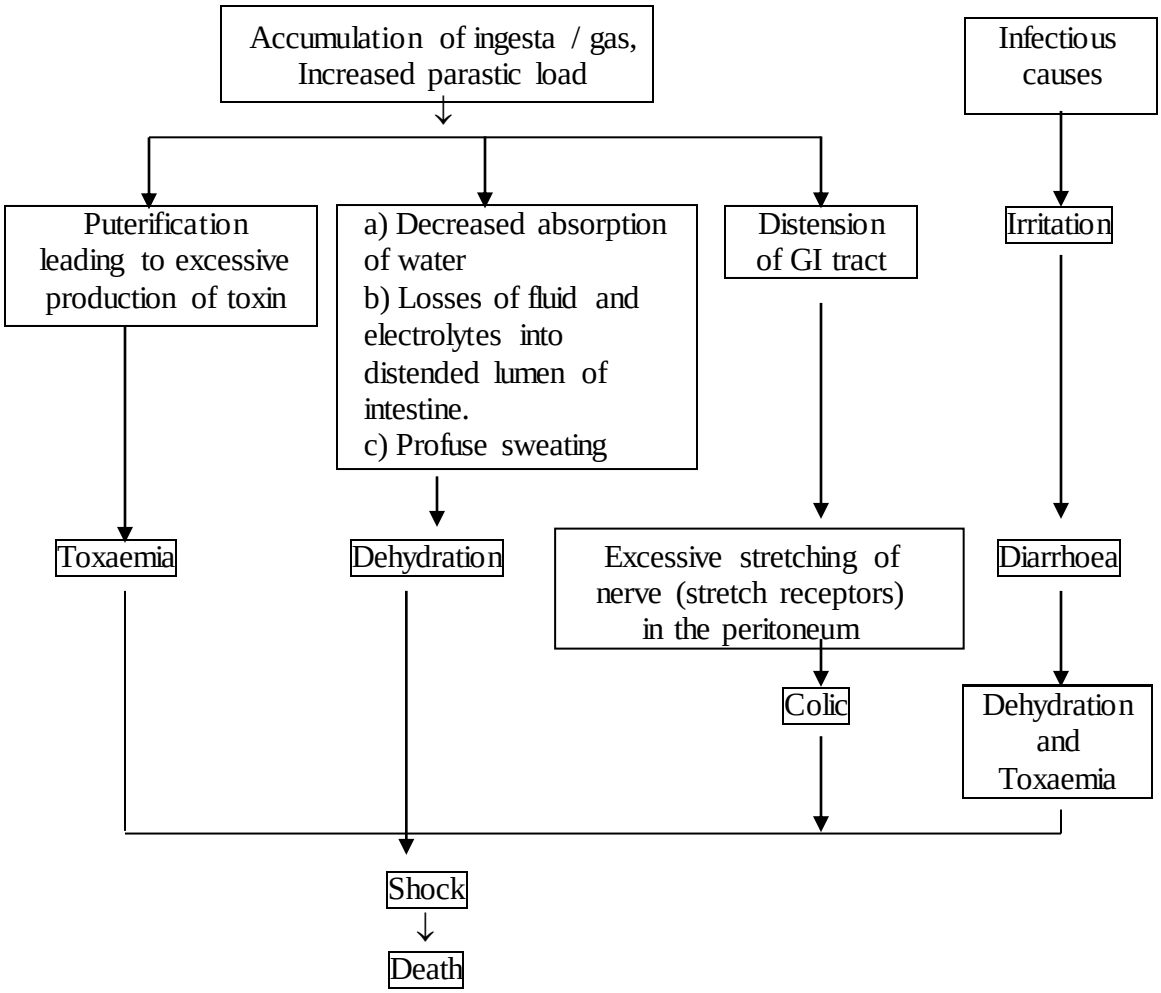
iv) Strangulation.

v) Enteroliths.

vi) Peritoneal adhesions.

vii) Caecal dilatation and torsion.

Pathogenesis :



Clinical Signs :

1. Sudden / acute intense attack of pain.
   - Animal shows restlessness, kicking at the belly, rolling on ground, pawing, stamping.
   - Looking at flank.
   - Animals repeatedly lie down and gets up.
   - Pain is intermittent in Spasmodic colic and continuous in tympanic and other type of colic.
2. Distension of abdomen.
3. Diarrhoea / constipation.
4. Signs of anorexia and dehydration.
5. Oligouria / anuria, painful micturition.
6. Rise in body temperature, rapid pulse rate and deep breathing.
7. Lip curling (Flehman response) and profuse sweating all over the body particularly in horses.
8. Mucus membranes are congested / cyanotic.
9. Intestinal sound is increased or may be absent / reduced in impaction.

Diagnosis :

1. History of overfeeding change in feed , feeding of poor quality roughages, reduced water intake.

2. Signs like restlessness, kicking at belly, rolling on ground, frequent sitting and getting up.
3. Auscultation and percussion :
   - It gives idea about motility and accumulation of gas, fluid in it. The most critical area for auscultation is high up in the right flank over the dorsal sac of caecum.
   - Tympanic colic metallic tinkling sound.
   - Spasmodic colic and enteritis – continuous borborygmi.
   - Impaction / paralytic ileus – absence of sounds.
4. Rectal examination : Rectal palpation should be done for displacement and distension. It may be due to fluids, faeces and foreign body. The empty rectum with a smear of mucous and blood indicated complete intestinal obstruction.
5. Abdominocentesis : Collection of fluid for colour, odour, opacity, lab. examination of total protein estimation, WBC, TLC, Staining for bacteria.
6. Clinical pathology : Increase PCV, total plasma protein, estimation of PCV and TPP will help in assessment of severity of dehydration and shock.

Prognosis :

1. Pulse rate less than 60 / min – favourable.
2. Pulse rate ranging between 80 – 100 / min – Guarded.
3. Pulse rate greater than 100/min – unfavourable.

Treatment :

1. Hydration therapy : Replacement of fluid lost – depending upon dehydration fluid should be given.
2. Relief from pain to prevent self inflicted injuries due to production in colicky horses.
   i) Use of pain killers :
      a) Inj. Novalgin @ 20-60 ml i/m or
      b) Inj fortwin (pentazocin) @ 0.5 – 1 mg/ kg i/v
      c) Dyprone @ 10mg/kg body weight.
         i) The combination of pentazocin @ 0.6mg/kg i/v and Analgin is commonly used in many stud farms.
         ii) Use of antispasmodic ex. Inj. Cyclopam/ spasmovet @ 0.5 mg/kg i/m.
         iii) Use of tranquilizers / sedatives in severe cases of colic e.x. Inj. Siquil @ 0.1 mg/kg i/v, i/m, in cattle.
              0.2 – 0.3 mg/kg i/v, i/m in horses.
              Inj. Largactil @ 1-3 mg/kg i/m, i/v
              Chloral hydrate @ 30-60 gm orally.
3. Lubrication and purgation : In impaction faecal softening agents such as
   a) Mineral oil ex. liquid paraffin – 500 - 1000 ml orally.
   b) Dioctyl sodium sulfosuccinate @ 10-20 mg/kg
      1-2 lit warm orally.
   c) Mild soap water enema.
   d) Use of parasympathomimetics : Neostigmine @ 0.02 mg/ kg S/C.
4. Corticosteroids to prevent shock : Inj. Dexona @ 0.5 – 1 mg/kg I/M, I/V.
5. Use of antihistaminics : Inj. Avil @ 0.5-1 mg/kg i/m.
6. A course of oral antibiotics to stop bacterial fermentation in GI tract.
   a) Sulfa trimethoprim @ 15 mg PO BID.
   b) Metronidazole @ 15 mg/kg body weight i/v.
   c) Ciprofloxacin @ 10 mg/kg PO, BID.
7. Use of antihelmintics : To remove endoparasites, particularly in chronic intermittent colic (Verminous arterits)

ex.: Fenbendazole @ 10 mg/kg orally, ivermectin @ 0.2 mg/kg S/C.

8. Witheld food for 12-24 hrs.

Prevention :

1. Regular deworming to control endoparasites.
2. Avoid excess feeding of very coarse hay/ straw.
3. Avoid over feeding of grains / brans.
4. Avoid sudden change in type of feed.

PRINCIPLES OF TREATMENT IN ALIMENTARY TRACT DISORDERS

A) Specific treatment :

Correction of primary causes :

1. Antibiotics
   - e.g. Gentamycin @ 2mg / kg i/m, i/v
     Enrofloxacin @ 2.5 – 5 mg/kg i/m
2. Antihelmintics
   - e.g. Albendazole @ 5-10 mg/kg orally.
     Fenbendazole @ 5-10 mg/kg orally
3. Antifungals
   - e.g. Griseofulvin @ 10-20 mg/kg orally daily for 3-4 days.
     Ketoconazole @ 5-10 mg/kg orally daily.
4. Cocidiostats
   - e.g. Sulphadimidine @ 150 mg/kg PO for 5 days.
     Furazolidone @ 10 mg/kg orally for 5 days.

B) Supportive and symptomatic treatment :

1. Correction of abnormal motility

i) When motility is increased then give –

a) Antispasmodics :
   e.g. Inj. atropine sulphate @ 0.03 – 0.06 mg/kg, i/m, s/c.
   Inj. Dicyclomin @ 0.5 mg/kg i/m

b) Use of antidiarrhoeals / astringents.
   e.g. Neblon powder @ 30-50 gm orally.

ii) When motility is decreased the give –

a) Parasympathomimetic drugs
   e.g. Inj. Neostigmine @ 20-40 µg/kg i/m

b) Purgatives
   e.g. Magnesium sulphate @ 250-350 gm orally.

iii) Rumenotorics in ruminants
   e.g. Bovirum bolus @ 2-4 boli daily orally for 2-3 days

2. Replacement of fluids and electrolytes lost :

Parenteral administration of large quantity of fluids to replace lost fluids and electrolytes.

a) Sodium bicarbonate 1.3% or 5% - in acidosis.
b) Ringers lactate solution to compensate electrolyte losses.
c) Dextrose Normal Saline I/V.

3. Relief of distension :

a) Use of spasmolytics to relieve spasm
   e.g. Dicyclomine @ o.5 mg/kg i/m.
b) Surgical intervention in case of intestinal or gastric accidents.

4. Reconstitution of rumen flora and correction of rumen pH :

a) Cud transplantation for reconstitution of rumen flora.
b) Rumen pH can be corrected by administration of
   i) Alkalizing agents like sodium bicarbonate or magnesium carbonate in acidic indigestion.
   ii) Acidifying agents like Glacial acetic acid in alkaline indigestion.

5. Relief of pain :

a) Use of analgesic :
   e.g. Inj. Analgin @ 33-50 mg/kg I/M
   Inj. Meloxicam @ 0.2 – 0.5 mg/kg I/M

6. Relief of tenesmus

This is most difficult :

a) Long acting epidural anaesthesia.
   e.g. Lignocaine HCl @ 5-10 ml.
b) Sedatives
   e.g. Inj. Siquil @ 0.1 mg/kg I/M, I/V

7. Use of antiemetics in gastritis / vomition :

e.g. Inj. Siquil @ 0.1 mg/kg I/V, I/M
Inj. Reglan @ 0.1-0.3 mg/kg I/M, I/V

GASTRIC ULCERATION

Definition : Are less commonly observed than erosions and seen more common in dogs than the cats.

Causes :

1. Drugs : NSAIDS, corticosteroids.
2. Primary gastric diseases – chronic gastritis, gastric dilatation and neoplasia.
3. Stress factors – severe illness, environmental stress.
4. Neurologic disease.
5. Metabolic disorders (renal disease, liver disease)
6. Gastric hyperacidity.

Clinical Signs :

- Acute or chronic vomiting with or without haematemesis – most common.
- Anorexia, abdominal pain, melena, anaemia, oedema.

Diagnosis :

1. History
2. Clinical Signs
3. Endoscopy is most sensitive method for diagnosis.
4. Barium meal X-ray.

Treatment :

The principles of medical therapy are to –

1. Remove the cause if possible.
2. Maintain mucosal perfusion.
3. Decrease gastric acidity.
4. Protect ulcer.

Prevention :

1. Withheld food to avoid stimulation of gastric acid and pepsin secretion.
2. Antiemetics in case of vomition.
3. Drugs :
   a. H_2 receptors antagonists to inhibit acid secretion by binding to the histamine H_2 receptor sites.
      - Ranitidine is more potent and lasts longer than cimetidine. Dose: @ 2mg/kg BID

- Fomotidine may be more potent than either ranitidine or cimetidne. Dose : @ 0.5-1 mg/kg BID.

b. Prostaglandin E Analogs :
   - They inhibit adenylate cyclose and there by reducing hydrogen ion secretion.
   - In addition these drugs increases gastric mucosal blood flow.
     e.g. Misoprostol @ 0.5 – 2.3 µg/lb BID or TID
   - It is drug of choice in NSAID induced gastric ulcers.

c. Proton pump inhibitors
   - They block hydrogen ion secretion by inhibiting Hydrogen ions, potassium ions and ATPase located at the apical membrane.
     e.g. Omeprazole. 0.66 to 2mg/kg once in a day.

d. Sucralfate :
   - This substance tightly adheres to the ulcerated tissue and protects it from acid and pepsin.
     Dose : 0.5 to 1 gm 2-4 times per day.

e. Miscellaneous drugs :
   Oral antacids – aluminium hydroxide, magnesium hydroxide.
   They are safe and effective neutralizing acid and inactivating pepsin.
   - Antiulcer therapy should be continued for 4-6 weeks.
   - Gastric dilatation refers to distention of the stomach caused by swallowed air, fluid or food.
   - Gastric dilatation is generally accompanied by volvulus (GDV).
   - Signs : Acute progressive distending abdomen nonproductive retching, hypersalivation, restlessness, depression, weakness and abdominal pain.

Chapter - 4

DISEASES OF LIVER

1) HEPATITIS

- Definition: "It means inflammation of the hepatic cells /liver "
  It may be acute or chronic in nature .
  Chronic hepatitis leads to cirrhosis / fibrosis of liver .

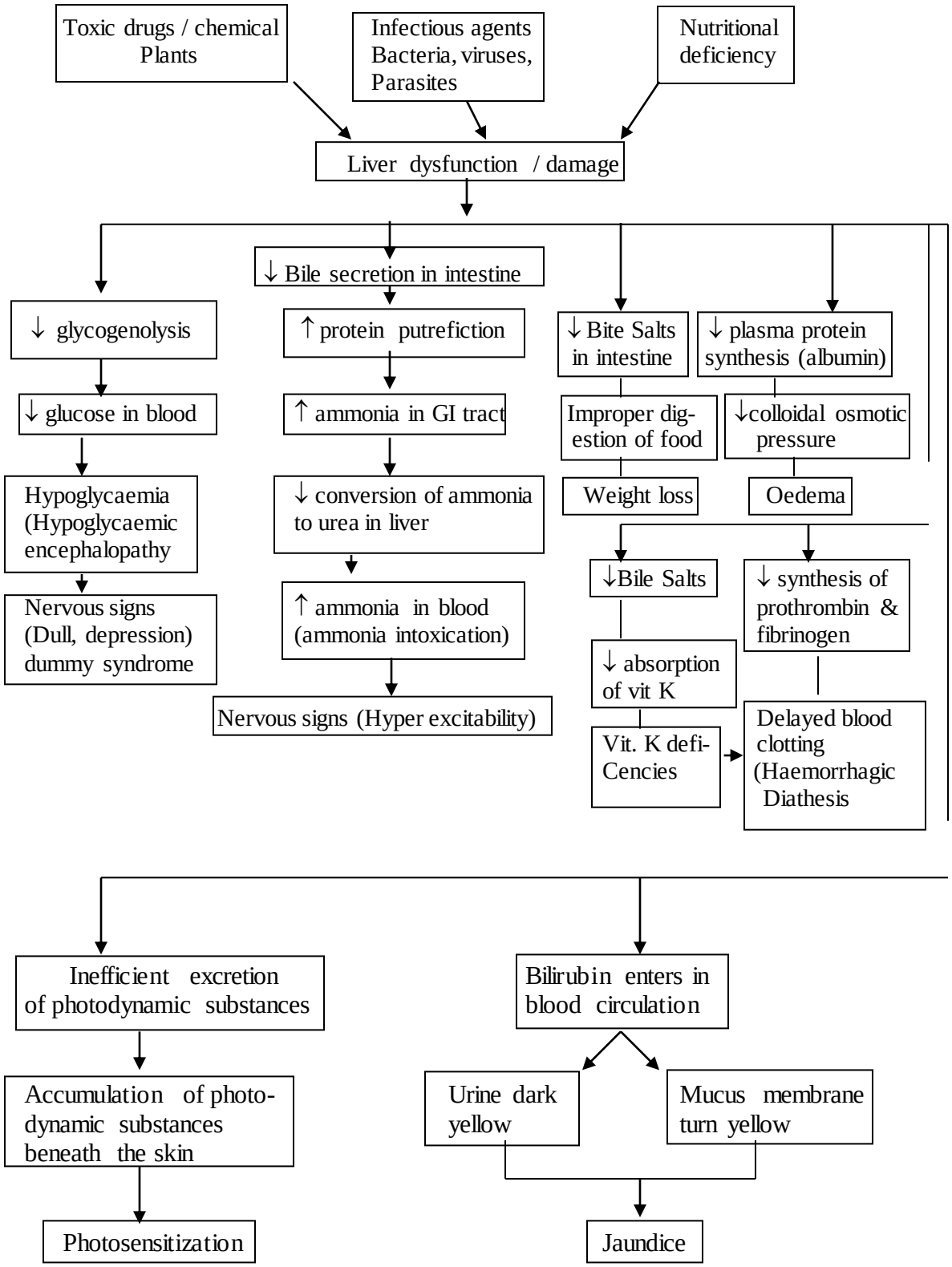
Etiology :-

1) Toxic hepatitis :-
   a) Inorganic poisons – cu,p,As,pb Hg,CCl4
   b) Organic poisons – gossypol,coal tar, alchol
   c) Poisonous plants – *Senecio, Crotalaria, Tribulus, Lantana etc.*
   d) Fungal toxisns – Aspergillus, penicillium, Fasarium
   e) Toxic algae .
   f) Drugs – tetracycline ,paracetamol, hexachloropheone
2) Infective hepatitis :-
   a)Bacteria :- *Leptospira, Listeria, Clostridium novyi*
   (Infectous necrotic hepatitis), *Salmonella, E-coli, Streptococci , Staphylococci.*
   b) Viruses :- Equine infectious anaemia, Infectious canine hepatitis, Feline panleukopenia, Equine rhinopneumonitis, Equine viral arteritis.

c) Chlamydia :- Rift valley fever
d) Parasites :- Acute or chronic liver fluke infestation migrating larvae of *Ascaris spp*.

3) Nutritional hepatitis (Trophopathic) :-
    a) Deficiency of methionine, cystine and choline
    b) Deficiency of vit. E and selenium
    c) High fat diet
4) Congestive heart failure leading to increase pressure in the sinusoids of the liver.
5) Miscellaneous causes :- Indirect hepatic damage caused by diabetes mellitus, ketosis,pregnancy toxaemia, fatty cow syndrome, hypothyroidism .

Pathogenesis :



Clinical signs :-

1) Anorexia
2) Dummy syndrome /Hepatic encephalopathy :- Aimless wandering, staggering, head pressing, mental depression, blindness.
3) Muscular weakness, weight loss, rough body coat
4) Jaundice – dark urine, yellow mucus membrane
5) Recumbency and coma
6) Photosensitization may develop .
7) Shedding of hair and wool coat may occur
8) Vommition , constipation followed by diarrhoea, steatorrhoea, abdominal pain etc
9) Ascites /oedema (bottle jaw /brisket oedema)
10) Increase tendency to bleed
11) Signs of ammonia intoxication –Hyperexcitability

Diagnosis :-

1) History : Access to poisons
2) Clinical signs e.g Anorexia ,mental depression, jaundice dark urine etc .
3) Liver function tests :-
    a) Serum Vanden Bergh test - positive (Biphasic) ↑
    b) Serum Bilirubin (Normal in cattle 0 - 1.9mg %) ↑
    c) Blood clotting time (Normal in cattle 7min) ↓
    d) Total serum protein (Normal in cattle 6 - 8 gm%)
    e) SGOT –60-150μ/L ↑
4) Liver biopsy
5) Radiography / ultrasonography.
6) Urine analysis i.e. Fouchet's test and Hay's sulphur test is positive .

Treatment :-

I) General care :

i. Give complete rest till complete recovery
ii. Provide fat and salt free diet.
iii. Provide carbohydrate rich diet /plenty of glucose
iv. Provide high quality protein diet.

II) Specific treatment :-

Antibiotics in bacterial and viral infections :- eg, Neomycin @ 10-20mg /kg orally .Amoxycillin/Ampicillin @ 10mg /kg IM, IV, orally
Antihelmintics in parasitic infection E.g. Fenbendazole @ 5-10mg /kg orally –Ascarids ozyclozanide (Distodin) @ 10-15mg /kg orally – for flucks

III) supportive treatment :-

a) Glucose / dextrose (5%) orally in dogs or parenterally to provides energy
b) prevent catabolism of proteins
c) promote detoxification mechanism
d) Liver tonics – e.g. Inj. Belamyl @ 5-10ml I/m in large animal Neohepatics @ 1-2ml in dog, Liv 52, Livogen syrup in small animals
e) Diuretics in ascites /oedema e.g. Ridema /lasix @ 1-2mg /kg I/M, I/V-LA, 5mg/kg I/M ,I/V -SA.
f) Steroids to reduce inflammation and catabolism e.g . Dexona @ 0.04mg /kg IM
g) Lipotropic agents e.g. choline and methionine - they prevent accumulation of fat in liver

h) Calcium therapy to prevent guanidine intoxication e.g. Calcium gluconate 10%solution @ 10ml I/V- in dogs.

i) Mild purgative like magnesium sulphate may be given for bowel cleansing.

2) JAUNDICE

Synonym : *Icterus*

Definition :-

" Jaundice means yellowish discolouration of all visible mucus membranes, body tissues and body fluids i.e. both secretions and excretions.

Classification :-

It is classified into three forms:

1) Prehepatic /Haemolytic jaundice
2) Hepatic /Toxic jaundice
   i) Posthepatic / obstructive jaundice .
   ii) Prehepatic / Haemolytic jaundice :-

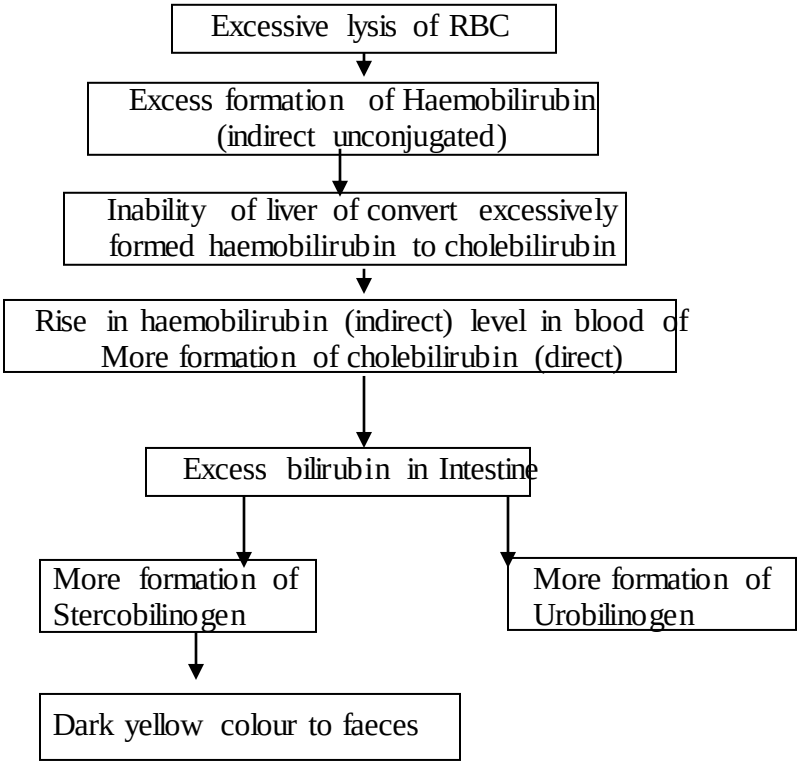
a) It is due to excessive lysis of RBC's

b) It occurs prior to passing of blood to liver so called as prehepatic jaundice

Causes :-

1) Haemoprotozoan infections :- Theileriasis , Babesiosis , Anaplasmosis
2) Bacterial infections :- Leptospirosis,Bacillary haemoglobinuria
3) Viral infections :- Equine infectious anaemia
4) Deficiency diseases :- phosphorus deficiency, Cu deficiency
5) Metabolic diseases :- post partrient haemoglobinuria
6) Poisonings :- snake bite, copper poisoning ,onion poisoning, water intoxication
7) Isoimmune haemolytic anaemia

Pathogenesis of haemolytic jaundice :



II) Hepatic /Toxic Jaundice :-

- It occurs due to damage to hepatic cells .
- It is usually due to certain toxins and hence also called as toxic jaundice .

Causes :-

1) Toxins :- (Toxic hepatitis)

a) Inorganic – Cu, P ,As, Pb ,Hg,CCl_4 etc.
b) Organic –Gossypol,coal tar, alcohol etc
c) Plants –Senecio, Crotalaria, Fribulus ,Lantana
d) Fungi – Aspergillus , penicillum , fusarium
e) Algal toxins
f) Drugs :- Tetracycline ,paracetamol etc

2) Infectitious agnts:-

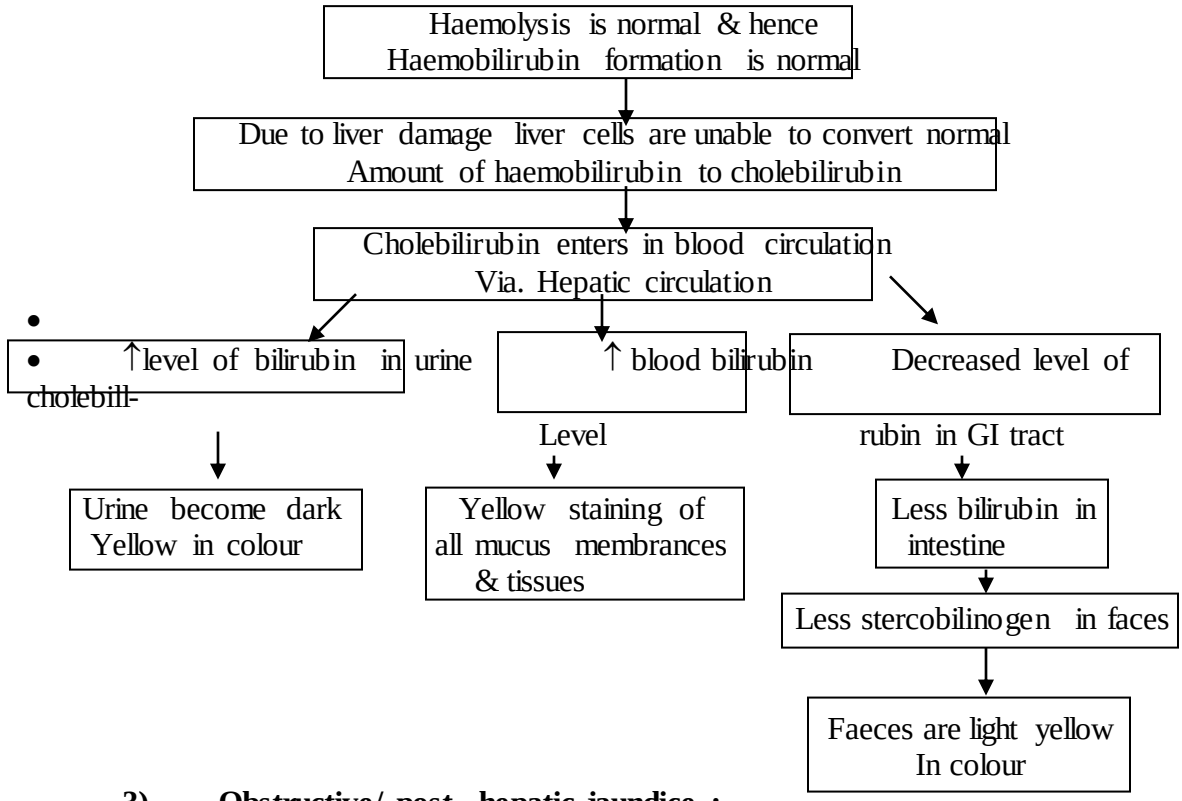
a) Bacteria :- *Leptospira, Clostridium novyi, E-coli, Salmonella , Septicemic listeriosis*
b) Virus :- *Equine infectious anaemia, Infectious canine hepatitis, , Feline panleukopenia*
c) Chlamydia :- Rift valley fever
d) Parasitic causes :-
Acute /chronic liver fluke infestation, migrating larvae of *Ascaris spp*

3) Nutritional causes :- (Trophopathic)
a) Deficiency of methionine and choline.
b) Deficiency of vit E and selenium

4) Congestive heart failure leading to increase in hydrostatic pressure in the sinusoids of the liver .

