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**Addis Ababa University**  
**College of Veterinary Medicine and Agriculture**  
**Department of Clinical Studies**  
**Second Semester Course Outline for the course Veterinary General Medicine**  
**(Vetm3132)**

2019/2020 Academic Year

**Course credit:** 7ECTS/4CrHr

**Prerequisite:** Vet Parasitology I&II, Vet Microbiology I &II, Vet Pathology I&II, Vet. Pharmacology and therapeutics I&II and Vet Clinical diagnosis

**Course Coordinator:** Dr. Sisay Girma, Assistant Professor

**Course Description:**

General systemic states: toxemia, septicemia, bacteremia, fever, hyperthermia, hypothermia, heat stroke, dehydration, electrolytes and acid-base imbalances, allergies. Diseases of alimentary system: stomatitis, pharyngitis, esophageal disorders, simple indigestion, ruminal acidosis and alkalosis, ruminal tympany, traumatic reticuloperitonitis/pericarditis, impaction of abomasum, neonatal infections, enteritis, equine colic, gastritis and principle of GIT treatment. Hepatobiliary System, Hepatitis, jaundice and cholelithiasis. Cardiovascular system: arrhythmias, myocarditis, endocarditis, valvular heart disease, circulatory failure, thrombosis and embolism, congenital cardiac defects and shock, edema. Hematopoietic and hemolymphatic system: anemia, leukemia, leucopenia. Respiratory system: pneumonia, bronchitis and pleurisy. Diseases of the urinary, nervous and musculoskeletal systems.

**Learning Outcomes**

**At the end of the course, students should be:**

- ✚ Familiar with the commonly used clinical instruments and drugs;
- ✚ Equipped with methods of clinical examinations and diagnosis on sick animals;
- ✚ Able to recognize the syndromes common to all diseases affecting all body systems; and
- ✚ Able to understand and appreciate the harmful microbial, genetic and environmental influences on animal health and production

**The full course outline is described in the following table**

<b>Time</b>	<b>Diseases/Topics</b>	<b>Instructor</b>
Week 1	<b>Hyperthermia, Hypothermia, Fever</b>	Sisay G.
Week 2	<b>Septicemia, Viremia, Localized infection and pain</b>	Sisay G.
Week 3	<b>Toxemia</b>	Sisay G.
Week 3	<b>Disturbance of fluid and electrolyte imbalance</b>	Sisay G.
Week 4	<b>Allergy/Hypersensitivity</b>	Sisay G.
<b>Week 4</b>	<b>Test One</b>	<b>Sisay G.</b>
Week 5	<b>Diseases of the Respiratory system</b>	Sisay G.
	(A). Pneumonia (B). Pneumothorax / Emphesyma (C). Pleurisy	
Week 6	<b>Diseases of the Alimentary tract</b>	Sisay G.
	(A). Stomatitis, Pharyngitis, Esophagitis and Esophageal obstruction (B). Hypermotility and Hypomotility (C). Simple indigestion (D). Acute carbohydrate engorgement	
Week 7	<b>Diseases of the Alimentary tract</b>	Sisay G.
	(E).Distention, Ruminal Tympani (F). Enteritis, Diarrhea and Vomiting (G). Equine colic (H). Acute intestinal obstruction	
Week 8	<b>Principles of Treatment of the GIT diseases</b>	Sisay G
	(1). Fluid and electrolyte therapy (2). Relief of distension (3). Restoration Rumen flora and correction of Acidosis and Alkalosis	
<b>Week 8</b>	<b>Test Two</b>	<b>Sisay G.</b>
Week 9	<b>Diseases of the new born (Neonatal infection)</b>	Dr. Abdi F
Week 10	<b>Diseases of the Cardiovascular system</b>	Dr. Abdi F
	(A). Principle of circulatory failure (B). Manifestation of circulatory failure (C). Pericarditis /TRP (D). Arterial thrombosis	
Week 11	<b>Diseases of the Blood and Blood Forming Organs</b>	Dr. Abdi F
	(1). Shock (2). Edema (3). Anemia	

Week 12	<b>Disease of the Urinary system</b> (A). Renal ischemia (B). Nephritis (C). Cystitis (D). Urolithiasis	Dr. Abdi F
Week 13	<b>Diseases of the Nervous system</b> (A). Manifestation of disease of nerves system (B). Encephalitis (C). Encephalomalacia (D). Meningitis	Dr. Abdi F
Week 14	<b>Disease of the Musculoskeletal system</b> (A). Myopathy (B). Myositis (C). Arthritis and synovitis	Dr. Abdi F
Week 15	<b>Diseases of the Skin and conjunctiva</b> (A). Photosensitization (B). Dermatitis (C). Ocular disease	Dr. Abdi F
<b>Week 16</b>	<b>Revision</b>	<b>Students</b>
<b>Final Examination</b>		<b>Dr. Abdi F</b>

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**References:**

1. Rdstits, O.M, Gay, C.C., Hinchcliff W.K., Constable, P.D. (2007). Veterinary Medicine: A text book of the disease of cattle, horses, sheep, pigs and goats, 10th ed. Elsever, london.
2. The Merk Veterinary manual (2000).Merck &CO.INC White house station, New jersey, USA
3. Seifert, H.S.M, (1996). Tropical animal health George-August university, kluver, Academic publication Dordrecht, Germany
4. Bradfor, p.s, (1996). Large Animal internal medicine, 2nd, edn. St. louis, Missouri USA
5. Howard (1981). Current veterinary therapy; Food Animal practice. WB saunders, Philadelphia USA.
6. Hungerford. r.G. (1975). Diseases of Livestock 9th ed. Sydney, Australia
7. Parker, W.H (1980). Health and disease in farm animals/3rd ed. pajamos press, oxford England
8. Sewll, M.M.H. and Brockles by, D.W. (1990). Handbook on animal Diseases in tropics, 4th ed. Baillere tindall, London.



**ADDIS ABABA UNIVERSITY**



**CVMA**

**Department of Clinical Studies**

**Doctor of Veterinary Medicine (DVM), Year III,  
Semester: II, Academic Year: 2019/2020**

**Veterinary General Medicine (Vetm3132)**

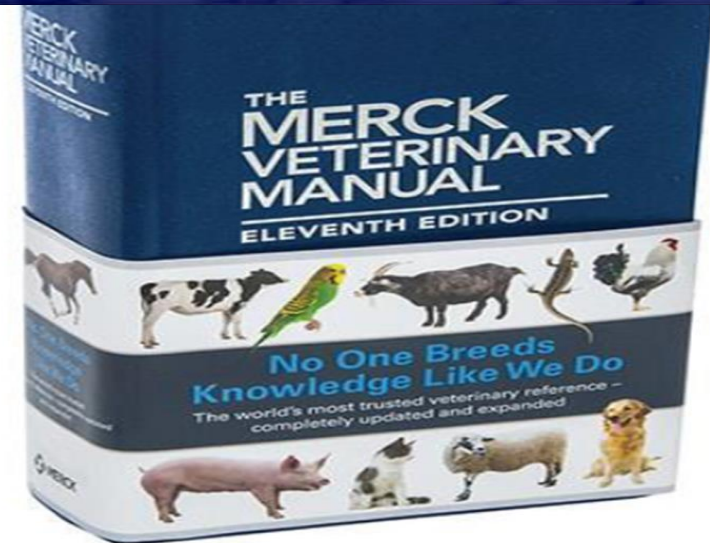
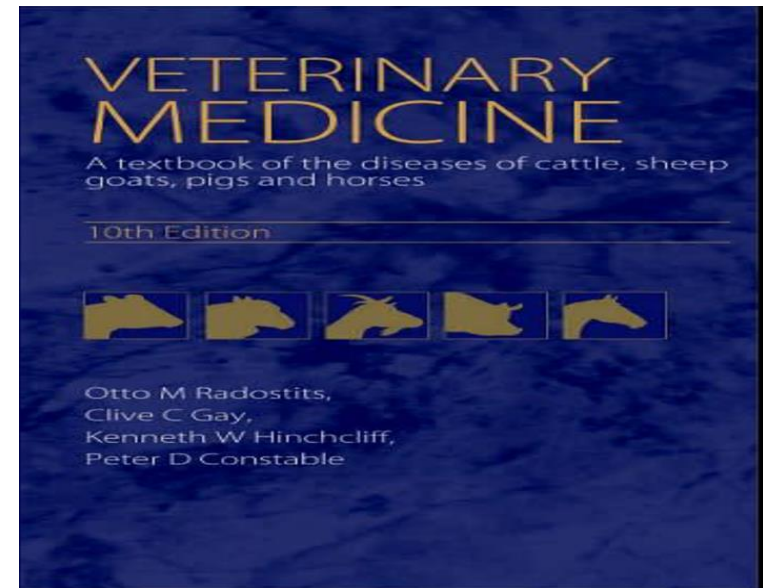
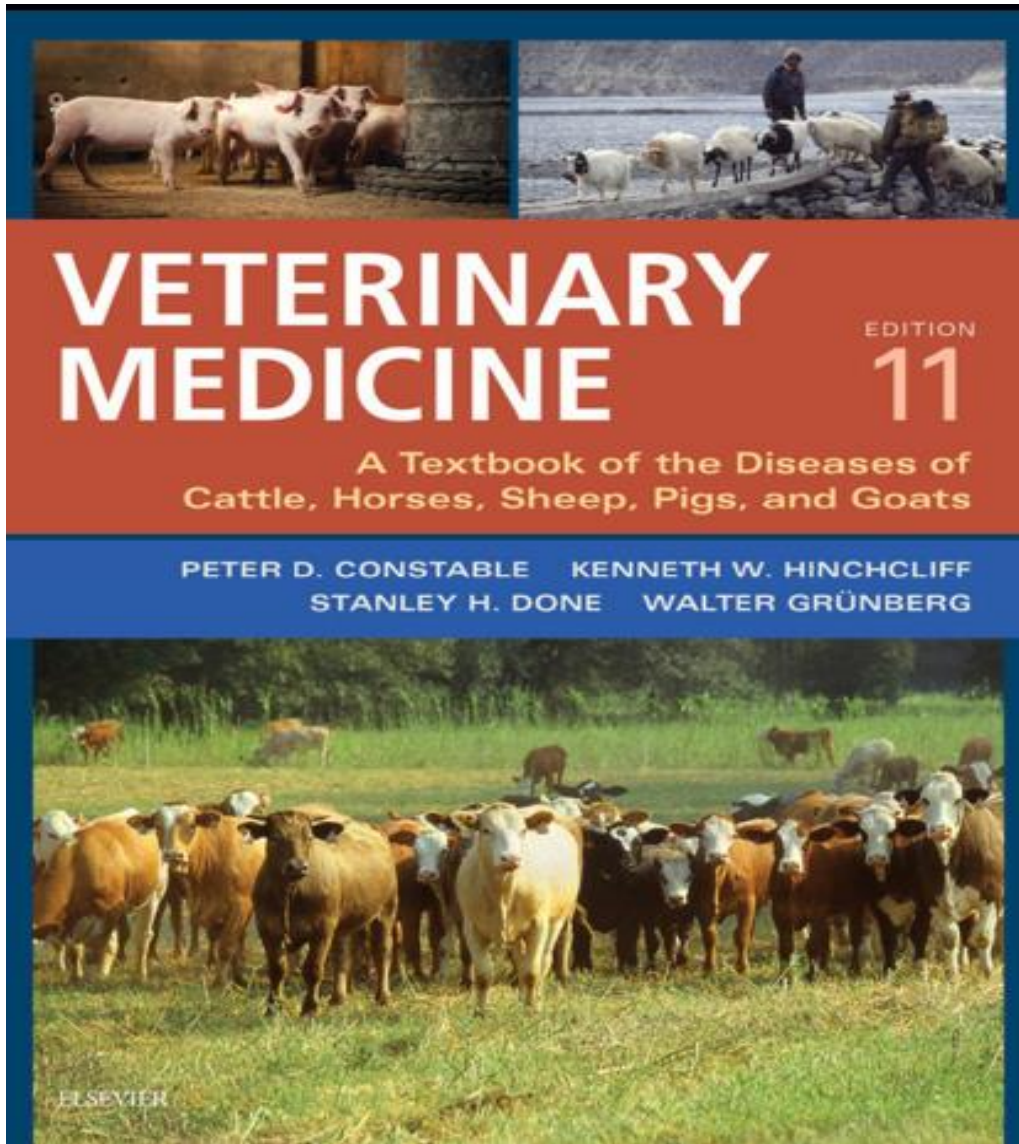


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**Bishoftu, Ethiopia, 19 February, 2020**

# Recommended Text Books



# Chapter one:- General Systemic States

- **General Systemic States** are states which **common to so many diseases** and they are **considered the base** which contribute to the **effects of many diseases**
    - I.e. There are several general systemic states that contribute to **the effects of many diseases**
    - **Because** these systemic states are common to many diseases
  - **General Systemic States** (i.e. General Systemic alterations) **Includes:-**
    - ✓ **Fever**
    - ✓ **Hyperthermia**
    - ✓ **Hypothermia**
    - ✓ **Toxemia**
    - ✓ **Septicemia / Viremia**
    - ✓ **Pain**
    - ✓ **Disturbances of body fluids, Electrolytes and Acid-Base balance**
- Abnormal temperature,  
i.e. characterized by significant changes in  
body temperature

# Body Temperature

- Farm animals maintain a **relatively constant body core temperature (homeothermy)** during **extreme ranges of thermal environments**
- This **homeothermic state** (i.e. constant body core temperature) is achieved by physiologic and behavioral mechanisms that modify **either rates of heat loss** from the body or **the rate at which heat is produced** by the metabolism of feed or body energy reserves
- For the **body temperature to remain constant** in changing thermal environments, **the rate of heat loss** must **equal the rate of heat gain**
- The **balance between heat gain** and **heat loss** is controlled by the heat-regulating functions of the **hypothalamus**



- The **body temperature** is a reflection of the **balance between**:
  - **Heat gain from the environment** (radiation, conduction, and convection) or **caused by metabolic activity** (maintenance, exercise, growth, lactation, gestation, and feeding) and
  - **Heat loss to the environment** (radiation, conduction, convection, and evaporation) or **caused by metabolic activity** (milk removal, fecal elimination, and urinary elimination)
- Absorption of heat from the environment occurs when the **external temperature rises above** that of the body
- **Heat production occurs** as a result of metabolic activity and the digestion of feed, muscular movement, and the maintenance of muscle tone
- **Shivering thermogenesis** is a response to sudden exposure to cold and is a major contributor to enhanced heat production

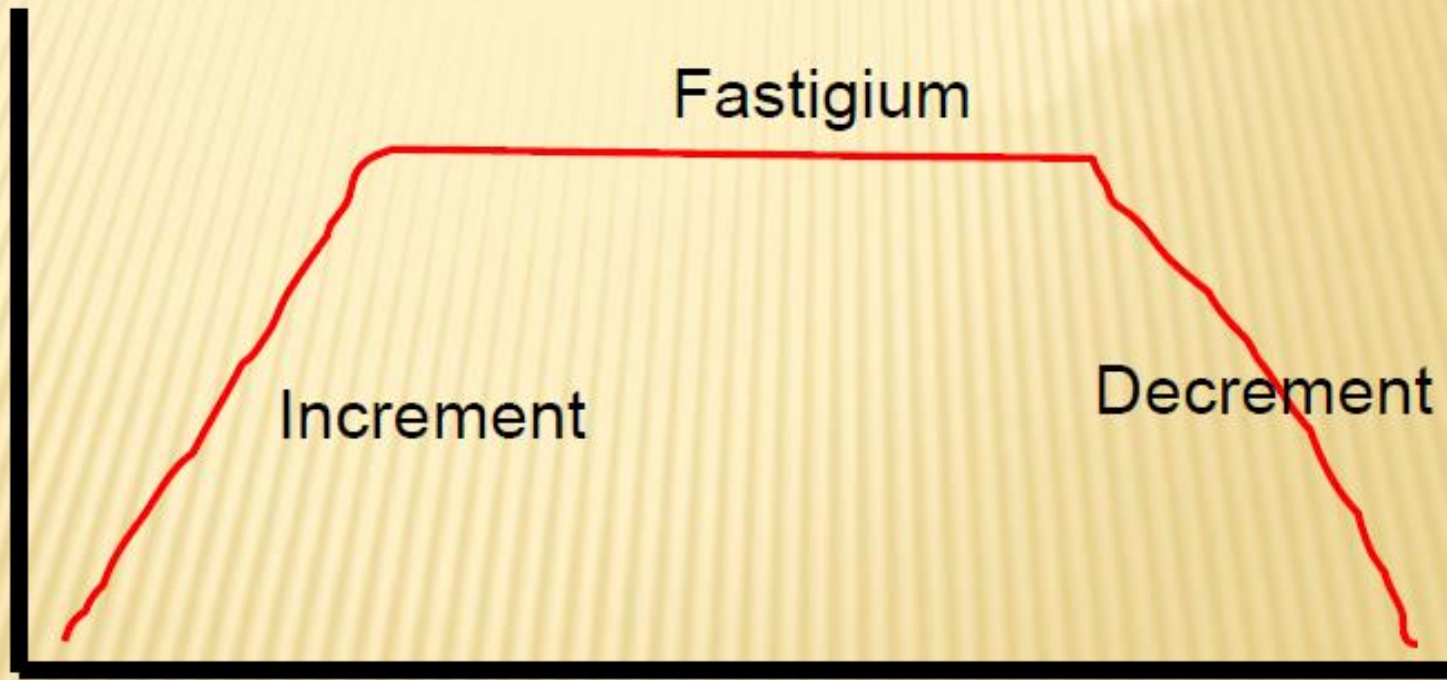
# ABNORMAL TEMPERATURE

- ✘ Fever
- ✘ Hyperthermia
- ✘ Hypothermia

characterized by physiologically significant changes in core body temperature

# FEVER (PYREXIA)

## STAGES OF FEVER



# Fever (Pyrexia):

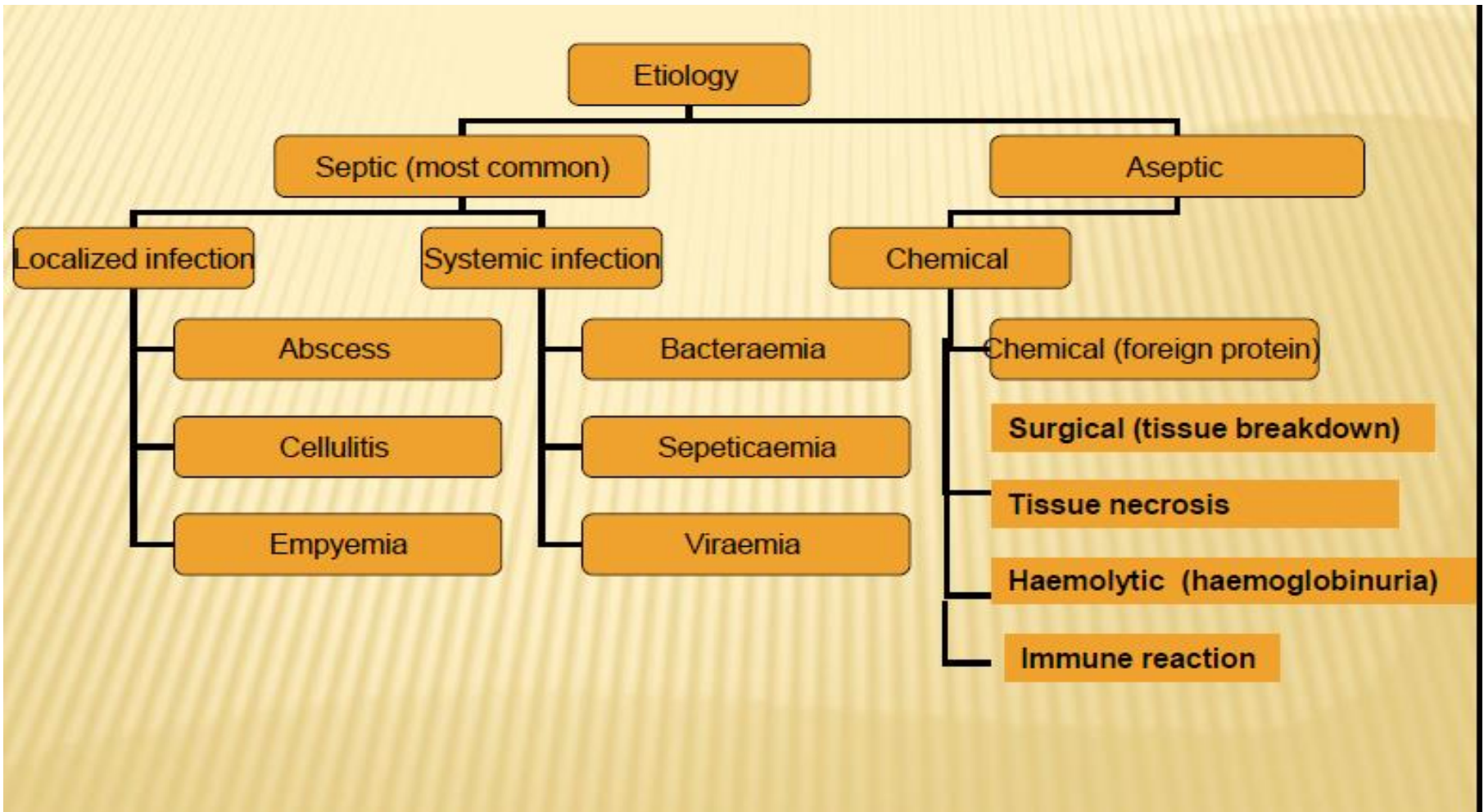
- Is **rise of core body temperature above normal limit** independent to the **effect of the ambient conditions**
- i.e. Fever is **an elevation of body temperature** that exceeds the normal daily variation and occurs in **conjunction with an increase** in the hypothalamic set point
  - i.e. It is an elevation of **core body temperature** above that normally maintained by an animal and is independent of the effects of ambient conditions on body temperature
- It is important to realize that fever is **a combination of hyperthermia and infection or inflammation** that results from an **elevated set point for temperature regulation**

# The normal temperature of different animals:

Animals	Range °C	Average	Animal	Range °C	Average
Horse (adult)	37.2 - 38	37.6	Camel	35 - 38.6	36.8
Foal	37.5 - 38.5	38	Pig (adult)	37.8 - 38.9	38.3
ox	37.8 - 39.2	38.5	Piglet	38.9 - 40	38.4
Calf	38.5 - 39.8	39.2	Small dog	38.6 - 39.2	38.9
Sheep	38.9 - 40	39.5	Large dog	37.5 - 38.6	38
Goat	38.6 - 40.2	39.5	Guinea pig	37.5 - 39.4	38.4
Rabbit	39.9 - 40.5	39.8			
Cat	37.8 - 39.2	38.5			

# Etiology of Fever

- Fevers may be **septic**, the more common type, or **aseptic**, depending on whether or not infection is present



## Septic Fevers:-

- These include infection with **bacteria, viruses, protozoa, or fungi** as:
  - **Localized infection** such as abscess, cellulitis, and empyema
  - Intermittently systemic, as in bacteremia and endocarditis
  - Consistently systemic, as in septicemia

## Aseptic Fevers:-

- |  |   |
|--|---|
| <ul style="list-style-type: none"><li>▪ <b>Chemical fevers</b>, caused by injection of foreign protein</li><li>▪ <b>Surgical fever</b>, caused by breakdown of tissue and blood</li><li>▪ <b>Fever from tissue necrosis</b>, e.g., breakdown of muscle after injection of necrotizing material</li></ul> | <ul style="list-style-type: none"><li>▪ <b>Severe hemolytic crises</b> (hemoglobinemia)</li><li>▪ Extensive infarction</li><li>▪ Immune reactions such as anaphylaxis and angioneurotic edema</li></ul> |
|--|---|

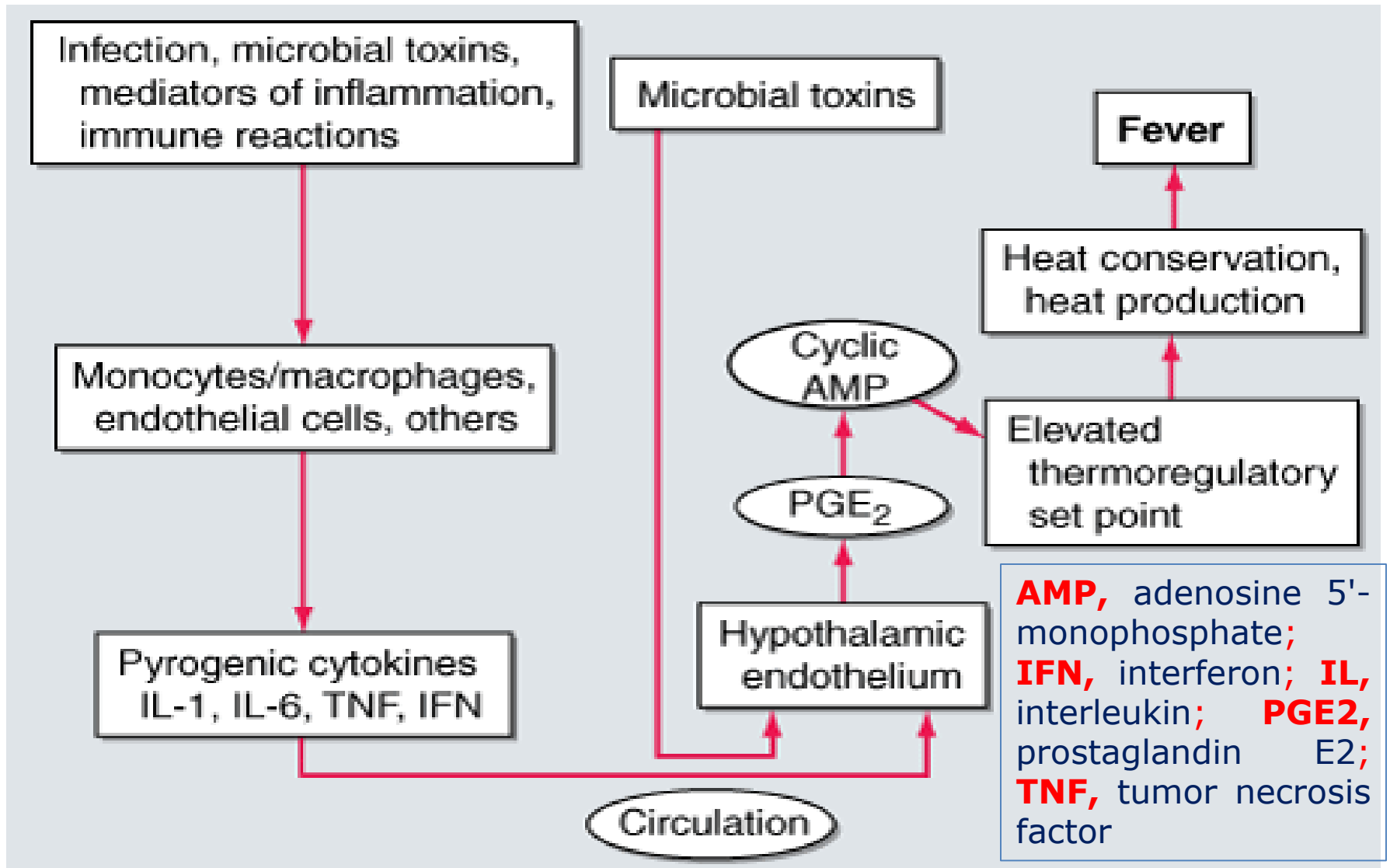
# Pathogenesis of Fever

- Fever may be **provoked** by **many stimuli**, most often, they are **bacteria** and **their endotoxins**, **viruses**, **protozoa**, **immune reactions**, these substances are commonly called **exogenic pyrogens**
  - **Exogenous Pyrogens**- Derived from **outside the patient** and include **microbes** and **their products**, e.g. **lipopolysaccharide endotoxin** produced by all gram negative bacteria, **enterotoxins** produced by staphylococcus and Streptococcal toxins
- Most fevers are mediated through the action of **endogenous pyrogens** produced by **granulocytes**, **monocytes**, and **macrophages**
  - i.e. **Cells stimulated** by exogenic pyrogens to produce cytokines called **endogenic pyrogens**
- **Endogenous Pyrogens**- **Cytokines** are small proteins that regulate immune, inflammatory, and hematopoietic processes produced from the patient, e.g. **IL-1,IL-6, tumor necrosis factor(TNF), ciliary neutrophilic factor(CNF) and interferon (INF)** produced especially by monocytes and macrophages but also by endothelial cells



- Endogenous pyrogens centrally affect the **thermosensitive neurons** in the hypothalamus increase the production of heat and decrease in heat loss
  - **E.g. Interleukin 1** initiates fever by increasing the synthesis of **prostaglandins**, especially **PG E 2** in the **anterior hypothalamus**
  - i.e. The **most important and best known endogenous pyrogen** is **interleukin-1 (IL-1)**, produced by monocytes and macrophages
  - This raises the **thermostatic set point** and induces the mechanism of heat conservation and heat production until the blood and core temp. are elevated to match the hypothalamic set point
  - Therefore, **antipyretics containing prostaglandin synthetase inhibitor** lower fever by **blocking the PG synthesis**

- Pathways of fever production are depicted in the figure below:



# Stages of Fever

## (1). Increment stage (chill period)

- Cutaneous vasoconstriction
- Absence of sweating
- Cold skin and extremities
- Reduced respiration
- Muscle shivering
- Reduced respiration
- Oliguria
- Rectal temperature is elevated

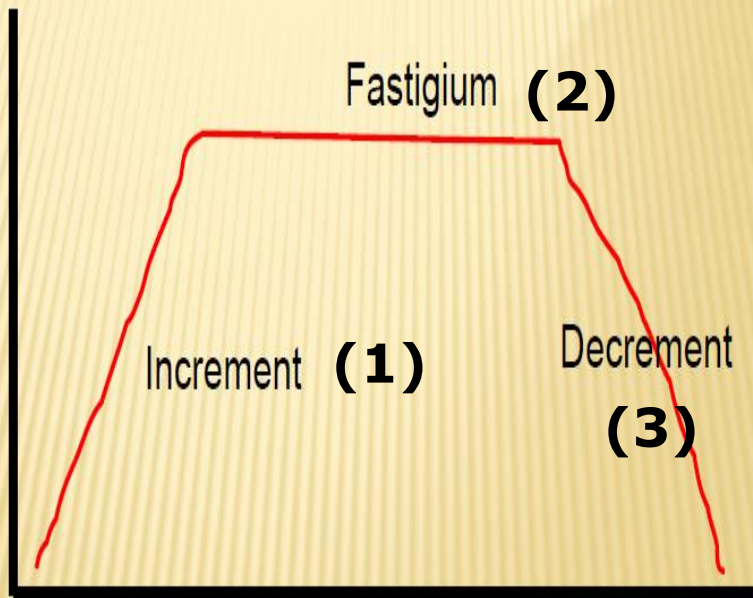
## (3). Decrement stage

- Excess **heat is dissipated** via
  - Vasodilatation
  - Sweating
  - Muscle flaccidity

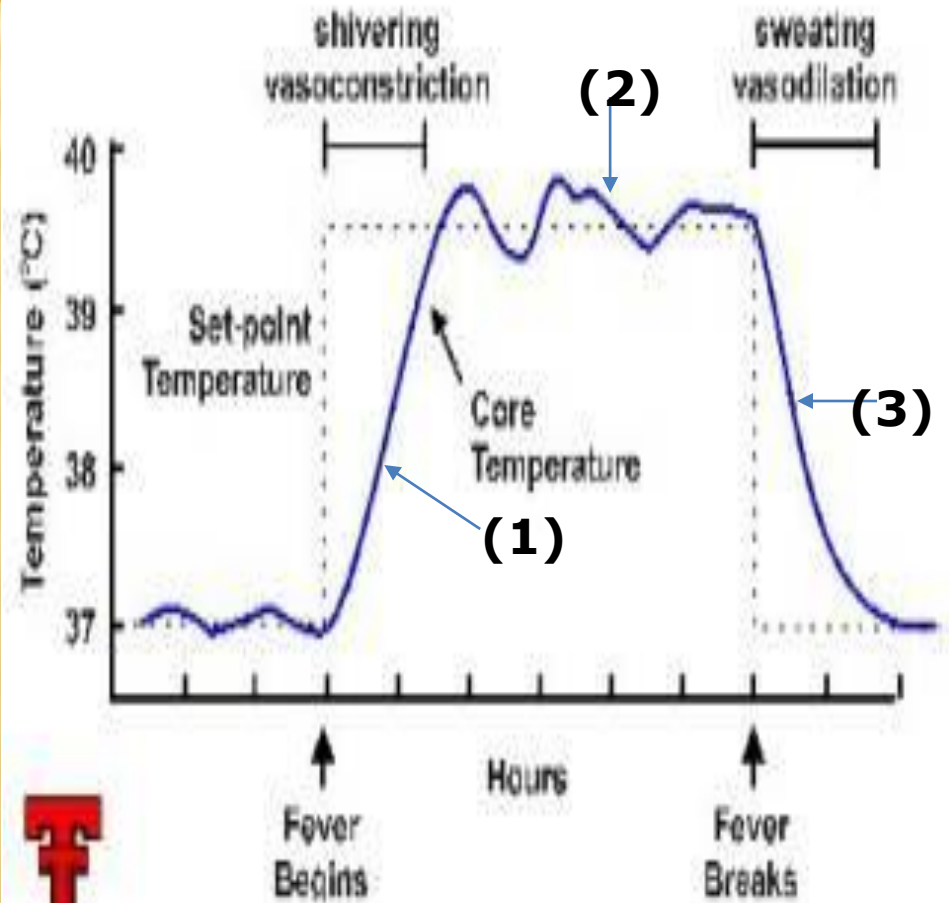
## (2). Fastigium stage (state of constant temp.)

- The period of heat increment raises temperature to the new thermostatic level
- Mechanisms of heat dissipation and production return to normal
- Cutaneous vasodilation to dissipate heat
- Sweating occurs
- Diuresis (increased urine production)
- Decreased ruminal motility
- Increased metabolism and tissue wasting

# STAGES OF FEVER



# Time Course of Typical Fever

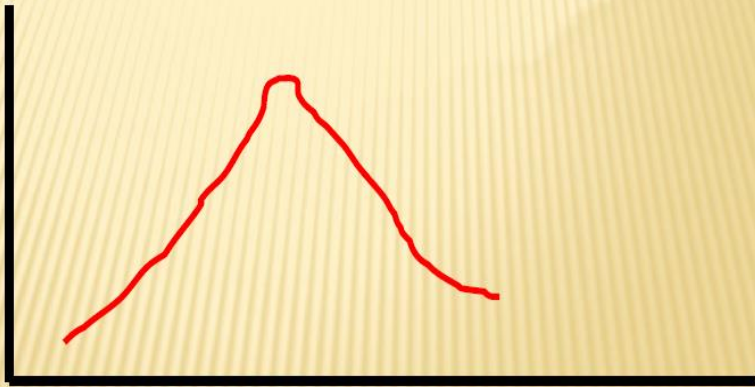


# Forms (Types) of Fever

- **Transient Fever:** fever for **short periods** (hours)
- **Continuous or sustained Fever:** Temperature remains above normal throughout the day and **does not fluctuate** more than 1 °C in 24 hours (i.e. **without diurnal variations**), e.g. typhoid, urinary tract infection
- **Remittant Fever-** Temperature remains above normal throughout the day and **fluctuates more than 1°C in 24 hours** (i.e. **with diurnal variation**), e.g., infective endocarditis, acute tonsillitis
- **Intermittent Fever-** fever is present only for several hours and always touches the baseline at some time of the day
  - i.e. when fever peaks last for 2 3 days and interspersed with normal period

- **Recurrent Fever (biphasic fever)**: peaks of fever for about 6 days interspersed with equal period of normal temp
  - Fever that is constant for several days (5 to 7 days), spontaneously reduce for 1 or 2 days, and then increase again
  - Biphasic fever fevers can be seen in such infections as yellow fever, and influenza
- **Atypical Fever**: when temperature variations are irregular

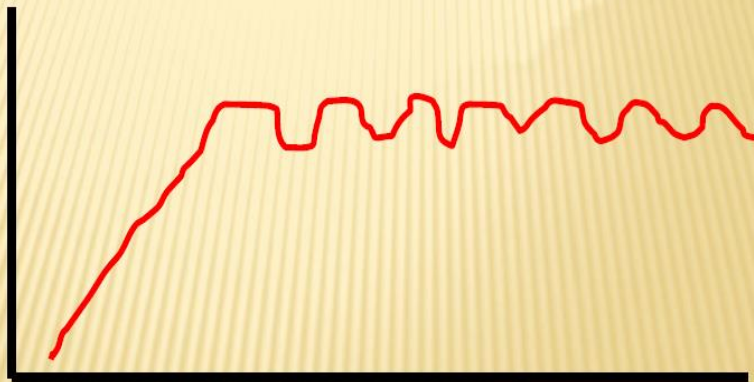
## TRANSIENT FEVER OR SIMPLE FEVER



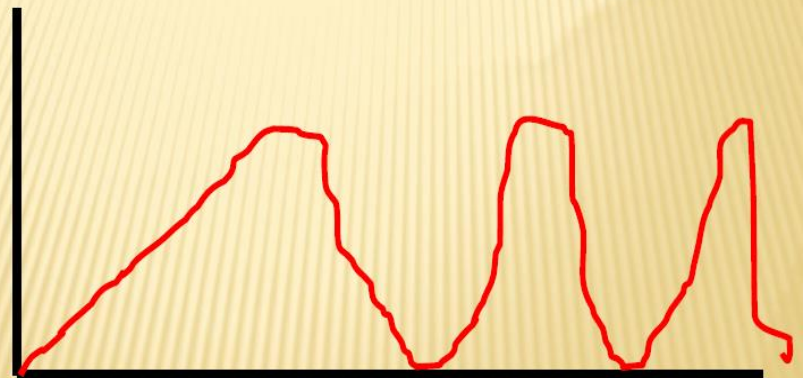
## CONTINUOUS OR SUSTAINED



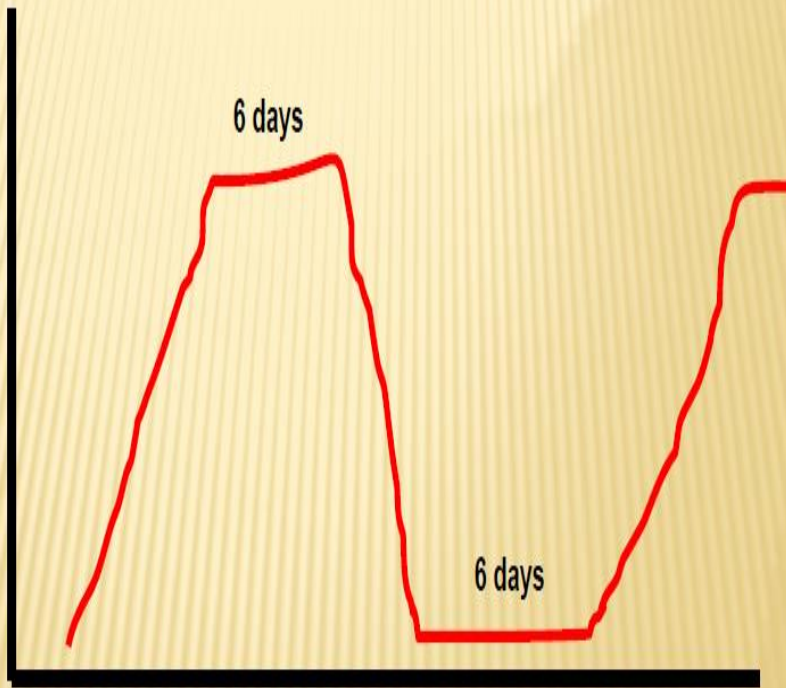
## REMITTENT FEVER



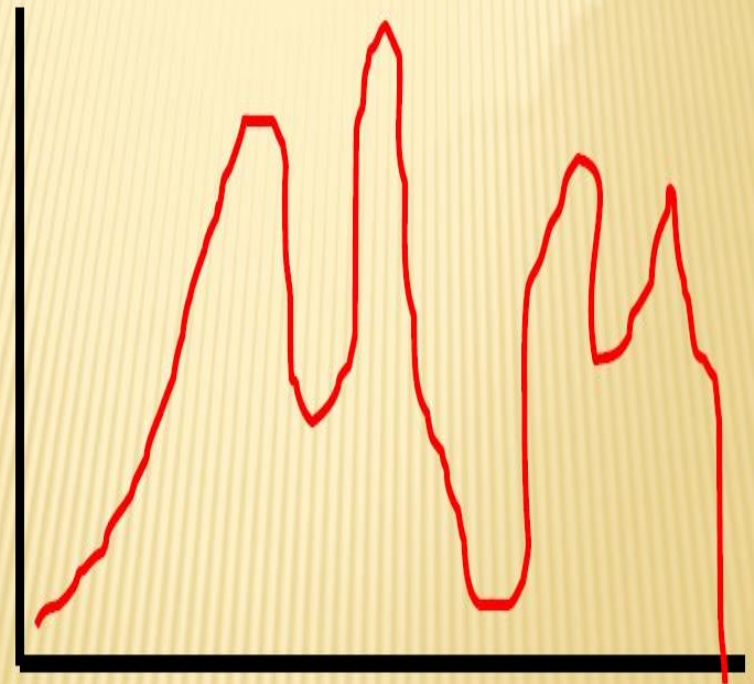
## INTERMITTENT FEVER



## RECURRENT FEVER (**biphasic fever**)



## ATYPICAL FEVER (VARIATIONS ARE IRREGULAR)

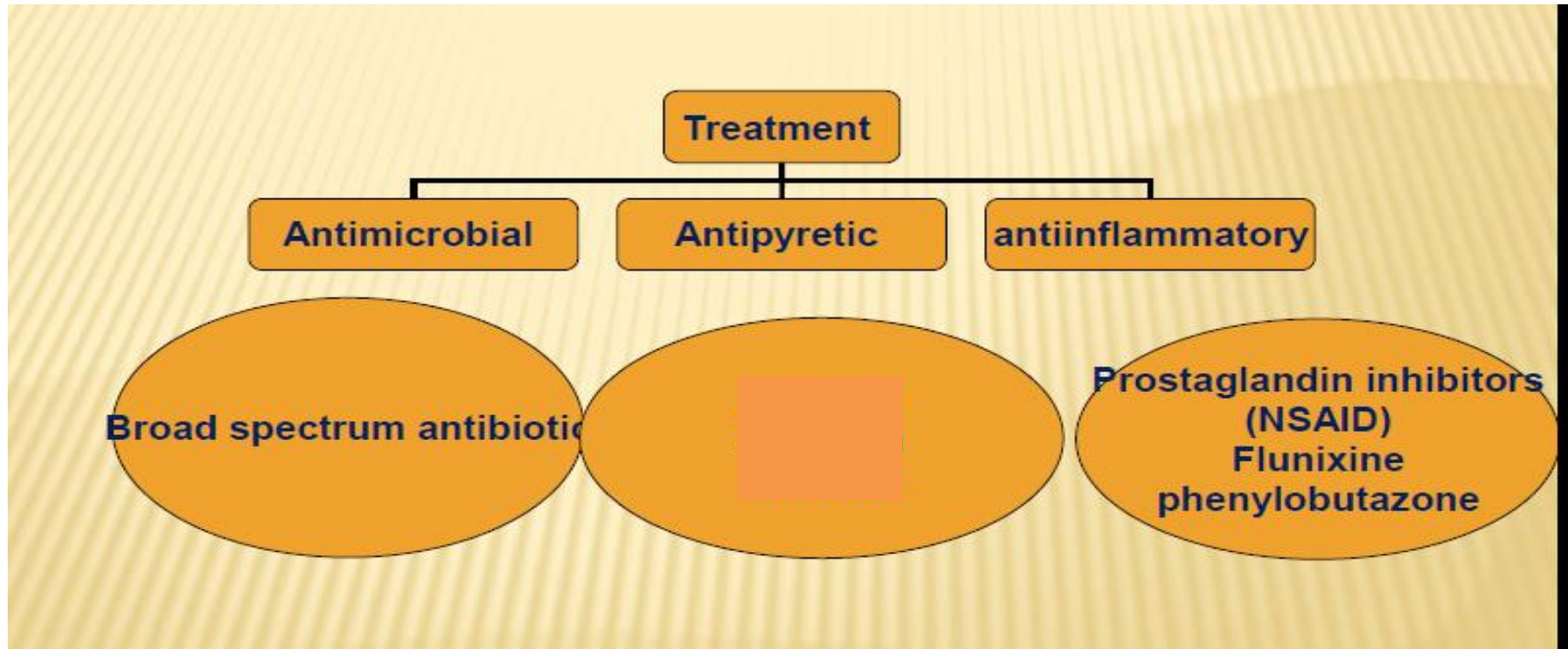




# Clinical Findings of Fever

- ✗ Elevation of body temperature
- ✗ Increased heart rate with diminution of pulse amplitude
- ✗ Hyperpnea (increased respiratory rate)
- ✗ Increased thirst
- ✗ Oliguria (reduced amount of urine) with sometimes albuminuria
- ✗ Scanty faeces
- ✗ Anorexia
- ✗ Depression and muscle weakness

# Treatment of Fever



- If the fever is high enough to cause discomfort or inappetence, or is so high that death from hyperthermia is possible, then nonsteroidal anti-inflammatory drugs (NSAIDs) should be administered
- Most NSAIDs, such as flunixin meglumine, are inhibitors of prostaglandin synthesis and act centrally to lower the thermoregulatory set point
- Rectal temperatures start to decline within 30 minutes of parenteral NSAID administration

# Thank You!!

Questions?



# **HYPERTHERMIA (Heat stroke or Heat exhaustion)**

- **Hyperthermia** is an elevated body temperature (i.e. core body temperature) caused by excessive heat production or absorption, or deficient heat loss
  - (i.e. **Hyperthermia** occurs when the body produces or absorbs more heat than it can dissipate)
- **Hyperthermia** is characterized by an uncontrolled increase in body temperature that exceeds the body's ability to lose heat
- The setting of the hypothalamic thermoregulatory center is unchanged
- In contrast to fever in infections, hyperthermia does not involve pyrogenic molecules
- **Hyperthermia** differs from fever in the mechanism that causes the elevated body temperatures: a fever is caused by a change in the body's temperature set-point, in contrast, hyperthermia occurs when the body temperature rises without a change in the body's temperature setpoint

# Causes of Hyperthermia

- The **most common causes** of **hyperthermia** are **heat stroke & heat exhaustion**
- **Heatstroke & heat exhaustion** are illnesses caused by **exposure to extreme heat** (i.e. **both** result from **overexposure** to extremely hot weather)
  - **Heat stroke** is defined as **a core body temperature** of **40.5 °C (105°F)** with **associated central nervous system dysfunction** in the setting of **large environmental heat load** that cannot be dissipated
  - Heat stroke is due to **an environmental exposure to heat**, resulting in **an abnormally high body temperature**
  - **Heat stroke** is an acute condition of **hyperthermia** that is caused by **prolonged exposure** to **excessive heat** or heat and humidity (i.e. **exposure to high environmental temperature** & high environmental humidity)
  - The **heat regulating mechanisms** of the body **eventually become** overwhelmed and **unable to effectively deal with the heat**, causing the **body temperature to climb uncontrollably**

- **Heatstroke** also called **sunstroke**, is the **most serious heat related illness** and it occurs when the body's temperature is **40.5 °C** or **higher**, and it is **life-threatening**
- **Heat exhaustion** is **less serious** than heatstroke, **if left untreated**, **heat exhaustion can progress** into **heatstroke**

## Comparative Features of Heat Exhaustion and Heat Stroke

Feature	Heat Exhaustion	Heat Stroke
Body Temperature	<39°C	41°C
CNS Dysfunction	Mild	Severe
Sweat Production	Yes	Occasionally
Dehydration	Yes	Yes
Multiorgan Dysfunction (e.g., rhabdomyolysis, acute renal failure)	No	Yes

- **Heat stroke (heat exhaustion)** is the **most common** encountered **clinical entity**

## Other causes of hyperthermia are:

- **Neurogenic hyperthermia:** Damage to hypothalamus, **e.g.**, spontaneous hemorrhage, may cause hyperthermia
  - i.e. It is central nervous system damage- Cerebral hemorrhage, hypothalamic injury
- **Dehydration:** Caused by **insufficient tissue fluids** to accommodate heat loss by evaporation
- **Excessive muscular activity:** For example, strychnine poisoning
- **Drug induced hyperthermia:** is relatively rare side effect of many drugs, particularly those that affect the central nervous system

# Clinical findings of Hyperthermia

- **In most species** the first observable **clinical reaction** (manifestation) to **hyperthermia occurs** when the **rectal temperature** is **increased by 3 to 4°C (4–7°F)** above normal
  - i.e. In most instances the **temperature exceeds 42°C (107°F)** and may **reach 43.5°C (110°F)**
- **An increase in heart** and **respiratory rates** with **a weak pulse, sweating,** and **salivation occur initially,** followed by a marked absence of sweating
- The animal may be **restless** but **soon becomes dull,** stumbles while walking, and **tends to lie down**
- In the early stages there is **increased thirst** and **the animal seeks cool places,** often lying in water or attempting to splash itself



- **Additional increases in rectal temperature** lead to labored respiration and general distress is evident
- Beyond this point the respirations become shallow and irregular, the pulse becomes very rapid and weak, and these signs are usually accompanied by collapse, convulsions, and terminal coma
- **Death occurs in most species** when the core temperature exceeds the normal value by approximately **5 to 7°C** (8–10°F)
- **Abortion may occur** if the period of hyperthermia is prolonged,

# Diagnosis of Hyperthermia

- **Hyperthermia** is generally **diagnosed in the presence** of **an unexpectedly high body temperature** and a history that suggests **hyperthermia instead of a fever**
  - Most commonly **this means that** the **elevated temperature** has **appeared in the individual's exposed to high environmental temperature & high environmental humidity (heat stroke)**
- The **absence of signs and symptoms more commonly related to infection-related fevers**, are also considered **in making the diagnosis**
- **If fever-reducing drugs lower the body temperature, even if the temperature does not return entirely to normal, then hyperthermia is excluded**

# Treatment of Fever

- **Rapid reduction of body temperature** by **physical means** accomplished by **adequate drinking water**, **together with shade** (i.e. affected animals should be **immediately placed in the shade**)
- Because **affected animals may not be interested in or capable of drinking**, the **intravenous administration of fluids** such as **0.9% NaCl** is indicated in animals that are weak, recumbent, or dehydrated
- **Antipyretics** are of no use

# HYPOTHERMIA



- **Hypothermia** is the decrease in body temperature **than normal** and it occurs **due to excess loss of heat** or when there is insufficient production of heat
- Hypothermia occurs when **an organism's temperature drops below that required** for **normal metabolism**
- **Hypothermia** is caused by prolonged exposure to low environmental temperatures
  - i.e. Hypothermia **is the condition of the body** where the **core temperature of the body is below 35° C** (e.g. in humans)
- It is the **major cause** of **morbidity** and **mortality in newborn farm animals** within the first few days of life
- It is a significant problem in **neonates at birth** and beyond ( 15%)

# Causes of Hypothermia

## (i). Situations causing excessive heat loss (Excessive loss of heat)

- it is due to exposure of animal to excessively cold temperatures environment that causes heat loss

## (ii). Insufficient heat production (Poor metabolic heat production)

- If animal body has insufficient body reserves of energy and insufficient feed intake result in insufficient heat production
- Hypothermia also occurs secondary to many diseases in which there may be a decrease in the ability to shiver and skeletal muscle contraction associated with decreased cardiac output, decreased peripheral perfusion and shock
  - Examples include, acute ruminal acidosis, Milk fever and the reduction of metabolic activity that occurs in the terminal stages of many diseases

### (iii). Combination of both

- Combination of excessive heat loss and insufficient heat production if animal take insufficient energy intake or starvation of newborn farm animals in a cold environment can be a major cause of hypothermia
- When newborn expose to cold temperature environment results in body temperature decreases
- The entire body, especially the extremities, becomes cold and the rectal temperature is below 37°C and may drop to 30°C in newborn