5) Miscellaneous causes – Indirect hepatic damage caused by Diabetes mellitus , ketosis , pregnancy toxaemia, fatty cow syndrome

Pathogenesis :



3) Obstructive/ post –hepatic jaundice :-

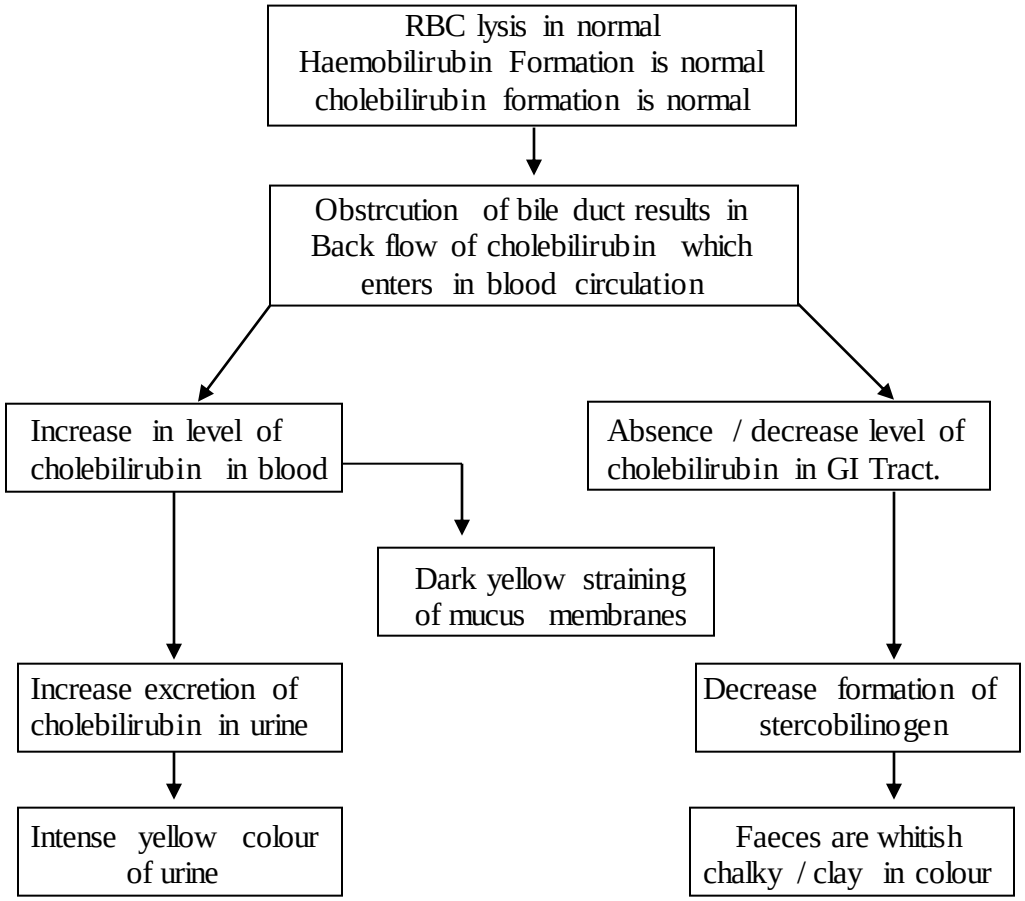
- It is due to obstruction of bile duct
- It is due to defect in bile duct which is next in sequence to liver so it is also called post – hepatic jaundice

Causes :-

It is due to obstruction of bile duct caused by

1) Cholangitis (Inflammation of bile duct)
2) Cholecystitis (Inflammation of gall bladder)
3) Cholelithiasis (gall / bile stones)
4) Parasites e.g *Dicrocoeilium*, *Toxocara* , *Mature liver flukes* etc.
5) Neoplasms cysts, Abscess in bile duct or exterior to bile duct which may cause compete or partial obstruction .

Pathogenesis of post hepatic/obstructive jaundice:



Clinical signs :-

1) Yellowish discolouration of mucus membrane (markedly visible in sclera).
2) Light yellow, dark yellow or greenish yellow urine. Red urine in haemolytic jaundice
3) Dark yellow to clay colour faeces (hypopigmentation) faeces may contain fat i.e steatorrhoea.
4) Muscular weakness
5) Mental depression
6) Loss of body weight (emaciation)
7) Loss of appetite.
8) Vomition in dogs and pigs
9) Constipation followed by diarrohea
10) Severe anaemia in haemolytic jaundice
11) Sometimes oedema, bottle jaw, ascites
12) Excitement, coma and death

Diagnosis :

1)Differential Diagnosis of jaundice:-

Sr. No.	Parameter	Haemolytic	Toxic	Obstructive
01	Erythrolysis	Increased	Normal	Normal
02	Liver	Normal	Normal	Normal
03	Bile duct	Normal	Normal	Abnormal
04	Serum indirect bilirubin	Very high	Increased	Normal
05	Serum direct bilirubin	Normal	Increased	Very high
06	Urine bilirubin	Absent	Increased	Very high
07	Urobilinogen	Increased	Normal to decreased	Decreased to absent
08	Urine colour	Light yellow	Intense	Intense
09	Stercobilinogen	Increased	Normal to decreased	Decreased/absent
10	Colour of faeces	Hyperpigmented	Normal to pale	Hypopigmented chalky / clay colour
11	Colour of mucus membrance	Slight to moderate yellow	Moderate yellow	Intense yellow
12	Colour of serum	Slight to moderate yellow / reddish	Slight to moderate yellow	Intense yellow
13	Icterus index (8.4 – 9.8 normal in cattle)	Low of moderate	Moderate	High
14	Vanden Bergh's test	Indirect	Biphasic	Direct
15	Liver function test.	Negative	Positive	Negative (initially)

II) Diagnosis

Clinical signs :- Yellow discolouration of mucus membrane, Vomition, constipation followed by diarrhoea, urine looks deep yellow to brownish or greenish yellow.

Liver function tests :-

- Vanden Berghs reaction – Indirect, direct or biphasic
- Blood clotting time is increased
- Serum bilirubin level is increased

Urine analysis :- Urine samples are positive for bile pigments and bile salts.

Treatment :-

1) **General care:-**
   a) Give complete rest
   b) Provide fat and salt free diet
   c) Provide carbohydrate rich diet

d) Provide high quality protein diet

2) Specific treatment :-

a) Antibiotics orally of parenterally e.g . Amoxycillin / ampicillin @ 5-10mg /kg IM, IV, Orally.

b) Antiprotozoan drugs :- e.g. Berenil @ 5.5-7.0mg /kg IM

c) Antihelmintics e.g . Flukicides like distodin @10-15 mg /kg PO Fenbendazole @50-10mg /kg PO

d) Surgical intervention in gall stone, tumor, neoplasm etc

3) Supportive treatment :-

i) Dextrose 5% or 10% orally /parentrally

ii) Liver tonics e.g. a) Belamyl @ 5-10ml IM in larger animals
b) Neohepatex @ 1-2ml IM in dogs
c) Livogen /liv 52 syrup orally

iii) Steroids- e.g. Dexamethasone @ 0.04 mg /kg IM

iv) Lipotrophic agents e.g. choline, methionine

v) Calcium therapy to prevent guanidin intoxication @ 10ml IV as 10% solution in dog

vi) Mild purgatives in toxic jaundice e.g . magnesium sulphate @ 250-350 gm orally in large animals

vii) Diuretics in ascites /edema e.g .ridema /lasix (furasemide) @ 1-2 mg/kg IM /IV.

3. ASCITES

Definition :-

" Ascites means accumulation of fluid in the peritoneal sac"

It is slow producing /chronic disease

It is more common in dogs as compared to other animals

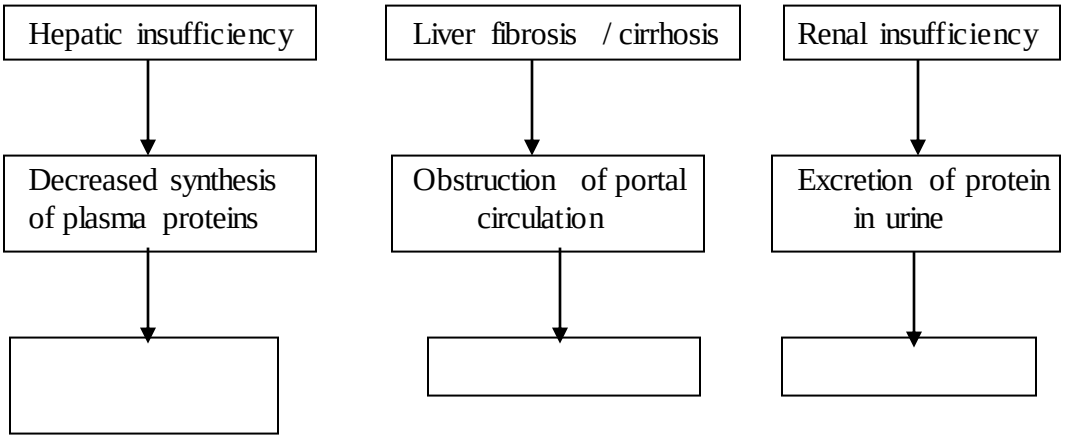
Etiology :-

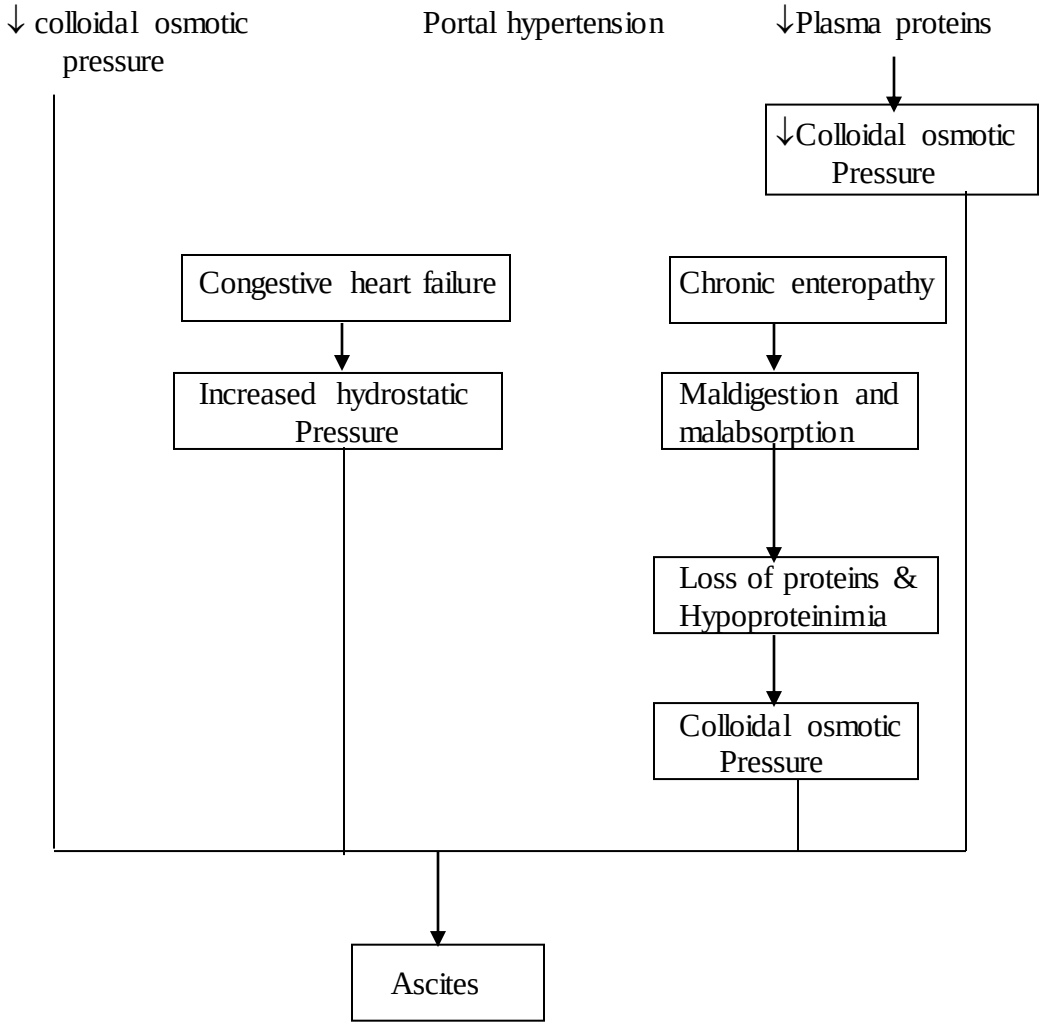
1) Acute or chronic hepatic insufficiency
2) Fibrosis or cirrhosis of liver
3) Congestive heart failure
4) Renal damage
5) Malabsorption and maldigesation of protein (Enteropathy)
6) Compression of mesenteric and lymphatic vessels by large tumor, cyst, tubercular nodules and enlarged lymphnode

Symptoms :-

1) Enlargement of abdomen on lower /ventral side
2) Hollow Hanks with prominent spines
3) Prominent abdominal vessels are seen
4) Fluid thrill or fluid wave on tactile percussion
5) Gradual loss of appetite and body weight
6) Tachycardia, dyspnoea and anaemia.

Pathogenesis





Diagnosis :-

1) Clinical signs :- Enlargement of lower abdomen .
2) Abdominocentesis :- Reveals clear fluid
3) Liver /cardiac / renal function tests / urinalysis.
4) Faecal examination :- Gross and microscopic
5) Examination of peritoneal fluid :- Colour, smell, culture.
6) Blood tests:-
    a) Hb, PCV, TEC decreases
    b) Total serum protein also decreases

Differential diagnosis:-

It is differentiated from condition characterised by enlargement of abdomen

1) Fluid filled cyst :-
    a) Careful palpation behind portal fissure may reveal rounded end of cyst
    b) No change in abdomen when anima turned in different posture
2) Ruptured bladder :-
    a) Common in males
    b) Ammonical smell to breath and sweat
    c) History of cessation of urination
    d) Evidence of urine on abdomenocentosis
3) Hydrops amnion :-
    a) Disorder present only in pregnent females.
    b) Slow enlargement of abdomen.
    c) No fluid on abdominocentosis
4) Peritonitis :-
    a) Signs of pain

b) Sero-sanguinous /purulent fluid on abdomenocentsis

Treatment :-

1) Correction of the primary cause
2) Provision of good quality protein diet
3) Give low sodium or sodium salt free diet
4) Use of diuretics e.g . Frusamide @ 1-2 mg /kg IM in LA& 4-5mg/ kg IM in dogs
5) Corticosteroids e.g Dexona @ 0.04 mg/kg IM
   Liver tonics e.g .Neohepatex @ 1-2ml IM
6) Abdominocentasis :- Complete drainage of fluid should not be done as there is a possibility of shock .

Principles of treatment of liver diseases

1) General care :-

a) Give complete rest to patient
b) Provide carbohydrate rich diet
c) Provide salt and fat free diet
d) Provide high quality protein diet

2) Specific treatment : -

a) Identification of cause and use of specific therapy
b) Antibiotics in bacterial and viral infections orally and parentrally e.g. Gentamicin @ 1-2mg /kg IM, IV, Ampicillin @ 5-10 mg /kg IM/IV orally.
c) Antiprotozoan drugs in haemoprotozoan infectcion eg Berenil @ 5.5 - 7.0 mg /kg IM as 20% solution.
   a) Anthelmintics flukicides in parasitic infection e.g. Fenbendazole @ 5-10mg/ kg orally - Round worms
      Oxyclozanide @10mg/ kg orally- flukes
   b) Surgical intervention in case of bile duct obstruction due to bile stone and other reason.
   c) In case of poisoning specific with antidote should be used e.g. Chlorinated hydrocarbon arsenic lead poisoning

3) Supportive treatment

a) Dextrose 25% solution @ 500-1000 ml for Large animal and 250 ml for dog IV
b) Liver tonics eg inj Belamyl @ 5-10ml IM large animal Neohepatics @ 1- 2ml IM in small animal
c) Vit.C,Vit A should be supplied
d) Diuretics in ascites and edema e.g . Ridema @ 1-2mg 1kg IM/ IV
e) Steroid to reduce inflammation and catabolism e.g .Dexona @ 0.04mg /kg IM
f) Lipotrophic agents e.g .choline and methionine
g) Calcium therapy in toxicities and to prevent guanidine intoxication e.g. calboral @ 100-300ml for large animal IV and 10 ml IV for dog as 10% solution.
h) Mild purgatives / laxatives for evacuation of GI contents e.g. sweet oil @ 250-500ml orally.

DISEASES OF RESPIRATORY SYSTEM

1. RHINITIS

Synonyms : Nasal Catarach, Coryza

Definition : "It is inflammation of nasal mucous membrane.
- It may be acute or chronic."

Etiology :

A) Predisposing factors :

i) Sudden change in climate – exposure to hot humid (rainy) / cold/chilled/ weather.
ii) Overexertion, hard work, prolonged strenous exercise, long distance transportation.
iii) Malnutrition / undernutrition, debility.
iv) Inhalation of dust/dirt, smoke, fumes, gas and chemical vapours.

B) Exciting / actual causes :

i)	Bacteria -	H.S. in cattle Strangles and glanders in horses. *Streptococcus, Staphylococcus, Spherophorus necrophorus.*
ii)	Viruses –	Cattle :- RP, MD, IBR and BMC Dog :- CD and Canine influenza. Goat :- RP, PPR, Goat pox Sheep :- BT, Sheep pox, PPR, RP
iii)	Parasites –	Small ruminants : *Oestrus ovis* Ruminants : - *Schistosoma nasalis*
iv)	Fungi –	*Aspergillus, Mucor, Rhizopus.*
v)	Allergic –	Dust, smoke, pollen grain, fumes.
vi)	Trauma -	Foreign bodies, stucking of awns, thistles etc., passing of stomach tube thorough nose in horse.

Chronic Rhinitis – Causes :

i) Nasal schistosomiasis in cattle – *Schistosoma nasalis* in cattle.
ii) Nasal polyps – *Rhinosporidium seebari* (fungi) in cattle.
iii) Nasal tumors
iv) Pus in air sinuses – sinusitis.

Clinical Signs :

- Nasal discharge – serous / mucoid / purulent-profuse- unilateral / bilateral –scanty - continuous discharge Orange to yellow coloured or greenish in colour / intermittent.
- Nasal mucosa shows erythema, erosion or ulcerations.
- Sneezing especially in allergic rhinitis.
- Shaking of head and rubbing of muzzle and nose on the ground.
- Difficully in breathing.
- Fever, dullness, depression and partial anorexia may be present.

Snoring respiration is characteristic of nasal polyp/ tumor / granuloma

Difference between Allergic rhinitis and Infectious rhinitis.

- **Allergic rhinitis :** Profuse watery nasal discharge, sneezing, ocular signs, short duration, eosinophilia and allergic history.
- **Infectious rhinitis :** Mucocopurulent nasal discharge, slight sneezing, marked nasal congestion, absence of occular signs and duration about 1 week.

Diagnosis :

1. History – Sudden change in climate, exposure to allergens.
2. Clinical signs – Nasal discharge, sneezing.
3. Nasal swab for isolation of bacteria / fungi.
4. Nasal washings for parasitic eggs.

Differential diagnosis :

1. RP, PPR, BMC, BT :-Nasal + oral lesions.
2. IBR – High persistent fever.

3. Bronchitis / pneumonia : Bilateral nasal discharge, coughing, abnormal lung sounds, severe signs.
4. Pharyngitis/choke : Nasal discharge may contain regurgitated feed material.

Treatment :

A) Specific treatment :

i) Antibiotics *viz.* Streptopenicillin / sulpha / amoxicillin / quinolones to check bacterial infection.

ii) Nasal granuloma : Inj. Anthiomaline 20 ml i/m followed by 15 ml i/m on two occasions at an interval of 3 days.

iii) Rhinosporidiasis / nasal polyp / tumor : Surgical removal.

iv) Fungal infection : Thiabendazole @ 20mg/kg orally for 6 weeks.

v) Nasal bots (*oestrous ovis*) : Inj. Ivermectin @ 1ml/50 kg s/c.

B) Supportive treatment :

i) Antihistaminics e.g. Avil/ Cadistin @ 0.5 – 1mg/kg i/m

ii) Nasal decongestants e.g. otrivin (oxmetazoline) drops, phenylephrine HCl

iii) Antiseptic inhalations : Vicks vaporub, Tr. Benzoin, Eucalyphus oil. Turpentine oil 30 ml in bucketful boiling water.

2. EPISTAXIS

Synonym : Nasal bleeding, Nasal haemorrhage.

Definition : It means bleeding from nasal vessels or from those of the accessory nasal cavities.

- It is not a disease but symptom of local or systemic disorders.
- Some breeds of dogs and race horse are very much prone to epistaxis.

Causes :

1. Hereditary predisposition - Bleeding occurs without any apparent cause
2. Injury due to foreign bodies or trauma is the most common cause of bleeding.
3. Nasal tumors / neoplasms
4. Nasal polyps (Rhinosporidiosis)
5. Nasal granuloma (Nasal schistosomiasis)
6. Ulceration of nasal mucosa : Glanders, mycotic infections of gutteral pouch in horse.
7. Purpura haemorrhagica in horse.
8. Poisoning : Bracken fern, sweet clover, copper, phosphorus, mercury and snake venom.
9. Maggots in nasal cavity.
10. Idiopathic thrombocytopaenia, vitamin K deficiency.
11. Ehrlichosis in dog.

Clinical Signs :

- Nasal bleeding may be unilateral or bilateral.
- It may appear drop by drop or in thin stream or in large quantity
  Large quantity bleeding is known as rhinorrhagia
- It is bright red in colour.
- Respiratory distress.

Diagnosis :

- History of injury
- Clinical signs.

It should be differentiated from bleeding from lungs (haemoptysis) and stomach (Haematemesis)

Treatment :

1. Rest
2. Correction of specific cause.
3. Cold water or ice packs should be held on frontal and nasal bones.
4. Plugging of one or both nostrils with cotton swab soaked in adrenaline/epinephrine or instillation of diluted adrenaline in nostrils.
5. Styptics : Stadren, Vit.K, calcium preparations.
6. Blood transfusion in severe cases.

3. LARYNGITIS, TRACHEITIS AND BRONCHITIS

Causes :

1) Predisposing factors : Similar to rhinitis.

2) Exciting causes :

1. Bacteria : *Pasteurella, Klebsiella pneumoniae, Spherophorus necrophorus, Streptococci, Staphylococci, Corynebacterium pyogenes, Haemophilus* in pig, calf diphtheria.
   Bordetella bronchiseptica is primary cause of infectious tracheobronchitis or kennel cough in dogs. Strangles in horses.
2. Viruses : IBR in cattle & buffaloes.
   Swine influenza – pig
   Equine influenza, equine herpes virus – horse.
   Canine influenza, Canine distemper – dog.
3. Allergerns – Dust, smoke, fumes, pollen grains.

Clinical signs :

1. Fever, anorexia, dullness, depression.
2. Nasal discharge - bilateral, scanty, serous, mucoid or muco-purulent may be foul smelling.
3. Cough – Usually dry and harsh.
4. Wheezing and snoring sound during inspiration.
5. Inspiratory dyspnoea.
6. Exposure to hot, humid or chilled atmosphere, dust, excitement and exercise aggrevate symptoms (coughing).

Diagnosis :

1. History – Sudden change of climate, stress.
2. Clinical signs – Coughing, nasal discharge, wheezing / snoring sound.
3. Culture of nasal discharge / tracheal exudate.
4. DLC count – Neutrophilia in bacterial infection.
   Eosinophilia in allergic condition.

Treatment :

1. Good care, rest and protection from cold weather.
2. Antibiotics : Streptopenicillin, gentamycin, Enrofloxacin, Amoxycillin.
3. Antihistaminics : Pheniramine / chlorpheniramine maleate.
4. Corticosteroids : Dexamethasone, prednisolone.
5. Expectorants : Ammonium chloride + Camphor + Glycerrhiza mixture.
6. Antiseptic inhalations : Tr. Benzoin, oil eucalyptus, Turpentine oil.

Chronic laryngitis : Sodium iodide (5-10%) @ 2 – 3 gm/100 lb body weight I/V for 5-7 days.

4. PNEUMONIA

Synonym : Pneumonitis

Definition : It is inflammation of the pulmonary or lung parenchyma.

- It is usually accompanied by bronchitis and pleurisy.
- Pneumonia accompanied by bronchitis is called as bronchopneumonia.
- Pneumonia accompanied by pleurisy is called as pleuro-pneumonia.

Etiology :

1) Predisposing factors :

- Sudden change in climate – exposure to rainy or cold weather.
- Overexertion, hard work, prolonged strenous exercise, long distance transportation.
- Malnutrition / under nutrition, debility.
- Inhalation of dust, smoke and chemical vapours.

2) Exciting / actual causes :

1. Bacteria - Usually causes bronchopneumonia

- *Pasteurella, Klebsiella pneumoniae, E. Coli, Salmonella, Pseudomonas*.
- *Streptococci, Staphylococci, Corynebacterium pyogenes*.
- *Mycobacterium tuberculosis* - Tuberculosis.
- CBPP – bovine, CCPP – caprines *Mycoplasma mycoides*
- *Haemophillus suis* pig.
- *Bordetella bronchiseptica* in dog.
- *Sphareophorus necrophorus* (Calf diphtheria) – calves.

2. Viruses Usually causes interstitial pneumonia

- Viral pneumonia – calves.
- Parainfluenza -3, adenovirus 1, 2 & 3, rhinovirus, reovirus – cattle.
- Swine influenza swine.
- Equine influenza, Equine rhinopneumonitis, Equine viral arteritis – equines.
- Maedi & Jagsiekte, RP, PPR, Pox, BT – sheep
- PPR, RP, Pox – Goat.
- Canine distemper, Canine influenza – Dogs.

3. Parasites (Verminous pneumonia) :

- Lung worms : D. filaria – Sheep and goat.
  Dictyocalus viviparus – Cattle.
- Ascarid larvae – Calves.
- *Toxocara canis, Ancylostoma caninum* – Dog.

4. Fungi (Mycotic pneumonia) : Aspergillosis, Histoplasmosis, Blastomycosis.

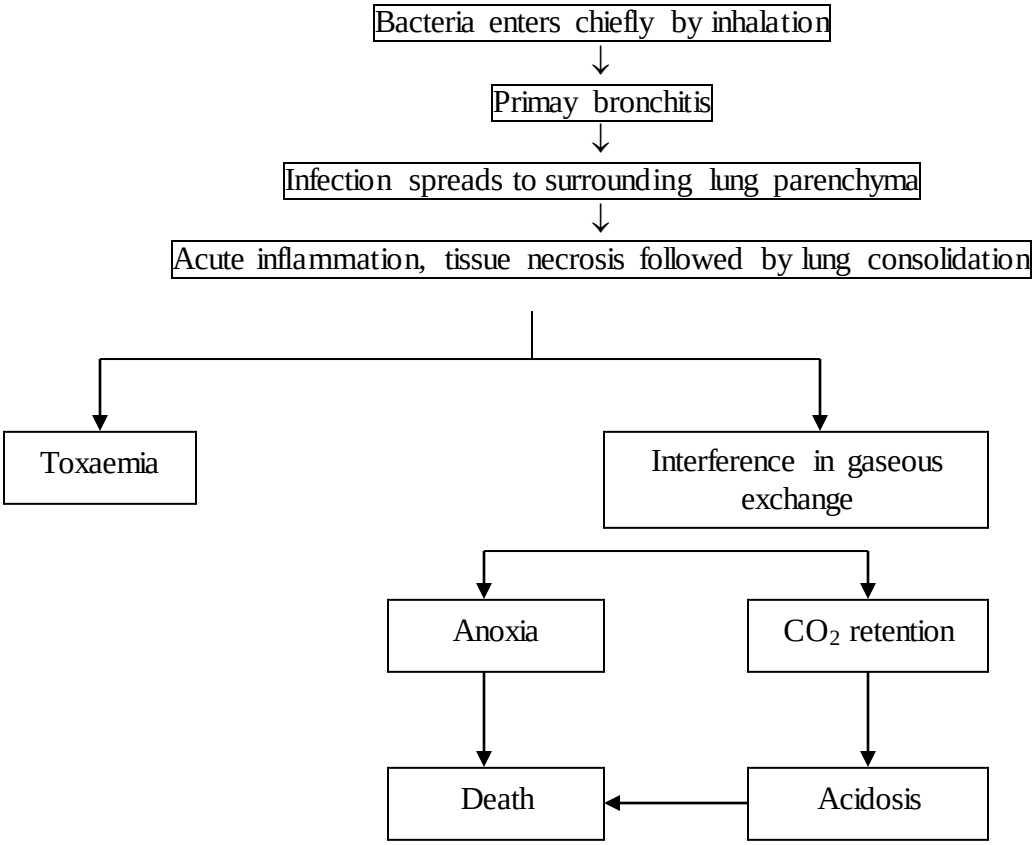
5. Foreign body Pneumonia : This is common in ruminants. Penetrating foreign bodies.

6. Aspiration pneumonia :

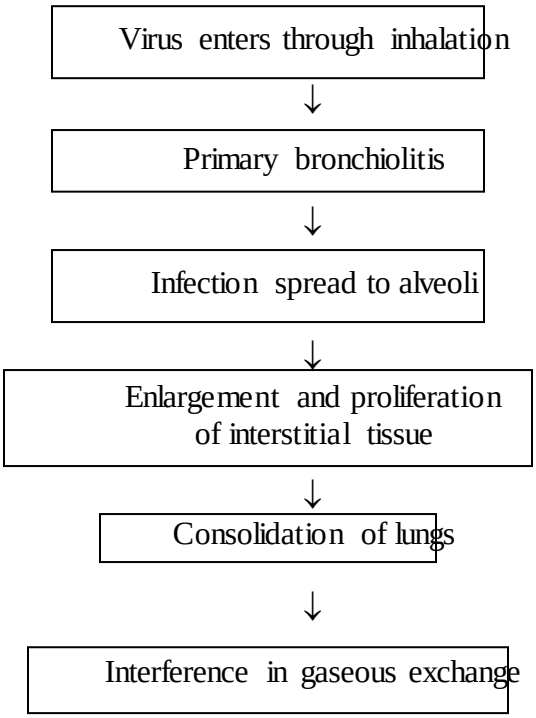
- Drenching of drugs / liquids through nostrils.
- Regurgitation of feed material in pharyngitis, choke / pharyngeal nerve paralysis etc.

Pathogenesis :

I) Bacterial pneumonia :



II) Viral pneumonia :



In viral pneumonia tissue necrosis and toxaemia are absent.

Clinical Signs :

- Fever, anorexia, dullness, depression, suspended rumination.
- Tachycardia.

- Respiration - Rapid shallow – early stage.
  - Dyspnoea – later stage
- Coughing - Moist, productive – Bronchopneumonia (Bacterial)
  Dry, unproductive – Interstitial pneumonia (Viral)
- Nasal discharge : Bilateral
- Chest pain : Reluctance to move and lie down.
  Stand with abducted elbows with extension of head and neck.
- On percussion : Dull sound may be heard.
- On auscultation : Early stage – Loud breath sound.
  Later stage – Moist/ dry rales.
  Consolidation – increased audibility of heart sounds.

Diagnosis :

1. History- Stress of strenous exercise, exposure to extreme climate.
2. Clinical signs : Fever, dyspnoea, coughing, nasal discharge.
3. Haematology : a) Leucocytosis with neutrophilia in bacterial infection.
   b) Leucopaenia in viral infection.
   c) Eosinophilia in allergic conditions/verminous pneumonia.
4. Radiographic examination : Increased radiopacity in pneumonia T. B. nodules, foreign body can be detected
5. Culture of traheal / bronchial exudates : This will help in identification of causative agent as well as in selection of antibiotic for treatment.
6. Examination of faeces to detect parasitic egg.
7. Serological tests.

Differential diagnosis : Refere Table 5.1 and 5.2

Treatment :

1) General care :

- Rest
- Provide warm, well ventilated, draft free accommodation.
- Provide ample fresh water and light nourishing diet.
- Avoid exposure to inclement weather
- Avoid premature return to work in bullocks.

2) Specific treatment :

1. Use of broadspectrum antibacterials / antibiotics.
   viz. Streptopenicillin, Gentamycin, Enrofloxacin, Amoxycillin, ampicillin, sulpha.
2. Use of anthelmintics.
   viz. Levamisole, tetramisole, fenbendazole, ivermectin.
3. Use of antifungals :
   Viz.: Amphotericin @ 0.5 mg /kg body weight in 500 ml
   5% dextrose slow I/V (dog and cat) or Ketoconazole @ 10 mg/kg orally TID.

3) Supportive treatment :

1) Antihistaminics e.g. Anistamin
2) Corticosteroids e.g. Dexona.
3) Antipyretics, analgesics and NSAID e.g. Meloxicam, Ketoprofen.
4) Bronchodilators and antispasmodics : Adrenaline, atropine sulphate, aminophylline, deriphylline.
5) Expectorants may have value in chronic cases.
6) Respiratory stimulants (e.g. Nikethamide) serve no useful purpose. e.g. Nikethamide – Dog 20-40 mg/kg body weight i/m, i/v.
7) Vit. C and Ca therapy to rejuvenate the lung parenchyma.
8) Oxygen therapy is life saving in severe cases. However, not practicable in veterinary practice under field conditions.

9) Fluid therapy in anorectic animals.

5. PLEURISY

Synonym : Pleuritis

Definition : It means inflammation of pleura

- It is usually bilateral in horses and unilateral in other species.

Classification : i) According to presence of fluid a) moist (b) dry pleurisy.

ii) According to course of disease a) acute b) subacute c) chronic

iii) According to etiology : a) Primary b) Secondary

Etiology :

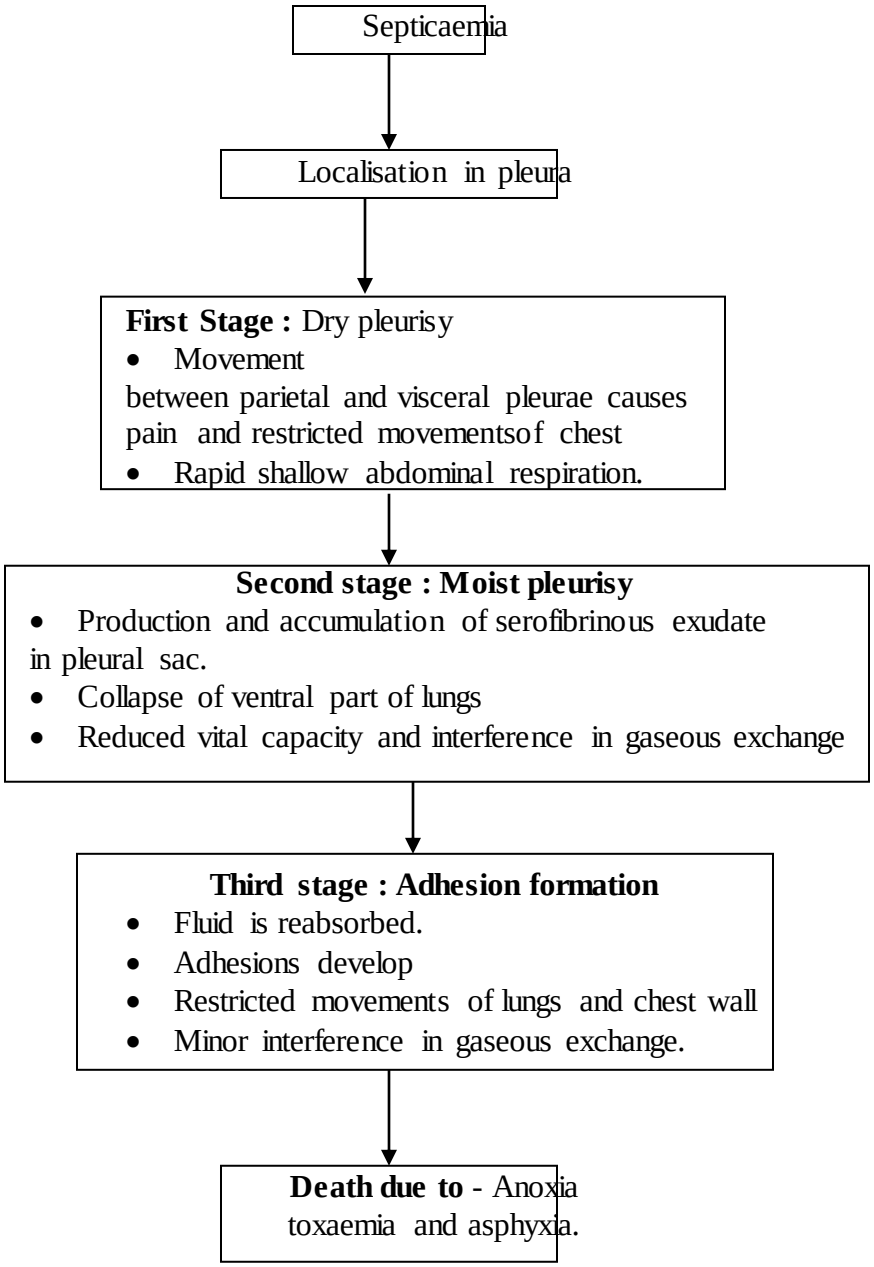
A) Predisposing factors : Similar to pneumonia.

B) Actual causes or Primary causes :

1) Foreign body and Traumatic perforation of thoracic wall
2) Bacteria :
   - Pasteurella multocida & P. haemolytica – All species.
   - CBPP & Tuberculosis – Cattle and buffaloes.
   - CCPP – Sheep and goat.
   - Haemophilus influenzae – Pig
   - Strangles – Horse
3) Viruses – Canine distemper – Dog.
   Equine influenza – Horses
4) Mycotic infections.
5) Neoplasms of thorax.
6) Parasites – Extensive lung worm invasion and Strongyle larvae

Most of the organisms responsible for pneumonia can cause pleurisy in animals.

Pathogenesis :



Clinical Signs :

- High fever accompanied by Anorexia, dullness, depression, suspended rumination.
- Tachycardia.
- Rapid, shallow, abdominal breathing – Dry pleurisy (Early stage)
- Dyspnoea (inspiratory) - Moist pleurisy (Late stage)
- Respiration markedly abdominal with restricted movement of chest wall.
- Groaning or expiratory grunt.
- Chest pain is outstanding clinical sign.
  - Disinclination to move and lie down.
  - Stands with abducted elbows and extended head and neck.
- On auscultation – pleuritic frictional rub in early stage.
- Dry sound close to the surface on inspiration and expiration.
- Fluid/splashing sounds in ventral thorax – moist pleurisy
- On percussion – dull sound.

Diagnosis :

1. History : Sudden change in weather, exertion.
2. Clinical signs : Dyspnoea, abdominal respiration, chest pain, frictional rub.
3. Haemogram – Leucocytosis with neutrophilia.
4. Examination of pleural fluid – TLC increases upto 40,000 to 1 lakh/dl
   Pleural fluid examination :
   i) Colour : Grey – Empyema, straw – exudate, Red – haemothorax. Milky – chylothorax (Normal : Pale)
   ii) Specific gravity : Above 1.016 (Normal upto 1.015)
   iii) Proteins : Above 4 gm % (Normal 3 g %)
   iv) Cytology : Neutropheal – Bacterial infection. Lymphocyte – Tuberoulosis.
5. Culture of pleural fluid
6. Radiographic examination – evidence of pleural adhesions and fluid.

Differential diagnosis : Refer Table No. 5.1

Pneumonia :

Parameter	**Pneumonia**	**Pleurisy**
Coughing	Present	Absent
Chest pain	Less marked	More marked
Ascultation	Moist/dry rales	Plueuritic rub/ Splashing sounds

Treatment :

1. Prolonged antibiotic therapy : Streptopenicillin, Metronidazole, Amoxycillin
2. Thoracocentesis - Site : 6^{th} or 7^{th} intercostal space. Aspiration of fluid from thoracic cavity.
3. Use of corticosteroids to reduce pleural effusions.
4. NSAIDS (Analgesics, Antipyretics, Antiinflammatory drugs) to reduce pain pyrexia and inflammation. e.g. Ketoprofen
5. Diuretics to remove excess of pleural fluids.
6. Multivitamins to rejuvenate the animal.

Note : Prognosis is always poor

6. PULMONARY EMPHYSEMA

Synonym : Broken wind or Heaves

Definition : Pulmonary emphysema is distension of lung caused by overdistension of alveoli with ruptured alveolar walls with or without escape of air into the interstitial spaces.

- It may occur secondary to some primary lesions which effectively traps excess amount of air in the alveoli.

Classification :

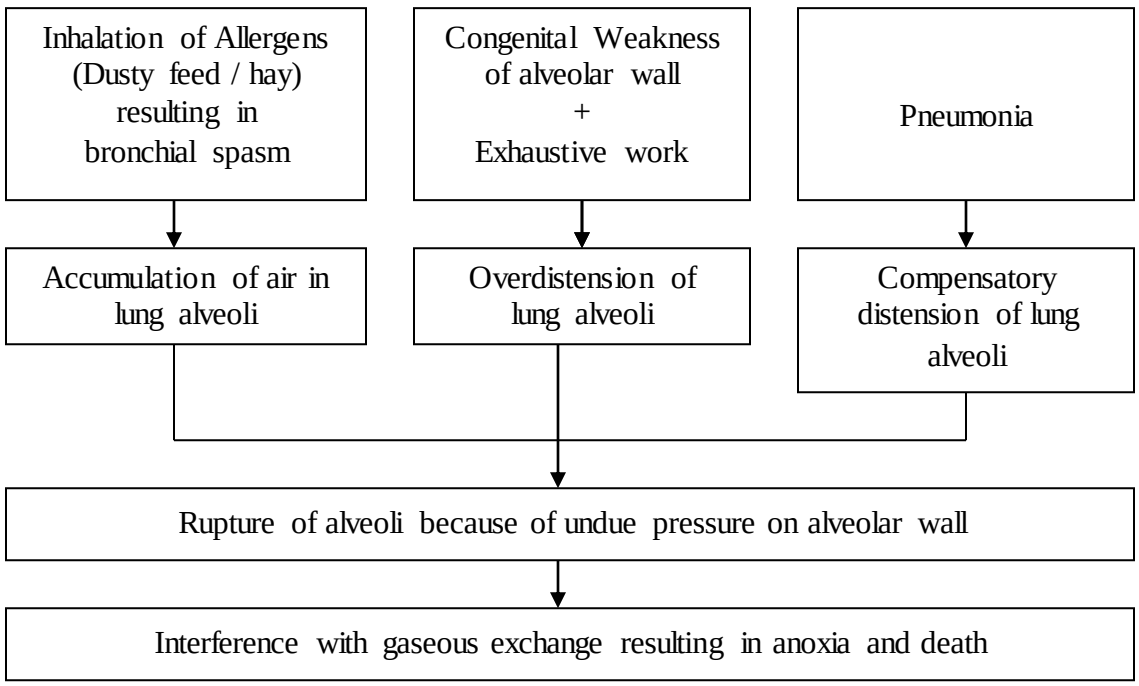
It is of two types viz. Alveolar and interstitial emphysema.

	Alveolar emphysema		**Interstitial emphysema**
1	The air accumulates in the alveoli of lung	1	Air accumulates in interstitial and interlobular spaces.
2.	It is either focal (acute) or diffuse (chronic) in nature.	2.	It is usually focal in nature
3	Acute alveolar emphysema is found in all species but chronic alveolar emphysema (Heaves/broken wind) is common in horses	3	It is common in cattle and dog
4	It is usually due to pulmonary sensitization	4	It is compensatory and mainly seen in pneumonic condition

Etiology :

i) Excess feeding of dusty hay/ fodder.
ii) Prolonged exhaustive work.
iii) Lung diseases : Chronic bronchitis, Atypical interstitial pneumonia, Verminous pneumonia, Pulmonary abscess, Pneumothorax.
iv) Chlorine gas poisoning.
v) Poisoning by the plants like *Zeieria arbore scens* and fungus Periconia species.

Pathogenesis :



Clinical findings :

i) Marked expansion of the chest wall.
ii) Expiratory dyspnoea.
iii) Poor exercise tolerance.
iv) Nasal discharge is bilateral, mucopurulent and more marked after drinking of water.
v) Hyperresonant or tympanic sound on percussion.
vi) Loud crackling sound on auscultation.

Diagnosis :

1. History – Chronic cough, pneumonia, feeding of dusty feed / forage, exhaustive work.
2. Clinical findings – Expiratory dyspnoea, Hyperresonance and crackling sounds.
3. X-ray examination reveals increased radiodensity of lungs.
4. Clinical pathology – DLC indicative of eosinophilia.

Differential Diagnosis : Refer Table 5.1

Treatment :

1. Give rest.
2. Animal should be kept in well ventillated room.
3. Use of antihistaminic preparations
   e.g. Inj. Avil @ 0.5 – 1 mg/kg body weight i/m or
   Inj Phenargan @ 1mg/kg body weight i/m
4. Corticosteroids :
   e.g. Dexamethasone @ 0.04 mg/kg body weight i/m
5. Use of bronchodilators :
   e.g. Inj. Deriphylline @ 1-2 mg/kg i/v
6. Use of antispasmodics in severe cases.
   e.g. Inj Atropine sulphate @ 0.03 – 0.06 mg/kg i/m
7. A course of broad sprectum antibiotics :
   e.g. Inj Streptopenicillin @ 10mg/kg i/m for 5-7 days or
   Inj Amoxycillin @ 5-10 mg/kg i/m for 5-7 days.
8. Trachaeatomy to save the life of animal in severe cases.

7. PNEUMOTHORAX

Definition : It means accumulation of air into pleural space.

Causes :

1. Rupture of bronchi or lungs.
2. Rupture of oesophagus.
3. Injury to chest wall – automobile accident, horn thrust etc.
4. Sub-diaphragmatic abscess.
5. Growth of gas producing organisms into pleura.

Clinical Signs :

- Dyspnoea
- Respiration – abdominal and painful.
- Buldging of the affected side of chest.
- No movement of the affected side of chest.
- Percussion- Hyperresonant sounds
- Auscultation – absence of lung sounds

Treatment :

- Removal of air through needle / canula.
- In open type - close the wound and treat with antibiotics.
- In closed type – give antibiotics.

8. HYDROTHORAX

Definition : It means accumulation of (transudate) fluid in thoracic cavity.

Etiology :

1. Right sided congestive heart failure.
2. Renal disorders.
3. Liver diseases.
4. Protein loosing Enteropathy.
5. Pulmonary / pleural neoplasia

Clinical Signs:

Signs are similar to pleurisy.

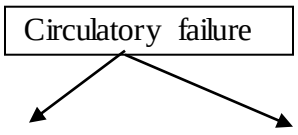
- Fluid accumulates in both the sides.
- Dyspnoea.
- Fever is absent
- Oedema in outer dependant body parts viz. Ventral abdomen.
- On percussion – Dull sound.
- On auscultation – splashing sounds.

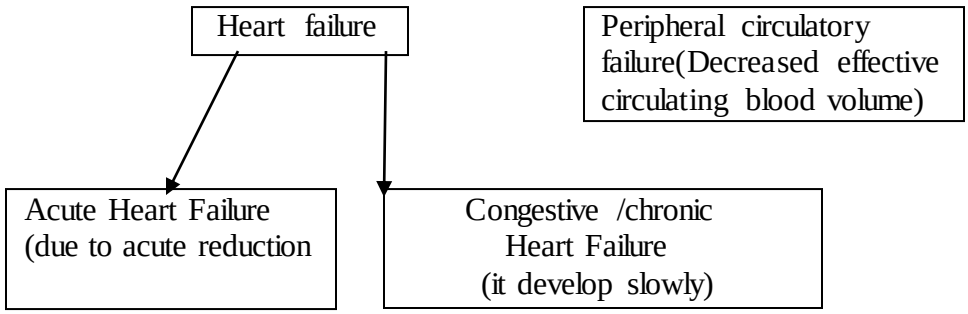
Treatment :

1. Correction of primary cause.
2. Repeated aspiration of fluid.
3. Use of diuretics.

Empyema – Presence of pus in the pleural space.

Chapter 6
DISEASES OF CARDIOVASCULAR SYSTEM





Failure of heart as pump results from:

- Defect in filling of heart
- Abnormality in the myocardium or
- Excessive workload or combination of three

Signs suggestive of cardiac insufficiency:

- Dyspnoea
- Fatigue
- Prolonged elevation in heart rate following exercise.

1) Congestive Heart Failure (CHF)

Definition:

It is defined as chronic heart failure characterized by congestion either in the pulmonary venous circulation or systemic venous circulation or both.

- CHF may be left or right sided.
- Left sided CHF is characterized by pulmonary congestion.
- Right sided CHF is characterized by systemic venous congestion.

Etiology:

1) Valvular diseases: (Increased flow / pressure load)

- Valvular stenosis or insufficiency resulting from endocarditis
- Aortic or pulmonary valve stenosis increases pressure load.
- Aortic and mitral valve insufficiency increases volume or flow load.

2) Myocardial diseases : Decreased cardiac output due to myocardial weakness

- Myocarditis, B.Q., T.B., and FMD in cattle. Strangles, equine influenza, SAHS and Equine infectious anemia in horses, parvovirus, CD in dog, B.T. in sheep.
- Myocardial degeneration due to toxic (As, Hg, Pb) or nutritional causes (vit.E, Se, Cu, Co, Fe deficiency)
- Myocardial asthenia due to anoxia or toxaemia.

3) Pericardial diseases (Incomplete filling of the heart) :

- Pericarditis.
- Hydropericardium
- Cardiac tamponade (acute compression of heart by rapid accumulation of blood).

4) Pulmonary hypertension :

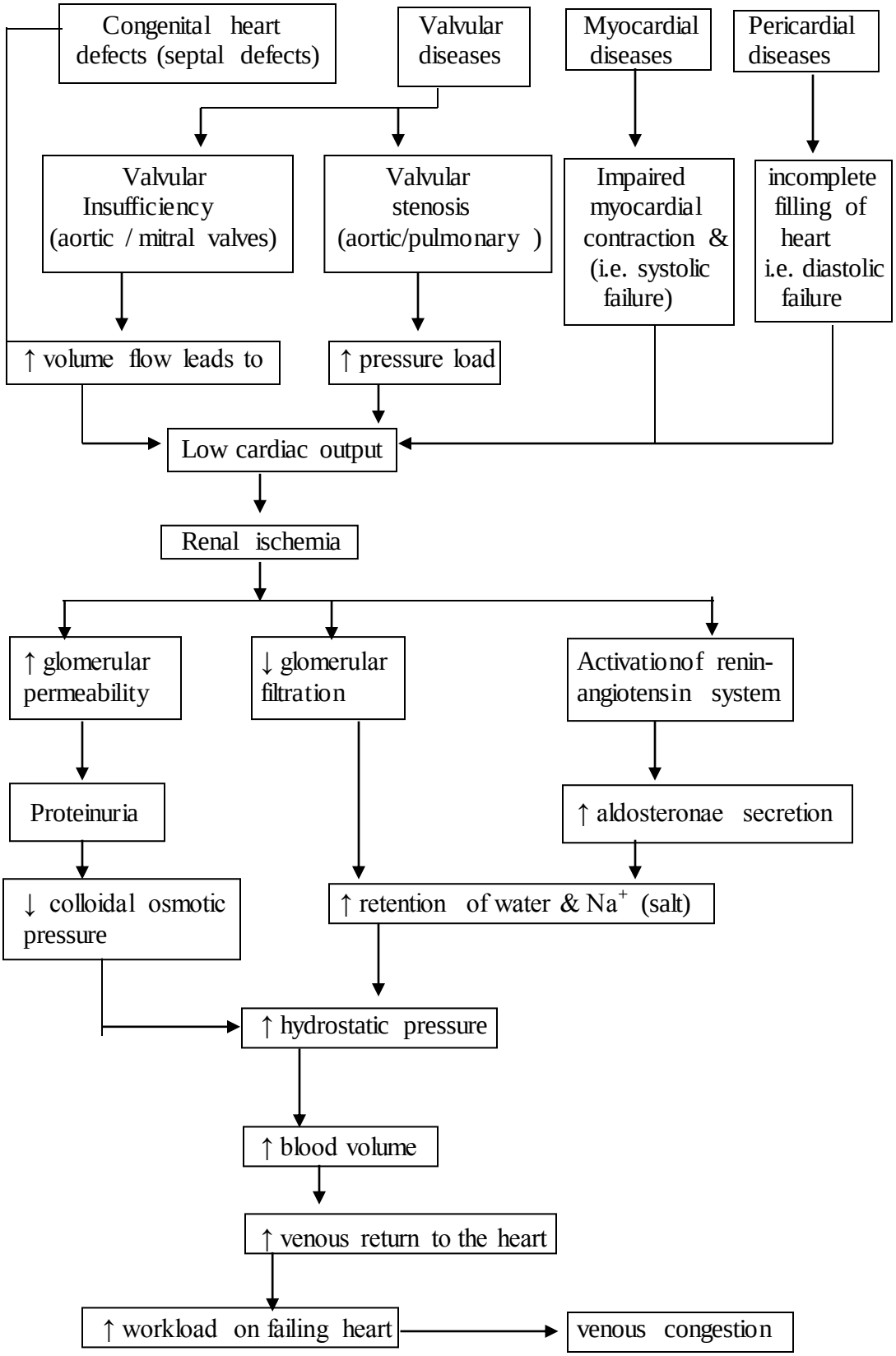
- Pulmonary emphysema : increased air pressure.
- Fibrosis of lungs : blood circulation is hampered.

5) Congenital defects producing shunts : (increase volume of load)

- Interventricular septal defect, most common defect in cattle, sheep and horses.
- Patent ductus arteriosus second most common defect in horses.