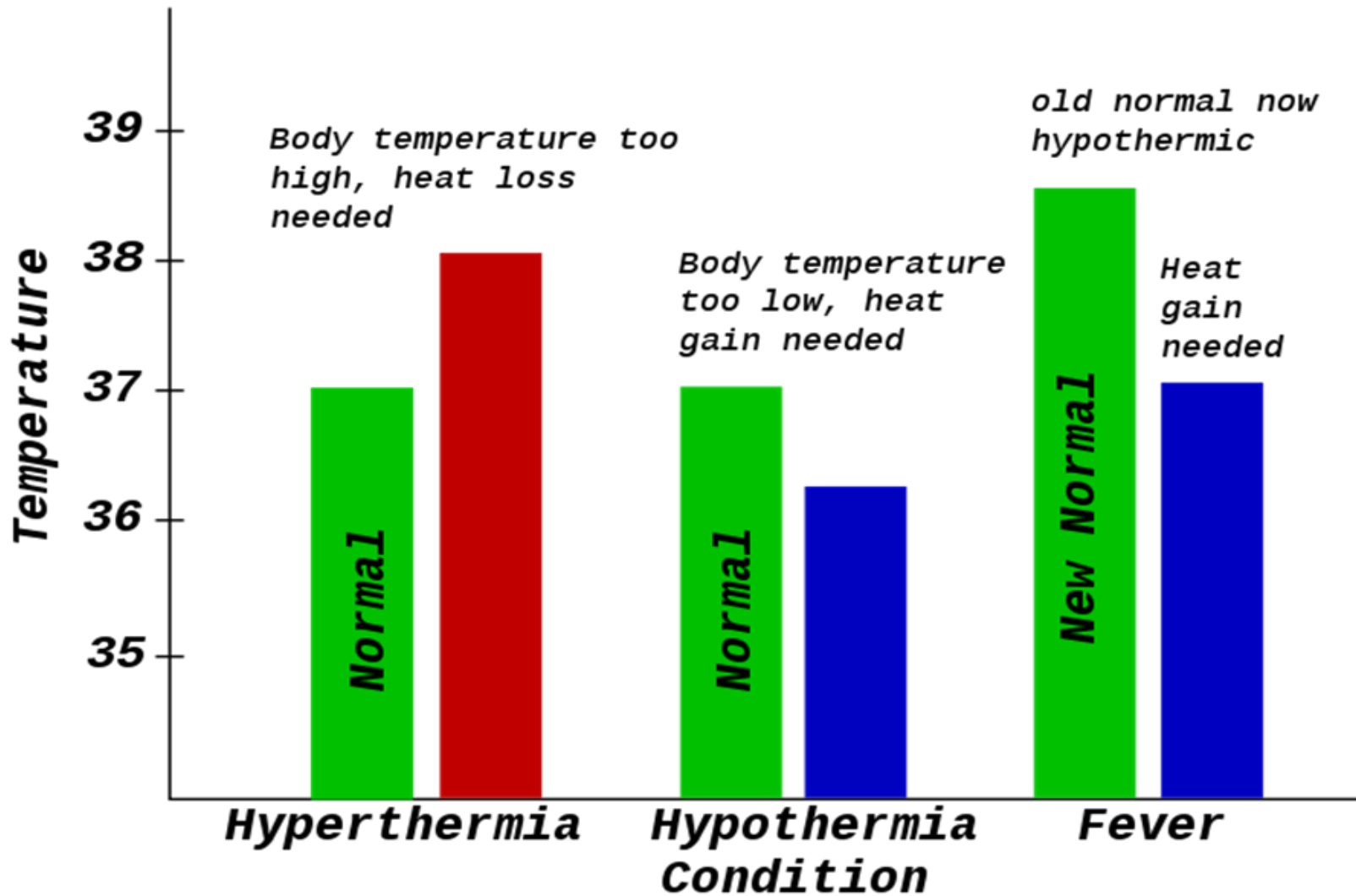
# Risk factors for neonatal hypothermia

i.e. **Why are newborns prone to develop hypothermia?**

- **Limited heat generating mechanisms** (e.g. Nonshivering Thermogenesis)
  - Functional brown adipose tissue is present in newborn calves, lambs, and kids, and its primary function is to generate heat by nonshivering thermogenesis
- **vulnerability** to getting exposed, being dependent others
- **Dystocia:**
  - Dystocia may result in a weak calf that has weak teat-seeking activity, a poor suck reflex and a poor appetite for colostrum, resulting in colostrum deprivation and hypogammaglobulinemia
- **Outdoor Parturition**
  - In Calves un-attending parturition of cow or buffalo during winter can lead to hypothermia in newborn those calves born outdoors during cold weather are susceptible to hypothermia
- **Wetting of the birth coat** (wetness of the fleece)



# A summary of the differences between hyperthermia, hypothermia, and fever



## Hyperthermia: Characterized on the left:-

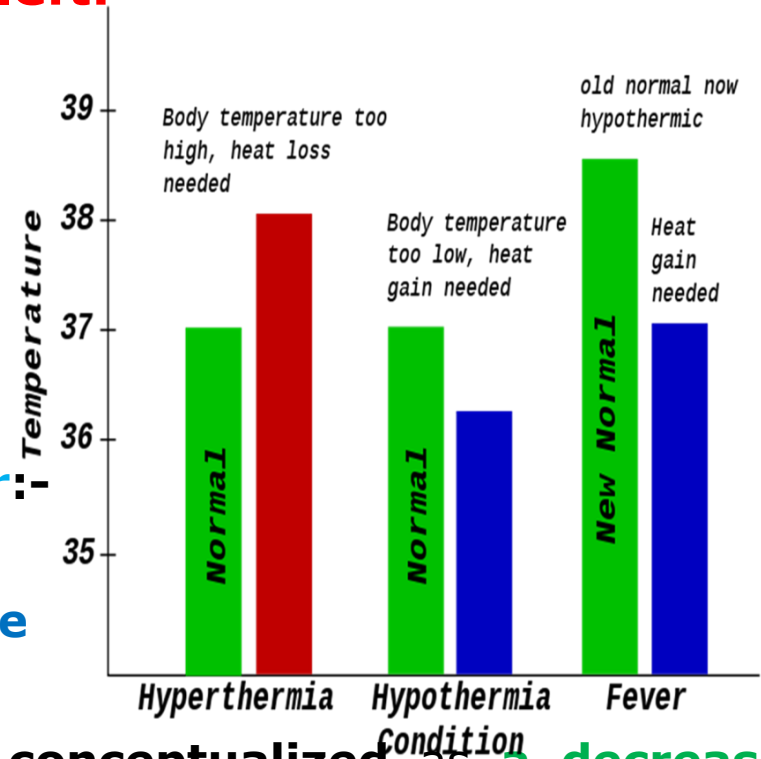
- **Normal body temperature** (**thermoregulatory set-point**) is shown in **green**, while the **hyperthermic temperature** is shown in **red**
- As can be seen, **hyperthermia** can be considered an **increase above the thermoregulatory set-point**

## Hypothermia: Characterized in the center:-

- **Normal body temperature** is shown in **green**, while **hypothermic temperature** is shown in **blue**
- As can be seen, **hypothermia** can be **conceptualized as a decrease below the thermoregulatory set-point as shown in green**

## Fever: Characterized on the right:-

- **Normal body temperature** is shown in **green** (It reads "New Normal" because the thermoregulatory set-point has risen)
- This has **caused what was the normal body temperature** (in **blue**) to be considered hypothermic



# Pathogenesis of Hypothermia

- Sudden exposure of neonatal animals at birth and during the first few days of life to cold ambient temperature results in **subnormal body temperature** and shivering as well as decreased cardiac output, heart rate, and blood pressure
  - This results in muscular weakness and mental depression, respiratory failure, recumbency, and a state of collapse and, eventually, coma and death
- The entire body, especially the extremities, becomes cold and the rectal temperature is below  $37^{\circ}\text{C}$  and may drop to  $30^{\circ}\text{C}$  or lower in neonates
- Nonshivering-induced thermogenesis may occur, resulting in depletion of brown adipose tissue deposits

# Clinical findings of Hypothermia

- A decrease in body temperature to below 37°C represents hypothermia for most farm animal species,
  - Weakness,
  - Decreased activity,
  - Cold extremities, and varying degrees of shock are common
  - Bradycardia,
  - Weak arterial pulse, and collapse of the major veins are characteristic
  - The mucous membranes of the oral cavity are cool and there is a lack of saliva

# Treatment & Prevention of Hypothermia

- Early recognition and treatment of animals with diseases leading to hypothermia is also necessary
- **Severe hypothermia** (<37°C) are dried off and given an intraperitoneal injection of 20% glucose (39°C)
- Immersion of hypothermic lambs in water at 38°C

## ■ Measures to minimize excessive heat loss include:-

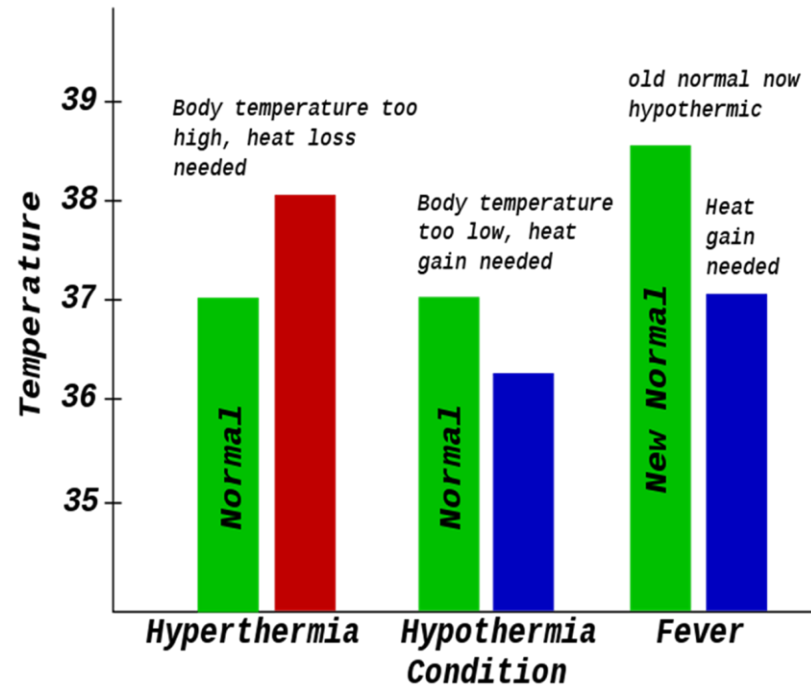
- Providing a dry environment for calving and lambing
- Providing a protective shelter for animal for calving and during the first week after birth can reduce mortality from Hypothermia
- The provision of adequate surveillance and assistance at the time of lambing or calving is necessary to minimize the incidence of dystocia and its consequences for the neonate
- The ingestion of adequate quantities of colostrum, beginning as soon after birth as possible, is important in order to provide immunoglobulins and energy sources for the neonate

## Quiz: #1 (5 points)

**(Q1).** Briefly **explain the differences** between **hyperthermia**, **hypothermia**, and **fever** (3 points)

**(Q2).** **Why are newborns prone to hypothermia?** (2 points)

Q1



Q2

- Outdoor Parturition
- Limited heat generating mechanisms (Non shivering thermogenesis)
- Dystocia
- vulnerability to getting exposed, being dependent others



# Thank You!!

Any question??



# SEPTICEMIA / VIREMIA

- **Septicemia** is a **potentially life-threatening infection** in which large amounts of **bacteria** are **present in the blood**
- It is commonly referred to as **blood poisoning** (i.e. Septicemia is also known as blood poisoning)
  - i.e. **Septicemia** is presence of **microbes** or **toxins in the blood**
- **Septicemia** is the **disease State** compounded (constitute) of **toxemia**, **fever** and the **presence of large numbers** of **infectious microorganisms** including **bacteria**, **viruses** and **protozoa** in the **blood stream**

- **Septicemia** is the acute invasion of the systemic circulation by pathogenic (virulent) bacteria accompanied by sepsis and septic shock with possible bacterial localization in various body systems or organs
- i.e. Septicemia is defined as having bacteria in the bloodstream that cause sepsis
- Septicemia is a common cause of morbidity and mortality in newborn farm animals that have not received a sufficient quantity of colostrum in the first 24 hours after birth

# Sepsis:-

- **Sepsis** is a suspected or proven bacterial infection in conjunction **with the presence** of **systemic inflammatory response syndrome (SIRS)**,
  - SIRS is defined as systemic inflammation in response to **injury**, being caused by **infectious agents** (e.g., **bacteria, viruses, protozoa, fungi**) or by **noninfectious causes** (e.g., trauma, toxins, hyperthermia, burns)
  - i.e. SIRS is **Infectious / Noninfectious**
- **Thus, Sepsis** is SIRS with proven or suspected **microbial etiology**
- **Sepsis** results in a complex set of interactions **between** the **inciting microbes** and the **host immune response** which triggers the **inflammatory cascade**
- **Bacterial infections** are the **most common** cause of **sepsis**, but **fungal, viral and protozoan infections** can lead to **sepsis**

## Severe sepsis:-

- **Severe sepsis** is sepsis accompanied by organ dysfunction (it is sepsis with acute organ dysfunction)
- i.e. Severe Sepsis is sepsis with signs of one or more organ dysfunction
- The acute organ dysfunction can manifest in any organ, and frequently manifest clinically as shock, respiratory failure, acute kidney injury, hematologic or metabolic disturbances

## Septic shock:-

- **Septic shock** is defined as severe sepsis with hypotension (mean arterial blood pressure <65 mm Hg) for at least 1 h, despite adequate fluid resuscitation (i.e. despite aggressive intravenous fluid therapy)
- i.e. Septic shock is a form of severe sepsis where the organ dysfunction involves the cardiovascular system (e.g. hypotension)

# Differences Between Bacteremia and Septicemia

- Bacteremia is different from septicemia in that bacteremia is not accompanied by sepsis or septic shock
- In bacteremia, bacteria are present in the bloodstream for only transitory periods and do not produce clinical signs
- i.e. In bacteremia, it is presence of bacteria in blood and evidenced by positive blood culture
- **In septicemia**, the pathogen is present throughout the course of the disease and is directly responsible for initiation of the disease process
- i.e. In septicemia the causative agent is present throughout the course of the disease and is directly responsible for the appeared clinical signs
- **In short, Bacteremia** is the simple presence of bacteria in the blood while **Septicemia** is the **presence and multiplication of bacteria** in the blood

S.N.	<b>Bacteremia</b>	<b>Septicemia</b>
1.	Bacteremia is the <b>simple presence of bacteria in the blood</b>	Septicemia is the <b>presence and multiplication of bacteria</b> in the blood
2.	Bacteremia is <b>not as dangerous as Septicemia</b>	Septicemia is a <b>potentially life-threatening infection</b>
3.	<b>Less amount of bacteria</b> are present in blood	<b>Large amounts of bacteria</b> are present in the blood
4.	This may occur through <b>a wound or infection</b> , or through <b>a surgical procedure or injection</b>	It can arise from <b>infections throughout the body</b> , including <b>infections in the lungs, abdomen, urinary tract &amp; Skin</b>
5.	<b>Toxins are not produced</b>	<b>Toxins</b> may be <b>produced by bacteria</b>
6.	Bacteremia usually causes <b>no symptoms or it may produce mild fever</b>	It shows symptoms like <b>chills, fever, very fast respiration and/or heart rate</b>
7.	<b>It can resolve without treatment</b>	<b>Untreated septicemia can quickly</b> progress to <b>sepsis</b>
8.	Rapidly removed from the <b>bloodstream by the immune system</b>	<b>Antibiotics</b> will be used to treat the bacterial infection that is causing septicemia
9.	<b>Caused by</b> <i>Staphylococcus, Streptococcus, Pseudomonas, Haemophilus, E. coli</i> , dental procedures, herpes (including herpetic whitlow), urinary tract infections, peritonitis	<i>Staphylococci</i> , are thought to cause <b>more than 50% of cases of sepsis</b> , Other commonly implicated bacteria include <i>Streptococcus pyogenes, Escherichia coli, Pseudomonas aeruginosa, Klebsiella</i> species and even <i>Candida</i> spp.

- **Viremia** is the **invasion of the systemic circulation** by **pathogenic viruses** with localization in various body tissues and in which the **lesions produced are characteristic of the specific virus**
- **Many infections associated** with rickettsias, protozoa, and fungi are also spread hematogenously throughout the body **but usually do not initiate a systemic inflammatory response syndrome**



# ETIOLOGY

- Many different infectious agents can result in septicemia or viremia
  - i.e. Septicemia can be a response to any class of microorganism

## Causes of Septicemia in all species of animals are:

- Anthrax, Pasteurellosis, Salmonellosis
- Pseudomonas
- Rift valley fever
- Leptosporidia, sarcosporidia

### Cattle, sheep, pig:

- *Pasteurella multocida*,
- *Pasteurella haemolytica*

### Horse:

*Pasteurella haemolytica*

### Pig:

- Hog cholera, African swine fever
- *Streptococcus zooepidemicus*
- *Erysipelothrix insidiosa*

- **Neonatal septicemias** are caused most commonly by gram-negative bacteria (*E. coli*, *Salmonella*, *P. multocida*)
- E.g., in **Calves**
  - Bacteremia and septicemia are often associated with ***Escherichia coli*** and ***Salmonella spp.***
  - ***E. coli*** is most frequently isolated from the blood of calves
- **Piglets**
  - Septicemia caused by ***E. coli*** is possible along with septicemia with localization in the joints, endocardium, and meninges associated with *Streptococcus suis*
- **Lambs**
  - Septicemia associated with ***E. coli*** occurs most frequently
- **Colostrum-deprived newborn animals** are highly susceptible to septicemia

## Two mechanisms operate in septicemia:-

(1). **The Exotoxins or Endotoxins** produced by infectious agents produce a **profound toxemia** and **high fever** leading to shock:

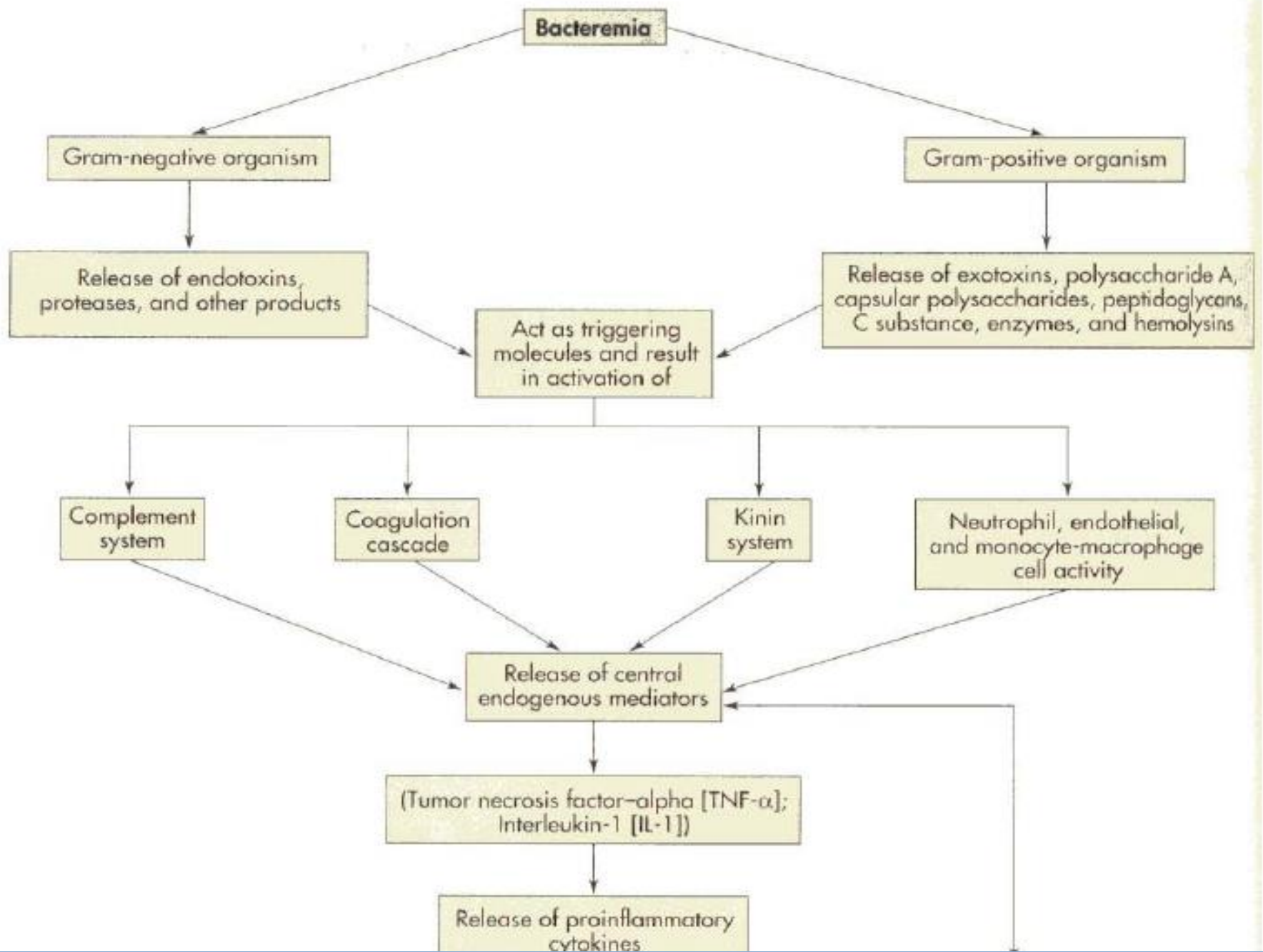
- This is because of **their** (**exotoxins & endotoxins**) **initiation** of the release of **host mediator** and due the **rapidity** by which **they multiply** and the **rapid spread** to all body tissues

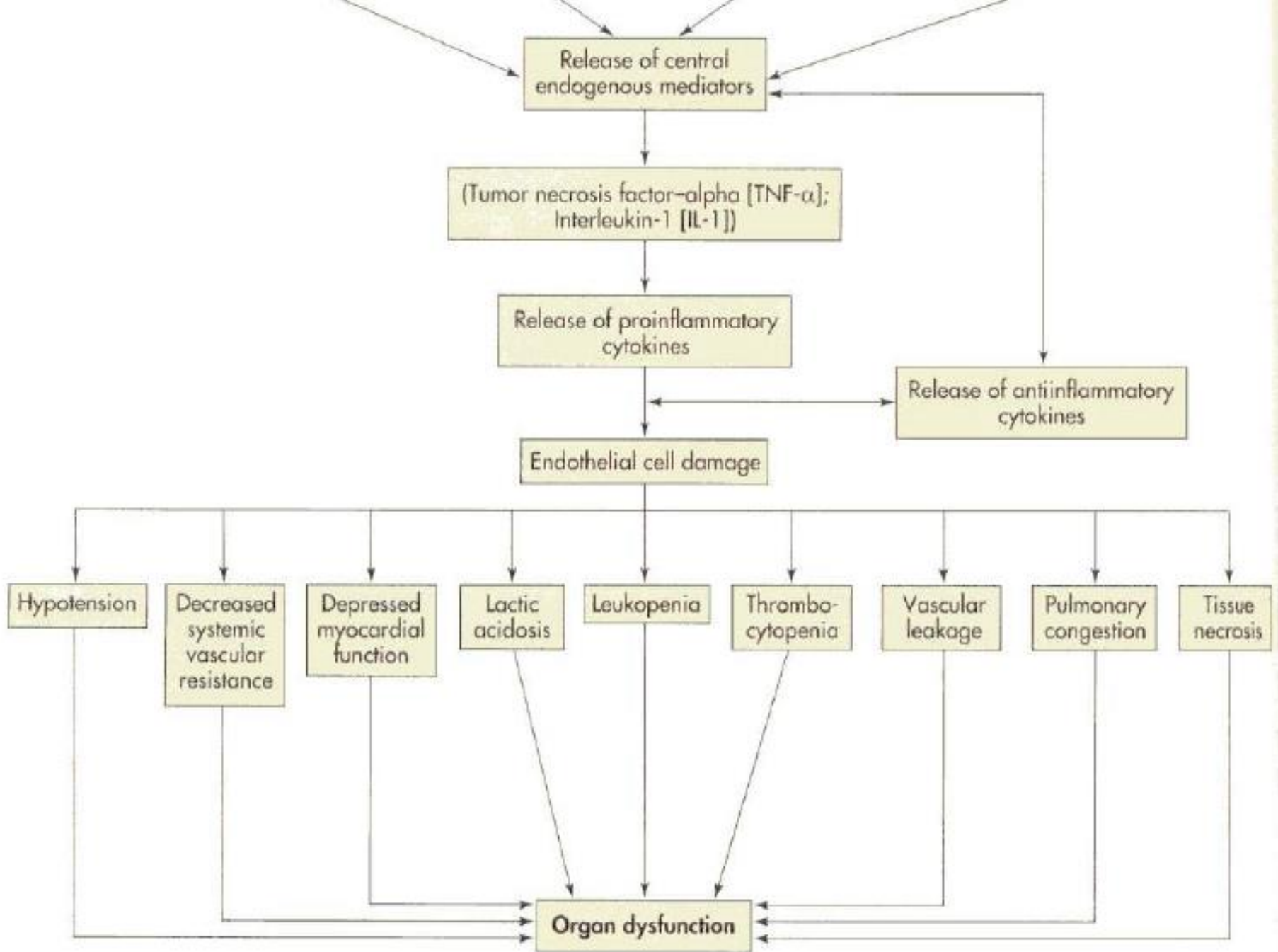
(2) **Localization of certain pathogens** occurs in many organs and may produce **serious lesions in animals**, which survive the toxemia