The common causes of CHF in :			
a.	Cattle and Buffaloes	:	Traumatic pericarditis
b.	Dogs	:	Valvular endocarditis

Pathogenesis



Clinical findings :

Right sided CHF :

- Subcutaneous oedema of ventral surface of the blood viz. jaw, neck, brisket.
- Jugular pulsation.
- Distension of Jugular vein.
- Tachycardia.
- Abnormal heart sounds- pericardial rub, Muffled splashing sounds
- Absolute dullness in cardiac area on percussion.
- Oliguria.
- Faeces normal – initial stages, profuse diarrhoea in late stage.
- Poor appetite, depression and staggering gait.

Left sided CHF :

- Increased rate and depth of respiration at rest.
- Decreased exercise tolerance.
- Coughing
- Dyspnoea and cyanosis during terminal stages.
- Tachycardia and cardiac murmur on cardiac auscultation.
- On auscultation of lung : moist rales.
- Dullness on percussion of ventral lungs.

Prognosis : unfavorable in most cases in large animals.

Diagnosis :

1) Clinical signs :- Brisket oedema and engorgement of jugular vein.
2) ECG
3) Clinical pathology : ↑ SGPT, ↑Urea, Albuminuria
4) Echocordiagraphy
5) Phonocardiagram
6) Phonocardiography

Treatment :

The line of treatment is as follows :

The principles of treatment in CHF are as follows :

1. Improvement of contractility by administration of positive inotropic agents such as cardiac glycosides.
2. Reduction of the effect of altered preload by diuretic agents.
3. Attempts to reduce demands on cardiac output by restriction of activity.
4. Rest
5. Provide salt free diet.
6. Avoid constipation by giving laxative.
7. Use of diuretics e.g. Furesenamide 40 – 80 mg PO in Dog.
8. Mannitol 20% @ 200- 300 ml i/v dog.
9. Use of corticosteroids : Prednisolone 20 mg orally daily for dogs.
10. Venesection in severe cases.
11. Cardiac tonics.
12. Tr. Digitalis @ 15-30 ml / 100 lb orally in horses.
    1. 0.3 -1 ml orally in dog.
    - It is ineffective in ruminants.

13. Digoxi – It is most commonly used cardiac glycoside Dose @ 0.02 – 0.03 mg/ lb i/v
    - Digitalis therapy has limitations in ruminants.
14. Dopamine :
15. Pericardiocentesis in pericarditis
16. Antibiotics in bacterial infections.
17. Pain killers to reduce chest pain.

2. PERICARDITIS

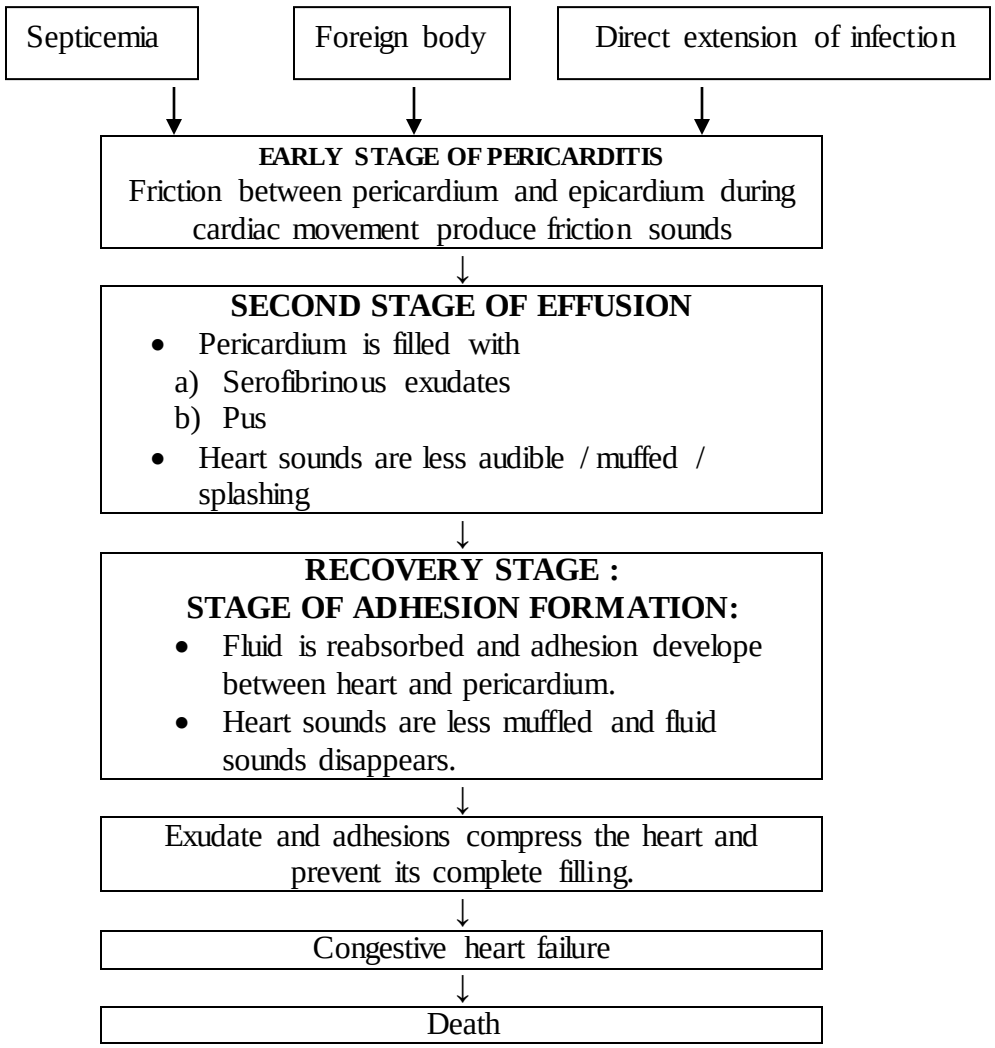
Definition : It is defined as inflammation of pericardium.

- Pericarditis may be traumatic or infective.
- Traumatic pericarditis is common in adult cattles / buffaloes whereas infectious pericarditis occurs in other animal species.
- Pericarditis may be acute or chronic in nature.

Etiology :

1. Trauma : Foreign body is the major cause of pericarditis in bovines. Sewing needle is very commonly recovered from cases of pericarditis.
2. Bacteria : *Pasterulla, E. Coli, Pseudomonas, Haemophilus, Salmonella, Staphylococci, Sterptococci, Mycoplasma and Mycobacterium spp.*
3. Viruses : Influenza – in horses.
   CD - in dogs
   Glasser's disease – in pig
4. Fungal : Coccidiomycosis.
5. Extension of infection from Pneumonia, Pleurisy, Pulmonary T. B., Myocarditis, Endocarditis.

Pathogenesis :



Clinical Signs :

- Fever (103^0 -106^0 F), poor appetite, ruminalstasis, drop in milk yield.
- Respiration sounds, shallow and abnormal.
- Jugular pulsation and engorgement of jugular vein.
- Brisket oedema.
- Chest pain : Arched back, abducted elbows with extension of head and neck. Disinclination to move and lie down. Prefers standing position.
- On ausculation : - Heart rate is increased (2-4 times).
  - Heart sounds are indistinctly heard.
  - Abnormal sounds muffled/ frictional rub / splashing sounds could be heard.
- On percussion : Increase area of cardiac dullness and evidence of pain.
- Death usually occurs within 1 – 3 weeks.
- Complete recovery is not common.

Diagnosis :

1. History of pica / recurrent tympany / ruminal dysfunction. No response to treatment.
2. Clinical Signs : Jugular pulsation, brisket oedema, chest pain, tachycardia, abnormal heart sounds.
3. Hematology : Leucocytosis with neutrophillia.
4. X-ray examination : Cardiac enlargement, foreign body.
5. ECG : Electrocardiographic changes include decreased amplitude of ECG complex.
6. Echocardiography : This is the most valuable technique to establish diagnosis of pericarditis and helps in differential diagnosis of effusive and fibrinous pericarditis.

Differential Diagnosis :

1. Pneumonia : Nasal discharge, cough, rales and heart rate and sounds – normal to increased.
2. Pleurisy : Pleuritic rub and heart rate and sounds – normal to increased.
3. Traumatic reticulitis : Heart and lung sounds are normal.

Treatment :

1. Give rest.
2. Provide salt free diet.
3. Broad spectrum antibiotics : For prolonged period e.g. combination of Penicillin and Gentamicin.
4. Analgesics and Antiinflammatory drugs.
5. Diuretics.
6. Pericardiocentosis to relieve the fluid pressure in pericardium.
7. Local irrigation with antibiotics may be tried.
8. Thoracotomy and pericardiotomy.

   * Treatment success rate is usually low in cattle.

3. Myocarditis and cardiomyopathy

Definition - Myocarditis is inflammation of myocardium (heart muscles).

It may be acute or chronic in nature.

Etiology :

It may be primary in origin and secondary to some other diseases.

A) Myocarditis :

1) Bacteria : B.Q. and T. B. in cattle and strangles in horses. Staphylococus aureus infection.
2) Viruses : FMD in cattle. SAHS and equine infectious anemia in horses. CD and parvovirus infection in dog. Encephalomyocarditis virus infection in pigs. Blue tongue in sheep.
3) Fungus : *Cryptococcus neoformans*
4) Parasites : Lysticerosis, Toxoplasma, Migrating larvae of *Strongylus spp*.
5) Infection may extend from endocardium and pericardium.

B) Myocardial degeneration (Cardiomyopathy)

1) Nutritional deficiency :

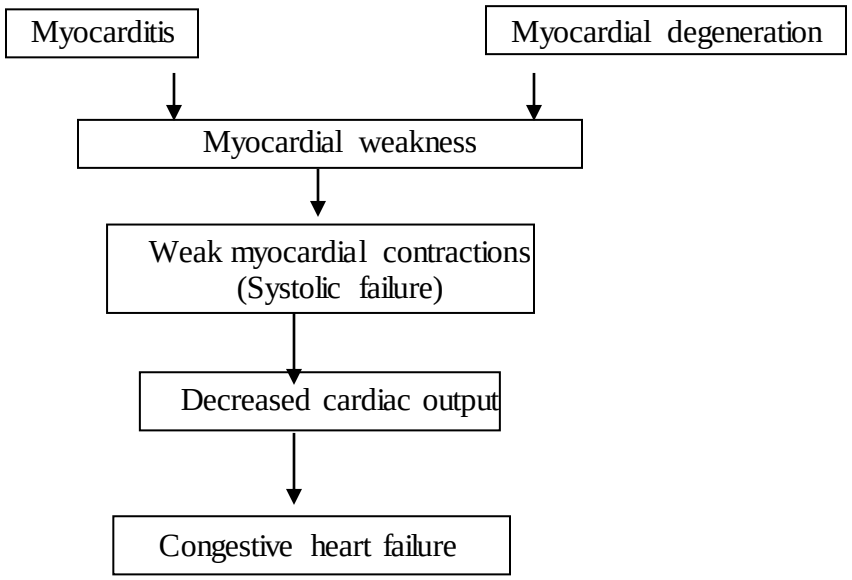
a) Vit. E and selenium in large animals.
b) Iron deficiency in piglets and veal calf.
c) Copper/cobalt deficiency in lambs.
d) Copper deficiency in cattle.

2) Toxicities :

a. Inorganic poisons: Aresenic, mercury, lead, copper, selenium, thallium.
b. Organic poison: Gossypol
c. Drug : xylazine
d. Snake venom :

Infectious agents are responsible for myocarditis whereas nutritional deficiencies and poisonings are responsible for myocardial degeneration.

Pathogenesis:



Myocardial diseases usually results in early conduction disturbances and arrythmias.

Clinical findings :

- Decreased exercise tolerance is the usual initial presenting signs in early stage or mild cases.
- Tachycardia
- Dysponea
- Weakness and early exhaustion
- On auscultation cardiac arrhythmia and increased cardiac area.
- sudden collapse and death due to acute heart failure in severe cases and in the late stage.

Diagnosis :

1) Clinical signs : Tachycardia, dyspnoea, cardiac arrhythmia
2) Clinical pathology : Elevated level of SGOT, SGPT and lactic dehydrogenase activity.
3) ECG
4) Echocardiography
5) Bacteriological and virological examination.
6) Toxicological and nutritional analysis.

Differential diagnosis :

1) Endocarditis :

- Cardiac murmurs on auscultation.

2) Pericarditis :

- On auscultation pericardial rub/muffled/ splashing heart souds.
- Jugular pulsation and brisket oedema.

Treatment :

1) Correction of primary cause with proper measures.
2) Use of cardiac tonics viz. Tr. digitalis @ 15-30ml/100 lb orally in horses @ 0.3 – 1 ml orally in dog.
3) Use of antibiotics viz. streptopenicillin @ 10 mg/kg i/m
   * *Response to treatment is poor.*

4. ENDOCARDITIS

Definition: Inflammation of endocardium is called as endocarditis.

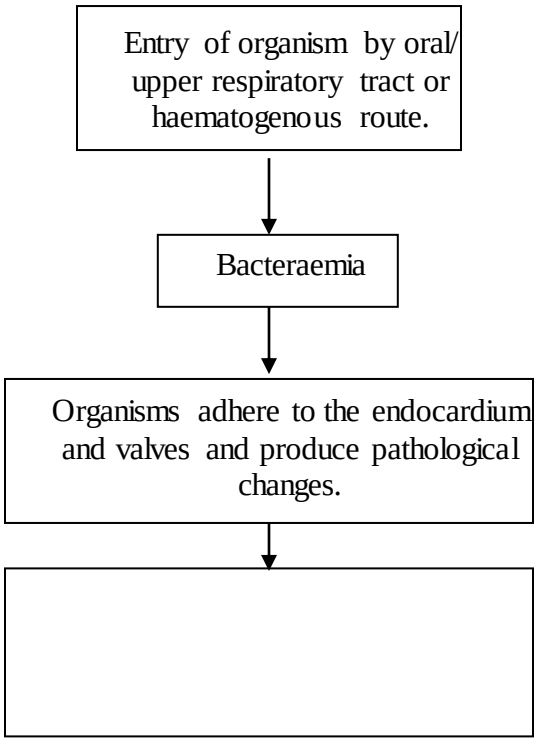
- It affects the lining of heart as well as valves within it.
- It may be acute or chronic in nature.

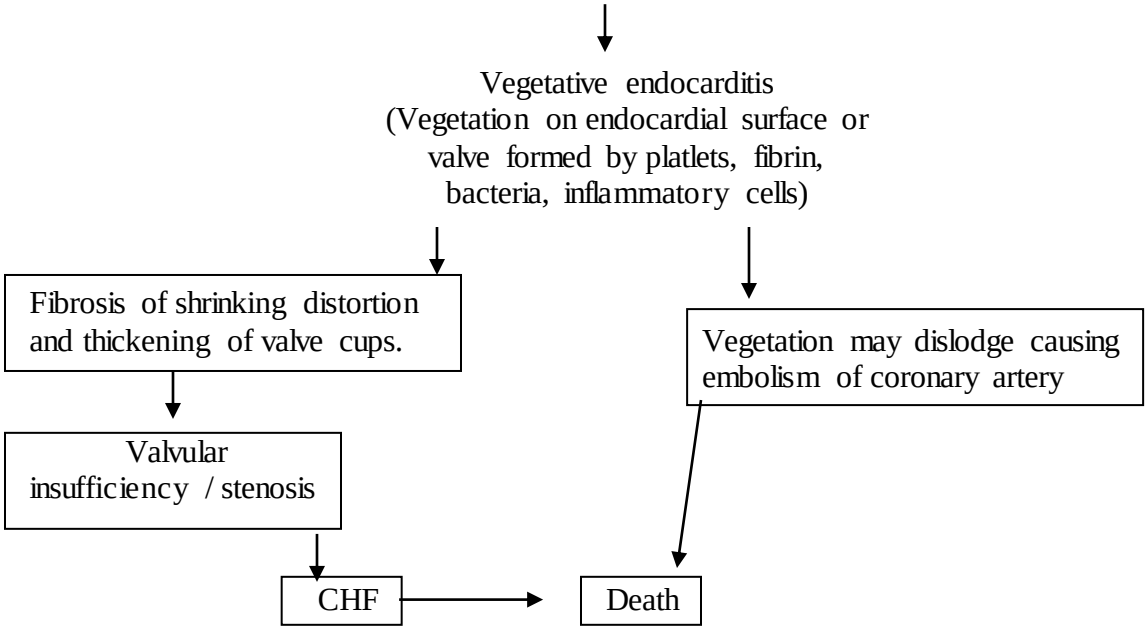
Etiology :

Endocarditis in large animals occurs most commonly secondary to chronic infection at some distant site and a persistent bacteremia e.g. mastitis, metritis, TRP, arthritis.

The common infectious causes of endocarditis in animals incude. *Corynebacterium pyogens, Clostridium chauvoei, Myoplasma mycoides, Streptococci, Strophylococci, Pseudomonas, E. coli. Pasteurella, Actinobacillus.*

Pathogenesis :





Sr.No	Species	Valve predilection
1	Cattle	RAV valve followed by LAV valve
2	Horse	Aortic valve followed by LAV and RAV valves
3	Sheep and swine	AV valves

Clinical Findings :

- Moderate fluctuating fever.
- Intermittent lameness due to pain in joints and limbs.
- Progressive loss of body weight and weakness.
- Pale mucosae (due to depression of bone marrow caused by toxins)
- Persistent tachycardia and a pounding heart beat.
- On auscultation cardiac murmurs.
- Thrill on palpation of cardiac area.
- Course is usually long (Several weeks or months)

Diagnosis :

1) History : Low grade infection / septic process viz. Mastitis, Metritis, TRP, arthritis poor body condition, intermittent/ shifting leg lameness.
2) Clinical signs. viz. presence of cardiac murmurs or persistent tachycardia.
3) Clinical pathology : Non regenerative anaemia, leucocytosis, neutrophilia and hypergammaglobulinemia indicative of chronic bacterial infection.
4) Echocardiography and ECG.
5) PM lesions : Large cauliflower like lesions on valves.

Differential diagnosis :

1) Pericarditis : Muffling/ splashing sound/ pericardial friction rub.
2) Pleurisy : Pleuritic rub is heard.

Treatment :

- It is not successful because of difficulty encountered in controlling infection.
- The drug should be selected on the basis of drug sensitivity parenteral administration of procaine benzyl penicillin in large doses. i.e. @20,000 iu/kg. daily in combination with gentamicin for 7-10 days is effective.
- Ampicillin or macrolide antibiotics viz. erythromycin is more effective.

- *Prolonged treatment is required*
- *Treatment is expensive*
- *Relapse is common*

- *Prognosis is poor.*

5. ANAEMIA

Definition : It is defined as qualitative and quantitative decrease in blood erythrocytes.

Etiology :

A) Excessive loss of blood (Harmorrhagic anaemia) :

1) Trauma to blood vessels : Accidents, surgical operations.
2) Bracken fern poisoning in cattle : Increase fragility of blood vessels and bone marrow suppression.
3) Sweet clover poisoning in cattle and sheep : Sweet clover contains coumarin which is an anticoagulant.
4) Warfarin poisoning in day A roderticide which is antagonistic to vit. K.
5) Purpura haemorrhagica in horses.
6) Thrombocytopenia caused by drugs, snake venom and mycotoxins.
7) Deficiency of vit. K, Vit. C deficiency produce thrombocytopeaia and prothrombia.
8) Gastric abomasal ulcers, epistaxis, haemoptysis.
9) Enzootic haematuria in cattle.
10) Pyelonephritis with bleeding in cattle.
11) Coccidiosis in calves.
12) Endoparasites.
    a) Cattle, buffalo, goat and sheep. *Haemonchus spp. Bunostomum spp. Fasciola spp.*
    b) Dog : *Ancylostoma caninum.*
    c) Horses : *Strongylus sp.*
13) Ectoparasites : Lice, ticks, fleas.
14) Hemophilia Type A in male dog and swine.

B) Increased destruction of erythrocytes (Haemolytic anaemina) :

1) Bacterial :
    a) Leptospirosis in dog and cattle.
    b) Bacillary haemoglobinuria in cattle.
2) Viral : a) Equine infectious anemia in horses.
    b) Feline infectious anemia in cat.
3) Protozoa :
    a) Babesiosis in all species.
    b) Anaplasmosis in ruminants.
    c) Theileriasis in dog.
    d) Ehrlichosis in dog.
4) Deficiency diseases :
    a) Post-partutient haemoglobinuria
    b) Copper deficiency.
5) Poisoning :
    a) Onion poisoning (n- propyl disulphide- toxic)
    b) Castor seed poisoning rape kale (saponin – toxic)
    d) Copper lead napthanene, arsenic.
6) Snake bite : Snake venom contans haemolytic enzyme. Lecithinase.
7) Water intoxication : Drinking of large quantity of cold water in calves.
8) Hypersensitivity to drug viz. sulfanilamide aspirin etc.
9) Isommune haemolytic anaenia in calves and foal
10) Autoimmune haemolytic anaemia is recorded in calves and horse but is rare.

C) Insufficient / decreased production of erythrocytes.

I) Nutritional anaemia :

Nutritional deficiencies of

i) Iron : Required for haemoglobin formation.

ii) Copper : Required for haemoglobin formation

iii) Cobalt : Required for utilization of iron for haemoglobin synthesis.

iv) Vit. B_{12} : Required for synthesis of RNA and DNA.

v) Folic acid : Pyridoxin, nicotinic acid and riboflavin

vii) Protein ; It is constituent of haemoglobin

II) Toxic Inhibition anaemia :

Bone marrow is normal but unable to utilize the haematinics resulting in decreased production of erythrocytes.

i) Chronic Interstital nephritis (Uraemia – suppression)

ii) Chronic infections : T.B., Brucellosis, Johne's disease

iii) Intestinal parasitism

iv) Ionising radiation

v) Chemical drugs e.g. Chloramphenicol, Streptomycin, sulpha drugs.

III) Aplastic anaemia :

This occurs due to aplasia of bone marrow.

1) Primary or idiopathic – it is rare

2) Secondary a) Chronic haemorrhage e.g. intestinal parasitism, gastric ulcers

b) Ionising radiation

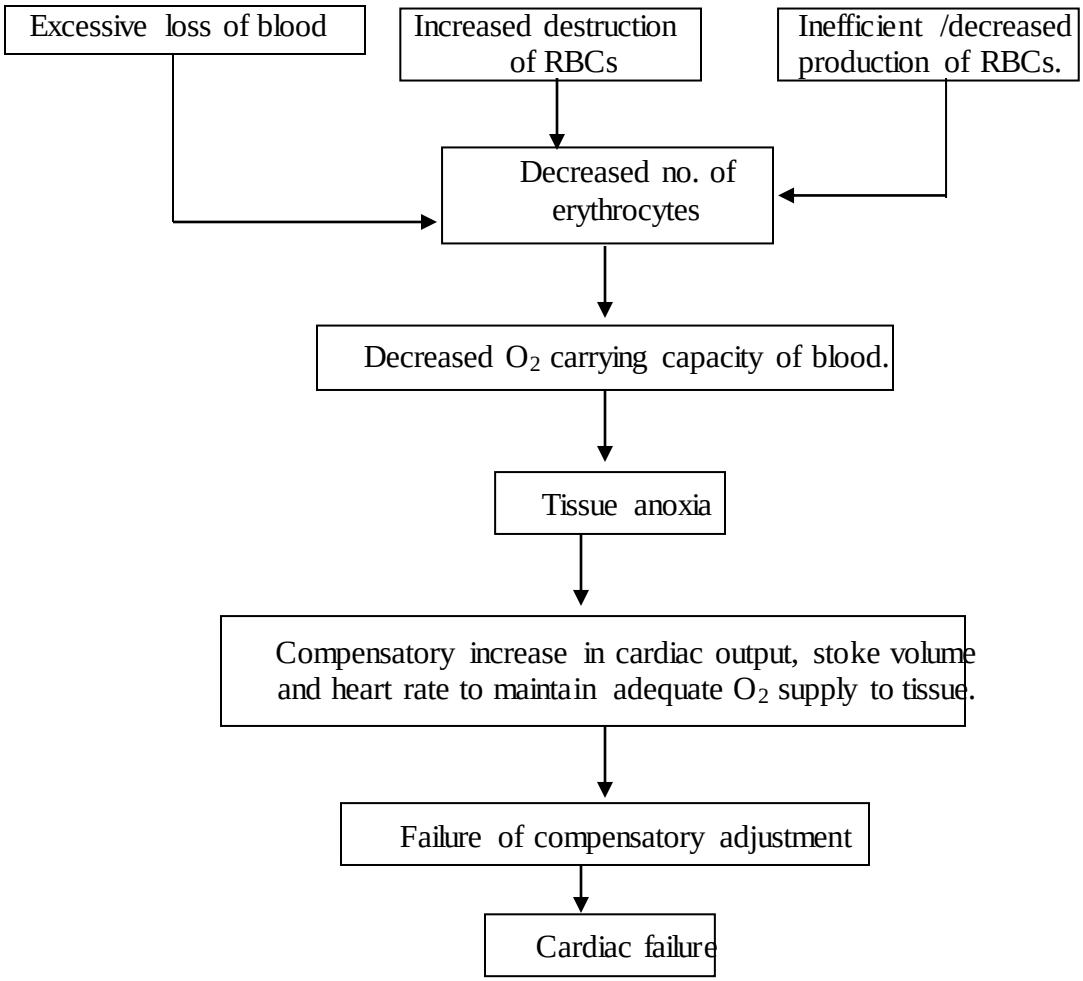
IV) Myelopthisic anaemia :

This is results from replacement of bone marrow by other tissues.

1) Primary : Neoplasms of reticuloendothelial system.

2) Secondary : Metastasis of other tumors. Osteodystrophias.

Pathogenesis :



Clinical signs :

- Pallor of mucosae is the outstanding clinical signs..
- Muscullar weakness.
- Dullness and depression
- Partial to complete anorexia.
- Tachycardia increased intensity of heart sounds initially and decreased intensity in
- later stages.
- Dyspnoea in later stages.
- Jaundic haemoglobinuria, hematuria, oedema and/or bleeding may be present.
- Hemic murmurs which is systolic in timing and waxes and wanes each respiratory cycle reaching at its maximum at the peak of inspiration.

Daignosis :

1) History : Haemorrhage, haematuria, haemoglobinuria, malnutrition.
2) Clinical signs : Pallor of mucsosae and weakness
3) Haematology : decrease PCV, TEC
    a) Iron and Copper deficiency : Microcytic hypochromic anaemia
    b) CO/Vit B_{12} deficiency macrocytic normochromic anaemia.
    c) Haemolytic : Macrocytic normochromic followed by microcytic hypochromic
    d) Haemorrhage : Normocytic normochromic anaemia
    e) Aplastic aneamia : Normocytic normochromic non- regenerative anaemia.
4) Blood smear may be positive for haemprotozons.
5) Serum analysis : Decrease in Fe, Cu, CO, Vit B_{12} total proteins.
6) Fecal examination : May reveal parasitic eggs.

7) Evidence of ectoparasites.
8) Urine analysis : Benridiae test positive in haematuria of harmoglobine cases.

Haemolytic anaemias are characterized by normal total protein, bilirubinemia and bilirubinuria

Immune mediated haemolytic anaemia show increased erythrocyte fiagility erythrocyte agglutination and a positive antiglobulin (Coomb's test)

A myeloid erythroid ratio less than 0.5 and a reticulocyte count greater than 5% are indicative of erythrocyte regeneration and ratios > 0.93 with normal reticulocyte count are indicative of non regenerative or hyposlastic anaemia.

Regenerative anaemias are caused by haemorrhage or haemolysis in the presence of normal bone marrow

Non regenerative anaemias are caused by primary or secondary bone marrow disorders in red cell production.

Low plasma protein is indicative of acute or chronic haemorrhage or malnutrition
Normal or high plasma protein is indicative of haemolytic anaemia.

Treatment:

I) Specific treatment to correct primary cause :

i) Antibiotics in bacterial infections. e.g. *Streptopenicillin* @ 10-30 mg /kg b/m
ii) Antiprotozoan drugs : a) Babesiosis : Inj. berenil single dose@ 3.5 – 7 mg/kg IM
b) Theileriasis inj Berenil (two dose) + inj. Oxyletracyclin @ 10-20 mg/kg for 5-7 days I/V
c) Anaplasmosis : Inj. oxytetracyline @ 10-20 mg/kg I/V for 5-7 days
iii)Ectoparasiticides : e.g Butox liquid@ 2-4 ml/lit spray
iv) Anthelmintics : e.g Albendazole @ 5-10 mg /kg orally.
v) Styptics in haemorrhage e.g Stadren @ 10 ml IM inj Vit. C, Vit. K, of calcium preparation parenterally for control of bleeding
vi) Supplementation of diet with protein, Fe, Cu, CO, Vit. B_{12}
vii) Polyvalent antisnake venon in snake bite cases.

2) Supportive treatment :

I] a) Haematinic mixture : Ferrous sulphate (5gm) + Cobalt sulphate (0.2gm) + Copper sulphate (0.2 gms in jaggery orally)
b) Injectable iron preparations e.g Imferon / Feritas @ 5-10 ml I/M in large animal, @ 1-2 ml in small animal on alternate days.
ii) Liver extract B-complex
e.g Inj. Belamyl / livogen @ 5-10 ml I/M in large animals
& @ 1-2 ml IM in small animals
iii) Plasma expanders viz. Harmacel/ dextran @ 10-15 ml/kg IV
Renal perfusion should be maintained in haemolytic anaemia by giving fluid therapy
v) Cases of autoimmune haemolytic anaemia respond well to corticostesroids.
vi) Blood transfusion in severe cases.

6. LYMPHANGITIS

Definition : It is denotes inflammation and enlargement of lymph vessels.
It is usually associated with lymphadenitis.

Etiology :

1) Glanders in horses.

2) Ulcerative lymphangitis due to *corynebacterium pseudotuberculosis* and streptococcus zooepdermicus in cattle and horses and caseous lymphadenitis in sheep and goat.
3) Skin farcy caused by *Nocardia spp*.
4) Cutaneous tuberculosis
5) Epizootics lymphangitis due to fungus i.e. *Histoplasma farcinosum*

Symptoms :
1) Ulcers usually exists at the site of infection.
2) Lymph vessels are enlarged, thickened and tortous and usually have secondary ulcer.
3) Local oedema may results from lymphatic obstruction.

Diagnosis :
1) symptoms
2) Examination of discharge for the presence of bacteria or fungi.

Treatment :

1) Early treatment is essential to prevent spread.
2) Remove foci of infection by surgical excision.
3) Give specific medical treatment
a) Antibiotics e.g. *inj*. *Streptopenicillin* @ 10-30 mg/kg I/M
b) Sodium iodide as 10% solution 1 gm/12kg body weight I/V

7. LYMPHADENITIS
(Lymphadenopathy)

Definition: Inflammation of lymphnodes means lymphadenitis.

- It most commonly occurs in response to a distal infection in the regions of body served by lymphnodes.
- Lymphadenopathy may be due to infection or neoplasms of lymph node.

Etiology:

A) Infectious causes :

1) Bacterial diseases :

a) Caseous lymphadenitis of sheep caused by *Cornybacterium pseudotuberculosis.*
b) Ulcerative lymphsngitis in horses and cattle due to *Cornybacterium pseudotuberculosis.*
c) Strangles in horses by streptococci equi.
d) Lymphadenitis by *Streptococci* zooepidemicus
e) Cervical adenitis of pigs caused by *Streptococci, Actinomycosis* and *Pasteurella multocida*.
f) Skin tuberculosis.
g) Actinobacillosis.
h) Pasteurella multocida
i) Tick pyaemia in sheep caused by *Staphylococcus aureus*.

2) Viral diseases

a) BMC
b) Ephemeral fever
c) Bovine encephalomyelitis

3) Protozoan diseases :

a) East Coast Fever / Thieleriosis

B) Neoplasm of lymphnodes

Lymphosarcoma

Symptoms :

1) Enlargement of lymphnodes. On palpation there may be pain and heat in acute cases.
2) Secondary signs : Respiration is difficult in case of enlargement of retropharyengeal lymphnodes. Oesophageal obstruction in enlargement of mediastinal lymphnodes.
3) Lymphosarcoma : Painless swellings of lymphnodes Chronic course

Diagnosis :

1) Clinical signs : Enlargement of lymphnodes
2) Needle biopsy for cytology and culture.

Differential diagnosis of lymphadenopathy

1) Theileriasis :

- History of tick infestation common in young animals and cross breed cattle.
- Persistent high fever accompanied by anaemia.
- Jaundice, blood smear positive for theileriasis and Koch blue bodies.

2) Bovine ephemeral fever :

- It usually occurs during rainy season in cattle and buffaloes.
- Transmitted by *Culicoides.*
- Three days fever; muscle stiffness.
- Recovery within three days.

3) BMC :

- Sporadic.
- High persistent fever followed by diarrhoea.

- Oral / ocular lesions in late stage.

4) TB :
- Chronic disease.
- No response to antibiotics and NSAIDS.
- Lymphnode biopsy positive for acid fast organism.

5) Actinobacillosis & pasteurellosis :
- Good response to antibiotics and antiinflammatory drugs in acute cases.

6) Lymphosarcoma :
- Chronic disease.
- Neoplasm, Painless and cold swelling of lymphnode.
- Histopathological examination help in confirmation.

Treatment :

1) In acute cases :
   - Antibiotics e.g. *Streptopenicillin* @ 10-30 mg/kg IM for 5 – 7 days.
   - Antiinflammatory and analgesics : e.g. inj Meloxicam @ 0.2-0.5 mg/kg IV for 5-7 days.
2) In chronic cases :
   - Use of counter irritants in chronic cases viz. massage by iodine ointment and hot foementation.

8. OEDEMA

Definition :- It means excessive accumulation of fluid in the tissue spaces.

Etiology:

1. Increased hydrostatic pressure :

a) Congestive heart failure resulting from pericarditis , valvular diseases and myocarditis .
b) Portal hypertension due to hepatic fibrosis causes ascites in small animals .
c) Periparturient udder oedema :
- It is Physiological oedema.
- It is due to pressure on mammary vein by large foetus in late pregnancy.
- It is common in high yielding particularly first calvers in cattle.

2. Decreased plasma colloidal osmotic pressure :

It is due to hypoproteinemia caused by

a) Malnutrition: Low protein diet results in inadequate plasma protein synthesis.
b) Liver disorders : Failure of synthesis of plasma proteins.
c) Renal diseases : Continued loss of proteins through urine.
d) Chronic haemorrhage : Continued blood loss results in hypoproteinaemia.

The common causes of chronic blood loss are as follows :

i. Heavy infestation: blood suking endoparasites .
   e.g. *Fasciola spp, Bunostomu spp, Haemonchus spp,* in ruminants .
   Ancylostoma in dog and *Strongyles* in horses .
ii. Heavy ectoparasitic tick infestation.
iii. Gastric / Abomasal ulcers :

e) Protein losing enteropathy (malabsorption): This condition is seen in chronic intestinal disease .(e.g. Oesophagostomiasis, Johne's disease which results in leakage of protein from body in to GI tract)

3.Obstruction of lymph / blood vessels:

a. Pressure on lymph / blood vessels from outside by cyst , abscess, tumors etc.

b. Lymphangitis , phlebitis , venous thrombosis .
c. Blockage of lymph vessels by parasites e.g. filaria.

4. Damage to blood vessels :

a. Bacterial diseases :- H.S., Anthrax , gas gangrene , Malignant oedema.
b. Viral diseases : Equine infectious anaemia, Equine viral arteritis .
c. Allergic conditions :- Release of vasodilators viz. Histamine resulting in increased capillary permeability favoring passage of fluids and proteins to the interstitial spaces.
d. Snake bite.
e. Some plant poisonings.
f. Anaemic anoxia.
g. Vitamin A deficiency.

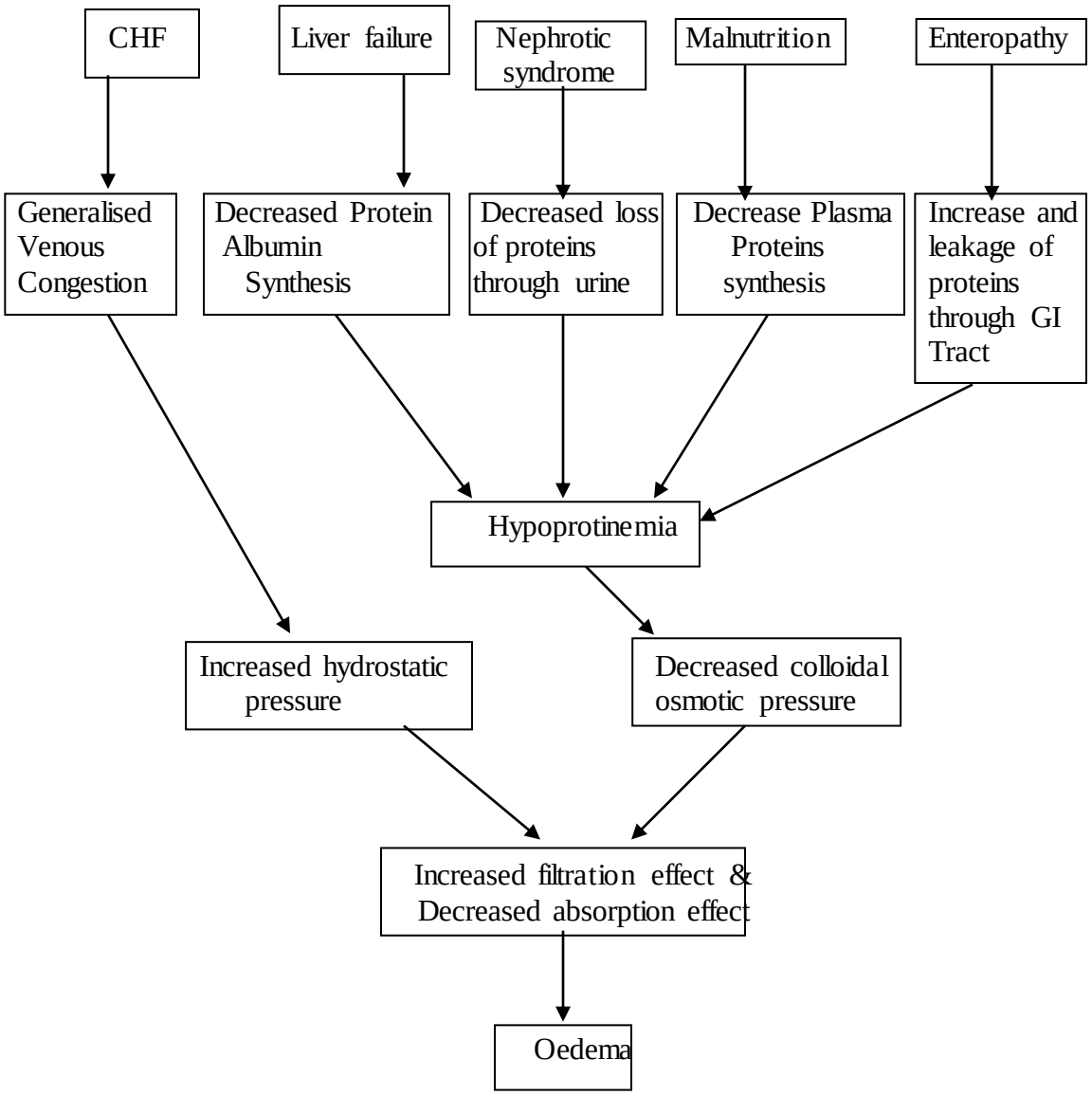
Pathogenesis:

To understand how and why fluid accumulates in the tissue spaces, it is necessary to consider capillary and interstitial fluid dynamics.

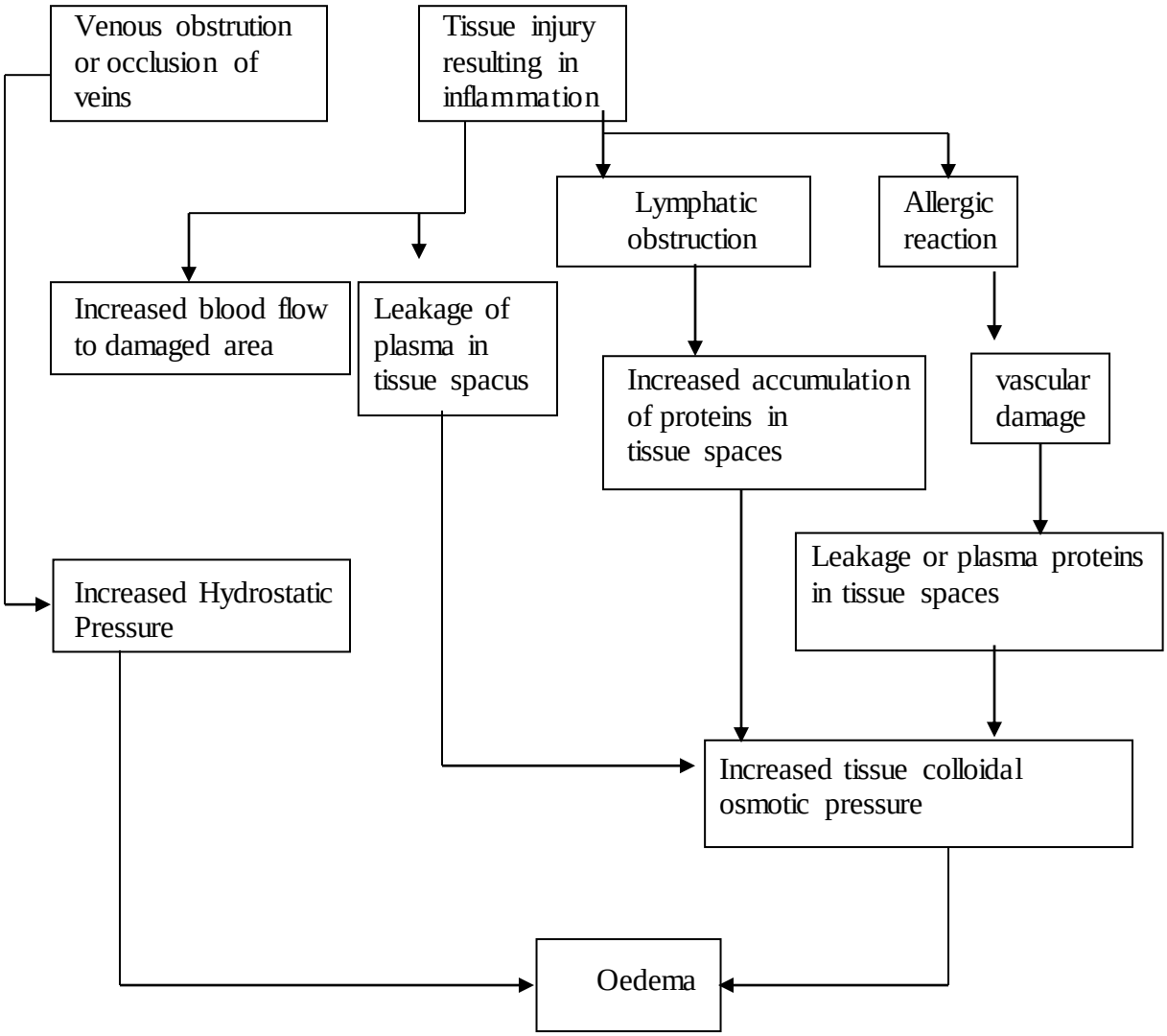
Blood capillary has two ends.

Arterial end	**Venous end**
1. Hydrostatic pressure:	
a. Blood – 45mm Hg	a. Blood -15mm Hg
b. Tissue fluid - 1-2mm Hg	b. Tissue fluid 1-2mm Hg
:. Filtration effect = 45-2=43mm Hg	Filtration effect =15-2=13mm Hg
2. Colloidal osmotic pressure :	
a. Blood plasma : 30mm Hg	a. Blood plasma 30 mm Hg
b. Tissue fluid : 2-3 mm Hg	b. Tissue fluid 2-3mm Hg
Absorption effect = 30-3=27mm Hg	Absorption effect 30-3=27mm Hg
3.Net filtration effect =16mm Hg	Net absorption effect =14mm Hg
4. At arterial end hydrostatic pressure is more than colloidal osmotic pressure so fluid pass into tissue spaces.	At venous end colloidal osmotic pressure is more than hydrostatic pressure so fluid enters in to vessels.

Causes and Pathogenesis of Generalized oedema:



Causes and pathogenesis of local oedema:



Clinical findings :

- Oedema may be local or generalised
- Oedema may be inflammatory or non – inflammatory.

1. Oedematous swellings are soft painless, pits on pressure.
2. Ascites : Distension of abdomen, fluid thrill on percussion . It is common in dogs.
3. Hydrothorax :- Dyspnoea, muffled or splashing sound on auscultation of lower thorax.
4. Hydropericardicum :- Muffled or splashing heart sounds, tachycardia.
5. Cerebral oedema :- Nervous singes .
6. Brisket oedema :- Swelling of brisket region .
7. Anasarca :- Oedema of indermandibular space , brisket ventral aspect of thorax and abdomen .
8. Allergic oedema:- Urticaria, itching and exudation.

Diagnosis:-

1.History of undernutrition , diarrhoea, bleeding, insect bite.
2. Symptoms of involvement of liver , heart , kidney & intestine.
3. Biochemistry :- Hypoproteinemia.
4. Haematology :- Decrease in Hb .PCV& TEC (anaemia), Eosinophilia in allergic oedema .

5. Urine analysis: Proteinuria in kidney disorders.
6. Faecal examination:- Parastic eggs in endoparasitism
7. Liver function test (e.g. Icterous index , Vanden bergh test , Fouchets test positive) serum protein decrease (normal :6-8gl/dl)

Differential diagnosis :-

- Pleurisy :- Abdominal respiration , Inspiratory dyspnoea, chest pain , rubbing sound in lung area.
- Pericarditis : - Tachycardia, tugular pulsation , brisket oedema, muffled/ splashing heart sounds.
- Uroperitoneum :- History of complete cessation of urination and on abdomeanocentesis. There is evidence of urine.
- Peritonitis:- Reluctance to move , arching of back , serosangiunous or purulent fluid on abdomencentesis X-RAY positive for foreign body.

Treatment:-

a) Allergic oedema- antihistaminics :
(e.g. inj. Avil @ 0.5 -1mg/kg IM) and corticosteroids ex .inj. dexona @ 0.04. mg/kg IM.

b) Cardiac oedema : Rest, salt free diet, diuretics (e.g. inj Frusonamide @ 1-2 mg/kg IM) and cardiac tonics (digoxia@1-2mg/kg IM)

c). hepatic oedema: rest, salt-free diet, diuretic, liver extract, dextrose 10% oral antibiotics

d) Renal oedema: Rest, salt free diet, antibiotics, corticosteroids
e.g. nutritional oedema, haematinics, (Iron, copper, cobalt) Multivitamns (Vit.A, Vit. B_{12}) protein rich diet and plasma expanders.

f) Parasitic oedema : Anthelmintics (e.g. Albendazole @ 5 -10mg/kg PO) Ectoparasiticides (e.g. Butox 2ml/lit spray) protein supplements and haematinics.

g) Mammary oedema: Rest, diuretics, Protien rich diet

h) Inflammatory oedema: Antinflammatory drugs and antibiotics

CHAPTER-7

DISEASES OF URINARY SYSTEM

Diseases of the bladder and urethra are more common and more important than diseases of kidneys in farm animals.

Principles of renal insufficiency :

- The kidneys excrete the end products of tissue metabolism and maintain fluid and electrolyte balance including acid-base balance by selective excretion of these substances.
- Glomerulus is a semipermeable filter which allows easy passage of water and low molecular weight solute but restricts passage of high molecular weight substances like plasma proteins.
- Glomerular filtrate is identical to plasma except that it contains little proteins or lipids.
- The priniciple mechanism that regulates water reabsorption by the renal tubules is antidiuretic hormone (ADH) and vasopressin.

Renal insufficiency and Uraemia :

- ***Renal insufficiency*** :It Means partial loss of kidney function.

- ***Renal failure*** : It is the terminal stage of renal insufficiency.
- The inability to concentrate urine is clinically evident as polyuria which is characteristic of developing renal insufficiency.
- Renal failure is seen as the clinical state of uraemia and is biochemically characterised by an increase in blood urea nitrogen and creatinine (azotaemia).
- ***Uraemia*** : It is systemic state which occurs in the terminal stages of renal insufficiency and characterized by rise in NPN substances in blood.
- Renal insufficiency can occur due to abnormality in
  a) Rate of renal blood flow.
  b) The glomerular filtration rate.
  c) The efficiency of tubular reabsorption.
- Circulatory emergencies such as shock, dehydration, haemorrhage results in marked reduction in glomerular filtration.
- In haemoglobinuric nephrosis, glomerular filtration is unaffected but tubular reabsorption is markedly depressed.
- In interstitial nephritis , the primary and major lesion is tubular degeneration.

Causes :

***1. Prerenal causes* :**

- Congestive heart failure.
- Acute circulatory failure
- Haemoglobinuric and myoglobinuric nephrosis.
- Dehydration, shock, haemorrhage.

***2. Renal causes* :**

- Glomerulonephritis
- Interstitial nephritis
- Pyelonephritis
- Embolic nephritis.
-

***3. Post renal causes* :**

- Vesical or urethral calculi.
- Rupture of urinary bladder.

Clinical manifestations of urinary tract diseases :

The major clinical signs of urinary tract diseases –

1. Abnormalities in urine
2. Abnormal constituents of urine
3. Variation in daily urine flow
4. Abdominal pain and painful and difficult urination.
5. Abnormal size of kidneys and abnormalities of the bladder and urethra.
6. Acute and chronic renal failure.

1. Abnormalities in urine :

i. *Colour :*

i) Straw colour - Normal
ii) Clourless - Dilute
iii) Amber colour - Bilirubinuria
iv) Brown colour - Pyuria and haematuria.
v) Coffee colour - Haemoglobinuria
vi) Red colour - Haematuria

ii. *Transparency* :

i) Clear - Normal
ii) Turbid or cloudy - Increase cells, crystals and mucus.

iii. *Specific gravity* :

i) 1.015 – 1.035 - Normal
ii) Decreased - Polyuria
iii) Increased - Oliguria

2. Abnormal constituents of urine :

i) *Proteinuria* :It mean presence of protein in urine

- Normal urine contains only small amount of proteins.
- Proteinuria is usually observed in haemoglobinuria, myoglobinuria, haematuria, UTI – glomerulonephritis, renal infarction, tubular nephrosis and amyloidosis.
- Mild proteinuria is observed in fever.
- Presence of proteinuria and casts in urine indicates kidney damage.

ii*) Haematuria* :

- It is the presence of blood in the urine.
- ***Causes* :**
- *Renal* : Pyelonephritis, calculi, glomerulonephritis,
  Postrenal : Cystitis, urethritis, calculi, trauma.
  Extra urinary causes : Uterine infection, trauma to vagina and penis, prostate infection.

iii) *Haemoglobinuria* :

- It is presence of free haemoglobin in urine.
- *True haemoglobinuria* : Hb filtered by the glomeruli.
- *False haemoglobinuria* : Hb released by lysis of RBCs in dilute or aged urine.
- *Causes* **:** Babesiosis, PPH, leptospirosis, bacillary haemoglobinuria.

iv*) Pyuria* :

- It means presence of pus or leucocytes in urine.
- It indicates UTI.

v) Crystalluria :

- It means presence of crystals in urine.
- Crystals in the urine of herbivores animals have no special significance unless they occur in very large number and are associated with signs of irritation of the urinary tract.

vi) Glycosuria :

- It means presence of glucose in urine
- Glycosuria in combination with ketonuria occurs in diabetes mellitus only.

3. Variation in daily urine flow / output :

i) *Polyuria* : It means increase in volume of urine.

Causes :

- Diabetes insipidus.
- Diabetes mellitus
- Chronic interstitial nephritis
- Administration of diuretics drug including corticosteroids.

ii) *Oligouria* : It means reduction in daily urine output.

Causes : Dehydration, CHF

iii) *Anuria* :It means complete absence of urine.

***Causes* :** Complete urethral obstruction in terminal stages of nephritis.

iv) Pollakiuria : Frequent passage of urine with or without increase in the volume of urine excreted.

***Causes* :** Cystitis, calculi in the bladder.
Urethritis and partial obstruction of the urethra.

v) Dribbling : is a steady, intermittent passage of small volumes of urine.

***Causes* :** Incomplete obstructive urolithiasis, Persistent uraemia
.

4. Abnormal urination :

i) Dysuria : It means painful or difficult urination.

***Causes* :** Cystitis, vesical calculus and urethritis.

***Signs* :**

- Frequent passage of small amounts of urine.
- Grunting may occurs and animal may remain in the typical
- Posture after urination is completed.
- Dysuria is usually associated with pyuria and haematuria.

ii) *Stranguria* : It is slow and painful urination.

***Causes* :** Cystitis, visual calculus, urethritis, urethral obstruction.

***Signs* :**

- Animal strains to pass each drop of urine.
- Groaning and straining may precede and accompany urination in urethral obstruction.
- Groaning and straining occurs after urination has ceased in urethritis.

iii) *Urinary incontinence* : It is defined as the lack of voluntary control of micturition and characterised by constant dribbling of urine.

***Causes* :**

a) Neurogenic : Paralysis of bladder due to cerebral and spinal cord lesions.

b) Non-neurogenic : a) Distension of bladder- urethral obstruction.
b) Bladder not distended-.
- Reduced bladder capacity.
- Urethral incompetence.

Signs : Urine scalding of perineum or urinary burn due to frequent wetting of the skin with urine.

5. Abnormalities of Kidneys, ureters, bladder :

- Enlargement or decreased size of kidneys may be palpable on rectal examination or detected by ultrasonography.
- Abnormaliteis of bladder viz. distension, rupture, calculi may be palpable on rectal examination.

1. NEPHRITIS

Definition : It means inflammation of kidney.

Classification :

A) Glomerulor nephritis :

i) It means inflammation of the glomeruli.

ii)It is very rare in animals.

iii)It is usually acute in nature.

iv)It may be primary or secondary.

a. Primary : Disease affects glomeruli
b. Secondary : Disease occurs secondary to diseases affecting other body system.

Causes :

- It is considered as an autoimmune disease.
- *E.Coli, Streptococci, ICH virus, Dirofilaria immitis* (heart warm) may act as antigen.

B) Interstitial nephritis :

It means inflammation of interstitial tissue of kidney.

i) It is very common in dogs and rare in other animals.
ii) It may be acute or chronic in nature.

Causes :

i) Bacteria *: E.Coli, Leptospira, Canicola, L. icterohaemorrhagica*.
ii) Virus : ICH (adenovirus)
iii) Parasites : *Diaoctophyma renale* (kidney worm)
iv) Toxins : Lead, Arscenic, mercury.
v) Drugs : Sulphonamide, paracetamol.

Kidney parenchyma undergoes inflammatory changes which blocks the tubules together with cellular infiltrations.

C) Pyelonephritis :

i)It means inflammation of renal parenchyma and pelvis.
ii)It is chronic suppurative inflammation of kidney.
iii)It is common in cows but has been recorded in sheep, goat, horse, buffalo and dog.
i) It is common in recently parturiated and advance peregnant females.