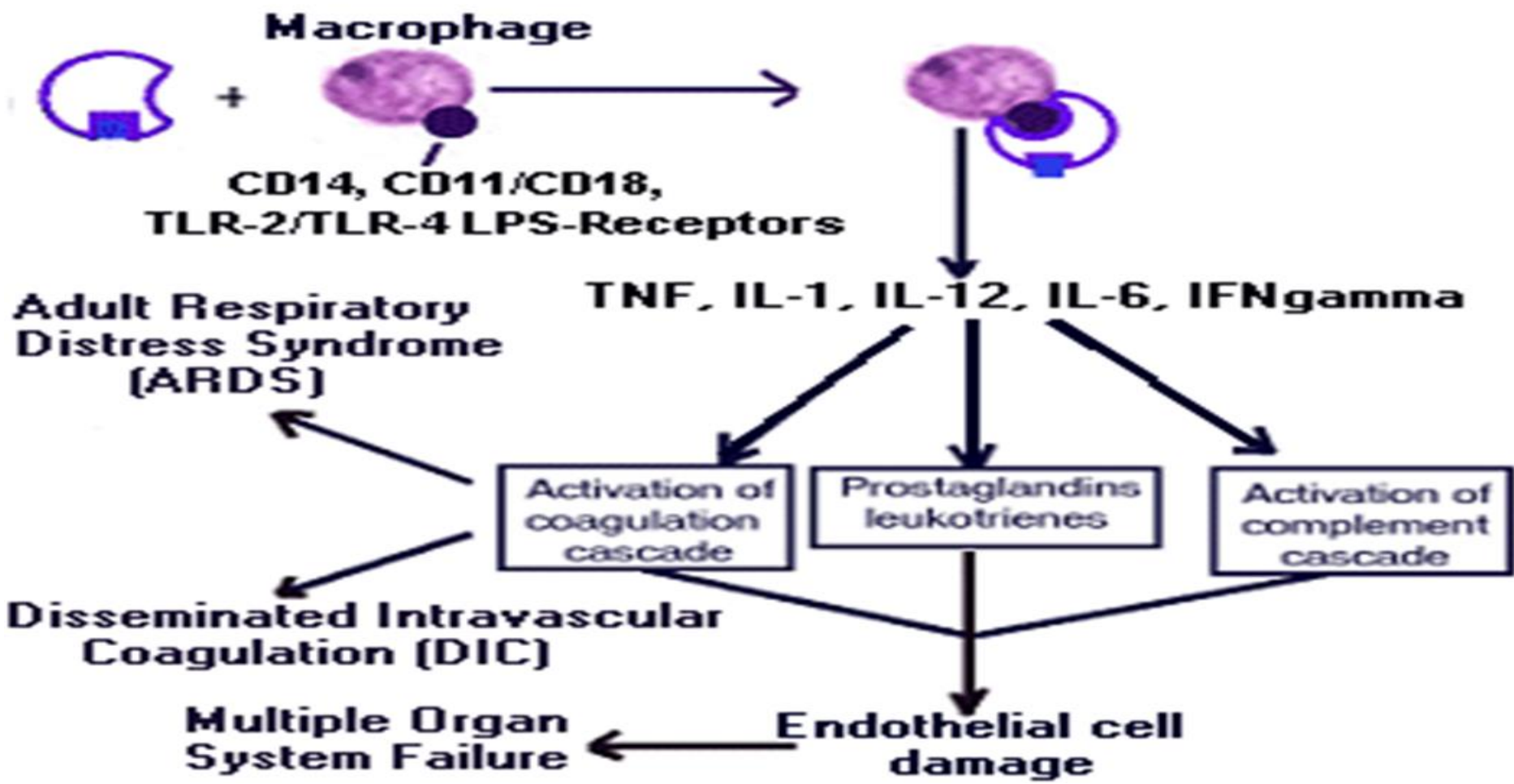
(3) And also cause **direct endothelial damage** and **hemorrhages** into tissues

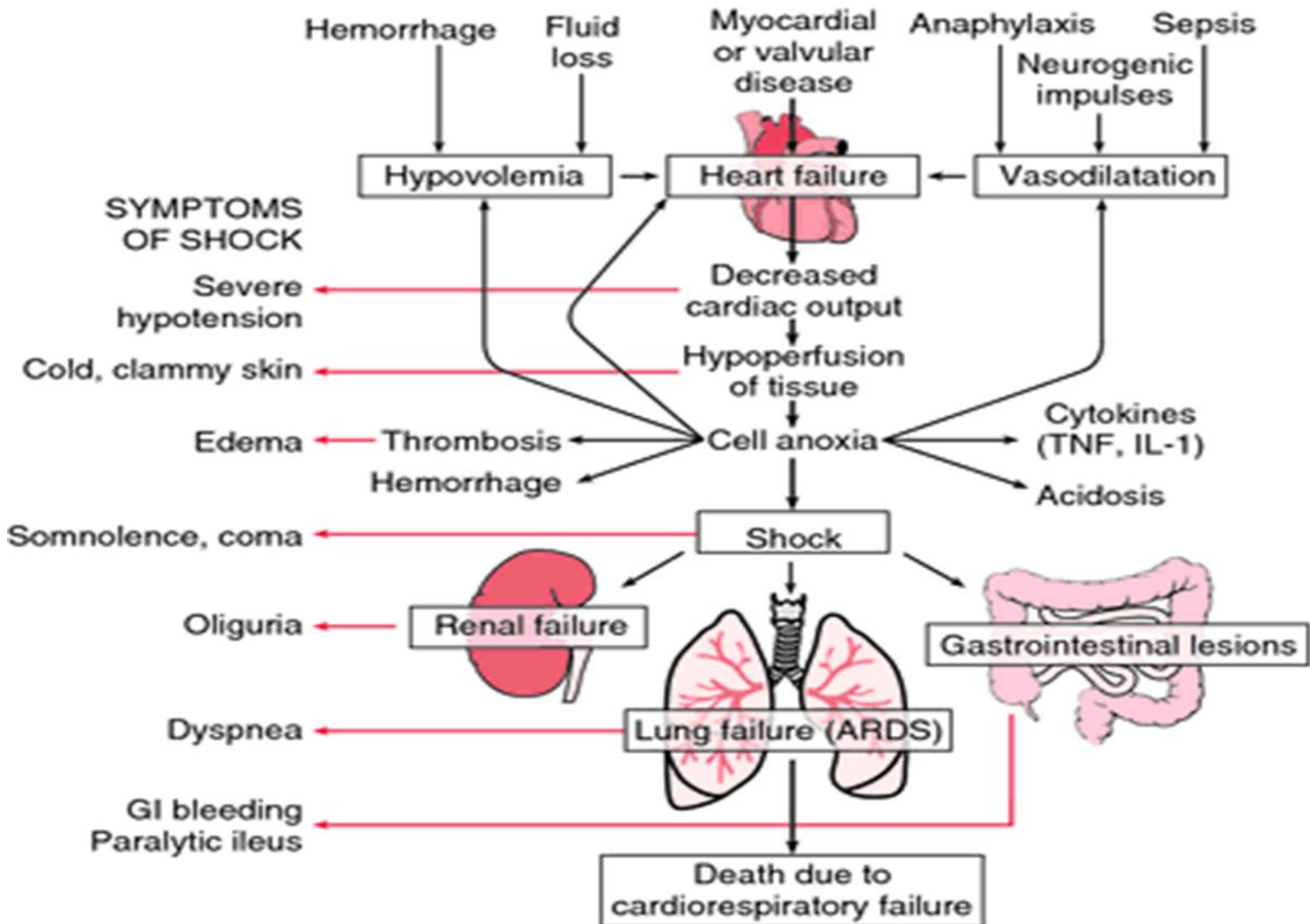
- **In Viremia**, Same general principles apply to a **viremia**, except that **toxins are not produced by virus**, the general signs, which occur, caused by the products of tissue cells killed by the multiplying virus

- **Disseminated intravascular coagulation** occurs in septicemia diseases especially that terminates fatally (common in **severe septicemic disease**)

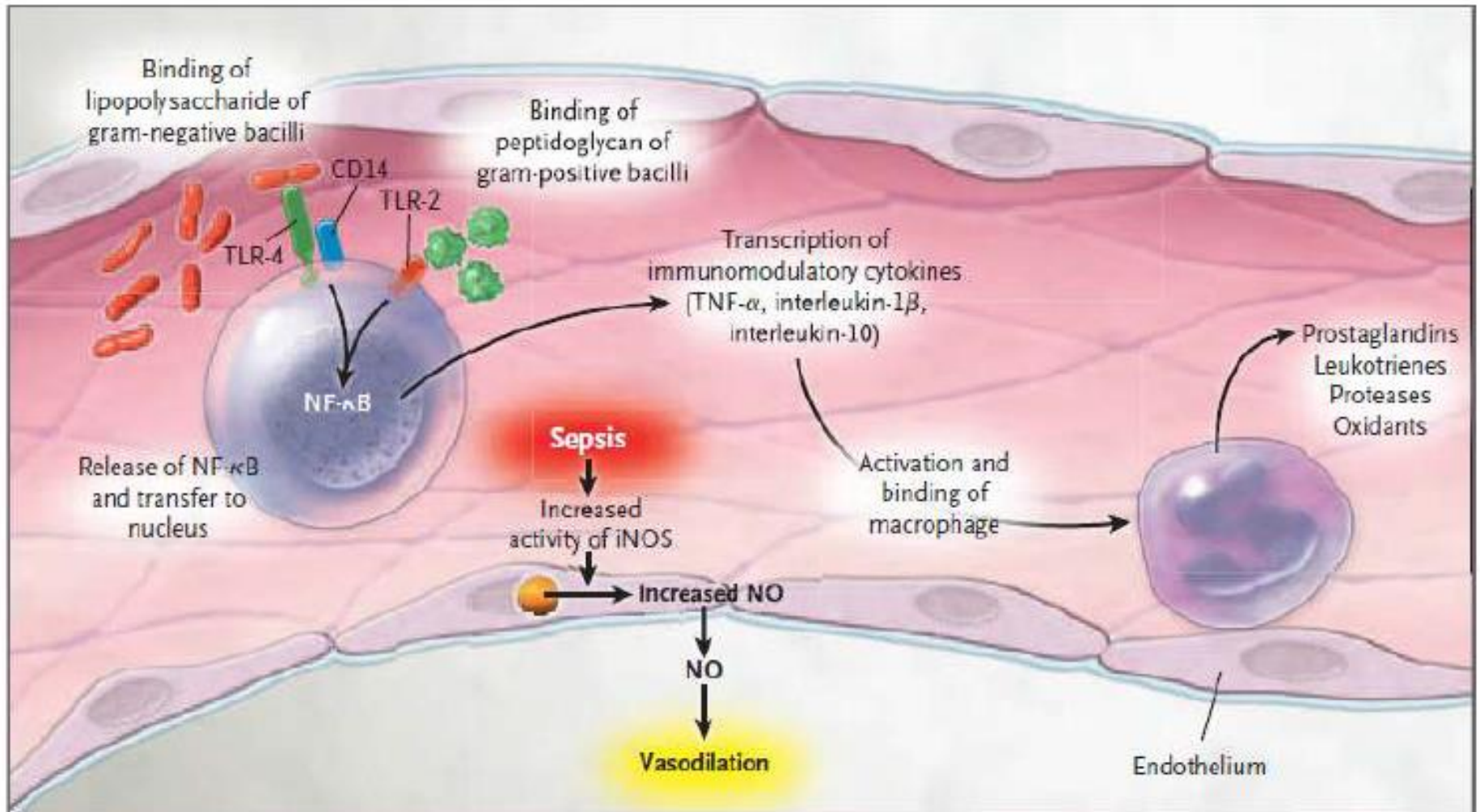








# Inflammatory response to sepsis





# CLINICAL FINDINGS

- The **major clinical findings in septicemia** are:
  - Fever,
  - Cardiovascular dysfunction (e.g. hypotension because of vasodilatation)
  - Shock,
  - Submucosal and subepidermal hemorrhages that are usually petechial and occasionally ecchymotic
    - ✓ The hemorrhages are best seen under the conjunctiva, mucosa of mouth and vulva
  - **Tachycardia** (rapid heartbeat), **tachypnea** (rapid breathing), and **shock-induced organ dysfunction** with cardiovascular hypotension, myocardial asthenia, and respiratory distress may occur in severe cases if the pathogen initiates the release of the host mediators, causing **SIRS**
- Specific signs may occur as the result of localization of the infection in joints, heart valves, meninges, eyes, or other organs

## Clinical findings of Neonatal Septicemia:

- Neonatal septicemia is common in all farm animal species from a few hours up to several days of age,
- The following features are common in Neonatal Septicemia:
  - Recumbency
  - Depression
  - Absence or marked depression of the suck reflex
  - Dehydration
  - Fever
  - Diarrhea
  - Injected or congested mucous membranes
  - Weakness
  - Rapid death
- Colostrum-deprived foals are commonly very ill and become comatose and die within several hours

- **Localized infections** in the **joints** and **lungs** are frequent in foals that survive for several days
- **Septic polyarthritis** is common and is characterized by **heat, pain, synovial distension,** and **lameness**
- **Pneumonia** is often observed and is characterized by **dyspnea** and **abnormal lung sounds**



## Blood Culture:

- Isolation of the causative bacteria from the bloodstream should be attempted by culture

## Hemogram:

- The presence of leukopenia (↓ number of WBCs) or leukocytosis (↑ number of WBCs) is an aid in diagnosis
- The type and degree of leukocytic response may be of prognostic significance, particularly the presence of band neutrophils, metamyelocytes, or toxic neutrophils

## Immunoglobulin Status:

- Low concentrations of serum protein and immunoglobulins are associated with failure of transfer of colostral immunoglobulins in newborn farm animals with consequent septicemia caused, most commonly, by gram-negative bacteria

## Serology:

# NECROPSY FINDINGS

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

- (1) Clinical signs, necropsy findings and clinical laboratory findings
- (2) Isolation of the causative agent from the blood stream

# TREATMENT

- The same general recommendations for treatment of fever (i.e. the principles of treatment are similar to fever)
- (2) Intravenous treatment with antibacterial drugs or sera and antitoxins is so urgent as soon as possible
  - i.e. Treatment should focus on broad-spectrum antimicrobial agents and general supportive measures
- (3) Hygienic precautions and prophylactic measures to prevent spread of the disease
- For neonatal septicemia the provision of a source of immunoglobulins is necessary
  - E.g. Provision of a source of immunoglobulins by plasma or blood transfusion is thought to be advantageous





# TOXEMIA

- **Toxemia** means **circulation of toxins** in the **blood stream**, the **toxins** are produced by **bacteria** or **parasites** or **body cells**
- Toxemia is a **clinical systemic state** caused by a **widespread activation** of **host defense mechanisms** to the **presence** of toxins produced by **bacteria** or **injury to tissue**
- **Toxemia does not include** the diseases caused by **toxic substances** produced by **plants** or **insects** or **ingested** organic or inorganic poisons

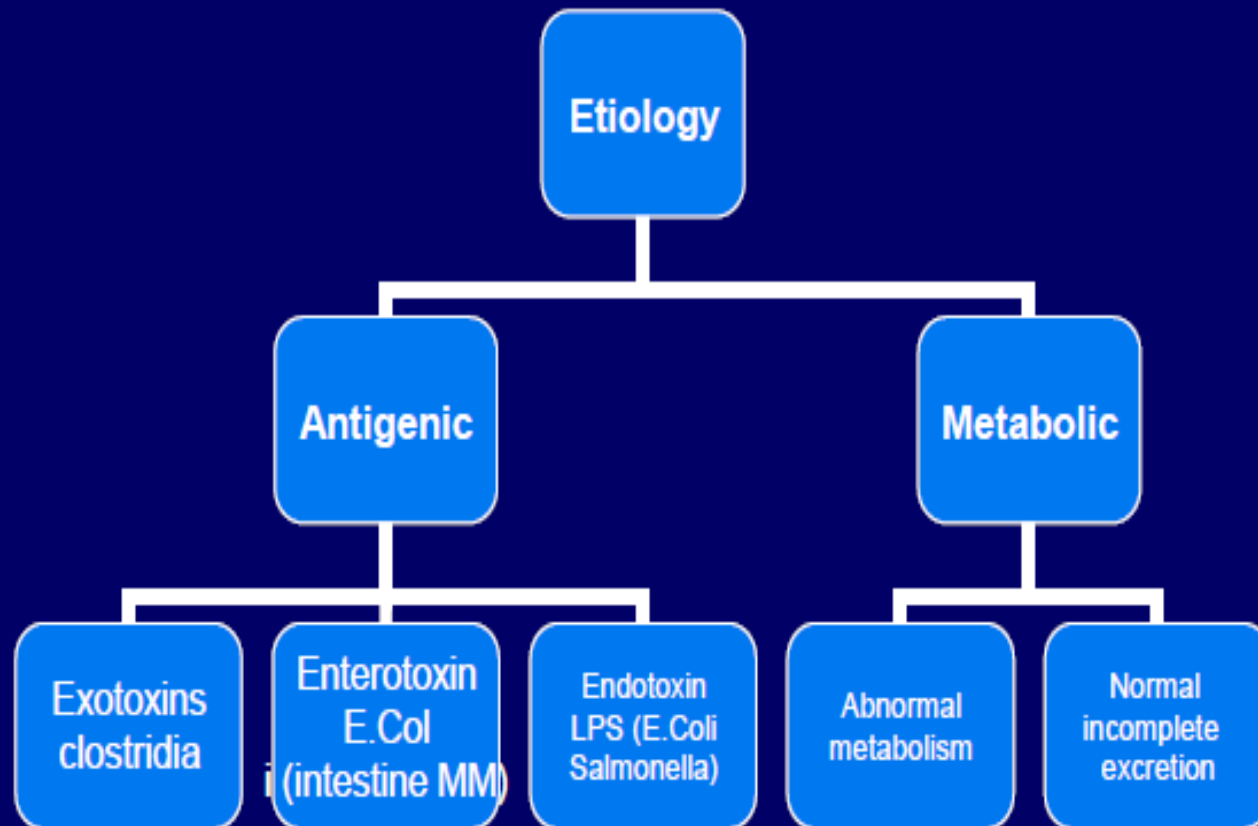
- The most common form of **toxemia** in animals (**large animals**) is **endotoxemia**, caused by the presence of **lipopolysaccharide** that form the **structural components of the cell-wall** of **gram-negative bacteria** in the blood and characterized clinically by **abnormalities of many body systems**
- The **endotoxins** are **liberated** (released) when the **cell** (**bacteria/microorganism**) die (i.e. undergoes cell's lysis)
- Endotoxins are **pyrogens**, which are **fever causing agents**
- **Gram-negative bacteria** such as **E. coli**, **Salmonella spp.**, and **Pasteurella spp.** cause many diseases of ruminants in which **endotoxemia is common**

- The **main abnormalities** of **endotoxemia** include the following:

- **Alteration of cardiopulmonary function** i.e. Marked alterations in cardiopulmonary function
- **Neutropenia, lymphocytopenia** and thrombocytopenia that may lead to coagulopathies
- **Decreased organ blood flow and metabolism**
- **Increased vascular permeability**
- **Decreased GIT motility**
- **Decreased perfusion of peripheral tissue** leading to shock
- **A high case fatality**

# ETIOLOGY

- Toxins can be classified as **antigenic** or **metabolic toxins**:-



- **Toxins** can be classified as **antigenic** or **metabolic** toxins:-

### **(A). Antigenic Toxins:-**

- Produced by **bacteria** and to less extent by **helminthes parasites**
- Both groups of pathogens act as **antigens** and **stimulate** the development of **antibodies**
- **Antigenic toxins** are divided (categorized) **into two**:- **Exotoxins & Endotoxins**

### **(B). Metabolic Toxins:-**

- These may be **accumulated** as a result of **incomplete elimination of toxic materials normally produced by body metabolism**, or by **abnormal metabolism**
- When the **normal mechanisms of detoxification** are **disrupted**, mainly in **hepatic dysfunction**, the toxins accumulate beyond the critical point and **resulted in a state of toxemia**, e.g. **ketone bodies** and **histamine**

# Exotoxins:-

- These are protein substances produced by bacteria that diffuse into the surrounding medium
- i.e. They are heat labile protein secreted by some species of bacteria
- Exotoxins diffuse into the surrounding medium and cause damage to the host by disrupting the normal functions of the cell or by directly destroying the cells
- Exotoxins are extremely dangerous to the extent that they can causes sever damage to the host
- There are many types of exotoxins but the most common ones are:
  - Botulinum Toxin,
  - Enterotoxin,
  - Tetanospasmin

- The important bacterial exotoxins are those produced by *Clostridium* spp. (botulism, tetanus, blackleg), for which commercial antitoxins are available
- Exotoxins may be ingested preformed, as in botulism, or produced in large quantities by heavy growth in the intestines, such as in enterotoxemia, or from growth in tissue (muscles), as in blackleg and black disease
- Exotoxins are released without requiring the cells to undergo lysis whereas for endotoxins to be released, the cell needs to undergo lysis
- Exotoxins are usually produced by gram positive bacteria while endotoxins are mostly produced by Gram-negative bacteria
- Exotoxins can be extremely lethal even in a small amount but endotoxins can only be lethal when present in large quantities



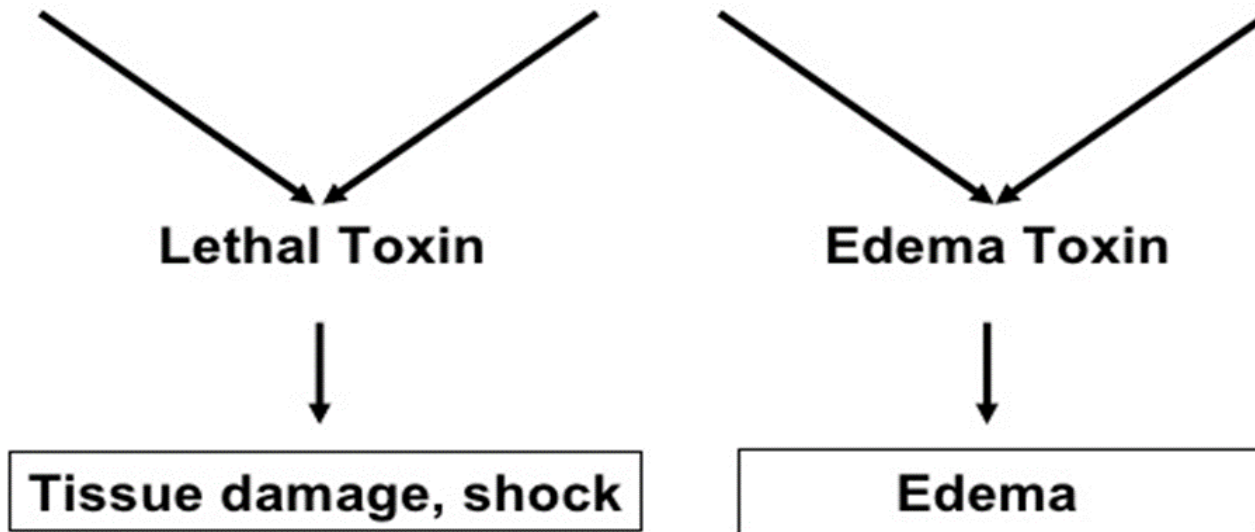
# Enterotoxins

- These are **exotoxins** that exert their effect **principally on the mucosa of the intestine**, causing disturbances of fluid and electrolyte balance.
- The **most typical example** is the enterotoxin released by **enterotoxigenic *E. coli***, which causes a hypersecretory diarrhea in neonatal farm animals.