Causes :

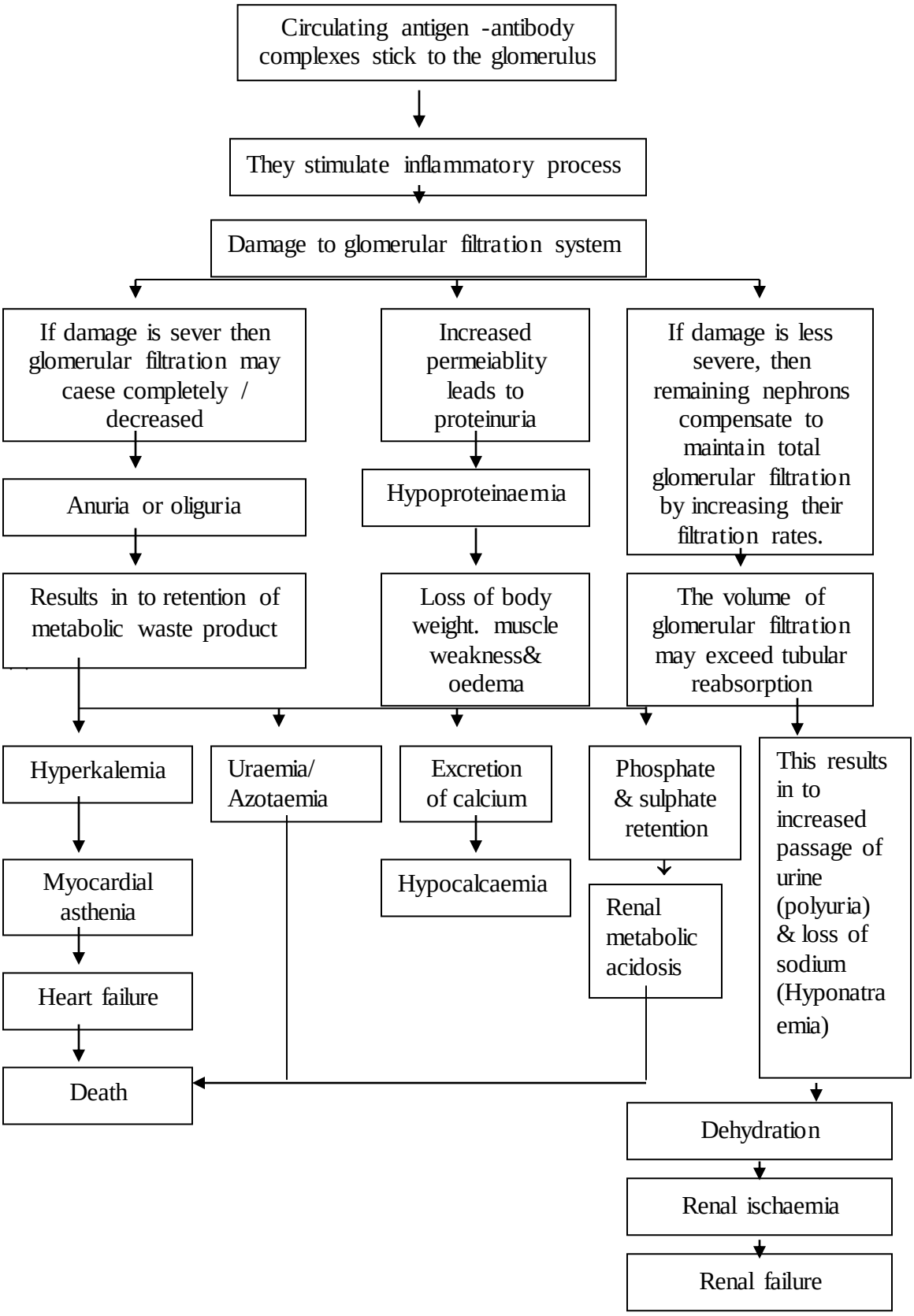
1) a) *Corynebacterium renale* (the most common) and *E. Coli* in cattle.
b) *Eubacterium suis* in pig.
2) Ascending infection from lower urinary tract or genital tract is the most common way of infection e.g. metritis, vaginitis, cystitis, urethritis.
3) Infection by haematogenous route e.g. septicaemia caused by P*seudomonas aeuroginosa* may also occur.

D) Embolic nephritis :

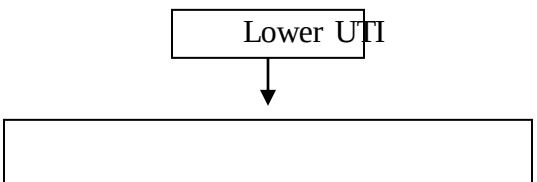
1) It means inflammation of kidney due to emboli
Emboli originate from the localised septic process.
a) Valvular endocarditis in all species.
b) Suppurative lesion in uterus, udder, naval region or peritoneal cavity in cattle.
2) Emboli associated with systemic infection.
a) Septicaemia in neonatal animals – Shigellosis in foal, Colibacillosis, Salmanellosis.
b) Erysipelas in pigs.
c) Septicemic strangles in horse

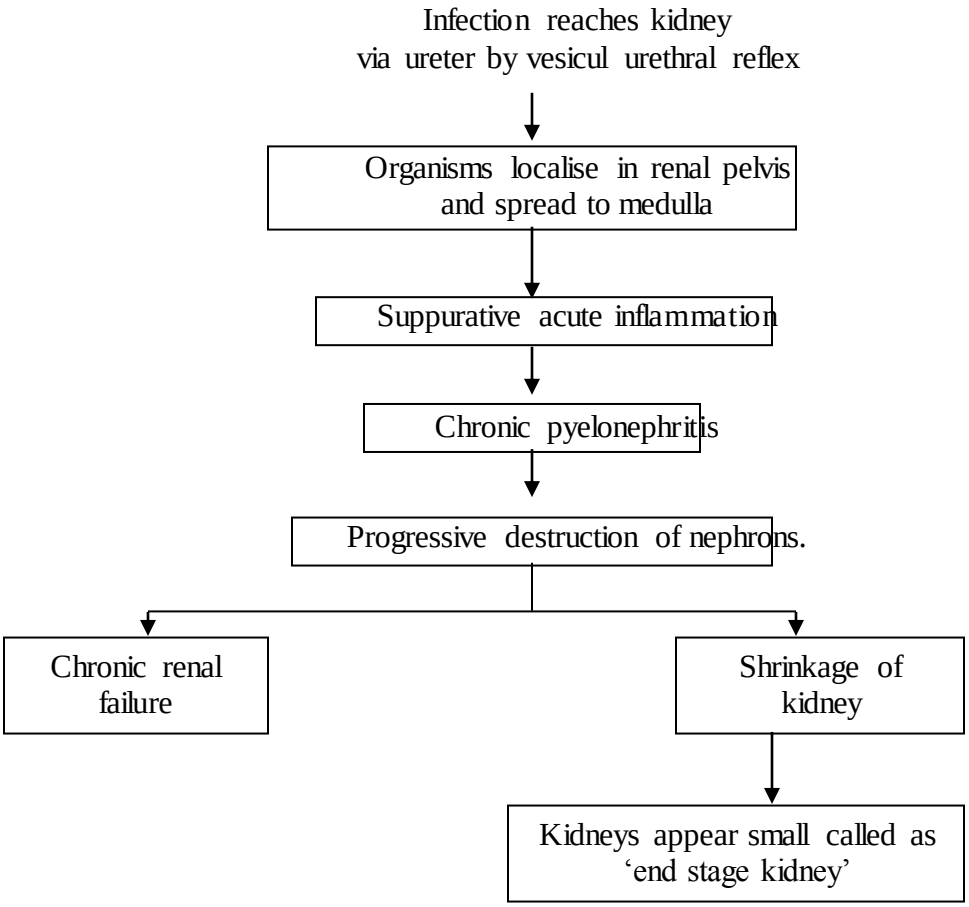
Pathogenesis :

A) Pathogenesis of Glomerulonephritis :



Pathogenesis of Pyelonephritis :




Infection reaches kidney via ureter by vesicul urethral reflex
Organisms localise in renal pelvis and spread to medulla
Suppurative acute inflammation
Chronic pyelonephritis
Progressive destruction of nephrons.
Chronic renal failure
Shrinkage of kidney
Kidneys appear small called as 'end stage kidney'

Clinical Signs :

1. Intermittent fever.
2. Slight to marked increased in heart and respiratory rates.
3. Anorexia/inappetence, suspended rumination, dullness, depression, muscle tremor.
4. Renal colic : Arching of back, stiff gait, tenderness of lumbar region.
5. Loss of body weight / muscle wasting i.e. emaciation.
6. Polydypsia and polyuria in chronic interstitial nephritis.
7. Anuria or oliguria in acute nephritis.
8. Turbid, foul smelling urine (pyuria) in pyelonephritis.
9. Red urine (Haematuria) in glomerulonephritis.
10. Frequent micturition and straining during urination i.e. stranguria.
11. Vomition in dog.
12. Anaemia
13. Dehydration
14. Oedema – puffy eye lids, anasarca.
15. Rectal palpation reveals enlargement and absence of lobulation of kidney-in pyelonephritis in bovine.

Clinical pathology :

1) Urinalysis:

i)Acute - High specific gravity and presence of RBC, WBC & cast, epithelial cells on microscopic examination.

ii) Chronic – low specific gravity with less cellular deposits.

iii)Proteinuria – It is cardinal sign of glomerulonephritis.

2) Blood biochemistry :

i) BUN – Increases (Normal: 6 – 27 mg/dl)

ii) Serum Creatinine – Increases (Normal: 1-2 mg/dl)

iii)Serum Total proteins – Decrease (Normal : 5.7 – 8.1 gm/dl)

Diagnosis :

1. History of metritis, retention of placenta, advance pregnancy / parturition in cows.
2. Clinical signs : Anuria/polyuria, abnormal constituents of urine, colic, dysuria, enlargement and loss of lobulations on per-rectal examination.
3. Urine analysis : Haematuria, pyuria, proteinuria.
   Microscopic examination reveals presence of epithelial casts and pus cells.
4. Serum biochemistry : Higher BUN and serum creatinine values.
5. Culture of urine for isolation of organism

Differential diagnosis of diseases characterised by polyuria and polydypsia :

	Characteristics	**Chronic interstitial nephritis**	**Diabetes mellitus**	**Diabetes insipidus**
1	Glycosuria	absent	present	absent
2	Specific gravity	low	high	low
3	Uraemia	present	absent	absent
4	Response to specific drugs	No response to Insulin/ADH	Response to insulin	Response to ADH

Treatment :

A) General care :

1. Give rest to the animal.
2. Provide proteins of higher biological value.
3. Provide low sodium diet.

4. Provide adequate drinking water.

B) Specific treatment :

- Antibiotics for 2-4 weeks.
  Selection of antibiotic depends upon urine pH:
- **a)** Drugs work well in alkaline pH. Sulpha–trimethoprim, Aminoglycosides (Contraindicated in pyelonephritis)
  b) Drugs work well in acidic pH : Tetracycline, nitrofuranin
  .
  c) Drugs not affected by pH: Ampicillin, Amoxycillin, Nalidixic acid, Quinolones.

C) Supportive and symptomatic treatment :

1. Urinary acidifires or alkalizers to change pH as altered pH reduces microbial population. e.g. NH_4Cl, Sodium acid phosphate.
2. Use of urinary antiseptics i.e. Hexamine (4gm) + sodium acid phosphate (30 gm) – orally.
3. Use of diuretics e.g. Furosemide @ 1mg/kg body weight i/m in cattle and 5mg/kg body weight IM for dog. It restores urine flow.
4. Use of steroids to prevent formation of immune complexes in glomerulonephritis e.g. Dexona @ o.o4mg/kg i/m.
5. Parenteral administration of B-complex may given e.g. Belamyl @ 5 ml i/m.
6. Antacids in case of gastritis due to uraemia e.g. Ranitidine @ 1-2 mg/kg I/v / orally.

Differential diagnosis of pyelonephritis in bovines :

	Character	Nephritis	Metritis	TRP
1	Urine	Abnormal through out urination	Abnormal in the beginning and at the end	Normal
2	Recurrent tympany	Absent	Absent	Usually present
3	Rectal palpation	Kidney enlarged, smooth and show loss of lobulation	Kidney normal uterus enlarged and full of pus	Kidneys and uterus normal. Rumen impacted or distended
4	BUN and creatinine	Increased	Normal	Normal
5	X-ray	No evidence of foreign body	No evidence of foreign body.	evidence of foreign body

2. **CYSTITIS**

Definition : Inflammation of urinary bladder is called as cystitis.

Etiology : ,1) Trauma : Calculi, improper catheterisation or direct injury, Corrosive chemicals, toxic substances,

2) **Bacteria :** *E.coli, Corynebacterium renale, Proteus spp*.
   Infection may be :
   a) Ascending infection from urethra or genital tract(vagina / uterus).
   b) Descending infection from suppurative nephritis.

Infection is mostly ascending type.

3) Urine stasis: Urolithiasis (calculi), neoplasms, structures of urethra, dystokia, prolapse of uterus

Urine statis favours microbial growth

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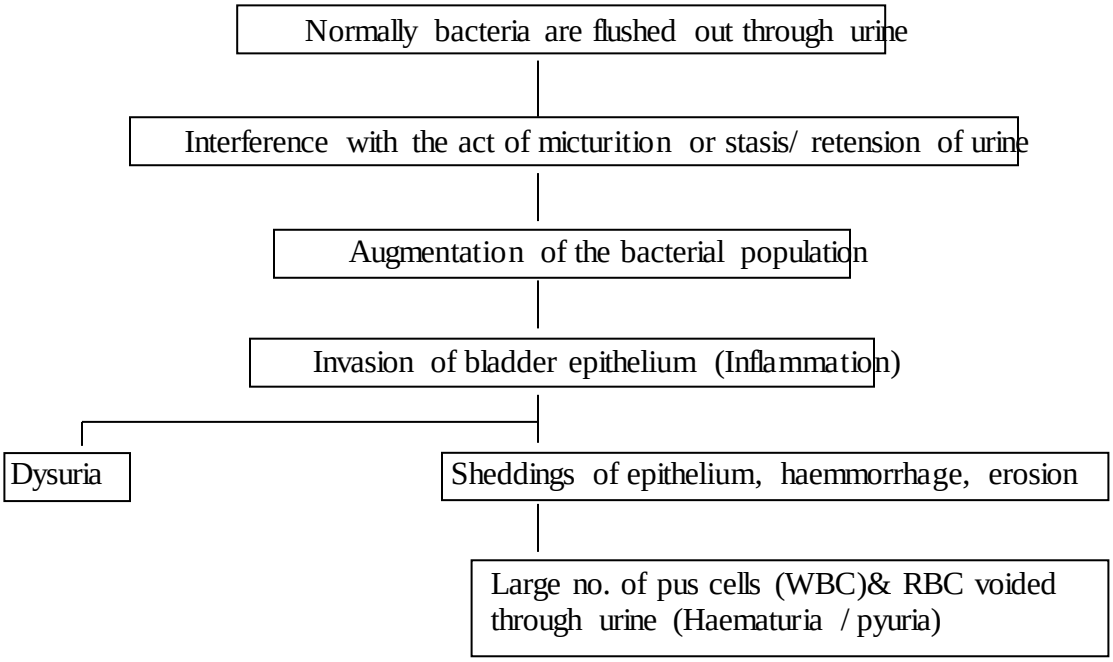
Pathogenesis :



Clinical signs :

A) Acute cystitis :

1. Mild fever, anorexia.
2. Dullness and depression
3. Frequent painful micturition (Dysuria / strangurea)
4. Pyuria (cloudy, turbid urine)
5. Haematuria (Red urine)
6. perrectal palpation of bladder reveals thick bladder wall and pain reaction.
7. Straddle gait.

B) Chronic cystitis :

- Signs are less severe.
- Bladder wall is thickened but less sensitive.

Diagnosis :

1. History of metritis, dystokia, prolapse of uterus or retention of placenta in females and urinary caculi in males.
2. Clinical signs : Abnormal urine and painful micturition.
3. Urine analysis : Red/turbid urine, presence of WBC, RBC, bacteria and bladder cells.
4. Culture of urine sample for isolation of organism.

Treatment :

A) Removal of primary cause :

1. Antibiotics e.g. streptopenicillin @ 10-30 mg/kg i/m for checking infection.
2. Surgical intervention in urolithiasis and neoplasms.

B) Supportive treatment :

1. Antispasmodics to give relief from pain. e.g. Dicyclomine @ 0.5 mg/kg i/m
2. Urinary sedative prevents painful micturition e.g. Tr. hyoscyamus @ 30-60 ml orally –.
3. Urinary antiseptics :e.g. A mixtureof Hexamine- 4gm+ Sodium acid phosphate – 60 gm + Trecle – g.s.

Mfd. Haust Sig. Once daily

4. Use of urinary acidifiers / alkalisers to change pH i.e. NH_4Cl or sodium acid phosphate @ 40mg/kg orally.
5. Fluid therapy in high doses to flush/wash urinary tract.

3. **URETHRITIS**

Definition :It means Inflammation of urethra.

Etiology :

1) Trauma due to –
   a) Calculi
   b) Catheterisation
   c) Probing with instrument.
2) Infection :
   a) Ascending infection from prepuce, vulva, vagina and uterus.
   b) Descending infection from bladder and kidneys.

Clinical findings :

1. Pain during urination (dysuria)
2. Animals remain in urinating posture for a long time.
3. Severe straining during urination (stranguria)
4. Dribbling of urine from urethral orifice (urinary incontinence)
5. Turbid / bloody urine (Haematuria or pyuria)
6. Sometimes mild fever, inappetance

Treatment :

Same as cystitis.

Principles of treatment of urinary tract disorders

A) General care :

1. Give rest to animal.
2. Provide protein of higher biological value.
3. Provide adequate drinking water.

B) Specific treatment :

Antibiotics for 2-4 weeks.

a) Drugs work well in alkaline pH.
   - Sulpha – trimethoprim, aminoglycosides.

b) Drugs work well in acidic pH
   - Tetracyline, Nitrofurantion.

c) Drugs not affected by pH
   - Ampicillin, Amoxycillin, Nalidixic acid and fluroquinolones.

C) Supportive treatment :

1. Urinary acidifiers or alkalizers to change pH as altered pH reduces microbial population e.g. NH_4Cl or sodium acid phosphate.
2. Use of urinray antiseptics i.e. Hexamine 4 gm, soidum acid phosphage – 60 gm orally.
3. Use of diuretics e.g. furosemide @ 1mg/kg i/m in cattle and 5mg/kg i/m in dog to restore urine volume in oliguria.
4. Use of steroids to prevent formation of immune complexes in glomerulonephritis e.g. Dexona @ 0.04 mg/kg i/m.
5. Parenteral B-complex may given e.g. Belamyl @ 5-10 ml I/M.
6. Antacids in case of gastritis due to uraemia. e.g. Ranitidine @ 1-2 mg/kg I/V orally.
7. Antispasmodics to give relief from pain e.g. Dicyclomine @ 0.5 mg/kg I/M.

8. Urinary sedative to prevent painful micturition. e.g. Tr. belladona, Tr. hyoscyamus @ 30-60 ml orally.
9. Fluid therapy in high doses to flush the urinary tract.

Chapter 8

DISEASES OF NERVOUS SYSTEM
Clinical Manifestations of diseases of Nervous system

I) MENTAL STATE

A) Excitation States :

1) Mania : The animal act in a bizarre way and appears to be unaware of its surrounding.

***Signs* :** Licking, chewing of foreign materials, abnormal voice, constant bellowing, walking into strange surroundings, drunken gait.

***Causes* :** Encephalitis (Rabies, pseudorabies) polioencephalamalacia, lead poisoning, pregnancy toxaemia, nervous ketosis, severe hepatic insufficiency.

2) Frenzy : It is state characterized by violent activity with little concern for surroundings and the Animal is difficult to control and dangerous to other animals and human attendants also.

***Causes* :** a) Nervous disorders : Pseudorabies, toxic and metabolic brain diseases.
b) Diseases of other body system : Severe colic in horses, extreme cutaneous irritation, photosensitization in cattle.

3) Aggressive behaviour : It is the state characterized by Aggresiveness and willingness to attack over other animal, human and inert objects cause Early stages of rabies and pseudo rabies in cattle.

B) Depressive States :

1) Syncope : It is sudden and transient loss of conciousness caused by inadequate oxygen / glucose concentration in the brain.

Causes : Acute heart failure, traumatic contusion, lightening stroke, electrocution, cerebral hemorrhages.

2) Narcolepsy / catalepsy : It is a syndrome characterized by excessive sleep.

3) Stupor : It means partial loss of conciousness.

4) Coma : It is complete loss of conciousness or terminal point of depression.

C) Intermediate States :

1) Head pressing : It is a syndrome characterized by the animal pushing its head against fixed objects or leaning into stanction.

2) Compulsive walking : It is the state in which Affected animals put their head down and walk, appear blind, walks into an object and shows head pressing.

***Causes* :** Toxic and metabolic brain diseases, encephalomyelitis.
Diseases characterized by incresed intracranial pressure.

3) Aimless wandering : This is the syndrome characterized by aimless walking, severe mental depression. apparent blindness, protruded tongue and continuous chewing movements.

II) INVOLUNTARY MOVEMENTS

1. Tremors : It is continuous, repeatative twitching of skeletal muscles. It is visible and palpable.

2. Tics : It is spasmodic twitching movements for longer intervals than tremors.
***Causes* :** Traumatic injury to spinal nerve.

3. Tetany : It is generalized symmetrical and continuous contraction of muscles

***Causes* :** Tetanus, grass tetany.

4. **Spasms :** It means involuntary contraction of group of muscles.
5. **Convulsions / seizures / fits / ictus :** These are violent muscular contractions affecting part / all of the body and occurs for a short period as a rule.

   * It is a spasm affecting whole body.

   i) ***Tonic convulsions* :** These are Characterised by prolonged muscular spasm without period of relaxation.

   ii) ***Clonic convulsions* :** These are manifested by repeated muscular spasm alternate with period of relaxation.

a) Intracranial	b)Extracranial
Encephalitis Meningitis	Brain hypoxia, Acute heart failure
Encephalomalacia Cerebral oedema	Toxic and metabolic disease of C. N. S.
Brain Tumor	Hepatic encephalopathy Hypoglycemia Hypomagnesemia Poision : In-organic, plant and mycotoxins

6. **Chorea :** It is a condition characterized by involuntary, fast and irregular action of some group of muscles mainly those of face and proximal part of extremities.

III) GAIT

The essential component of neurological gait abnormality are weakness ataxia.

1. **Weakness / paresis :** It is a state where the power of contraction of muscles is weaker than normal but not entirely lost. i.e. parturient paresis.

   Signs : Dragging of limbs, trembling of the limb when animal bears weight on a weak, limb stumbling and knuckling of fetlock while walking on slope or in circle.
2. **Ataxia :** It is inco-oardination of movement without spasticity, paresis / involuntary movement mainly the limbs and body.

   ***Signs* :** Swaying from side to side of pelvis, trunk, and sometimes whole body. This results in abducted foot placement, crossing of limbs or stepping of opposite foot.
3. **Hypermetria :** It is lack of direction and increased range of movement.
4. **Hypometria :** It is a stiff or spastic movements of the limbs, little flexion of joints particularly carpal and tarsal joints.
5. **Dysmetria :** It is a term which includes both hypometria and hypermetria e.g equine degenerative myeloencephalopathy.

IV) DISTURBANCES IN SENSATION :

1. **Anaesthesia :** Loss of sensation / sensitivity
2. **Hypoaesthesia** : Decreased sensitivity
3. **Hyperaesthesia :** Increased sensitivity

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1. MENINGITIS

Definition : "It means inflammation of meninges characterized by fever, hyperaesthesia and rigidity of muscles."

- It occurs most commonly as a complication of pre-existing disease.
- It is usually caused by bacterial infection.
- Meningeoencephalitis is common in neonatal farm animals.
- Inflammation of duramatter is called as pachymeningitis
- Meningitis are of 2 types.
  1. Spinal Meningitis : Meningitis of spinal cord
  2. Cerebral Meningitis : Meningitis of brain

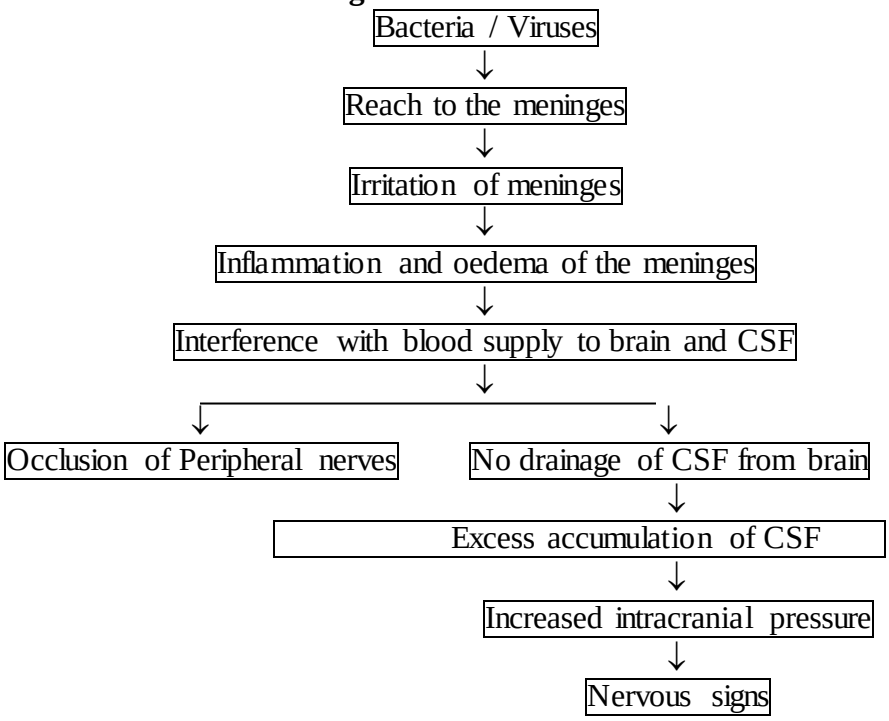
Etiology :

1. Bacterial causes :

1. Cattle : Listeriosis, streptococcal and staphylococcal infections, colibacillosis, pasteurellosis, lepiospirosis, corneybacterial infection and T.B.
2. Horse : Strangles.
3. Pigs : Glasser's disease *(Haemophilus suis*) Erysipelas, Salmonellosis.
4. Sheep and Goat : Pasteurellosis.

1. *Streptococcal and coliform septicaemia are probably the commonest causes of meningitis in neonatal farm animals.*
2. Viral causes :
   1. Cattle : sporadic bovine encephalomyelitis bovine malignant catarrh infectious bovine rhinotrachitis.
   2. Dog : Rabies, Canine distemper.
3. Misiellaneus causes :
   1. Middle ear infection. 2. Dehorning in cattle
   3. Bronchopneumonia. 4. Chronic sinusitis
   5. Naval ill 6. Subarachnoid haemorrhage
   7. Sharp penetrating wound. 8. Acute specific fever.

Pathogenesis :



Clinical signs :

- High fever $104 - 106^0$ F
- Slow and irregular – Biot's type / cheyne-stokes respiration

- Bradycardia.
- Rigidity of head and neck muscles, opisthotonus, locked jaw
- Excitation, restlessness, mania in the early stage
- Tremor, hyperesthesia and convulsions.
- Incordination in gait.
- Head pressing, circling movement in either direction.
- Blindness is common in cerebral meningitis.
- Depression, paralysis, recumbency and coma in later stages.

Clinical Pathology :

- **Examination of C. S. F. :**
  Turbid colour, high protein content, low level of glucose and increased cell count and Increased CSF Pressure.
- Culture of C. S. F. for isolation of organisms.

Diagnosis :

- History – circling movements
- Clinical signs
- Clinical pathology – Examination and culture of C.S.F.
- Blood, urine and stool Examination
- EEG and CT scan.
- X-ray : Space occupying lesions.

Differential diagnosis :

1. Tetanus, Poisoning ,Encephalitis Cerebral oedema, Spinal cord compression Encephalomalacia , Space occupying lesions , Brain trauma Chlorinated hydrocarbon poisoning and Hypovitaminosis – A.

(See details under encephalitis)

Treatment :

A) General care:

- Proper nurising and care
- Provide soft bedding to avoid bed sore formation
- Good Nutrition

B) Specific treatment :

Antibiotics :

Use of broad spectrum antibiotics at high doses to check bacterial infection for 5-7 days.
e.g.

i) Cefotaxime (@ 20 – 40 mg/kg IV/IM) is the most promising antimicrobial for treatment of meningitis.
ii) Sulphonamide (@ 100-200 mg/kg P O/ I V) is the drug of choice as it attains maximum concentration in C.S.F. (@ 100-200 mg/kg PO/ IV)
iii) Suplhatrimethoprim with or without gentamicin are also recommended.
iv) Ampicillin / Amoxycillin @ 10-20 mg/ kg I/m.
v) Chloramphenicol @ 4-11 mg/ kg I/m as it has been suggested crosses blood brain barrier

C) Symptomatic treatment :

1. Muscle relaxant and sedative to check spasms and convulsions.
   e.g.
   i) (Largactil) Chlorpromazine HCl @ 1 mg/ kg I/M ,I/V every 8-12 Hrs. interval.
   ii) Diazepam (Anxon/Calmpoze) @ 0.25 – 0.5 mg/kg I/M / I/V every 6 hours.
   iii) Chloral hydrate @ 20-30 mg P. O. for large animals.
   iv) Phenobarbitone @ 4-18 mg/kg P. O. in dog & cat.

2. Use of corticosteroids / Non steroidal anti-inflammatory drugs to reduce inflammation

e.g.: i. Dexamethasone (Dexona) @ 1 mg/kg I/M I/V
ii. Meloxicam @ 0.5 mg/kg I/M or I/V

3. Use of Hypertonic solution to reduce intracranial pressure :
e.g. i. Mannitol 10% or 20% @ 0.5 gm/kg I/v
ii. Dextrose 25% @ 0.5 gm/kg i/v

4. Use of parenteral fluids for nourishment and to check dehydration
e.g. Dextrose 5% or 10%. IV

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2.ENCEPHALITIS

Definition: "It is inflammation of brain characterised by hyperexcitability followed by paralysis and unconsciousness."

It may be primary or secondary lesion as a sequele of other diseases.

Etiology :

A. ***Bacterial agents*** **:** Listeriosis, Salmonellosis. Enterotoxaemia, Swine erysipelas, Necrobacillus infection.

B. Viral infection **:**

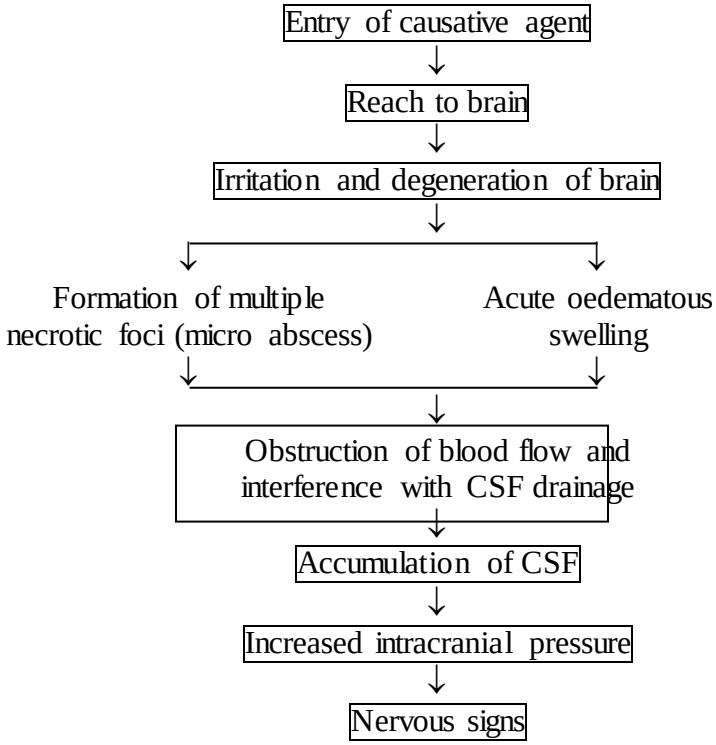
1. ***Cattle*** **: S**poradic bovine encephalomyelitis, Bovine encephalomyelitis, Bovine malignant catarrh. Bovine spongiform encephalopathy.
2. ***Horse*** **:** Infectious equine encephalomyelitis, Borna disease, Equine herpes viruses.
3. ***Pig*** **:** Pseudo rabies, viral encephalitis, Hog cholera, African swine fever.
4. ***Dog.*** **:** Canine Distemper.
5. ***Sheep*** **:** Louping ill, Scrapies.
6. ***Goat*** **:** Caprine viral leuko encephalomyelitis.

C. ***Parasitic causes*** **:** Migrating larvae of parasites e.g. *Oestrus ovis, Setaria spp. Multiceps multiceps*. Nervous coccidiosis, Cerebral babesiosis, Sarcocystosis.

D. ***Fungal :*** Cryptococcosis.

E. ***Toxic agents*** **:** Lead, Arsenic, Salt poisoning, Ipomea plant.

Pathogenesis :



Clinical Signs :

1. High fever, anorexia, tachycardia, depression.
2. Stage of excitement characterised by bellowing, kicking, pawing, mania, aggressiveness, hyperaesthesia, blind charging, clonic convunsions, frothy salivation, champing of jaws and nystagmy.
3. Signs of mental depression and head pressing in between nervous episodes.
4. Circling movements, staggering gait, ataxia, compulsive walking, blindness
5. Spastic paralysis may be in either sides,Recumbency, inability to stand, stupor, coma and death.
6. Excessive drooling and pharyngeal paralysis in rabies.
7. Paraesthesia and hyperaesthesia in pseudorabies and scrapie.
8. Hypermetria, in bovine spongiform encephalophathy.
9. Progressive ascending paralysis characterized by loss of sensation and weakness in hindlimbs initially followed by weakness in forelimbs in rabies.
10. Unilateral facial paralysis or Unilateral deviation of neck in listeriosis.

Diagnosis :

1. History
2. Clinical signs – hyperexcitability followed by paralysis and unconciousness.
3. Examination of CSF : Turbid high protein, low glucose and high cell count.
4. Culture of CSF for isolation of organism.
5. X-ray, EEG, CT.
6. Blood, Urine and stool examination.

Differential diagnosis :

1. **Meningitis :**
   - Fever, hyperaesthesia
   - Rigidity of neck muscles and trismus (lock jaw)
2. **Encephalomalacia (Softening of brain tissue) :**
   - History of grain engorgements
   - Fever is absent.
   - Good response to vitamin B (thiamine) therapy.
   - Polioencephalomalacia means degenerative changes in gray matter.

- Leukoencephalomalacia means degenerative changes in white matter.

3. Space occupying lesions (Brain cyst,tumor, abscess)

- Long / chronic causes course.
- Progressive increase in duration and frequency of nervous episodes.
- Unilateral circling
- No response to treatment.

4. Brain trauma :

- History of injury to head viz hitting of stone, beating on skull, fighting.
- Sudden onset, no fever, Evidence of injury

5. Acute cerebral o`edema / Salt poisoning :

- History of excess salt consumption.
- Less excitement.
- No hyperaesthesia, no fever.
- May respond to diuretics.

6. Chlorinated hydrocarbon poisoning :

- History ingestion of poison.
- Acute onset and short course.
- Absence of rigidity of neck muscles

7. Lead poisoning :

- History of access to lead, viz. licking of paint.
- Acute onset, short course, no fever.
- Good response to Ca disodium EDTA @ 70mg/kg I/V for 2-3 days.

8. Hypovitaminosis 'A' :

- History of young age and feeding of vitamin A deficient diet.
- No fever.
- Good response to vit. A therapy.

9. Tetanus :

- Generalised rigidity of muscles.
- Evidence of umbilical infection / wound / uterine infection.

Treament :

A) General treatment :

- Good nursing.
- Good bedding to avoid bed sore formation.

B) Specific treatment :

- Antibiotic Therapy : Use of broad spectrum antibiotics at high dose rate for 5-7 days should be used.
  - i) Cefotaxime @ 15-20 mg/kg I/V
  - ii) Sulphadimidine @ 100-200 mg/kg I/V, P/O.
  - iii) Chloramphenicol @ 4-11 mg/kg I/M
  - iv) Ampricillin / Amoxicillin @ 5-10 mg/ kg I/M
  - v) Enrofloxacin @ 2.5 – 5 mg/kg I/M

C) Symptomatic treatment :

a. Sedatives (tranquilizers) to control of consvulsions.

- i) Triflupromazine HCl (Siquil) (@ 0.1 mg/kg I/V). every 8-12 hrs
- ii) Diazepam (Anxol) @ 0.25 – 0.5 mg/kg I/M.every 6 hrs interval.
- iii) Chlorpromazine HCl (Largactil) @ 1mg/kg I/M every 8-12 hrs interval.

b. Corticosteroids to reduce inflammation of Intracranial pressure

- i) Dexamethasone. @ 0.04 – 1mg/kg I/M

c. NSAID is to reduce inflammation and Intracranial pressure.

- i) Meloxicam (Melonex)@ 0.2-0.3 mg/kg I/M.

d. Hypertonic solutions to reduce intracranial pressure.

i) Mannitol 10% or 20% @ 0.5 – 2 gm/ kg I/V.
ii) Dextrose 25% or 50% @ 0.5gm/kg I/V

e. Parenteral fluid therapy, for nourishment and to check dehydration e. g. Dextrose 5 % or 10 % I/V.

3.EPILEPSY

Definition: It is proxymal, self limiting functional cerebral disturbances characterized by seizure, fits and convulsions leading to sudden loss of conciousness.

Classification :

1. According to etiology :

A. Idiopathic

B. Symptomatic

A. Idiopathic / congenital Epilepsy :

- Unknown etiology, hereditary origin must be due to metabolic disturbances.

B. Acquired / symptomatic Epilepsy :

Local causes : Trauma / injury to cranium.

- Space occupying lesions.
- Inflammation.
- Degenerative changes in cerebrum.
- Otitis media.

General Causes : Fever – Febrile fit / febrile convulsions.

- cardiac disorder – Aortic stenosis, pulmonary stenosis, myocardial infarction.
- Metabolic disturbances – diabetes mellitus, viraemia, hepatic coma, alkalosis, decreased Calcium, distemper, cryptococcosis, toxoplasmosis.
- Round / tape worm infestation.

2. According to nature of fit :

A. Focal epilepsy.

B. General epilepsy

A. Focal epilepsy : In this type, abnormal electrical disturbances is localized to particular part of brain.

Types :

1. Psychomotor epilepsy / Temporal lobe epilepsy.
2. Jacksonian epilepsy
3. Myoclonic epilepsy.
4. Akinetic attack / focal fits.

B. General epilepsy :

In this type abnormal electrical disturbance spread all over the brain.

Types :

1. Grand mal/major epilepsy : -

* Stage of aura * Unconciousness
* Convulsions * Post convulsive

2. Petil mal / minor epilepsy

3. Status epilepticus.

Symptoms :

A) Major epilepsy / Grand mal :

1. Stage of aura :

- It is the first stage.
- It is warning signal / indication that fit is coming. Animal shows expression that some danger is coming. It last for few seconds. (2 sec.)
- Animal remains concious but loss its voluntary power.
- Type of Aura depending on the site of abnormal electrical activity.
- Aura Motor Aura : Twitching and convulsion of the angle of mouth.
- Sensory Aura : Peculiar sensation on the body.

- Visual: Vacant look, visual hallucination

2. Stage of prodrome (Unconciousness)

Stage of aura is followed by stage of unconciousness, animal becomes still and unconcious.

3. Stage of convulsions :

It has two phases : a) Tonic phase
b) Clonic phase

a. Tonic Phase : Suddenly muscles of the whole body are thrown into tonic spasms and animal falls on the ground.

This phase is characterized by deviation of eyes, cry, cyanotic condition, rapid pulse, temporary stoppage of respiration and limb abnormalities.

b. Clonic phase : Tonic phase is followed by clonic phase. Clonic convulsions of all muscle groups of limbs. This phase is characterized by pedaling movements.

Initially – convulsions occurs at regular interval. Afterwards convulsions at longer intervals. Finally final jerk and animal goes into flaccid coma.

This stage is characterized by tongue bitting. Hypersalivation, blood stained froth through mouth, papillary dilatation.

This stage lasts for few seconds to few minutes.

4. Stage of post ictus (Post convulsive)

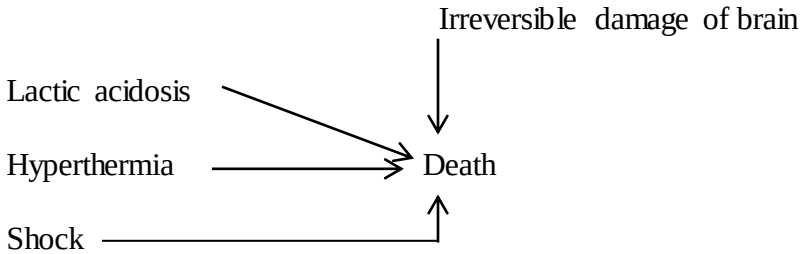
After clonic phase animal become concious again but no full nervous control characterized by flaccidity of muscles, depression, fatigueness, inco-ordination of gait, blindness. This stage lasts for few seconds to few days.

C. Minor epilepsy / Petit mal

- It is rare in animals.
- It lasts for 10-30 seconds.
- Characterized by blinking movements of the eyes. Jerking movements of head and limbs.

D. Status epilepticus :

Characterized by multiple and continuous fits seizures with either brief or total lack of inter ictal period.



Diagnosis :

1. History
2. Clinical signs.
3. Lab investigations.
   - Blood biochemistry - Sugar
     Urea
     Calcium
   - Urine
   - Stool – Protozoa examination
   - C.S. F. – Intracranial disturbances.
4. X-ray of skull : Space occupying lesions.
5. E.E.G. is helpful for diagnosing
   - Type of epilepsy
   - Site of lesion

- Differentiation between symptomatic and idiopathic epilepsy.
- Abnormal spikes in E.E.G.

Differential diagnosis :

1. Viral encephalitis – Canine distemper.
2. toxoplasmosis.
3. Cerebral neoplasia
4. Trauma
5. Cryptococosis
6. Hydrocephalus
7. Parasitism
8. Hypoglycaemia
9. Hypocalcaemia
10. Tetanus – 3rd eyelid prolapse, lock jaw.
11. Cyanide poisoning – bright red mucous membrane.
12. Chlorinated hydrocarbon poisoning.
13. Organophosphate poisoning.

Treatment :

A) General Treatment :

- Avoid constipation.
- Avoid trauma / accident so keep animal in a safe place.

B) Specific Treatment :

a) **Major epilepsy.**
Anticonvulsants : Phenobarbitone 1-5 mg/kg body weight upto 180 days or
Dilantia sodium @ 20-35 mg/kg/day or
Primidone @ 17-70 mg/kg/day 2 doses.

b) **Minor Epilepsy :** Sodium valproate / zarontia
Tips : First use – phenobarbitone. If no response use phenobarbitone + Dilatia sodium, if no response use Phenobarbitone + dilatia sodium + primidone.
(Gradually withdraw barbitone)
Sodium valproate / zarontia : one teaspoonful B. I. D. orally

Guidelines :

- Maximum dose should be give initially.
- Suddenly withdrawl should not be done.
- One drug should be replaced gradually to another drug
- Treatment should be continued for few days after last fit.
- More dose in evening and less in morning.

3. Status Epilepticus :

- Diazepam – 5-30 mg I/v
- Phenobarbitone - 4-20 mg/kg
- In dog Paraldehyde - 4-8 gm P/R
- If depress the cerebrum but not vital medullary centers.
- 50% Dextrose – hypoglycaemia calcium – Hypocalcaemia
- It reduces intracranial pressure.
- Corticosteroid : Dexamethasone / Betamethasone @ 1-4 mg / kg to maintain body temperature.
- Vit. B complex.
- Antiepileptic drugs :
- Bromides : No use now.
- Barbesaclone – 10mg/kg/ day for 15 days.
- Magnesia phosphorica – Magphos 6 × 3 tab/day for one month.
- Drugs contraindicated in epilepsy :

- Phenothiazien derivatives.
- Chlorpromazine (largactil)
- Triflupromazine (siquil)

5. Principles of Treatment of diseases of Nervous system :

General care :

1. Good nursing.
2. Avoid bed sore by providing good bedding.
3. Frequent turning of animal.
4. Massage of affected parts with liniment in paralysis.
5. Infrared therapy in paralysis
6. Nervine tonics ($B_1 + B_6 + B_{12}$) in paralysis.
7. Antiseptic inhalation – to avoid the respiratory tract infection.
8. Fluid and electrolyte therapy to reduce intra-cranial pressure.

Limitations in treatment of Nervous disorders :

1. The failure of nervous tissue in brain and spinal cord to regenerate.
2. Impermiability of blood, brain barrier to many antibiotics.

Specific Treatment :

1. Elimination and control of infection :

- Treatment of infection of CNS is limited because of blood brain barrier.
- Very limited information is available on the penetration of parenterally administered antibiotics into CNS of either normal farm animals or inflammation of the nervous system.
- The most useful antimicrobial for nervous system meningitis in farm animals are third generation cephalosporines, sulphatrimethoprim and gentamicin.
- In encephalitis / meningitis blood brain barrier is not likely to be intact or damaged and that parenterally administered drug will diffuse into the nervous tissue of CSF.
- The level of chloramphenicol required in CSF for bacterial activity of most gram negative enteric bacteria causing meningitis in farm animals are not attainable and therefore it is not considered to be drug of choice.
- Intrathecal injection of antimicrobials have been when parenteral therapy is unsuccessful. However intrathecal injections can cause rapid death and therefore not recommended.

Relative diffusion of antimicrobials in human :

1. Excellent with or without inflammation – sulphonamides 3rd generation – cephalosporin viz. cefotaxime.
2. Good only with injection – Ampicillin, penicillin.
3. Minimal or not good with infection – Tetracyclin, streptomykin, kanamycin, Gentamicin.
4. No passage with injection : Polymixin-B, colistin.

2. Decompression :

- Intracranial pressure is usually increased in most cases of encephalitis, but it is severe in acute cerebral oedema, space occupying lesions and hypovitaminosis.
- The combination of mannitol and corticosteroides is used for reducing intracranial pressure.
- Mannitol is given as a 20% solution @ 2gm/kg body weight I/V over 30-60 min period is a successful.
- Intracranial decompresant with an effect lasting for 4 hours.
- The effect of mannitol could be prolonged by I/V administration of dexamethasone 3 hrs after mannitol.

The danger with mannitol could be prevented by following mentioned tips :

- It should be repeated often.
- It must be given animal in shock.

- It should be given I/V slowly.
- Dexamethasone is to be given @ 1mg/kg body weight I/V. It is safe and has a good effect but does not decompress sufficiently.
- Hypertonic glucose given I/V is dangerous because initial temporary decompression is followed by and increase in CSF pressure after 4-6 hrs interval.

CNS Depressent :

- Use of sedatives, general anaesthetics agents, tranquilizers to check convulsions and spasms during acute phase.
- There drugs will control convulsion and therapy prevent the animal from injury itself, allow sometimes to examine properly. Assess the diagnosis and initiate treatment.
  - Diazepam @ 0.25 – 1 mg /Kg I/V or I/M 6 hrs interval.
  - Chloropromazine HCl @ 1mg/kg I/V or I/m at 8-12 hrs interval.
  - Triflupromazine @ 0.1mg/kg I/V or I/m at 8-12 hrs interval.
  - Phenobarbitone @ 4-6 mg/kg po for dog and cat.

CNS stimulant :

- They are useful during the period of depression.
- They are indicated only in nervous shock.
- After anaesthesia or other short term reversible anoxia such as cynide or nitrate poisoning.
- They exert only a transient improvement.

4. PARALYSIS

Definition: "It is a condition characterized by total inability to perform voluntary movements due to complete or incomplete loss of nervous control over the body function." It includes loss of sensory or motor power or both.

Classification :

A. Anatomical Classification :

A. Cerebral paralysis	**B. Spinal paralysis**	**C. Peripheral paralysis**
Involvement of brain	Involvement of spinal cord	Involvement of peripheral nerves
Causes :		
Apoplexy, Encephalitis, Meningitis, brain injury, contusion, tumor, cyst abscess, T.B. lesion in brain	Traumatic injury to spinal cord. Myelitis, Meningitis,encephalomylitis Abscess, cyst, tumor, T.B. lesions in spinal cord, Interverteral disc protrusion	Injury to nerve trunk Affection of nerve endings. Deficiency of Vit. B. complex, Drug toxicity

B. Classification of paralysis on the basis of muscle tone :

A. Spastic paralysis	**B. Flaccid paralysis**
1. Paralysis in contractile stage of muscles 2. It is characterized by increased tone of limb muscles and increased tendon jerks. 3. It is due to lesion of upper motor neuron.	1. Paralysis in relaxation stage of muscles. 2. It is characterized by decrease tone of limb muscles and absence of tendon jerks. 3. It is due to lesion of lower motor neuron.

C. Classification of paralysis on the basis of lesions :

A. Upper motor neuron paralysis	**B. Lower motor neuron paralysis**
Characterized by spastic paralysis of hindlimbs.	Characterised by flaccid paralysis of hindlimbs.

D. Clinical classification of paralysis :

1. **Monoplegia :** It means paralysis of one or few nerves. e.g. radial nerve paralysis.

2. **Diplegia :** It is the condition characterized by paralysis of affecting both sides of body.
3. **Hemiplegia :** It is the condition characterized by paralysis of one side of the body.
4. **Quadriplegia :** It is the condition characterized by paralysis of all four legs.
5. **Paraplegia :** It is the condition characterized by paralysis of hind limbs.

PERIPHERAL NERVE PARALYSIS :

Forelimbs :

1) Radial paralysis(Dropped elbow)

It is commonly observed in horse, cattle and occasionally in dogs.

Causes :

- Injury to first rib.
- Recumbency on hard /rough floor, deep abscess or tumors.

Clinical Signs : Inability to bear weight and animal stands wirh the help of knee and fetlock joint of forelegs.

2. Suprascapular paralysis : (Slipped shoulder) :

Causes : It is due to injury to suprascapular nerve innervating supra-spinatus and infra spinatus muscle.

Clinical Signs : Asymmetry of forelegs, slipping of shoulders outwards in time when foot is placed on ground.

HIND LIMB :

1. Obturator nerve paralysis : (Calving paralysis) :

Causes : It is either due to primary damage to obturator nerve or indirectly to spinal nerve. It is usually due to manual handling in dystokia.

Signs : Ataxia and inability to get up with hindlegs

2. Gluteal nerve Paralysis :

Causes : Injury to qluteal nerve

Clinical signs : Atropy of gluteal muscles.

3. Sciatic Nerve Paralysis (Thigh)

Signs : It is characterized by loss of power of muscle of thigh except those situated above and in front of stifle joint. The limb hangs loosely and there is forward jerk while the animal attempts to walk.

4. Tibial nerve paralysis:

It is paralysis of extensor of hock and flexoar muscles of digit. It is rare in animals.

Clinical Signs: Slight flexion of fetlock while sole is in opposition with the ground.

5. Peroneal nerve paralysis :

It is usually seen following parturition in cattle and horse.

Cause : Trauma / pressure

Clinical Signs : Flexion of fetlock and pastern of one or both hind limbs.

TREATMENT :

1. Good nursing.
2. Complete rest.
3. Provide good bedding made up of straw gunny bags, blankets with Avoid bed sore
4. frequent turning of animals.
5. Infrared therapy.
6. Massage of affected part by liniment.
7. Parenteral B. complex (B_1+B_6+B_{12}) till compete recovery.
8. Fluid and electrolytes parenterally e.g. DNS, Dextroxe 5% or 10% etc.
9. Give Antiseptic inhalation should be given to avoid respiratory tract infection
10. Antibiotics like chloramphenicol @ 4-11 mg/kg i/m should be given in case of paralysis of cerebral origin.

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CHAPTER 9
SKIN DISEASES

1. PRURITUS (Itching)

Definition. It is the sensation that gives rise to desire of scratching.
Causes:
1) Peripheral pruritis :
- i) Ectoparasites. : Mites, ticks, lice.
- ii) Ringworm
- iii) Photosensitization
- iv) Allergic dermatitis eczema, urticaria.
- iv) Pityriasis

2) Central Origin stimulation of itching center in the medulla
- i) Pseudorabies in pig.
- ii) Scrapie in sheeps.
- iii) Metabolic diseases disturbances, Diabetes mellitus, nervous form of ketosis, hepatitis, nephritis.

Treatment:
1) Specific Treatment
   a) Use of acaricides
   b) Antifungals
   c) Regulation of diet.
2) Supportive treatment:
   a) Sedative central origin
   b) Antihistaminics are poor antipruritic agents unless given in sedation doses
   c) Corticosteroides are very effective antipruritic agents
3) Application of soothing agent e.g. calamine lotion.

2. ALOPECIA

(Synonym: Baldness, Hypotrichosis.)

Definition: Loss of hairs fur or wool.
It may be seasonal or as a consequence to certain disease. It may be local or diffuse.
Causes: a) Congenital: Congenital hypotrichiasis and baldy calf syndrome
b) Acquired.
- i. Nutritional deficiency: Vitamin A, zinc, iodine.
- ii. Ectoparsites - Mites, ticks lice, fleas.
- iii. Sever endoparasite infestation in young animals.
- iv. Ringworm
- v. Leucania leucocephala (subabul) poisoning, Thallium poisoning
- vi. Hormonal imbalance
  - a) Hypothyroidism
  - b) Excess estrogen
  - c) Sertoli cell tumor
  - d) Hyperadrenocostism – causing syndrome
  - e) Hypoadrenocortism

Treatment:
1) Removal of primary cause: antibacterial, acaricides, antifungals.

2) Diet supplementation with iodine, protein, Ca and Vit. A
3) Castration may help in hair growth
4) Oral or parenteral administration of arsenical preparation.
5) Hormonal therapy : Methyl testosterone @ 1 mg/kg for male dog and diethyl stilbesterol @ 0.1 mg for female dog.

3. PITYRIASIS / DANDRUFF

Definition:

It is a condition characterized by presence of bran like scales on the skin surface.

Causes: a) Primary Pityriasis – is due to over production of keratinized epithelial cells. It also occurs due to deficiency of Vit. A, nicotinic acid, rboflavin and linoleinic acid.

b) Secondary Pityriasis due to excessive desquamation of epithelial cells.

i) Ectoparasites : Flea, lice and manage miles.

ii) Mycotic skin infection- Ringworm.

Clinical Signs: Primary : Accumulation of scales, no itching.

Secondary: Primary lesions and scales.

Treatment:

1) Correction of primary cause.
2) Thorough washing
3) Application of emolient ointment.

4. PARAKERATOSIS

Definition : It means incomplete or imperfect keratinisation of epithelial cells of skin.

Causes: 1) Inherited in cattle and pig.

2) Dietary deficiency of zinc.

Clinical Signs : Lesions may diffuse but are often confined to the flexor aspects of joints, large, soft, scale accumulate usually, crack and fissure and removal of crusts leaves. Rows red surf.