# Anthrax Toxins

**Lethal Factor      Protective Antigen      Edema Factor**



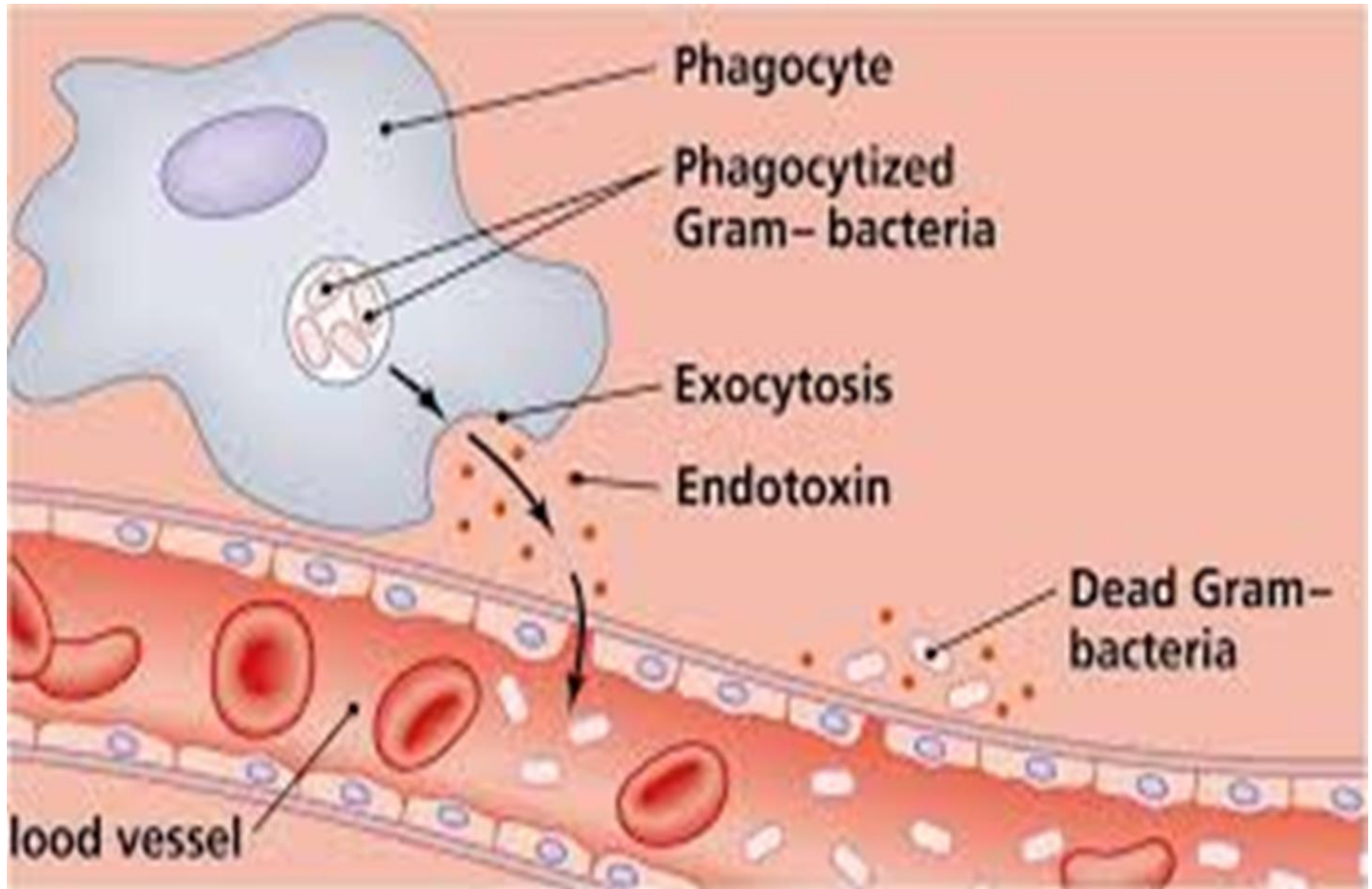


# Endotoxins

They are lipopolysaccharides (LPS) that form structural components of the wall of gram-negative bacteria

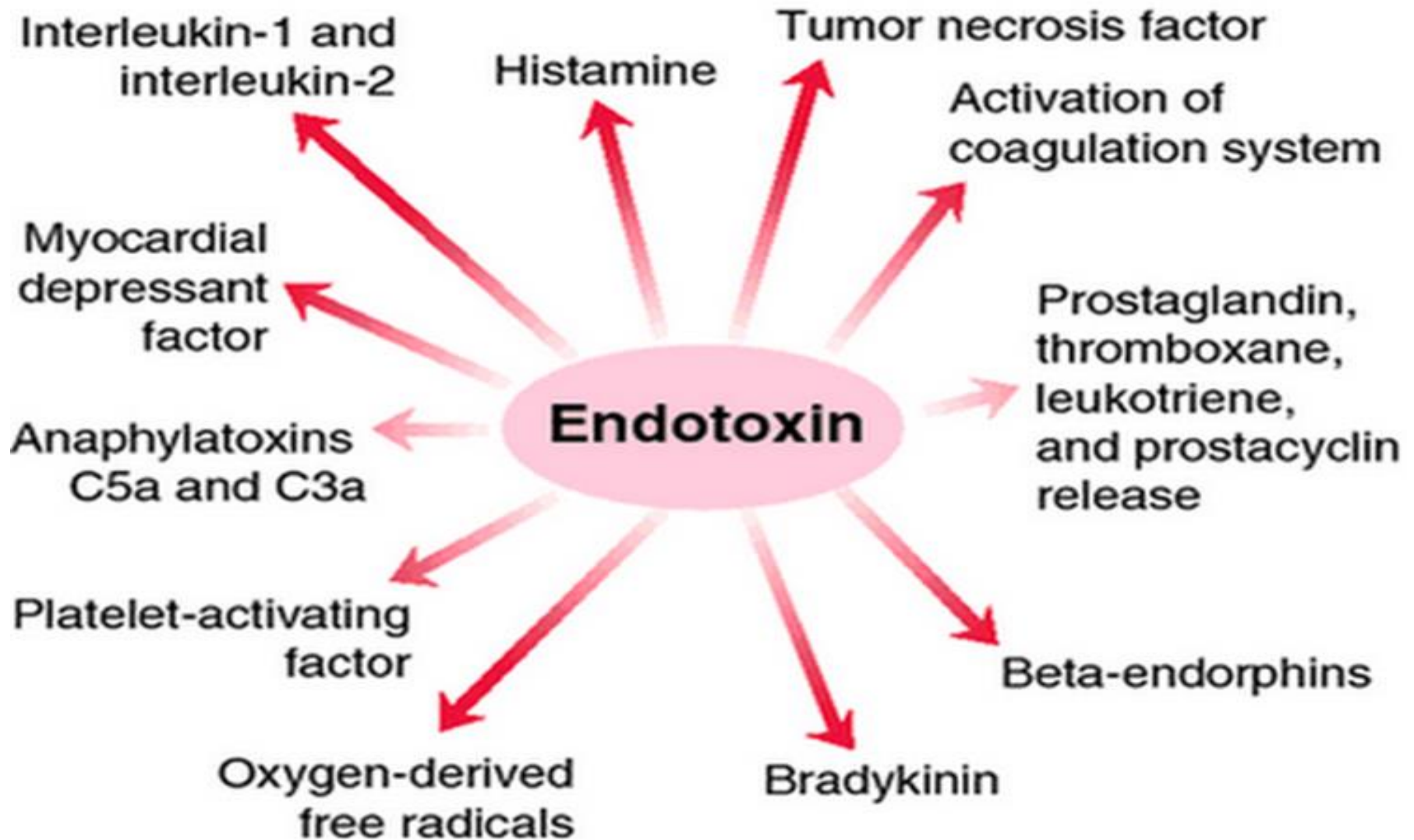
- ❑ The endotoxins of several species of Gram-negative bacteria are a major cause of morbidity and mortality in farm animals. They are heat stable
  - ❑ The endotoxins are lipo-polysaccharides found in the outer wall of the bacteria.
  - ❑ Endotoxins are released into the immediate surroundings when the bacteria undergo rapid proliferation with production of unused sections of bacterial cell wall or, most commonly, when the bacterial cell wall breaks.
  - ❑ Endotoxin gains access to the blood when there is a severe localized infection, such as a coliform mastitis in dairy cattle, or a disseminated infection, such as coliform septicemia in newborn calves.
- 
- ❑ The **best known endotoxins** are those of **E. coli**, which have been used extensively as **models for experimental endotoxemia**, and *Salmonella* spp.
  - ❑ Other examples: *Pasteurella* spp. etc

## e.g., Salmonella Endotoxin



## Endotoxins and their effect to the host:-

- Endotoxins have a non-specific effect on the body

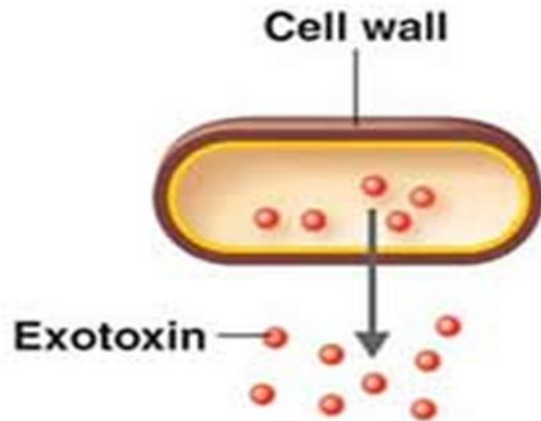


- Endotoxins can only be lethal when present in large quantities

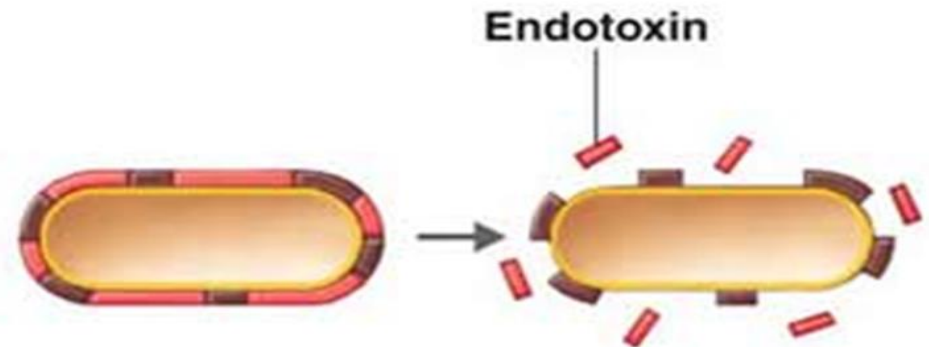
# Differences between Endotoxins and Exotoxins

- Endotoxins and exotoxins; although similarly elicit immune response do have a lot of differences

## Differences Between Exotoxins and Endotoxins



**(a) Exotoxins** are proteins produced inside pathogenic bacteria, most commonly gram-positive bacteria, as part of their growth and metabolism. The exotoxins are then secreted or released into the surrounding medium following lysis.



**(b) Endotoxins** are the lipid portions of lipopolysaccharides (LPSs) that are part of the outer membrane of the cell wall of gram-negative bacteria (lipid A; see Figure 4.13c). The endotoxins are liberated when the bacteria die and the cell wall breaks apart.

Characteristics	Exotoxins	Endotoxins
Source	Living gram positive and gram negative bacteria	Lysed gram negative bacteria
Location	Released from the cell	Part of cell
Chemical Composition	Protein	Lipopolysaccharide
Heat Sensitivity	Liabile (60-80C)	Stable (250C)
Immune Reactions	Strong	Weak
Conversion to Toxoids	Possible	Not possible
Vaccines	Readily available as toxoids	no vaccines available
Enzyme Activity	It has no enzymatic activity.	It has mostly enzymatic activity.
Molecular Weight	Its molecular weight is 10KDa.	Its molecular weight is 50-1000KDa.
Denaturing	On boiling it get denatured.	On boiling it cannot be denatured.
Specificity	Specific to particular bacterial strain	Non specific
Antigenicity	High	Poor
Examples	<i>Staphylococcus aureus</i> , <i>Bacillus cereus</i> , <i>Streptococcus pyogenes</i> , <i>Bacillus anthracis</i> .	<i>E.coli</i> , <i>Salmonella typhi</i> , <i>Shigella</i> , <i>Vibrio cholera</i> .

Toxicity

Immunogenicity

It is highly toxic  
They are immunogenic

It is moderately toxic  
weak immunogenicity b/c not capable of producing antitoxins



# Metabolic toxin

- ❑ These may accumulate as a result of incomplete elimination of toxic materials normally produced by body metabolism, or by abnormal metabolism
  - ❑ Normally, toxic products produced in the alimentary tract or tissues are excreted in the **urine and feces** or detoxified in the **plasma and liver**.
  - ❑ When these normal mechanisms are disrupted, particularly in hepatic dysfunction, the toxins may accumulate beyond a critical point and the syndrome of toxemia appears.
  - ❑ Examples: ketonemia (ketosis) and lactic acidemia
- ❑ The production of toxins by abnormal metabolism includes the production of histamine and histamine-like substances in damaged tissues
  - ❑ Ketonemia caused by a disproportionate fat metabolism, and lactic acidemia caused by acute ruminal acidosis (grain overload), are two common examples of toxemia caused by abnormal metabolism

**This will be a group presentation**

**Will be presented by Group\_\_\_\_\_?? On \_\_\_\_\_??(date)**

**I will form the group**





## Clinical signs of Acute toxemia

The **clinical findings** of **acute toxemia** in most nonspecific toxemias are **similar**:

- ❑ Depression, anorexia and muscular weakness are common in acute endotoxemia.
- ❑ Calves do not suck voluntarily and may not have a suck reflex.
- ❑ Scant feces are common but a low-volume diarrhea may also occur.
- ❑ The heart rate is increased and initially the intensity of the heart sounds is increased, but later as the toxemia worsens the intensity may decrease.
- ❑ The pulse is weak and rapid but regular.
- ❑ A fever is common in the early stages of endotoxemia but later the temperature may be normal or subnormal.
- ❑ Terminally, there is muscular weakness to the point of collapse and death occurs in a coma or with convulsions.





## Clinical signs of endotoxemia

- Endotoxemia is most commonly associated with bacteremia or septicemia due to infection with Gram-negative organisms, especially *E. coli*.
- The clinical findings of severe endotoxemia include:
  - Depression
  - Hyperthermia followed by hypothermia
  - Tachycardia followed by decreased cardiac output
  - Decreased systemic blood pressure
  - Cool skin and extremities
  - Diarrhea
  - Congested mucosae with an increased capillary refill time
  - Muscular weakness, leading to recumbency.



## Chronic toxemia

- Lethargy, separation from the group, inappetence, failure to grow or produce and emaciation are characteristic signs of chronic toxemia.

## Hematology:-

- Changes in total and differential leukocyte numbers occur in endotoxemia,
  - Leukocytosis and neutrophilia occur with mild toxemia
  - Leukopenia, neutropenia, and lymphopenia in sever toxemia

## Serum Biochemistry:-

- A low plasma glucose concentration, high serum urea concentration, and a low serum albumin and low total protein concentration are usually present

## Toxin Assay:

- Endotoxins are detected by the limulus lysate assay test

- Exotoxins are detected using the following methods
  - ELISA-based method
  - Precipitation
  - Neutralization

## Histopathology:-

- **Microscopically**, there is **degeneration** of the **parenchyma** of the **liver** and the **glomeruli** and **tubules** of the **kidney** and the **myocardium**
- There may also be **degeneration** or **necrosis** in the adrenal glands

# TREATMENT

## (1). Removal of foci of infection

- Removal of the **origin of the toxin if possible** and **provision of specific antitoxin** with supportive treatment

## (2). Administration of antimicrobial agents with a gram-negative spectrum

### Antimicrobial agent with Gm-ve spectrum

- Streptomycine
- Gentamycin
- Polymyxin B

## (3). Aggressive fluid and electrolyte therapy to combat the relative hypovolemia, systemic hypotension, hypoglycemia, and electrolyte and acid-base disturbances

### Aggressive fluid therapy IV to restore cardiac output and increase urine output.

- Lactated ringer solution
- Isotonic saline solution

## (4). Anti-inflammatory and antipyretic drugs

- NSAIDs: Flunixin meglumine at dose of 0.25mg/kg
- Corticosteroids Doses of 1 mg/kg BW

e.g., Prednisolone, methylprednisolone and dexamethasone

- These four treatments are routinely applied and are called goal-directed therapy



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# Questions

**What is the difference between antigenic and metabolic toxemia?**

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## GENERAL SYSTEMIC STATES

These are states which common to so many diseases and they are considered the base which contribute to the effects of many diseases

### Individual Student Assignment (10%):

Date of Submission: May 22, 2020  
Via Email: [girmasis@gmail.com](mailto:girmasis@gmail.com)

- Prepare PowerPoint slides (maximum of 15 slides) on the topics given below:
- It will be evaluated on the content & preciseness of the PowerPoint on your given topic
- Identify your topic as described below on the basis of your name alphabetical order

# 6 Students, (from A to B)

Pathogenesis of Toxemia

# 9 Students, (from D to G)

Pain & Pathophysiology of pain

# 10 Students, ( from H to M)

Disturbances of body fluids & Electrolytes

# 10 Students (from N to Z)

Disturbances of Acid-Base balance



# **DISEASES OF THE RESPIRATORY SYSTEM**

# function

## ❖ Gaseous exchanges:

## ❖ Acid base balance:

- $\uparrow \text{CO}_2 \rightarrow \text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{HCO}_3^- \leftrightarrow \text{H}^+ + \text{HCO}_3^-$ 
  - elimination of  $\text{CO}_2$  via alveolar ventilation allows very effective removal of  $\text{H}^+$  by shifting the equilibrium to the left
  - this allows  $\text{H}_2\text{CO}_3/\text{HCO}_3^-$  to act as a very effective buffer system
- Changes in the rate of ventilation allow very rapid adjustments in plasma pH, and ventilation is responsive to arterial pH
- Alveolar ventilation removes approximately 24,000 mEq of carbonic acid daily

## ❖ Acting as a blood reservoir

- ❖ Filtering and probably destroying emboli, metabolizing some bioactive substances like serotonin, prostaglandins, corticosteroids and leukotrienes
- ❖ The upper airways also provide for the sense of smell
- ❖ Play a role in temperature regulation in panting animals

# How respiratory system protects The entry of pathogenic particles deposited

Large airborne particles

↓ **deposited**

On the mucous lining of the nasal passage, larynx, Trachea & bronchi

↓ **After that Carried**

by mucociliary to pharynx

↓

swallowed or expectorated

Small particles

↓ **deposited**

alveoli

↓ **Phagocytized**

macrophages

# PRINCIPALES OF RESPIRATORY INSUFFICIENCY

- Anoxia
- Hypercapnea
- Respiratory failure

# ANOXIA

- ❖ It means failure of the tissues to receive an adequate supply of oxygen

## ❑ Types of anoxia:

### ❑ Anoxic anoxia:

- Occurs when there is defective oxygenation of the blood in the pulmonary circulation.
- It is usually caused by primary disease of the respiratory tract.

### ❑ Anemic Anoxia:

- Occurs when there is a deficiency of hemoglobin per unit volume of the blood.
- It is usually caused by anemia due to any cause."e.g."poisoning by nitrites or carbon monoxide .

### ❑ Stagnant Anoxia:

- Occurs when the rate of blood flow through the capillaries is reduced.
- It usually occurs in cases of congestive heart failure, peripheral circulatory failure
- & venous obstruction.

### ❑ Histotoxic Anoxia:

- Occurs when the blood is fully oxygenated, but because of the failure of the "tissue oxidation system," the tissues can not take up oxygen .
- It usually occurs as a result of cyanide poisoning.

# Complications of anoxia

- Increase in depth of respiratory movement “hyperpnea”.
- Stimulation of splenic contraction.
- Erythropoiesis in the bone marrow.
- Increased heart rate.
- Signs of dysfunction of various organs appears, cerebral anoxia, myocardial dysfunction, renal and hepatic dysfunctions as well as reduction in motility and secretory activity of alimentary tract

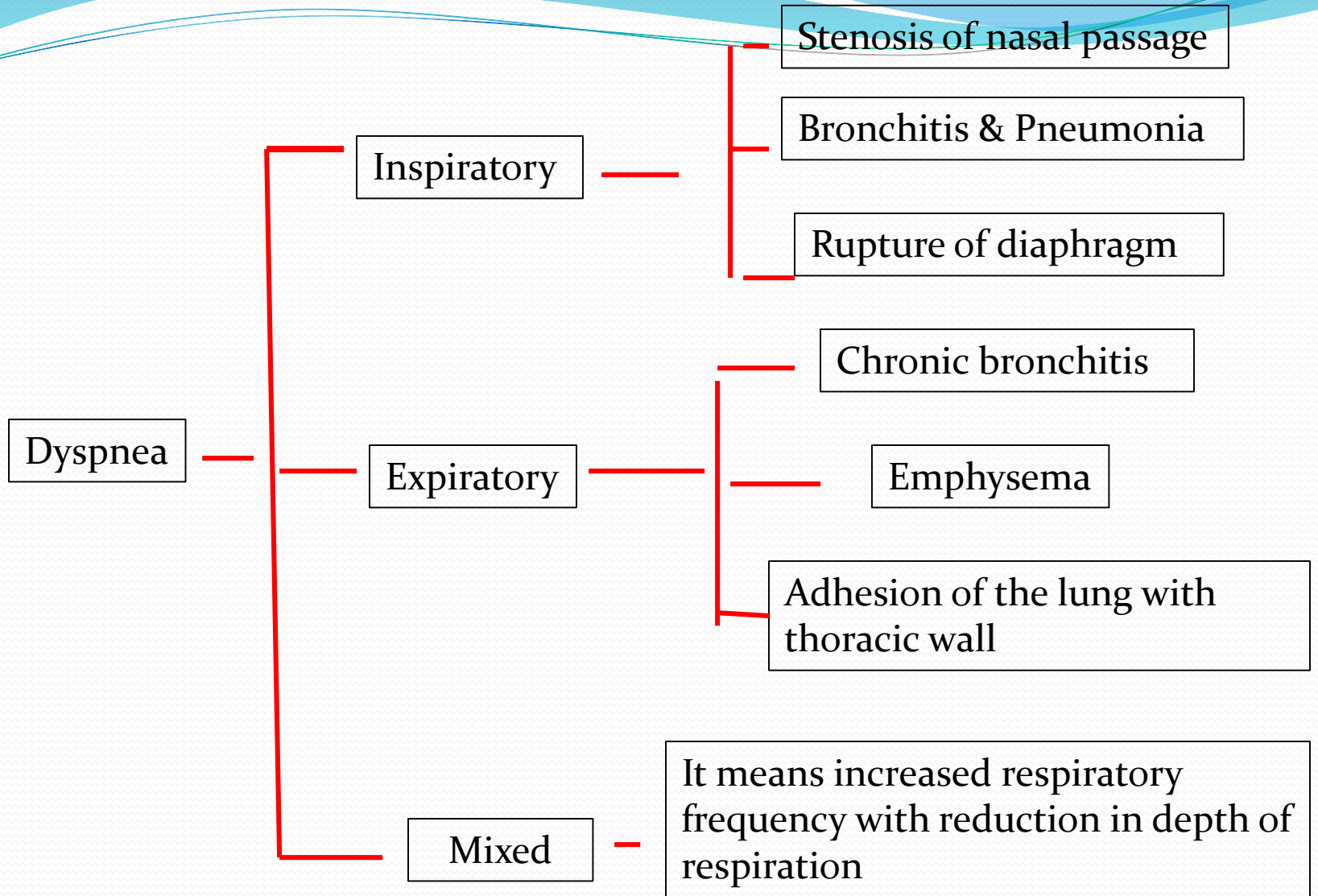
# RESPIRATORY FAILURE

- Respiratory failure is the terminal stage of respiratory insufficiency, in which the activity of respiratory center is diminished to the point where the movement of respiratory muscles is completely stopped.



# Types

- ❖ Tachypenic
- ❖ Dyspneic,
- ❖ Asphyxial
- ❖ Paralytic



# Asphexial respiratory failure

## □ Causes:

- Pneumonia .
- Pulmonary odema.
- Upper respiratory tract obstruction.

## □ Clinical signs:

- Hypercapnia → stimulate respiratory center → stimulation respiration
- Anoxia.
- Gaspig.
- Apnea → death

# Paralytic respiratory failure

## □ Causes:

- Poisoning with respiratory center depressants.
- Nervous shock.
- Acute heart failure.
- Hemorrhage.

## □ Clinical signs:

- Variable degree of dyspnea & gasping.
- Paralysis of the respiratory center → shallow respiration
- complete stop of respiration .

# Tachypneic respiratory failure

## □ Causes:

- Increased pulmonary ventilation "hypoxia " but no carbon dioxide retention
- "acapnio ".

## □ Clinical signs:

- Rapid & shallow respiration

# Treatment of respiratory failure

- ❖ In paralytic type → stimulants of respiratory center are given .
- ❖ In asphyxyial type → oxygen is provided .
- ❖ In tachypneic type → oxygen & CO<sub>2</sub> are provided

# Principal manifestations of respiratory insufficiency

- Abnormalities in the rate, depth, or ease of breathing
- Lethargy or exercise intolerance
- Abnormal posture
- Abnormal lung sounds
- Abnormal respiratory noises
- Coughing
- Cyanosis : not seen in case of anaemic anoxia
- Nasal discharge
- Epistaxis and hemoptysis

# Abnormalities in the rate, depth, or ease of breathing

- Polypnea - faster rate of breathing
- Tachypnea -an increased rate of breathing,
- Hyperpnea - an abnormal increase in the rate and depth of breathing
- Dyspnea - labored or difficult breathing



# Abnormal posture

- Stand with the head and neck held low and extended.
- Animals, except horses have open-mouthed breathing
- Severely affected animals, and those with pleuritic pain (horses or cattle with pleuritis) or severe respiratory distress, usually stand with elbows abducted

# Normal respiratory sounds on auscultation

- ❖ The normal respiratory sound heard over the respiratory area consists of vesicular sound & bronchial sound:-
- ❖ vesicular respiratory sound "vesicular murmur
- ❖ **Crepitant rales:** the sound produced by rubbing a tuft of hair held between fingers close to the ear.

It occurs in cases of:

- Bronchiolitis
- Early stages of pneumonia
- Pulmonary odema

# Frictional sound

❑ It resembles the sound produced by rubbing two pieces of leather against each other

or

❑ by pressing the finger against the ear & stretching the finger nail of other hand.

❑ It occur in cases of

- Preexudative stage of pleurisy.
- Pericarditis

## ❑ **Emphysematous sound (Harch sound), or (Sharp sound)**

- Resembles the sound produced by collection of a piece of paper between fingers & hand.

## ❑ **Girgling sound :**

- Resembles sound produced by gases & air bubbles, as in cases of diaphragmatic hernia (in the chest) & bloat (in the rumen)

# DISEASES OF THE UPPER RESPIRATORY TRACT

## □ RHINITIS "CORYZA OR NASAL CATTARAH"

### ➤ Definition:

- Rhinitis means inflammation of the mucous membrane of the nose

### ➤ Etiology:

- **Primary causes:**

- ❖ Inhalation of irritant vapour as ammonia or chlorides.
- ❖ presence of foreign bodies in the nose as dust particles.

- **Secondary causes:**

- ❖ Sudden exposure from hot to cold,
- ❖ Glanders.
- ❖ Strangles.
- ❖ Meliodosis of sheep.
- ❖ Necrotic rhinitis of sheep.

## Viral causes:

- -Malignant catarrhal fever of cattle.
- -Mucosal disease.
- -Render pest.
- -Blue tongue disease.
- -Infectious bovine rhinotracheitis.(I.B.R.).
- -Equine viral rhinopneumonietis.
- -Swine influenza.

## Parasitic causes:

- -*Ostrus ovis* of sheep.
- -Blood flukes as *S. Nasalis* of cattle.

Fungal Aspirigillosis of dogs

Allergic condition

# Clinical signs

- Redness and swelling of mucous membrane of the nose.
- Bilateral nasal discharge which usually begins watery in character then
- mucopurulent & purulent.
- Swelling of the submaxillary L.N.
- Difficulty of swallowing "dysphagia".
- Sometimes the discharge rises up and blocks the nose leading to a condition of
- "snorting".
- When there is irritation the animal rubs its nose against any hard objects.
- There may be lacrimation and bleeding.

# Differentiation between acute & chronic rhinitis

<u>Acute rhinitis</u>	<u>Chronic rhinitis</u>
1-Watery nasal discharge	1-Mucoid nasal discharge
2-Mucous memb. is swollen& redden	2-Mucous m. is swollen &bluish
3-Swelling of the submaxillary L.N	3-Swelling of submaxillaryL.N.followed by dysphagia
4-Stenosis of the nostril	4-The animal rubs its nose against objects



# Treatment

- Put the animal in a well ventilated space away from draughts.
- Complete rest of the animal & give only laxative, easily digestive food.
- Irrigate the nostrils with Sodium bicarbonate (1% solution).
- Apply medicated steam inhalation, but contraindicated to be used in milk and meat producing animals.
- Put Vaseline on the upper lip of the animal.

# EPISTAXIS

## □ Definition

- It means **bleeding from nostrils or from nasal sinuses**

## □ Etiology:

### • Primary cause:

- Bad use of the tracheal tube
- Traumatic injury of the nose, head, frontal nasal bore as well as sinuses
- May be congenital
- Over exhaustion of race horse
- May occur without any apparent cause

### • Secondary cause:

- Ulceration of the nose & septum nasi as in case of glanders in equine
- Puncture of parasite in infection diseases as anthrax & hemorrhagic septicemia

## • **Other causes:**

- Diseases of mucosa of the upper respiratory tract, nasal cavity, nasopharynx or guttural pouch
- Erosion of the mucosa occurs in glanders
- granulomatous & neoplastic lesions in the nasal cavities.
- entry of foreign body
- by accidental injury to the facial bones.
- purpura hemorrhagica
- sweet clover poisoning
- bracken fern poisoning
- congestive heart failure.

# Clinical findings

- There is bleeding from nostrils either unilateral or bilateral.
- Blood is bright red in color,
- It may be scanty & stop by itself or profuse & the bleeding is profuse.
- Pulse is rapid & weak
- pale mucous membrane
- Anemia
- loss of condition & death.

# Treatment

- Complete rest of the animal & keep it quite
- Apply cold compresses on the head particularly on the frontal nasal bone & nose.
- Apply astringent solution on the affected nostrils as:
  - Alum solution 2% or tannic acid 2%.
  - Piece of gauze soaked with adrenaline 2%.
- In case of bilateral bleeding apply tracheotomy & plug the two nostrils with a piece of gauze soaked in adrenaline
- Inject vitamin K to accelerate coagulation “  $\frac{1}{2}$  -3 ml /50kg .B.W.
- Inject calcium chloride 10% 100 cc s/c.
- Treat the cause if it is secondary epistaxis.
- Fluid therapy “ Glucose sol. 20% I/V about one litre” or normal physiological saline 500 ml I/V.

# Pneumonia

- Pneumonia is the inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleurisy.

# Causes

- **Cattle:**

- Pneumonic pasteurellosis (Shipping fever)
- *Pasteurella hemolytica*,
- *P. multocida*
- parainfluenza -3 virus (PI3).
- Enzootic pneumonia of calves
- Bovine Syncytial Virus (BSV),
- reoviruses,
- Bovine herpes virus 1 (BSV virus) plus *Chlamydia* spp.
- *Mycoplasma* spp.
- *Corynebacterium pyogenes*,
- *Strept* spp.
- *Actinobacillus actinoides*
- Contagious bovine pleuropneumonia caused by *Mycoplasma mycoides*.
- Lungworm pneumonia (*Dictyocaulus viviparus*).

## • **Horses:**

- Strep. Sp.
- E. Coli,
- Actinobacillus equi.
- adenoviruses or pneumocystis carini.
- Corynebacterium, Rhodococcus equi and equine herpesvirus.
- Dictycaulus arnifeldi and Parascaris equorum
- As a sequel to Strangles.
- equine viral rhinopneumonitis in adult animals.
- Glanders and epizootic lymphangitis (Histomonas farcinicus)



## • **Sheep:**

- Pneumonic pasteurellosis (*Pasteurella* sp.)
- PI3 or Chlamedia spp.
- Strept. Zooepidemicus, *Salmonella abortus ovis*.
- *Mycoplasma* spp. (Severe pneumonia).
- *Mycoplasma* sp.
- *Corynebacterium pseudotuberculosis* (sporadic).
- Melioidosis (*Pseudomonas pseudomallei*).
- Lungworm pneumonia (*Dictycolus filarial*).
- Progressive interstitial pneumonia

- **All species:**

- Toxoplasmosis (sporadic cases).
- Systemic mycosis lesion are focal only.
- Aspiration pneumonia.
- Secondary pneumonia caused by Strept. Spp.  
Corynbact.spp. dermatophilus sp.
- Interstitial pneumonia, pulmonary consolidation and fibrosis by toxins in plants

Causative agents (Dust particles, Bacteria, Virus etc.)

produce

Respiratory diseases

Impaired

Pulmonary defense Mechanism

1. Aerodynamic filtration
2. Mucociliary clearance
3. Cough reflex
4. Change the quality of mucous
5. Alveolar macrophages

Extension to the Visceral surface of the pleura

Deposited

Bronchiols

produce

Primary Bronchiolitis

Spread by bronchiols & lymphatics

Surrounding pulmonary parenchyma

produce

Septic foci

Form

Abscesses

Rupture

Pneumonia

Interfere

Gaseous exchange

As a result develop

Anoxia & Hypercapnia

Retention of CO<sub>2</sub>

Acidosis

produce

Polypnea, Dyspnea, tachypnea

Develop

Cynosis if large areas of the lung are affected

1. Pleuritis
2. Pleuropneumonia
3. Pleural effusion
4. Thoracic pain

Extension to the Visceral surface of the pleura

Accumulation of inflammatory exudates

Abnormal lung sound

# Clinical findings

- Rapid, shallow respiration
- dyspnea occurring in the later stages.
- Polypnoea.
- Cough
- Bronchopneumonia - moist painful cough,
- interstitial pneumonia -, dry, backing cough,
- nasal discharge may or may not be present s accompanying inflammation of the upper respiratory tract.
- Auscultation of the thorax before and after coughing may detect exudate in the air passages.
- the vascular murmur is increased.
- Moist rales develop in broncho-pneumonia as bronchiolar exudation increases
- in uncomplicated interstitial pneumonia, clear, harsh bronchial tones are audible.
- Fever
- anorexia,
- depression,
- an increase in pulse rate.

# Treatment

- Isolation of affected animals
- Affected animals should be housed in warm, well ventilated, draft-free accommodation, provided with ample, fresh water, and light nourishing food
- Antimicrobial agents e.g. Sulphadimidine(33.5%) solution @ 15-30ml/50kg B.W. I/V or S/Cly, oxtetracycline @ 10-20 mg/kg BW I/m or I/V for 7 days, Penicillin @ 20-40 lakh IU for LA for 5-7 days
- In parasitic Pneumonia- Levamisole, Fenbendazole
- In fungal- Grisovin, Amphotericin-@ 0.5 mg/Kg BW
- Corticosteroids for their anti-inflammatory e.g. Dexamethason
- O<sub>2</sub> supply
- Expectorants, e.g. Zeet Expectorant, Pot iodide
- VitC & Calcium

# Aspiration pneumonia (Drenching pneumonia, Foreign body Pneumonia, Regulation pneumonia, Inhalation pneumonia, Mechanical pneumonia)

- **Etiology:**

- Careless drenching.
- Careless passage of stomach tube during relieve of other diseases.
- Vomiting in ruminants and horses that followed by aspiration.
- Paralysis of the pharynx & larynx

# Symptoms:

- Death occurs quickly
- Signs of pneumonia are present
- Coughing
- Rales,
- foamy nasal discharge
- Protrusion of tongue
- fetid odors breath,
- Congested mucosa
- Rapid pulsation & febrile condition.
- The condition is usually followed by gangrenous pneumonia

# Treatment

- If the lesion is advanced, treatment is non effective,
- using broad spectrum antibiotic & sulfonamides.
- Care of the animals and supportive remedies & sedatives



# Lung worm infestation (Verminous broncho pneumonia)

- Lung worm infestation is an enzootic or epizootic affection manifested by bronchitis & bronchopneumonia & caused by infestation of lung by metastrongylides

# Etiology

- *Dictycaulus filarial* of sheep, goat, camel & occasionally cattle.
- *Dictycaulus viviparous* of cattle.
- *Dictycaulus oviparous* of horse & donkey.
- *Metastrongylus elongatus* of pigs & occasionally in sheep.
- *Prostrongylus rufescens*: sheep, goat & rabbit.
- *Prostrongylus commintatus*: sheep and goat.
- *Mallerious coppillaris*: sheep & lambs.
- *Strongylus abstruses*: cat.
- *Haemoststrongylus vasorum*: dog.

# Clinical findings

- Cough.
- Nasal discharge.
- Respiration is labored.
- Severe rise of body temperature reaching to 42 when the lung is involved.
- Emaciation.
- Anaemia.
- Frequent diarrhea.
- Enlargement & swelling of submaxillary region, lips & eye lids

# Treatment

- Carbolic acid 1% I,M. injection of 5 ml for sheep & goat  
20 ml for calves & equines.
- Phenothiazine.
- Copper sulphate.
- Levamisole injection

# Pulmonary emphysema

- Emphysema means over distension of alveoli without any change in the pulmonary tissues & with or without escape of air into the interstitial tissue causing reduction in the air space & loosing the elasticity of the alveoli.

# Classification

- ❑ **Acute alveolar emphysema**:- mostly in cattle & is “temporary distension of the alveoli”.
- ❑ **Interstitial emphysema**:- in which rupture of one or more alveoli & the air escape to the interlobular & intralobular spaces “mostly in cattle & is fatal”.
- ❑ **\*Chronic alveolar emphysema** or “Heaves Disease” in horses, overdilatation of the alveoli till complete loss of elasticity

# Etiology-Acute alveolar emphysema

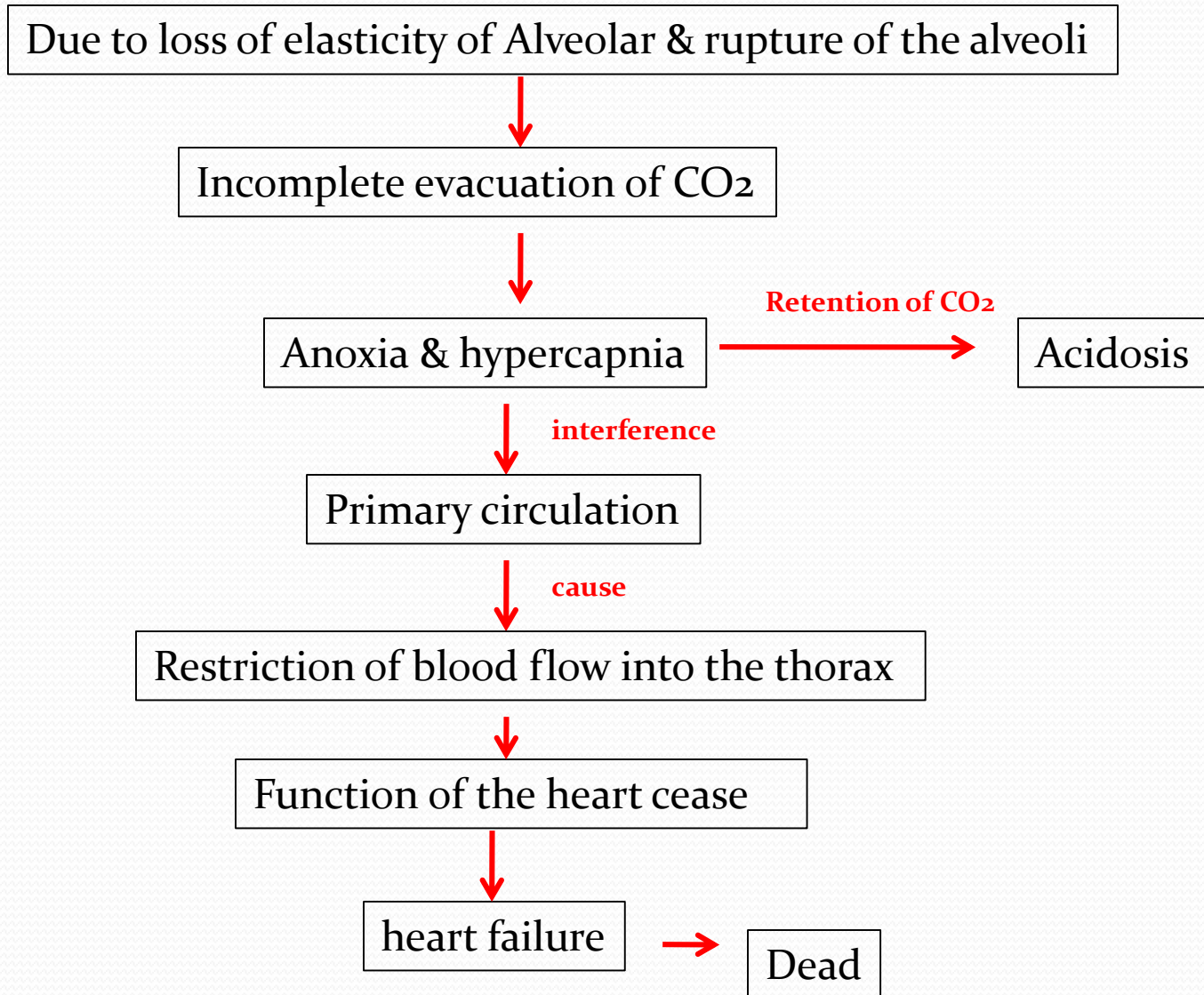
- atypical pneumonia
- parasitic pneumonia
- anaphylaxis.
- Persisting cough.
- Obstruction of the alveoli with food particles, foreign bodies, larvae of ascaris, dictyocoulus viviparous or exudates in case of bronchitis.
- Collapse of some parts of the lung “alveoli”.
- Traumatic reticuloperitonitis
- pulmonary abscess

# Interstitial emphysema

- Heavy infestation with lung worm.
- Damage of the lung tissue.
- In case of parturition over distention & lead to rupture of the alveoli



# Pathogenesis



# Clinical findings

- Marked restriction of the expansion of the chest wall
- Laboured breathing
- Prolonged expiration
- Dyspnea
- Increased respiration rate
- Dilation of nostril
- Extension of head & neck
- Anaemic
- Edema
- Death

# Treatment:

- Administration of Beladona 25-40 gm. daily for adult horses suffering from chronic alveolar emphysema.
- In interstitial emphysema ,Slaughter the cattle.
- Complete rest of the animal away from air droughts.
- Put the animal in a well ventilated space.
- Oxygen therapy may be recommended.
- Injection of Atropine , relieve of dyspnea specially if odema is present.
- Antihistaminics therapy in acute alveolar emphysema
- A course of antibiotic
- Steroid and Broncho dilators

- ❑ **Pneumothorax**”: It means entry of the air in the pleural cavity in a sufficient quantity to cause collapse of the lung.
- ❑ **Hydrothorax**: Means presence of non inflammatory fluid in the pleural sac.
- ❑ **Hemothorax**: Means presence of blood in the pleural cavity.

# Pleurisy

- It means an acute inflammation of the pleura causing pain during respiration movements & manifested clinically by shallow & rapid respiration and signs of pain.

# Etiology

- Traumatic perforation of the thoracic wall primary pleurisy.
- contagious bovine pleuropneumonia,
- *P. multocida* & T.B
- Equine pneumonia & Strangles of horse.
- Perforation of diaphragm by sharp foreign body.
- Primary peritonitis spread of infections infected pleurisy.
- Extension of infection from the lung.
- Complication of traumatic pericarditis.
- Rupture of esophagus due to severe obstruction.
- It is permanent lesion in contagious pleuropneumonia

# Pathogenesis

□ 1<sup>st</sup> stage:

❖ contact & movements between parietal & visceral pleura *cause*

- *pain*
- *respiratory* disturbance
- shallow & rapid respiration.

❖ 2nd stage

- production of serofibrinous inflammation exudates
- reducing the vital capacity of the lung
- interfering with gaseous exchange

❖ 3rd stage

- the fluid is absorbed & development of adhesion
- Restriction movement of the lung
- Interfere gaseous exchange

# Clinical findings:

- Shallow, rapid & painful respiration.
- Abdominal respiratory movements.
- There may be pneumonia accompanied by rales & increased vesicular sound.
- Dyspnea & toxemia may appear.
- Pleurisy is unilateral,
- Pain on percussion.
- Shallow & short painful cough.
- Death occurs due to combination of toxemia & anoxia..
- The animal stands with abducted elbow, to relieve pressure from lungs & pleura.
- Loss of appetite, dullness depression.



# Treatment:

- As pneumonia.
- Apply, broad spectrum antibiotics.
- Aspiration of fluids by paracentesis.
- To reduce pleural percussion of fluids give dexamethazone  
o .1 mg / k.g B.W.

# Digestive System: Ruminant Tympany (Bloat)

1



# Diseases of Digestive system

2

## □ **Ruminal Tympany (Bloat)**

- abnormal distension of the rumen and reticulum by excessive retention of the gases of fermentation
- **persistent foam** mixed with rumen contents or
- **free gas** separated

# ETIOLOGY

3

- 1. **Primary ruminal tympany (frothy bloat)**
  - ▣ Caused by the production of **a stable foam** in the rumen
  - ▣ essential feature: **coalescence of the small gas bubbles is inhibited**
  - ▣ **intraruminal pressure increases**

# A. Pasture bloat

4

- Leguminous or pasture bloat due to foaming qualities **soluble leaf proteins**
- **Frothiness** of the ruminal contents as vital factor
- **chloroplast particles** (readily colonized by rumen microflora)
- Gas bubbles trapped among the particles (prevent **coalescence of bubbles**)

# Pasture bloat

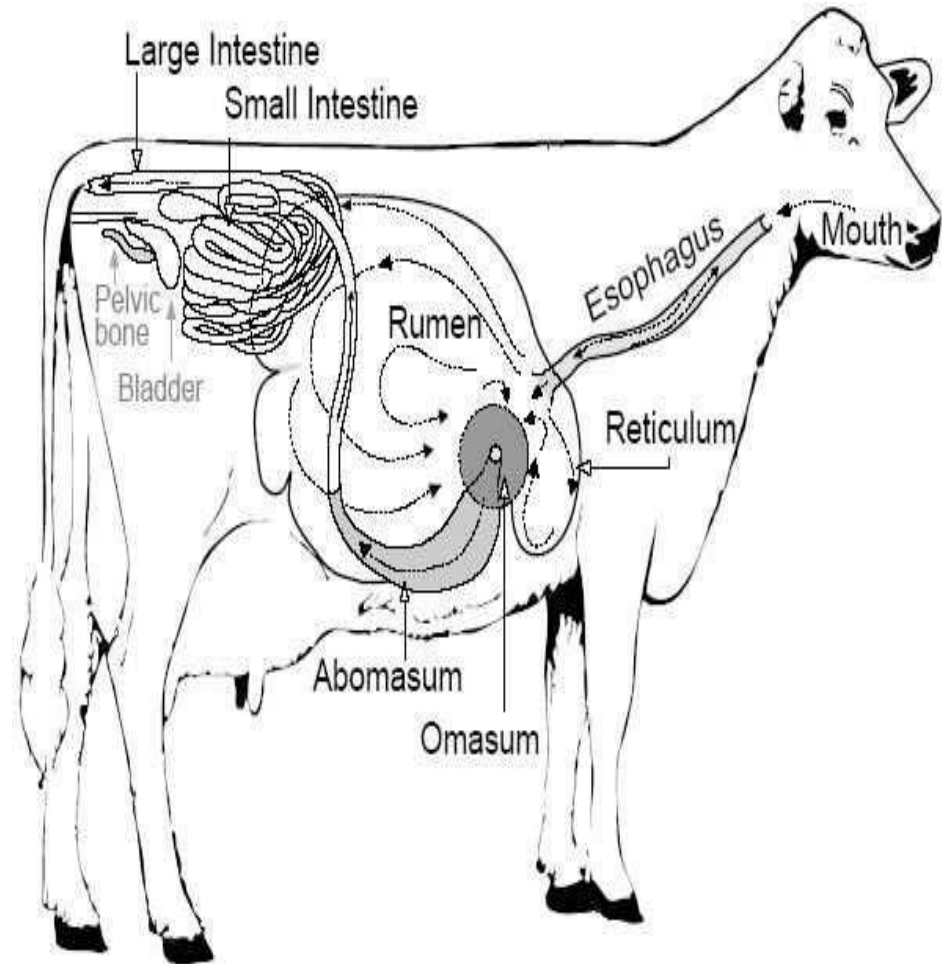
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- **Bloating forages:**
  - ▣ Alfalfa (*Medicago sativa*)
  - ▣ red clover (*Trifolium pratense*)
  - ▣ and white clover (*Trifolium repens*)
- **Crop maturity:**
- succulent pasture (in the prebloom stage) is the biggest single risk of bloat in cattle

## B. Feedlot bloat

6

- Caused by feeding **finely ground grain**
- Associated with high-level grain diets
- **viscosity of the ruminal fluid** increased due to **insoluble slime** by bacteria
- The slime may **entrap the gases** of fermentation



- **2. Secondary ruminal tympany (free-gas bloat)**

7

- Physical obstruction to eructation occurs in:

- **Esophageal obstruction**

- **foreign body**

- **stenosis** of the esophagus

- **Tuberculous lymphadenitis**

- **bovine leukosis**

- Interference with the **nerve pathways**

- A sudden marked change in **the pH of the rumen** contents due to either acidity or alkalinity

- **Hypocalcemia**



- **3. Chronic ruminal tympany**
- Chronic ruminal tympany occurs in calves up to 6 months of age:
  - ▣ Persistence of an enlarged thymus
  - ▣ Continued feeding on coarse indigestible roughage
  - ▣ Passage of unpalatable milk replacer into the rumen (instead of abomasum)
- Disappears spontaneously in time



# PATHOGENESIS

9

- Normally, gas bubbles **coalesce** rumen **fluid**
- Separate from the rumen contents to form **pockets of free gas**
- Finally eliminated by **eructation**
- A grass fed cow (**100L gas during the first hour of feeding**)
- A legume fed cow (**200L per hour**)
- **In frothy bloat, the gas bubbles remain dispersed throughout the rumen contents**

## □ PATHOGENESIS

10

Frothiness of the rumen contents interferes with **eructation reflex**

- Abnormal increase in the volume of the rumen & reticulum **cause distention**
- In free-gas bloat the animals **cannot eructate** the pockets of free gas because of abnormalities of the reticulorumen or esophagus

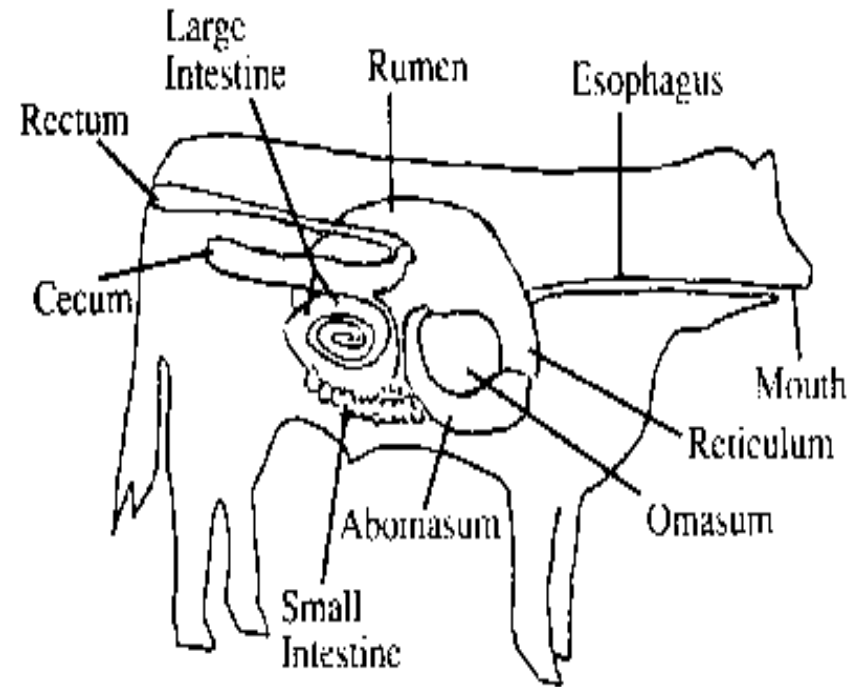
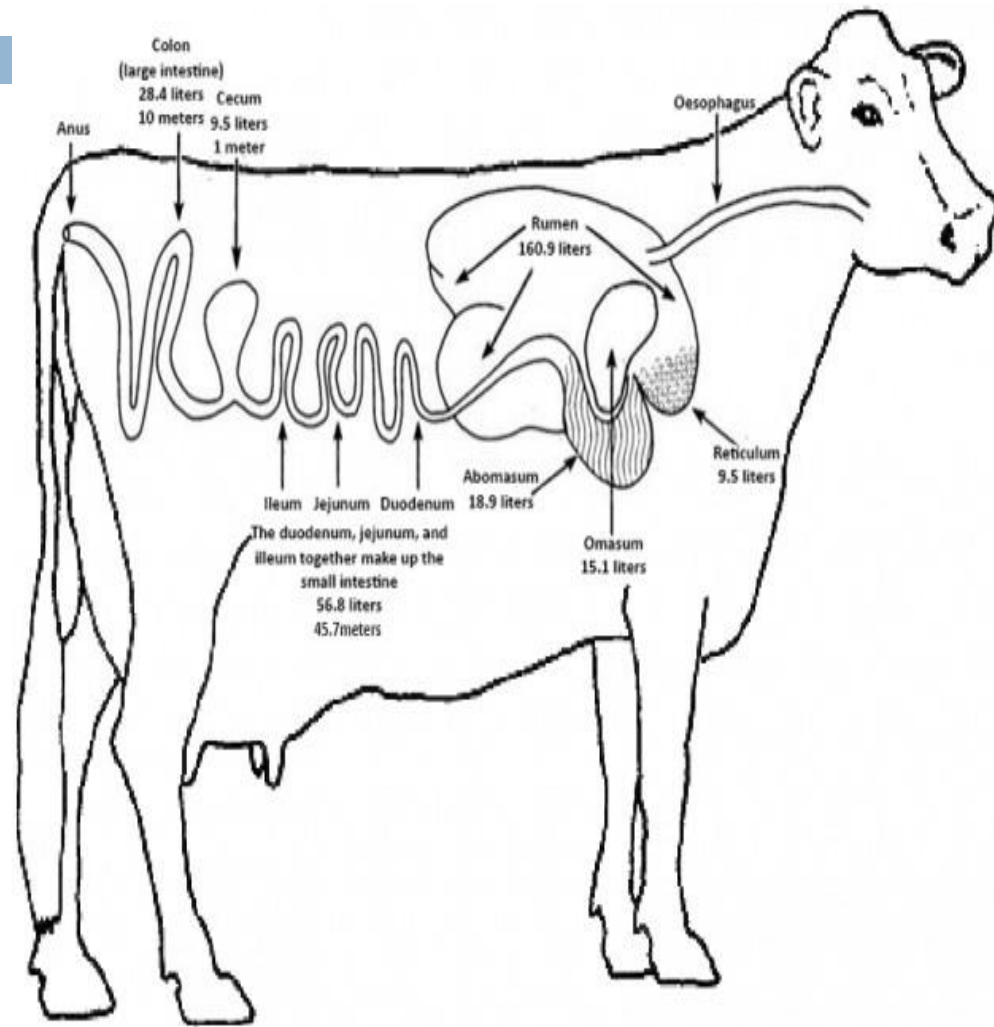