3) **Diagnosis :** Skin biopsy

Treatment:

Dietary supplementation of zinc.

Vigerous washing with soap water.

Application of white lotion /astringent lotion.

Composition of lead acetate -30 gm + Zinc sulphate- 24 gm + Aqua-600ml

5. HYPERKERATOSIS

Definition : It means excessive keratinization of epithelia cells. It is an increase in thickness of the stratum corneum

Causes:

1) Generalised:
   a) Chronic arsenic or chlorinated Napthalene posioing.
   b) vit A deficiency
2) Localised:

Local at pressure point (elbows) when animal lie on hard surface.

Clinical Sign:

- Thickening and corrugation of skin
- Skin is hairless dry and scaly
- Plugs of hyperkeratotic material can be removed leaving the underlying skin intact.

Diagnosis: Skin biopsy

Treatment:

- Correction of primary cause

- Use of keratolytics e.g. salicylic acid ointment.
- Vit. A supplementation.

6. IMPETIGO

Definition :

It is superficial eruption of thin walled vesicles which develops into pustules and surrounded by a zone of erythema.

Causes: Bacteria- *Streptococci, Corynebacterium*.
In animal it mainly causes by *Staphylococci*.

Clinical Signs:

- Vesicles appear on relatively hairless parts of the body
- Later on they become pustules.
- Pustules rupture to form scabs.
- Sucessive crops of vesicles/pustuls appear.
- Udder impetigo in cows lesions over udder and teats.
- Contagious pyoderma in baby pigs – lesions over face and neck

Diagnosis: Culture of vesicular fluid.
Differential Diagnosis: Cow pox and pseudo cowpox lesions restricted to the teats.
Treatment:

- Antiseptic skin wash and application of antiseptic ointment.
- Parenteral antibiotics e.g. penicillin, amoxycillin.

7. PACHYDERMA

Definition : It is thickening of skin affecting all layers.
Causes: There are no specific causes however it is mostly caused by nonspecific chronic or recurrent inflammation of skin.
Clinical Sign:

- Thickening usually confined to local areas.
- Skin is thick, tough and tight and can't be picked into folds
- Hair coat is thin or absent.
- No fissures in the skin / accumulation of cell debris / scales.

Diagnosis: Histopathological examination of skin biopsy.
Differential diagnosis: Presence of fissures in skin and accumulation of scales
Hyperkeratosis and Parakeratosis.
Treatment:

Administration of corticosteroids locally and parenterally is helpful in early stage of disease. Surgical removal when small area is involved.

8. SEBORRHOEA

Definition : It means excessive secretion of sebum on skin surface.
Causes:

1) Primary seborrhea is rare in animals.
2) Secondary seborrhea is always associated with dermatitis and skin irritation in large animals.
    a) Exudative epidermatitis of pigs (*Saphylococcus hyicus*)
    b) Greasy heel of horses.
    c) Greasy heel of cattle
    d) Flexural seborrhea of cattle.

Clinical Signs:

A) Greasy heel of cow :

- It occurs in cows; constantly graze on pasture in irrigated and muddy areas.
- Local swelling with deep fissuring and outpouring of foul smelling exudates on the back of pastern on all four feet but most severely in hindlimbs.
- Lamness.

B) Flexural seborrhea in cow:

- It is most common in young recently calved dairy cows.
- Serve inflammation with profuse outpouring of sebum in between two halves of udder and medial aspect of thigh

Treatment:

- Remove the animal to dry land.
- Wash the affected area with hot soap water.
- Apply astringent lotion (e.g. white lotion) daily
- Application of ointment containing antibiotic fungistat and corticosteroid viz gentamicin,clotrimazole and betamithazone.
- Broad-spectrum antibiotic for rapid recovery.

C) Greasy heel of horses :

- It occurs mostly on hind pasterns of horses which stand continuously in wet stables.
- Lameness and soreness to touch due to excoriations.
- Skin in thick greasy.

9. ACNE

Definition : It means infection of hair follicles by suppurative organisms.

Causes: *Staphylococci*

- Demodectic mange in cattle, goat.

Clinical Signs:

- Lesions begin as nodules and then develop into pustules and crust followed by loss of hair fibers.

Treatment:

- Wash the affected area with antiseptics.
- Apply antiseptic ointments /lotions locally.
- Antibiotics parenterally if excessive and of long duration.

10. ECZEMA

Definition : It is inflammatory reaction of the epidermal cells to exogenous or endogenous substances to which cells are sensitized.

Etiology :

A) Predisposing factors :-

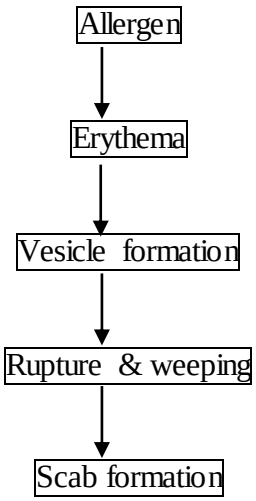
1) Inherited susceptibility
2) Constant scratching
3) Long term soiling
4) Repeated wetting
5) Hormonal imbalance, seasonal variation and hepatic disorders.

B) Actual causes :

1) Physical agents : Dust , cold , heat etc .
2) Chemical agents : Bathing & washing with highly chlorinated water & soap.
3) Food allergy : Ingestion of protein i.e milk , meat, egg, fish , pork etc.
4) Autointoxication due to overeating or bowel stenosis
5) Worm allergy : Digestion of internal parasites

6) External parasites : Fleas , flies , ticks etc .
7) Drug allergy : Penicillin, Ampicillin, Streptomycin, Antiseptics or disinfecting agents.

Pathogenesis :-



Clinical Signs :

- Eczema is common in dogs however rare in large animals.
- Lesions are usually seen over neck , back , rump & abdomen
- Eczema is of two types : moist & dry eczema.

a) Moist eczema :-

- Oozing of fluid (weeping skin)
- Matting of hairs surrounding patch of alopecia
- Pruritus .

b) Dry eczema :

- Dry thickened skin.
- Pruritus

Diagnosis :

1) History of change in diet , environment , administration of drugs.
2) Clinical signs : Weeping skin.
3) Clinical pathology - Eosinophilia
4) Evidence of internal & external parasites.
5) Response to antihistaminic drugs.

Differential diagnosis :

I) Mange :- Weeping absent , lesions usually dry, erythematous & scaly, skin scraping positive for mites , lesions commonly over head , neck & fore legs.

II) Ringworm :- Whitish asbestos like lesions over head , face & neck

- Itching is less maked
- Lesions are usually dry & scaly
- Weeping absent
- Skin scraping +ve for dermatophytes.

Treatment :

1)Avoid exposure to the sensitizing substances

a) Change in diet
b) Change in environment
c) Avoid wetting & unnecersary irritation
d) Removing of internal & external porasiles-
Deworming with Albendazole @ 5-10mg/kg orally & spraying with Butox @ 2-4ml/lit.

2) Antihistaminics e.g. inj. Avil @ 0.5-1mg/kg IM .
3) Corticosteroids e.g inj. Dexona @ 0.04 mg /kg IM , IV .
4) Sedatives if pruritus is severe
e.g. siquil @ 0.1mg/kg IM or IV .
5) Local dressing with astringent, antiseptic lotion in early stage & protective ointment in later stage. e.g. Himax or Ectosep ointment etc.
6) Antibiotics to check secondary bacterial infections e.g. streptopenicillin @ 10-30 mg/kg IM

11. URTICARIA

Definitions : It is allergic condition characterised by the appearance of wheals on the skin surface. It is commonly observed on horse, pig and rare in cattle, buffaloe, sheep, goat, dog and cat.

Etiology :

I) Primary Urticaria:

A)External causes ;-

i) Insect stings
ii) Contact with stinging plants
iii) Contact with irritant chemicals
iv) Exposure to excess heat or cold.

B) Internal causes :-

1) Digestive disturbances due to change in diet.
2) Milk allergy – prolonged unmilking or when cows are dried off.
3) Drug allergy – due to administration of drugs like penicillin
4) Parasites – Ascarids, tapeworms

II) Secondary Urticaria

- Respiratory tract infection in horses.
- Erysipelas in pigs.

Clinical signs :

1) Sudden appearance of wheals on skin
2) Itching / pruritus exudation / from skin surface more prominent in plant/insect bite
3) Slight fever a diarrhoea may be observe
4) Subsidence of wheals within 24-48 hours is usual but they may be persist for 3-4 days due to appearance of fresh lesions.

Diagnosis :-

1) History of change in diet environment, administration of drugs, unmilking in cows
2) Clinical signs – wheals & pruritus.
3) Clinical pathology – Eosinophilia
4) Response to antihistaminic drugs
5) Intradermal skin test are of little value.

Differential diagnosis:

Angioderma :

- It involves subcutaneous tissue rather than skin.
- Lesions are much larger and more diffuse.

Treatment:

1) Prevent exposure to allergens-A and change of diet and environment especially exposure to the insects or plants is standard practice.
2) Antihistaminic e.g. inj. Avil @ 0.5-1mg/kg I/M
3) Corticosteroids e.g. inj. Dexona @0.04mg/kg I/M
4) Anthelmintics to remove internal parasites e.g. Albendazole @5-10mg/kg orally.
5) Ectoparasiticides to kill ectoparasities e.g. Butox liquid @ 2-4ml/lit spraying
6) Local application of cooling lotions – sodium bicarbonate solution or white lotion 7) Laxatives orally
8) In large animal practice parenteral inj of calcium salts are used with apparently good results.

	Urticaria	Eczema
Species	1. Common in horse & pig	1. Common in dogs
Signs	2. Lesion usually generalized	2. Localized over dorsal part of body
	3. Wheal formation	3. Absent
	4. Usually no weeping	4. Weeping

12. MANGE (ACARODERMATITIS)

The various types of mange are as follows

- **1) Sarcoptic mange (Scabies)**
- **2) Demodectic mange (Folliculitis)**
- **3) Psorptic mange**
- 1) Sarcoptic mange (scabies)or Barn itch.
- **It occurs in all species. It is most important in pigs**
- Etiology: ***Sarcoptes scabei.***
- Predisposing factors :
    1) Malnutrition, debility
    2) Poor management , worm loads
    3) Unhygienic conditions
    4) Stress factors
    5) Frequent washing with alkaline soaps.
    6) The disease is most active in cold wet weather.

Clinical findings:-

1) Constant progressive lesions over head neck and forelimbs. They may be localized or generalized.
2) Pruritus i.e.severe itching.
3) Excoriations i.e. superficial abrasions on skin.
4) Alopecia i.e. loss of hair, wool or fur.
5) Erythema i.e. red coloration of skin
6) Dandruff i.e. scale formation.
7) hyperkeratosis i.e. thickening and wrinkling of skin in later stage
8) Emaciation

Diagnosis:-

1) Clinical signs : Alopecia , itching , erythema excoriations
2) Skin scraping examination
3) Response to acaricides
4) Pigs : Lesions commence on the trunk.
5) Sheep and goats : On the face.
6) Cattle : On inner surface of thighs, ventral side of neck, brisket and around root of tail
7) Horses and camel :- on the head and neck.
   The course of mange is rather more acute than other types of mange and may involve entire body surface in 6 weeks in cattle .

I) Demodectic mange : (Follicular mange)

It is more common in dog.

Etiology :

1) *Demodex canis* – Dog
2) *Demodex bovis* – Buffalo, cattle
3) *Demodex caprae* – Goat *D. phyloides* –pigs
4) *Demodex ovis* – shelp *D. equii* – horses

Predisposing factors :

1) Young age group (below 8-12months)
2) Malnutrition, debility
3) Worm load.
4) Bath with alkaline soaps
5) Stress factors
6) Hereditary factors

Clinical findings:

In dog:

It occurs in two forms in dog

1) Squamous form
2) Pustular form

1) Squamous form:

a) lesions generally occur over head and neck
b) Scaly skin lesions – dandruff
c) Seborrhoea –excessive secretion of sebum
d) Hyperkeratosis: - Thickening of skin
e) Alopecia –loss of hairs.

2) Pustular form

a) Affects almost any part of body
b) Pustular skin lesions
c) Alopecia
d) Erythema
e) Disagreeable odour

In ruminants :

Papules/nodules (pin head to tennis ball size) develop over head neck and shoulder region .

Diagnosis:-

1) History :- Young age group
2) Clinical signs and symptoms :- nodules over neck region in ruminants
3) Skin scraping examination in dogs or examination of pus smear prepared from nodules in bovines and caprines.

II) Psoroptic mange

Synonyms:- Sheep scab, Body mange, Ear mange

Etiology :- *Psoroptes ovis* in sheep but may also ocour is cattle ,horses and possibly donkey

It causes

1) Sheep scab in sheep.
2) Ear mange in goat , sheep , horses and rabbits.
3) Body mange in cattle and sheep

Clinical Findings :

1) Sheep : Lesions appear mostly over wither, shoulder sides and back, Itching, alopecia, scab formation.
2) Goats : Typical scabs appear on ear, nose and poll.

3) Cattle : Lesions over wither, neck and around base of tail.
Inching, alopecia and scabs formation.

Diagnosis :

1) Clinical signs and symptoms.
2) Skin scrapping examination.

Difference between Sarcoptic and Demodectic mange in dog.

	Sarcoptic	***Demodectic***
1) Cause	***Sarcoptic scabei***	***Demodex cauis***
2) Age	All age group	Mostly young pups (6-8 months)
3) Lesions	Head, Face, neck and foreleg	May be generalized (two forms – squamous and pustular form)
4) Response to treatment	Good	Slow and poor

Treatment :

Clip the affected area and wash it with shampoo or lukewarm water.

1) Specific treatment :

A) Use of topical acaricides :

e.g. a) Spraying of Butox liquid (Deltamethrin) 1.25% @ 4ml / lit at weekly interval on 1-2 occasions or till recovery OR
b) Clinar / Pelctocid / (Cypermethrin) @ 2ml/lit weekly on 1-2 occasions.
c) Ectodex/Taktik liquid i.e. Amitraz @ 6ml/lit at weekly interval on 1-2 occasions or till recovery OR
d) Application of Himax lotion or ointment / ectosep ointment locally daily for 10-15 days.

B) Use of injectable acaricides :

e.g. a) Ivermectin @ 0.4 mg/kg SC in dog, 0.3 mg/kg SC in pig, and 0.2 mg/kg SC in other species. Repeat dose after 10-14 days if requried. OR
b) Inj. Doramectin @ 0.2 mg/kg SC, repeat after 14 days if required.

2) Supportive treatment :

a) Antihistaminics to reduce itching e.g. Inj. Avil @ 0.5 – 1 mg/kg IM.
b) Use of sedatives in severe itching e.g. Phenargan @ 0.75 mg/kg IM.
c) Antibiotics e.g. Ampicillin, Amoxycillin @ 5-10 mg/kg IM IV
d) Vit. A and mineral supplimentation to promote healing of skin lesions.
e) Inj E-care-se @ 1ml/25kg IM Selenium and Vit. E is helpful in stimulation of immune system in chronic less / non-responsive cases of demodicosis.

CHORIOPTIC MANGE

Chorioptic mange is the commonest form of mange in cattle and horses.

Etiology : *Chorioptes bovis*.

Clinical Signs :

Leg mange in horses – Violent stamping of feet and rubbing of the back of hing pasterns on wire or stamps.

Tail mange in cattle – Small crusty scabs on tail, udder, thigh with little irritation.

Scrotal mange in sheep – Serum exudate is seen over scrotum.

13. RINGWORM

Synonym : *Dermatophytosis, Tinea infection*

Ringworm is the superficial fungal infection of epidermis viz. hair, nails, feathers, hooves.

- It is zoonotic disease animal ringworm is a major cause of human ringworm in rural area.
- It occurs in all animal species.
- Dermatophytosis is caused by dermatophytes.
- Tinea infection – Thread like appearance of mycelium fungi.

Etiology :

- **It is caused by dermatophytes i.e. Fungi which affect the superfacial keratinized layers of the body are called as dermatophytes.**
- **The dermatophytes are multicellular fungi made up of cells joined linearly to form filaments termed as hyphae. The hyphae form a langled mass called as mycelium**
- **C, B, S, G –** ***Trichophypon verrucosum, T. Mentagrophytes*****.**
- **Horse -** ***Trichophypon verrucosum, T. Mentagrophytes, T. equinum, mycrosporum gypseum.***
- Dog - ***T. Mentagrophytes, , mycrosporum gypseum, M. canis.***
- **Tricophytosis – Occurs in all animal species while Microsporosis occures in horses, pig and dog.**

Trichophyton	***Microsporum***
1) They infect hair, skin or both	1) They infect hairs of the skin
2) The macrocanidia are smooth and thin walled	2) Macrocanidia are rough and thick walled
3) Spores are arranged in long chain (ecto and endothrix)	3) Spores are arranged in mosaic pattern (ectothrix is outside the hair)

Predisposing factors :

1) Age - Young animals
2) Climate – Hot and humid conditions
3) Close confinement
4) Inadequate nutrition
5) Overcrowding –
6) Alkaline PH of skin.

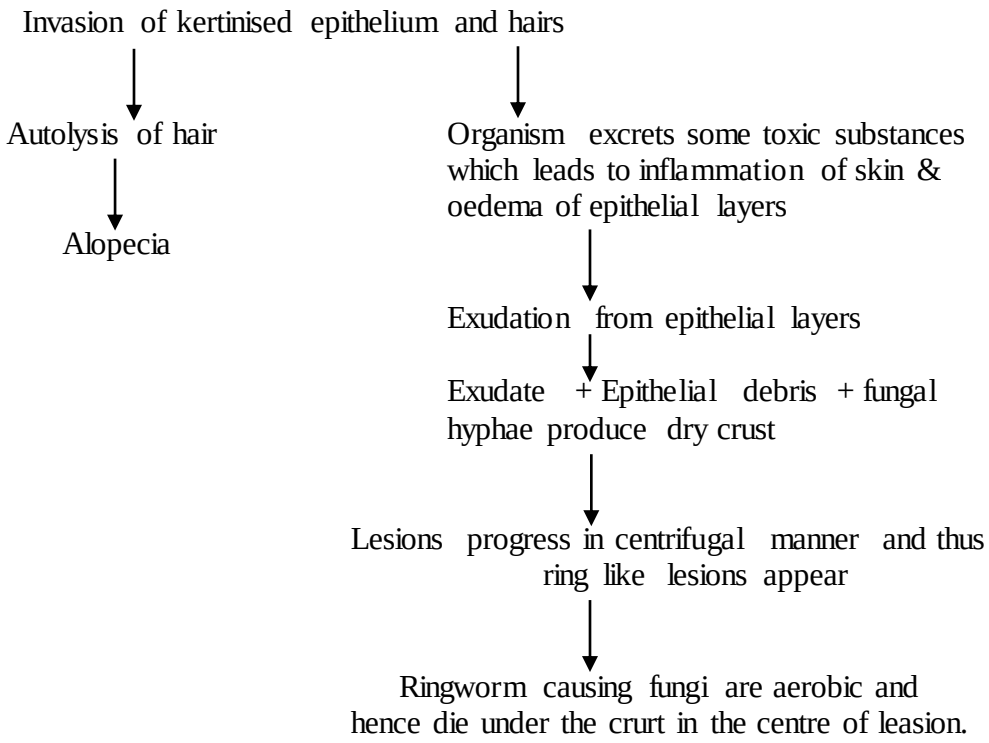
Transmission :

a) Direct contact with infected aninmals.
b) Indirect contact : Grooming kits, beading materials, fomites.
c) Animals shed/kennels/ byres/ stables remain infective for long time.

Pathogenesis :

Entry of fungus

↓



Clinical signs :

Discrete circular, whitish asbestos like lesions usually over head, around eyes, ear, neck, shoulder, and back

Lesions usually spread peripherally with central healing

Dandruff

Alopecia

Mild pruritus.

Vesicles and pustules may be seen as a result of secondary bacterial infection.

Skin shows mouth eaten appearence in untreated/ neglected cases.

Diagnosis :

1) Clinical signs : Withish circular lesions over head, face, neck.
2) Skin scrapping examination.
3) Cultural examination – Sabourauds agar.
4) Skin biopsy
5) Wood's light – M canis produce yellow green fluroscence when examined under wood's light.

Treatment :

1) Clip the hairs and clean the area and remove the crusts.
2) Local treatment / topical application
   a) Apply weak solution of Tr. Iodine
   b) White field ointment (Salycylic acid 39 + benzoic acid 69 + Vaseline)
   c) Salycylic acid ointment (2- 6%) or Benzoic acid ointment. (2 - 6%)
   d) Bordaux mixture : It is good for treatment of large number of animals and or if large areas are affected copper sulphate 1 kg dissolved in separte containers unslaked lime 2 kg and then mixed in earthenware container and made upto 180 litters. Spraying is done at weekly interval with hand spray
   e) Miconazole 2 % (zole / zole- f ointment)
   f) Clotrimazole 1% cream / solution spray (candid cream)
   g) Himax ointment.

h) 4% thiabendozole ointment.

3) Systemic treatment :

a) Griseofulvin (Tab Grisivion 125 mg@ 25 mg/kg orally daily for 3-4 weeks in dog, 5 mg/kg for 7 days in claves.

b) Ketconazole (Tab fungicide 200mg)@ 5-10 mg/kg orally daily.

c) Sodium iodide @ 1gm/14 kg BW in 10% solution at weekly interval

14. DERMATITIS

Definition :

It means inflammation of dermis and epidermis.

Etiology :

A) Infections causes :

1. Bacteria : *Streptococci, Staphylococci, E. coli, Pseudomonas, Mycobacterium tuberculosis* (skin TB), *Spherophorus necrophorus* (foot rot)

2. Viral : Pox in cattle, buffalo, sheep and goat.
Contagious ecthyma in goat.
RP. MD. FMD, BT in sheep.
Hog cholera in swine.
CD in dog

3) Parasites : Ectoparasites particularly mange mites
Cutaneus larva migrans (Ascarid + hookwosm larvae)
Cutaneous miasis (Maggots)
Humpsore and leg sore in cattle and buffalo (*Stephanofilaria*)

B) Non- infectious causes :

1) Physical : Trauma, injury, sunburn, extensive hot or cold.
2) Chemicals : Irritant chemicals, acids, alkalies.
3) Allergic : Allergens, insect or plant stings.
4) Nutritional : Vit. A , B complex, zinc deficiency.

Clinical Signs :

1) Bacterial infection - Pyoderma
2) Allergic – Urticaria or eczema
3) Mites – Mange
4) Fungi – Ringworm
5) Vit.A deficiency – Hyperkeratosis.
6) Zinc deficiency – Parakeratosis.

Diagnosis :

1) History of diet and environment
2) Clinical findings.
3) Examination of skin scrapping for mites/fungi
4) Examination of skin swab for bacteria.
5) Skin biopsy
6) Haematology – Leucocytosis in bacterial infection
Leucopaenia in viral infection
Eosinophilia in parasitic infection.

Treatment :

A) Removal of primary cause :

1) Removal of physical and chemical agents.
2) Supplementation of diet with vit. A, B-comples zinc and proteins.
3) Antibiotics - Parenterally and locally in bacterial pyoderma and also in other types of dermatitis to check secondary bacterial infection.
4) Antifungals - Ringworm
5) Acaricides – Mange

6) Antihistaminics and steroids in allergic conditions.

B) Supportive :

1) Antibiotics to check secondary bacterial infection.
2) Antihistaminics to control itching
3) Multivitamins to promote skin repairs
4) High protein diet to facilitate skin repair
5) Sedatives when pain and itching is severe.

CHAPTER 10

DISEASES OF EYE, EAR AND JOINTS

1. CONJUNCTIVITIS

Definition : It means inflammation of conjunctiva.

Etiology :

1) Bacteria : *Staphylococci, Streptococci, Mycoplasma, Morexella bovis, Leptospira pamona of bovis*
2) Viral : ICH, CD, IBR
3) Mycotic : *Apsergillus candida*
4) Parasitic : *Eyeworm (Thelazia), Filaria worm (setaria sp)*
5) Trauma : Foreign body, dust, smoke, thorn, grassblades.
6) Chemical: Irritant chemicals. Soap, tick dips, disinfectants.
7) Allergic : Foreign proteins, pollengrains, drugs

Clinical Sign:

- Acute or chronic
- Unilateral or bilateral.

Primary or secondary to systemic diseases

- Ocular discharge
- Blepharospasm due to pain
- Redness and swelling of mucosa
- Epiphora (Tear mark in chronic cases)
- Foreign body or parasites may be observed
- Systemic reaction in acute cases.

Diagnosis:

1) Clinical examination.
2) Opthalmoscopic examination
3) Culture.

Treatment:

1) Keeps animal away from light sources i.e. in dark.
2) Remove foreign body if any.
3) Eye wash – Boric acid lotion 1%
4) Antibiotics ointment / cream locally 2-3 times daily.
5) Antibiotics and corticosteroids in several cases.
6) Antihistaminic and steroids- allergic cases.
7) Mechanical removel of antihelmintics – parasites
8) Systemic antibiotics – acute cases.

2. OTITIS

- Otitis externa : inflammation of external ear.
- Otitis media : inflammation of middle ear
- Otitis interna : Inflammation of internal ear

Causes :

1) Trauma /irritation : grass blades, thorn, awas
2) Bacteria: *Streptococci, Staphylococci, E-coli, Pseudomonas, Proteus.*
3) Fungi : Otomycosis are not uncommon
4) Parasites : Otoacariosis caused by *Otodectes cynotis* in dog and cat.
    - Ear mites e.g. *Psoroptes sp.* in cattle and buffalo *Stephallo fileria zaheri – earsora* in buffalo.

Clinical Sign:

- Irritation is predominant clinical sign. Head shaking, ear rubbing and scratching.
- Lesions : Diffuse or discrete, ulcerative or granulomatous, uni or bilateral.
- Ear red, hot and painful.Rotation of head forwards affected side.
- Otorrhoea – purulent discharge from ear
- Fever, dullness and loss of appetite may be observed.

Diagnosis:

Clinical signs
Cultural examination of discharge.
Skin scraping examination
Otoscopic examination

Treatment:

1) Antibiotics in bacterial infection – systemic and aural.
2) Specific acaricides in otoacariasis
3) Cleaning of ear canal
4) Surgical drainage in chronic otorrhoea.

3. ARTHRITIS

Definition : Inflammation of synovial membrame and articular surfaces characterized by lameness and local pain, heat and swelling of the joint.

Etiology

It is mainly infectious in origin

1)Bacterial infections

Calves, lambs, foals : joint ill caused by *Cornybacterium pyogen, Staphylococcus Erysipelothrix insidsa, Salmonella.*

Cattle: Haemophilus, Mycoplasma, Brucella, Ephemeral fever.

Sheep : Mycoplasma

Pigs : Glasser's disease, Mycoplasma, Brucella.

Horses : Strangles, porysynovitis.

2) Trauma :

Clinical Findings:

1) Inflammation of synovial membrane causes pain and lameness in the affected limb.
2) Pain and heat of joint on palpation
3) In pyogenic infection rupture of joints may occure.
4) Stiff gait.
5) Fever,anorexia, dullness may be seen.

Diagnosis:

1) Signs: Swollen joints, lameness.

2) Increase in volume of synovial fluid.
3) Collection of synovial fluid for culture and sensitivity.
4) X-ray examination.

Treatment:

Acute infection arthritis should be treated as quick as possible.

1) Antibiotics: Parenterally for several days. eg. Inj.Dicristicin @10 mg/kg b/w. IM for 5 – 7 days. Antibiotics can also be given directly into joint capsule along with corticosteriods

e.g. Penicillin+hostacortin

2) Administration of NSAIDS

Meloxicam@ 0.2-0.5mg/kg IM/IV for 5 to 7 days.

3) Hot fomentation of affected joints may be carried out.