Figure 2. Digestive System of the Bovine

# PATHOGENESIS

11

- Rumen movements are initially stimulated by the distension
- The resulting hypermotility exacerbates the frothiness of the ruminal contents
- Terminally there is a loss of muscle tone and **ruminal motility**
- As the **intraruminal pressure increases**, occlusion of the vena cava, reduced lung capacity and death from **hypoxia**



# CLINICAL FINDINGS

## 12 Primary bloat:

- ▣ **Sudden death**
- ▣ Feedlot cattle die of bloat commonly found in the morning
- ▣ Obvious **distension** of the rumen
- ▣ Discomfort and the animal may **stand and lie down**
- ▣ **Kick** at its abdomen and **even roll**



# CLINICAL FINDINGS

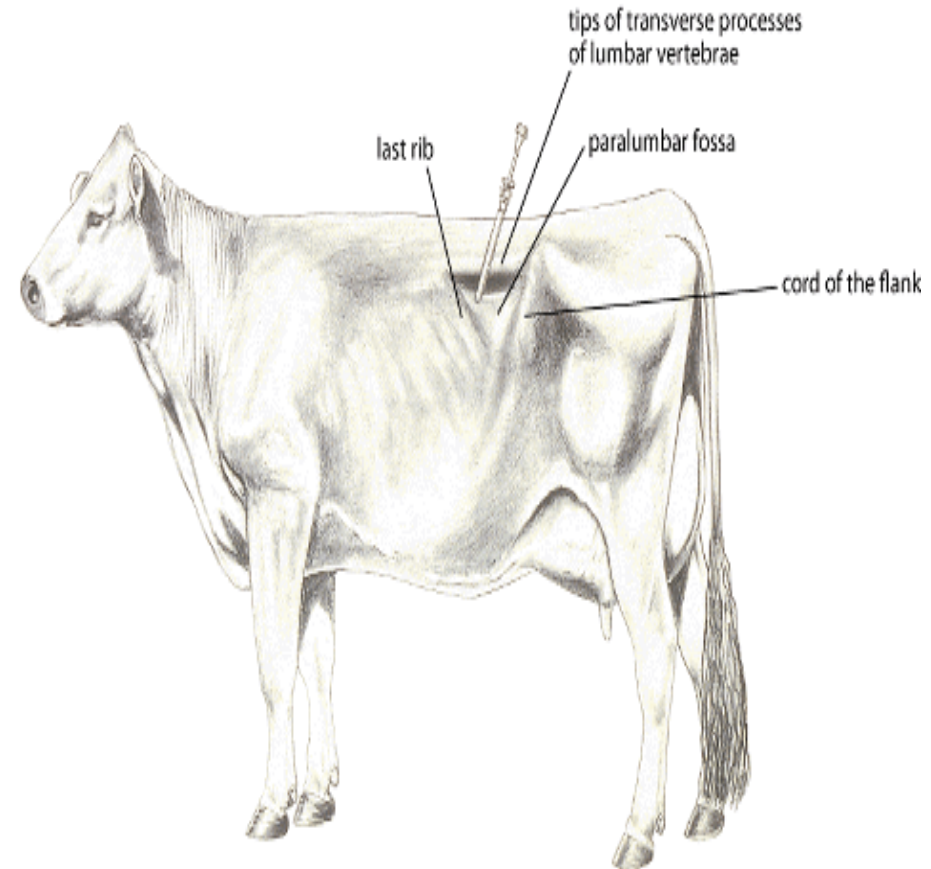
- ❑ **Frequent defecation and urination**
- ❑ **Dyspnea** accompanied by **mouth breathing, protrusion of the tongue, salivation** and extension of **the head**
- ❑ The respiratory rate is increased up to **60/min**
- ❑ Occasionally, **projectile vomiting** occurs



# CLINICAL FINDINGS

14

- In mild to moderate bloat:
  - ▣ the left **paralumbar fossa is distended**,
  - ▣ the animal is not in distress, and
  - ▣ 5-7 cm of skin over the left paralumbar fossa may be easily grasped and 'tented'



- In severe bloat:
  - ▣ **The skin over the left flank is very tense** and
  - ▣ cannot be grasped and tented
  - ▣ Low pitched tympanic sound
- Collapse and death almost without struggle occur quickly

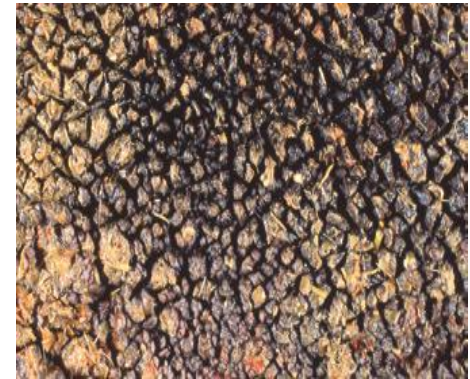




# NECROPSY FINDINGS

16

- **Protrusion and congestion of the tongue**
- **Marked congestion and hemorrhages of lymph nodes**
- **Friable kidneys** and mucosal hyperemia in the small intestine
- **The lungs are compressed**
- **The rumen is distended**
- A marked erythema is evident beneath the ruminal mucosa
- **The liver is pale** because of expulsion of blood from the organ
- Occasionally, the rumen or **diaphragm have ruptured**



# TREATMENT

17

- 1. **Emergency rumenotomy**
  - ▣ Using a sharp knife, a quick incision **10-20 cm in length** over the midpoint of the **left paralumbar fossa**
  - ▣ Once animals fall down - **death**
- 2. **Trocar and cannula**
- 3. **Promote salivation** or careful drenching with sodium bicarbonate (150-200 g in 1 L of water)
- 4. **Stomach tube**
- 5. **Antifoaming agents** (**tallow** at the level of 3-5 % of the total ration, 4 % salt, **Poloxalene**)

**3**

# CONTROL

19

- Condensed tannins in forages
- Alternative temperate forages
- Field management
- Grazing management
- Antifoaming agents
  - ▣ Oils and fats
  - ▣ Water-soluble feed supplements
  - ▣ Poloxalene
  - ▣ Alfasure
  - ▣ Alcohol ethoxylate detergents
  - ▣ Controlled-release monensin capsules
- 10-15% roughage



# **COLLEGE OF VETERINARY MEDICINE AND AGRICULTURE**

## **DEPARTMENT OF CLINICAL STUDIES**

**COURSE: VETERINARY GENERAL MEDICINE (Vetm 3132)**

# Diseases of the newborn (Neonatal infection)

# Objective

*At the end of this session students will be able to:-*

- ❖ Understand why the new born farm animals will require special management as compared to adult farm animals?*
- ❖ Describe major classifications of diseases of neonates, their treatment, control, and prevention*

# 1. Diseases of the newborn (Neonatal infection)

- This chapter considers diseases that occur during the first month of life in new born animals (neonates).
- **The new born require special attention because:**
  1. Underdeveloped immunology;
  2. Dependence on adequate colostrum containing adequate antibodies **at the right time**
  3. Dependence on frequent intake of readily available carbohydrate to maintain energy
  4. Unable to withstand fluctuating environmental temperature which will affect maintaining their normal body temperature and predisposes them to infectious diseases, dehydration and death.
- Thus any abnormality to the new born will seek rapid intervention with supportive therapy in the form of fluids, electrolytes and energy and nursing care.



- Neonatal/Newborn diseases in animal can be classified as

- 1. Fetal diseases**

- 2. Parturient diseases**

- 3. Post natal diseases**

- 1. Fetal diseases**

- Are diseases that occur in fetus **during intrauterine life**, e.g. congenital defects ( intrauterine growth retardation and neonatal neoplasia); prolonged gestation, intrauterine infections, abortion, fetal death with resorption or mummification or maceration.

- 2. Parturient diseases- occur during delivery**

- Commonly associated with dystocia, causing cerebral anoxia or fetal hypoxemia, and their consequences and predispositions to other diseases; injury to the skeleton or soft tissues.

### 3. Postnatal diseases- occur after birth (in neonate)

- These can be early postnatal diseases, delayed post natal diseases and late post natal diseases.

#### 1. Early postnatal diseases

- Occur **within 48 hours** of birth
- Deaths during this period are most likely acquired congenitally than **infectious disease**.
- Majority of diseases occurring during this periods are **noninfectious or metabolic** e.g. hypoglycemia and hypothermia due to poor mothering, hypothermia due to exposure to cold, low vigor in neonates due to malnutrition.
- **Infectious diseases** are often initiated during this period and manifest clinically at a later age because of their **incubation period**.

- Some infections like **navel infection, septicemic disease and enterotoxigenic colibacillosis**, have a short incubation period and can occur during this period.

## 2. Delayed postnatal diseases

- Occur in 2 - 7 days old neonates.
- May occur due to
  - Desertion by mother
  - Mammary incompetence resulting in starvation
  - The above reasons will cause failure of transfer of **colostral immunoglobulins**
  - And predispose the neonate to diseases like **colibacillosis, joint ill, lamb dysentery, septicemic disease**, most of the viral enteric infections in young animals, e.g. **rotavirus and corona virus**.

### 3. Late postnatal diseases

- Occur in newborn of **1- 4 weeks of age** .
- There is still some influence of **hypogammaglobulinemia**, with late onset **enteric diseases** and the development and severity of **respiratory disease** in this period.
- But other diseases not directly associated with failure of transfer of immunoglobulins such as **cryptosporidiosis, white muscle disease and enterotoxaemia** start to become important.

## 3.1. Neonatal infections

- Infection is a common cause of morbidity and mortality in neonates.
- There are a number of specific infectious pathogens that can cause disease.
- The acquisition of immunoglobulins from colostrum for passive antibody protection is important in ungulates (hoofed animals) **immediately after birth (within 12hrs)** because maternal immunoglobulins are not transferred transplacentally.
- **If not**, the newborns are vulnerable to infectious diseases during the neonatal period

# Common causes/etiology of neonatal infection in d/t spp of animals

Species of animals	Causative agents of infection
1. Calves	<p>1. Bacteremia and septicemia associated with <i>Escherichia coli</i>, <i>Listeria monocytogenes</i>, <i>Pasteurella</i> spp. <i>Streptococcus</i> or <i>Salmonella</i> spp.</p> <p>2. Enteritis associated with enterotoxigenic <i>E. coli</i>, <i>Salmonella</i> spp“ rotavirus and <b>corona virus</b>, <i>Cryptosporidium parvum</i> and <i>Clostridium perfringens</i> types A, B and C; and occasionally by the virus of infectious bovine rhinotrachitis and bovine virus diarrhea</p>
2. Piglets	<p>1. Septicemia with or without localization in joints, endocardium and meninges associated with <i>Streptococcus suis</i>, <i>Streptococcus equismilis</i>, <i>Streptococcus zooepidermicus</i> and <i>L. monocytogenes</i></p> <p>2. Bacteremia, septicemia and enteritis associated with <i>E. coli</i> transmissible gastroenteritis, Aujeszky's disease, swine pox, enterovirus infections, and vomiting and wasting disease are associated with viruses.</p>

## Species of animals

## Causative agents of infection

### 3. Piglets

3. Enteritis associated with *C. perfringens*, *Campylobacter* spp., rotavirus and *Coccidia* spp.
4. Arthritis and septicemia associated with *Erysipelothrix rhusiopathiae*.

### 4. Foals

1. Septicemia with localization associated with *E. coli*, *Actinobacillus equi*, *Klebsiella pneumoniae*,  $\alpha$ -hemolytic streptococci, *S. zooepidermicus*, *L. monocytogenes*, *Rhodococcus equi* and *Salmonella typhimurium*
2. Enteritis associated with *C. perfringens* types A, B, and C, *Clostridium difficile*, *R. equi*, *Salmonella* spp., *Strongyloides westeri*, *C. parvum* and rotavirus.

### 5. Lambs

1. Septicemia or bacteremia with localization in joints and/or synoviae and/or leptomeninges associated with *E. coli*, *L. monocytogenes*, streptococci, micrococci, *E. rhusiopathiae* and *Chlamyodophila* spp.
2. Enteritis associated with enterotoxigenic *E. coli*, *Salmonella* spp., rotavirus and corona virus and *C. parvum*

## Table cont'd---

### Species of animals

### Causative agents of infection

Lambs

3. Lamb dysentery associated with *C. perfringens* type B and C
4. Gas gangrene of the navel associated with *Clostridium septicum*, *Clostridium novyi* and *Clostridium chauvoei*
5. Pyemia associated with *Staphylococcus aureus*, *Fusobacterium necrophorum* and *Arcanobacterium pyogenes*
6. Pneumonia, polyserositis and peritonitis associated with *Pasteurella multocida* and *Mannheimia haemolytica*.



## Epidemiology

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

➤ Things to be considered in epidemiology of neonatal infection

## **1. Sources of infection: can be;**

- **Prenatal infection**
- **Post natal infection**

## **2. Routes of transmission**

## **3. Risk factors**

### **1. Prenatal infection**

- **Intrauterine infection (inutero).**
- Prenatal infection is mostly associated with abortion, still birth and weak neonates.

## 2. Post natal infection

- **Occur after birth**
  - Acquired from enteric or respiratory tract flora of the dam and/or other animals including others infected neonates;
  - The environment
  - Depending upon the specific agent, the source infection may be carrier animals or in the environment.

## 2. Routes of transmission

- Commonly by;
  - Ingestion
  - Aerosol
  - Via the umbilicus
  - Trauma
- The organisms are capable to invade through respiratory and intestinal epithelium to produce a bacteremia and septicemia.

### 3. Risk factors associated with neonatal infections

- Occurrence of neonatal infections generally depend on the following conditions;
  - ❑ **Level of immunity and immune response** (influenced by **Colostrum** intake and absorption of sufficient quantities of **immunoglobulin's** from the colostrum)
  - ❑ **Exposure pressure** (factors that influence cleanliness of the environment of the neonate.)
  - ❑ **Age of exposure**
  - ❑ **Virulence** (toxin production or other virulent factors of the agents like B-toxin of *E. coli* and *Cl. perfringens*)
  - ❑ **Animal risk factors** (that interfere with sucking drive and colostrum intake, such as cold stress and dystocia).

## Pathogenesis

- **Bacteremia and septicemia** are common features of neonatal exposure to infectious agents;
- Bacteremia with few or no systemic signs, followed by localization in various organs may cause;
  - ❑ If the portal of entry is the navel it causes **navel ill**.
  - ❑ Joints (producing a suppurative or non **suppurative arthritis**).
  - ❑ Eye to produce a **panophthalmitis**
  - ❑ In the heart valves to cause **valvular endocarditis**
  - ❑ In the meninges to produce a **meningitis**
  - ❑ Bladder ( cystitis)
  - ❑ Liver( hepatitis) and many other sites

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

## Clinical signs

- Depends on;
    - The virulence of the agent
    - The age of newborn
    - The immune status of the neonates
1. With organisms that have a **low propensity for toxemia** there is **fever, depression, anorexia and signs referable to localization.**

## **These include**

- Endocarditis with a heart murmur
- Panophthalmitis with pus in the anterior chamber of the eye
- Meningitis with rigidity, pain and convulsions and
- Polyarthrititis with lameness and swollen of joints

2. With more virulent organisms there are clinical signs of toxemia as well as bacteremia, including

- Fever
- Severe depression
- Prostration, coma and petechiation of mucosae
- Dehydration, acidosis and rapid death

## **Diagnosis**

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

- However, in outbreaks of a suspected infectious disease in young animals there is usually a need for more diagnostic microbiology and pathology
- The following methods are used in combination for the diagnosis of neonatal infection.

### **1. Epidemiological**

### **2. Clinical signs**

### **3. Pathological (Necropsy) findings**

- All the above methods of diagnosis give the tentative diagnosis of the disease (laboratory works are mandatory for confirmatory).



# Treatment

- Treatment of neonates depends on the nature of the condition ;
  - Commonly antibiotics (it depends on the agent),
  - supportive therapy (fluid and electrolytes—common in neonatal infection) and
  - other nursing cares.

# Common conditions of neonates

## 1. Omphalitis, omphalophlebitis and omphaloarteritis (Navel ill)

- It is infection of the **umbilicus and its associated structures**
- Common in new born farm animals particularly **in calves.**
- Anatomically the umbilical cord consists of
  - ✓ The amniotic membrane
  - ✓ The umbilical veins
  - ✓ The umbilical arteries
  - ✓ The urachus
- Normally the umbilical cord dries up within a week after birth, but if not appropriately disinfected immediately, the umbilicus may be infected and results in **Omphalitis, omphalophlebitis and omphaloarteritis (Navel ill)**
- Infection of the urachus may extend to bladder and cause **cystitis.**

### ❑ Omphalitis

- Infection of the **umbilical cord stump** (external aspects of the umbilicus ) in the newborn, commonly 2 - 5 days after birth in calves.

### ❑ Omhalophlebitis

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

### ❑ Omphaloarteritis

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

# Omphalitis in calves



## Etiology

- common bacteria identified in navel ill includes;
  - *E. coli*
  - *Proteus* species
  - *Staphylococcus* species
  - *Actinomyces pyogenes*

## Pathogenesis

- If the umbilicus is not well disinfected with appropriate disinfectant, the bacteria may enter to the umbilicus and may cause infection.
- Anaerobic bacteria like *C. tetani* may proliferate and cause lock jaw.
- Omphalphelibitis may cause liver abscess as it has connection with liver of fetus.
- Complication may results in infection of urachus results in cystitis.

## Clinical signs

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

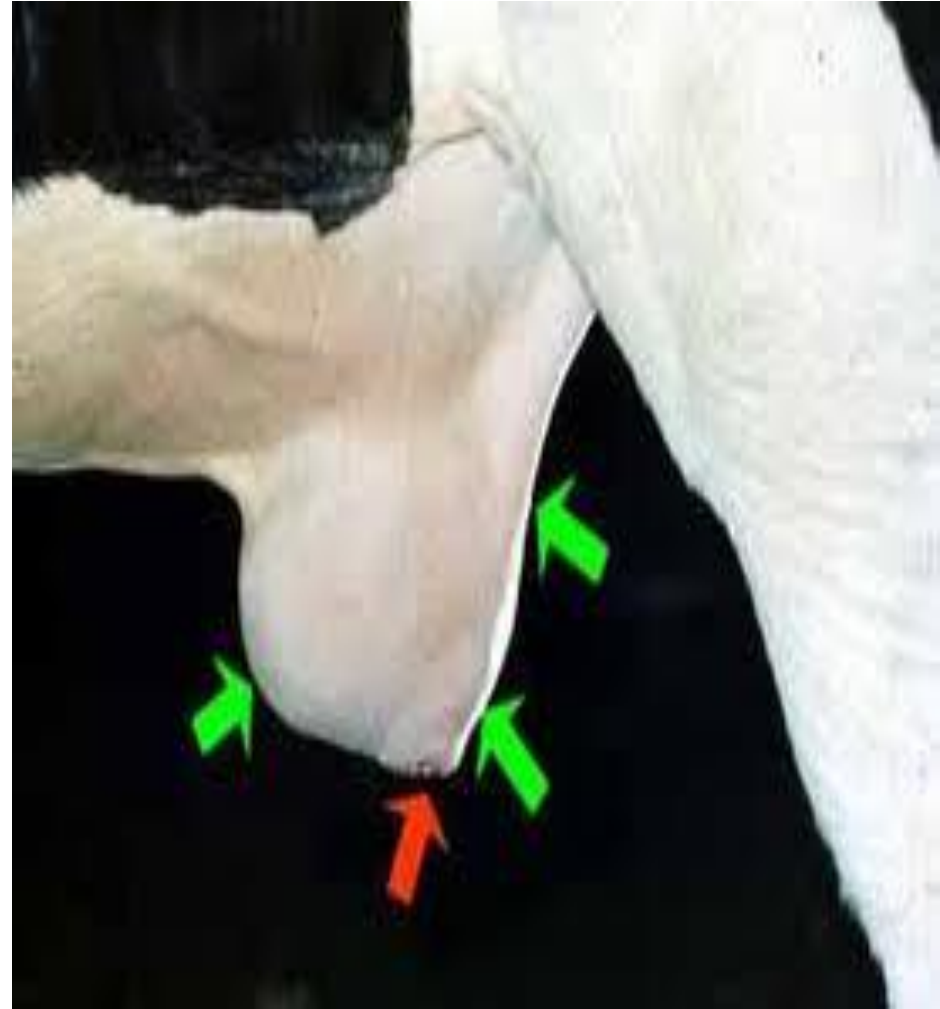
## Diagnosis

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

## Differential diagnosis

- You must differentiate from umbilical hernia.

## Umbilicus hernia in calves

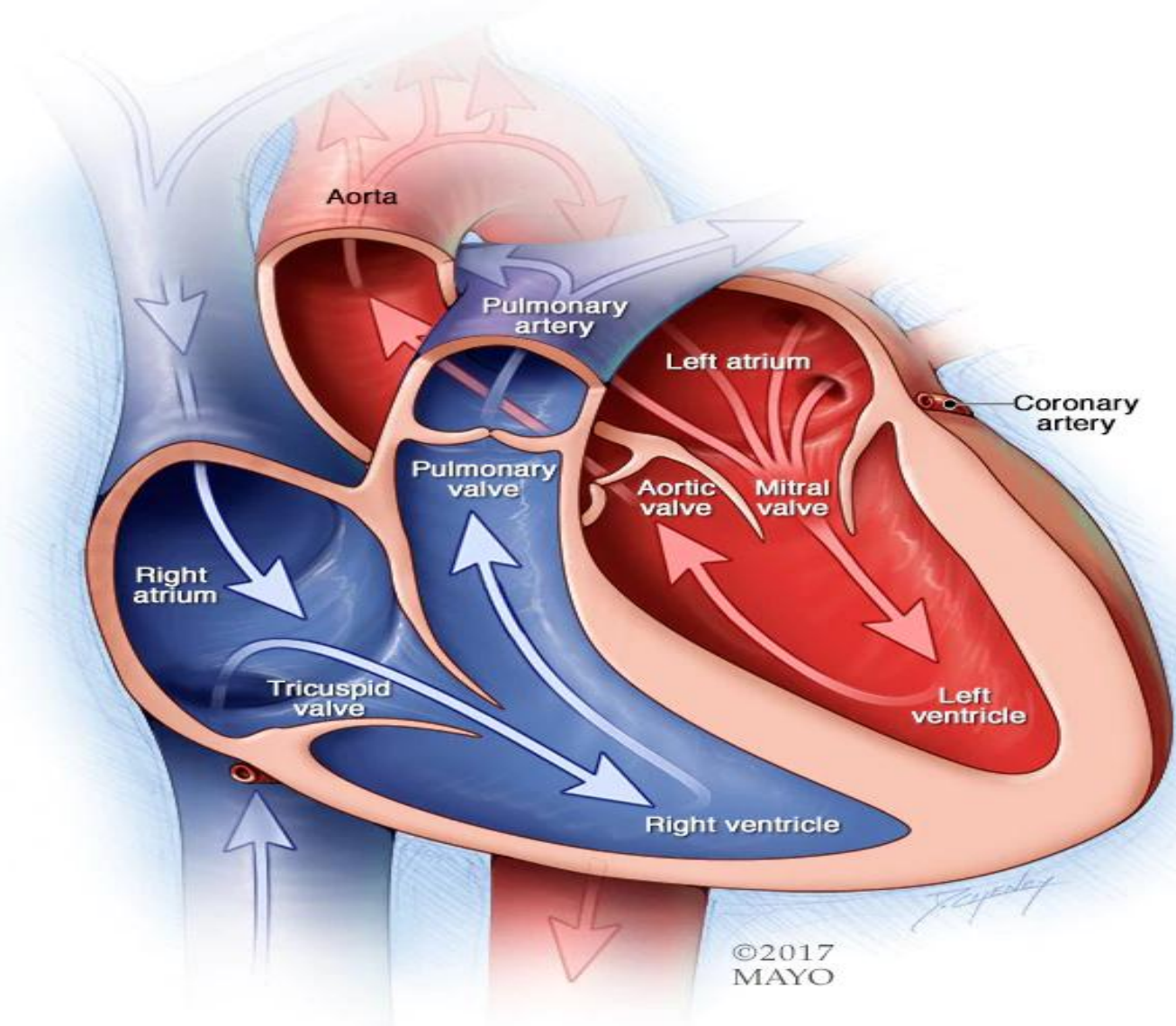


## Treatment and control

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



# Diseases of the cardiovascular system



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*What do you know about cardiovascular physiology????*

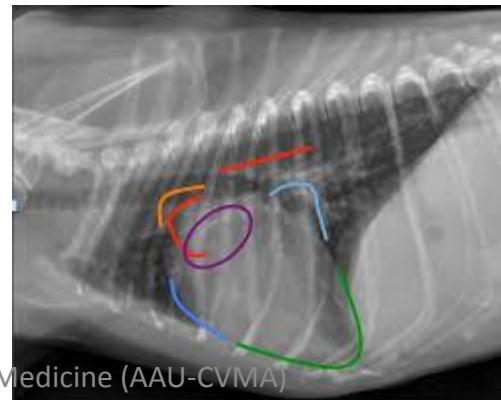
# 1. Diagnosis of cardiovascular system

- The diagnosis of **CVS** can be commence by reviewing medical history and signs
- Proceeded by conducting physical examinations:
  - **Auscultation** of heart and lungs
  - **Palpation** of abdomen and pulses
- Imaging is important step forward to **assess and confirm** the conditions in the heart and lungs.

# Imaging tools used for diagnosis of CVS:

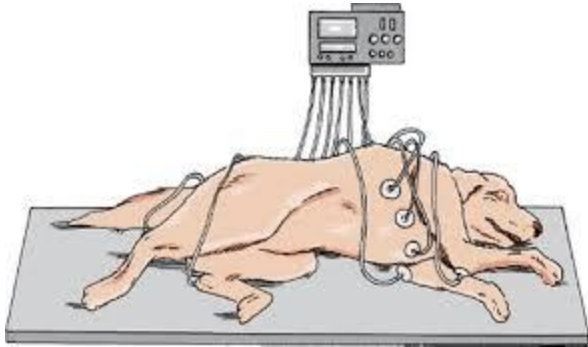
## 1. X-ray (radiography)

- Indicate generalized enlargement of the heart and presence of fluids in the lungs.
- Most heart problems can be highly suspected by physical examination and X-ray.



## 2. Electrocardiography:

- Used to record **electrical activity of the heart** or Dx of arrhythmias



### 3. Echocardiography:

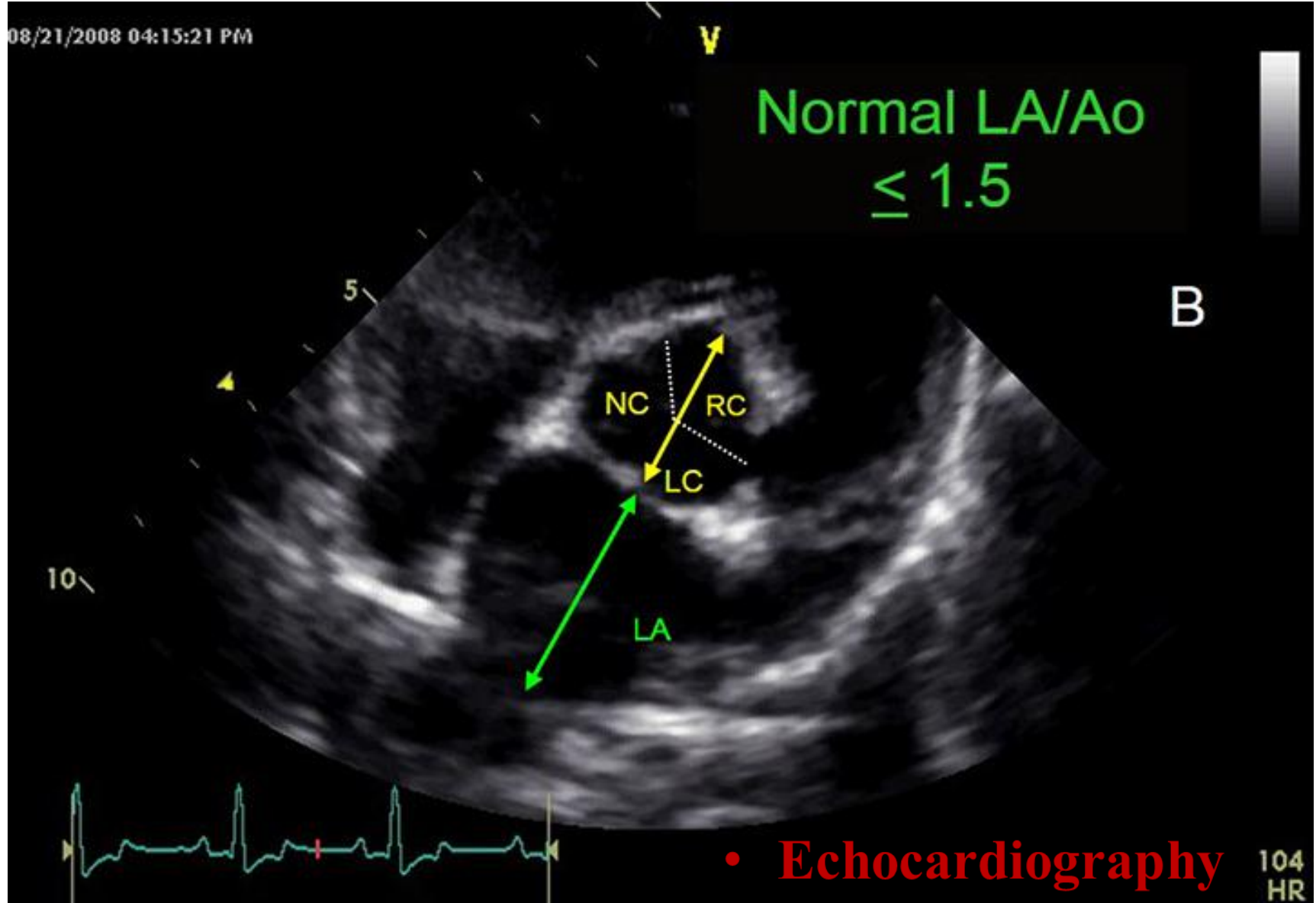
- Is a type of **ultrasonography** used to view the structure and function of heart in real time
- Excellent **to confirming tentative diagnosis** about CVS
- Used for;
  - Assessing severity of leaky heart valves or narrowed vessels
  - Evaluating chamber size and heart muscle function
  - Diagnosis of high blood pressure in the lungs
  - Identifying birth defects in the heart
  - Detecting heart tumors

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• **Echocardiography**

## 2. General manifestations of cardiovascular system diseases

- Exercise intolerance/tiredness
- Weakness
- Coughing
- Breathlessness/ Difficulty breathing
- Increased respiratory rate
- Abdominal distension
- Loss of consciousness (due to reduced blood flow to brain= fainting)
- Swelling of the legs
- Bluish discoloration of mucus membrane
- Loss of appetite and weight loss



### 3. Principles of Heart failure or Congestive heart failure (CHF)

- **Heart failure** is characterized by the heart's inability to meet body's demand (ineffective; **can't pump hard enough or can't fill enough**) and it is a progressive condition.
- Heart failure is not a disease rather, it is a complex pathophysiologic **syndrome** resulting in the **clinical manifestation** known as heart failure.
- It is consequence of variety of diseases.
- **Congestive heart failure** is an advanced pathophysiologic state of heart failure characterized by renal sodium retention, elevated venous pressures, and fluid accumulation in the lungs, subcutaneous tissues, or body cavities.

# Pathophysiology of heart failure

- The main pathophysiology of **HF** are **reduction in efficiency of heart muscle or myocardial failure and pressure/volume overload.**
- 1. **Myocardial failure:** results in myocardial loses normal systolic contractility and diastolic relaxation functions
- Mainly caused by **cardiomyopathy** due to myocardial infarction (**MI**) (heart muscle oxygen starved=> muscular atrophy and death )

## 2. Volume or Pressure overload:

- Common in dogs and responsible for left sided congestive heart failure
- Mainly caused due to **hypertension (pressure overload)** (increases the force of contraction needed to pump blood)
- Over time these increases in workload will produce changes to the heart like **hyper-function, exhaustion and myocardial damage**
- Which will affect its heart muscle contractility and relaxation efficiency.
- Both **cardiac inflow** (filling) and **cardiac output** (ejection) reduce=>

**HF**

# There are four functional classifications of heart failure:

## 1. Systolic myocardial failure

- It is a general *reduction in the ability of the heart muscle to contract*.
- It may be caused by trauma, infection, drugs or poisons, electric shock, heat stroke, or tumors.
- Some cases have no known cause.

## 2. Heart failure resulting from the *impedance (obstruction) to cardiac inflow* may result in a decrease in blood flow.

- This may be caused by
  - external compression of the heart (for example, fluid in the sac surrounding the heart)
  - diastolic dysfunction resulting in a stiff ventricle and reduced ventricular filling
  - abnormalities to physical structures of the heart.

### 3. Heart failure caused by pressure overload

- Pressure overload is a condition which occur when the ventricular systolic (pumping) pressure must be higher than normal to eject the stroke volume;
- Usually associated with hypertension (increased blood pressure throughout the **body** or in the **arteries of the lungs**) or obstruction to ejection as with aortic stenosis.
- **Long-term increases in stress** to the heart wall during contraction will result in stiffening of myocardium .

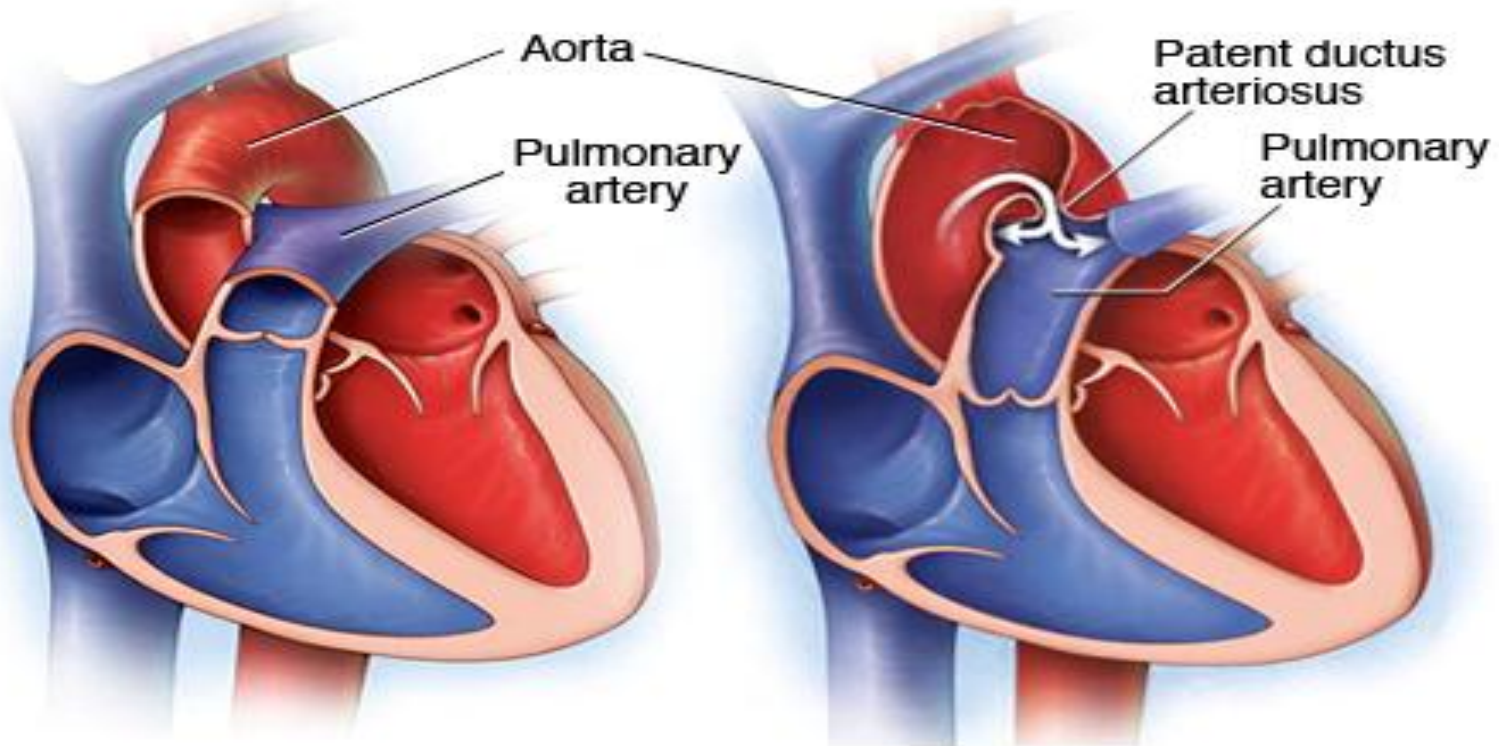
**4. Volume overload** is a condition in which the ventricular systolic (pumping) volume is higher than normal; usually associated with;

- Left-to-right shunt (patent ductus arteriosus (PDA), ventricular septal defect (VSD)),
- A regurgitate heart valve, or
- Chronic bradycardia.
- Any disease that increases volume of blood in the ventricle(s), thus increasing blood flow.



Normal heart

Patent ductus arteriosus



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## Patent ductus arteriosus

Patent ductus arteriosus, shown in the heart on the right, is an abnormal opening between the aorta and the pulmonary artery. A normal heart is shown on the left.



Type of heart failure	Description/signs
<p>Left-sided heart failure: blood is not effectively squeezed/pumped to the body (↓ blood to the body)</p>	<ul style="list-style-type: none"> <li>• Fluid back up in the lungs (pulmonary edema (crackles and dyspnea (shortness of breath))),</li> <li>• Reduced pulse due to poor tissue perfusion causing.</li> </ul>
<p>Right-sided heart failure: blood is not effectively squeezed from right ventricle to the lung (↓ blood in lungs)</p>	<ul style="list-style-type: none"> <li>• Fluid may back up into the body and manifested by JVD (Jugular distension); EDEMA/FLUID in the abdomen, legs and feet.</li> </ul>
<p>Systolic heart failure</p>	<ul style="list-style-type: none"> <li>• The left ventricle can't contract vigorously, indicating a pumping problem.</li> </ul>
<p>Diastolic heart failure (also called heart failure with preserved ejection fraction)</p>	<ul style="list-style-type: none"> <li>• The left ventricle can't relax or fill fully, indicating a filling problem.</li> </ul>

# Signs of Heart Failure

- Depend on the causes and the heart chamber affected;
  - **Left-sided congestive heart failure**; coughing, difficulty breathing are the most common signs. Fainting, low heart rate and low blood pressure and collapse.
  - **Right-sided congestive heart failure**; edema in different body parts (in the abdomen (ascites), the chest cavity, and the limbs).
  - **Biventricular failure**: signs attributable to both forms of congestive heart failure can be noted, although it is common for signs of one to outweigh the other.

# Medical treatments for CHF/HF

- Preferred drugs:

## 1. Positive inotropes:

- **Cardiac glycosides**-increases myocardium force of contraction by enhancing calcium influx into the myocardial cells.

- **Digoxin (most commonly used ),**

- **Digitoxin (not recommended in cats and horses) and**

- **Digitalis**

- » **Toxic effects are common in: hyperparathyroidism, renal insufficiency, old age and obese animals**

2. **Diuretics**-used to reduce edema secondary to CHF/HF or other causes
- Thiazides, loop diuretics (**furosemide**), potassium sparing diuretics
3. **Vasodilators**-effectively decrease myocardial workload
- For e.g. **ACE-inhibitors** (angiotensin converting enzyme inhibitors).
  - ACE converts angiotensin I to angiotensin II in the blood.
  - Angiotensin II-causes blood vessels to contract and narrow. Which will later lead to high blood pressure (hypertension).
  - **ACE inhibitors will reverse angiotensin II action**

# **Nutrition**

- low-sodium diet

# **Other Treatments**

- Oxygen
- Thoracentesis and abdominocentesis
- Bronchodilator
- Cough suppressants

# 4. Diseases of Heart

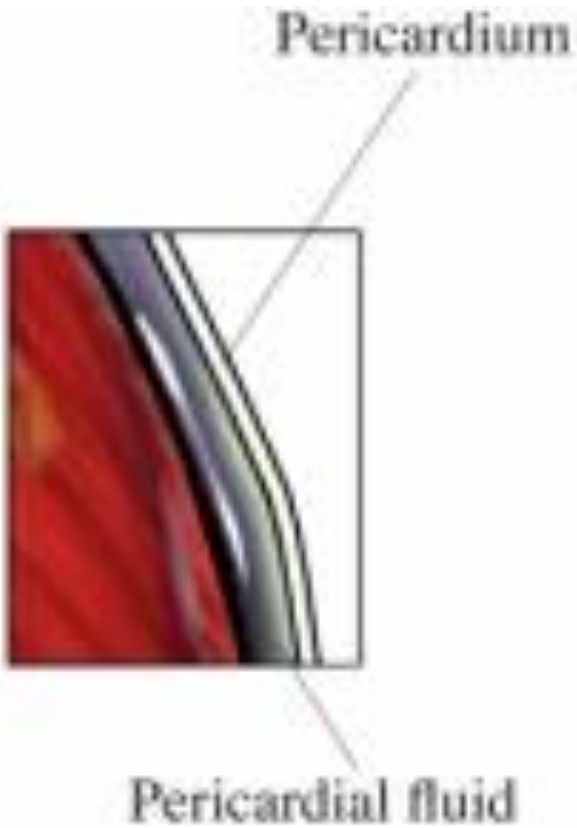
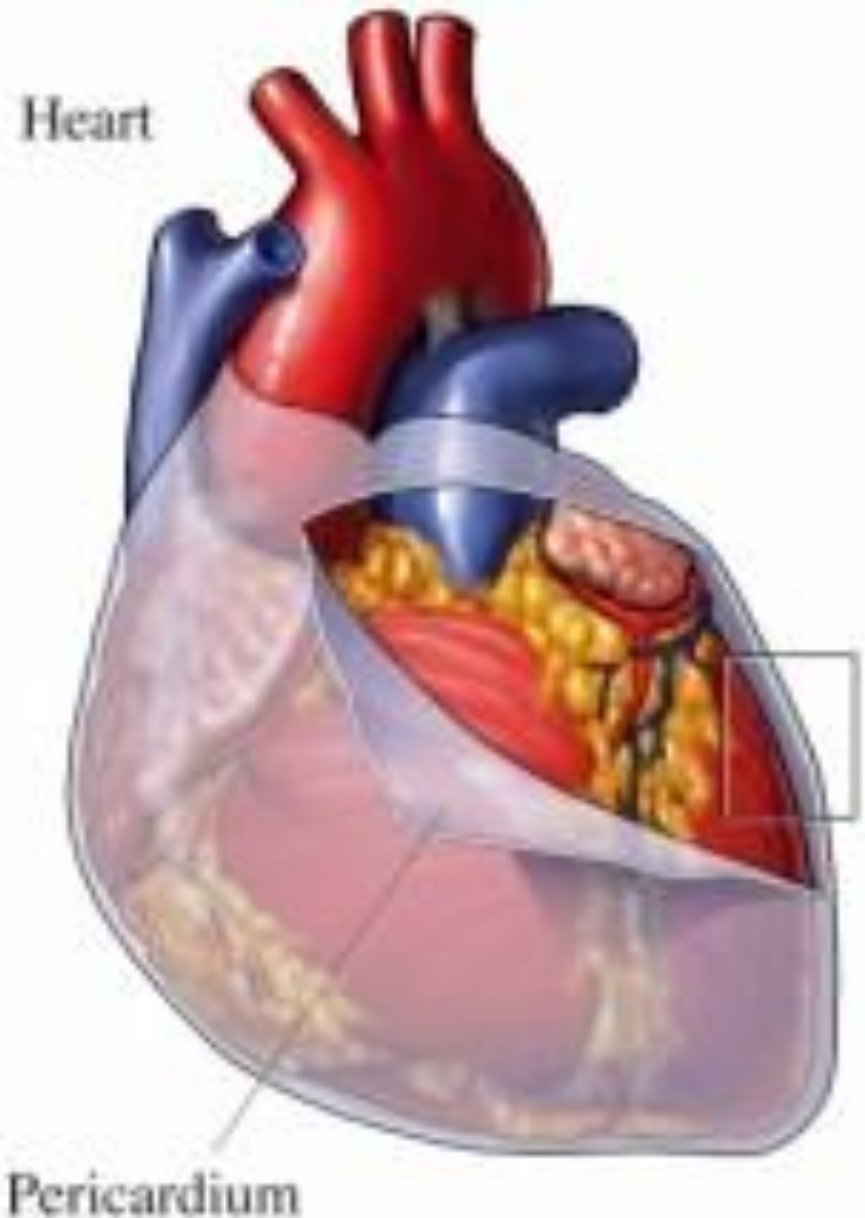
## 4.1. Pericarditis

- Is inflammation of the pericardial sac characterized by audible friction sound and muffling of the heart sound which leads to congestive heart failure.
- Pericarditis is not common but presents in three general forms:-
  1. Effusive
  2. Fibrinous
  3. Constrictive
  4. Traumatic
  5. Although combinations of one or more of the three forms can occur.

### 1. Effusive pericarditis

It is characterized by the accumulation of a protein rich fluid (transudate) within the pericardial sac.

# Pericardium of heart



## 2. Fibrinous pericarditis

- It is the subsequent of effusive pericarditis characterized by fibrin deposition which can lead to fibrinous pericarditis.

## 3. Constrictive pericarditis

- It is the subsequent of fibrinous pericarditis formed as the result of maturation of fibrin within the pericardial sac and it may reaches up to epicardium.

## 4. Traumatic pericarditis

- Pericarditis which is caused by perforation of the pericardial sac by an infected foreign bodies, occurs commonly in cattle.
- It is the extension from traumatic reticulitis or **TRP**.



# Some common bacteria and virus that cause pericarditis in domestic animals

Species of animals	Causative agent and disease of animals
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Cattle	
--------	--

	Mannheimia haemolytica, Black disease - if patients survive more than 24 hours
--	--

	Pasteurellosis , Staphylococcus aureus, Mycoplasma spp.
--	---

## Species

## Causative agent and disease of animals

Sporadic bovine encephalomyelitis, Haemophilus spp., including Histophilus somni, Tuberculosis, Pseudomonas aeruginosa, Mycoplasma spp., Klebsiella pneumoniae Actinobacillus suis, Idiopathic effusive (non septic) pericarditis.

Horses

Streptococcus spp., including S. equi, S. zooepidermicus and S. faecalis, Tuberculosis, Corynebacterium pseudotuberculosis, Actinobacillus equuli in association with EHV-1 infection  
Idiopathic effusive (non septic) pericarditis

## 4.2. TRP (Traumatic reticulopericarditis)

- **Traumatic reticulopericarditis or TRP** is the perforation of the wall of the reticulum initially (called **traumatic reticulitis**) and later piercing of the pericardium of the heart (called **pericarditis**) by a **sharp foreign body like nail, wire and other sharp metallic materials** ingested while grazing.
- The disease is common in large ruminant animals.
- But rarely in sheep and goat.

# Epidemiology

## Occurrence

- The disease is common in adult dairy cattle and feedlot animal.
- Because they are exposed frequently to this condition.
- However, it can also occur infrequently in yearlings , sheep and goat.

## Contributory factors

1. **The feeding behavior of ruminants:** - The non- discriminatory feeding behavior of cattle contributes the occurrence of TR.
2. **Advanced pregnancy :** - Pregnancy of cows is one of the contributory factor for occurrence of TRP.
  - Because the pressing effect of gravid uterus on reticulum pushes/ forced the perforated metal to other sites.

## Pathogenesis

- Lack of oral discrimination by cattle leads to the ingestion of foreign bodies that would be rejected by other species.
- Swallowed foreign bodies with feed may rarely lodge in rumen and omasum.
- But in most instances they pass the rumen and lodge in the reticulum.

## Why?

- This is because of the anatomical structure of reticulum mucosa which has honeycomb (net like structure).
- The honeycomb-like structure of the reticulum provides many sites for fixation of the foreign body.
- As the result many blunted metallic materials may lie there without causing harm.

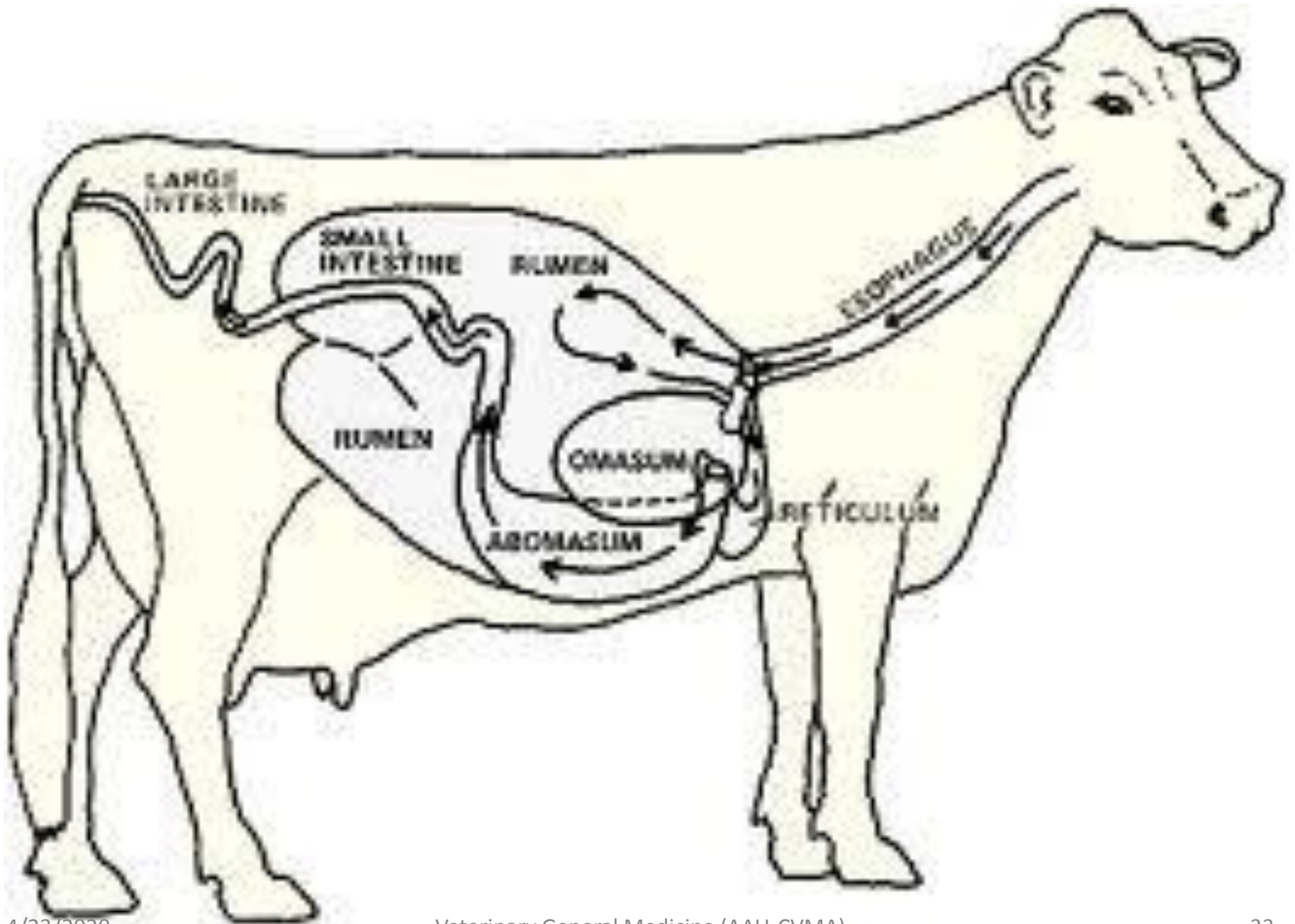
# The mucosa structure of fore stomachs of ruminant animals



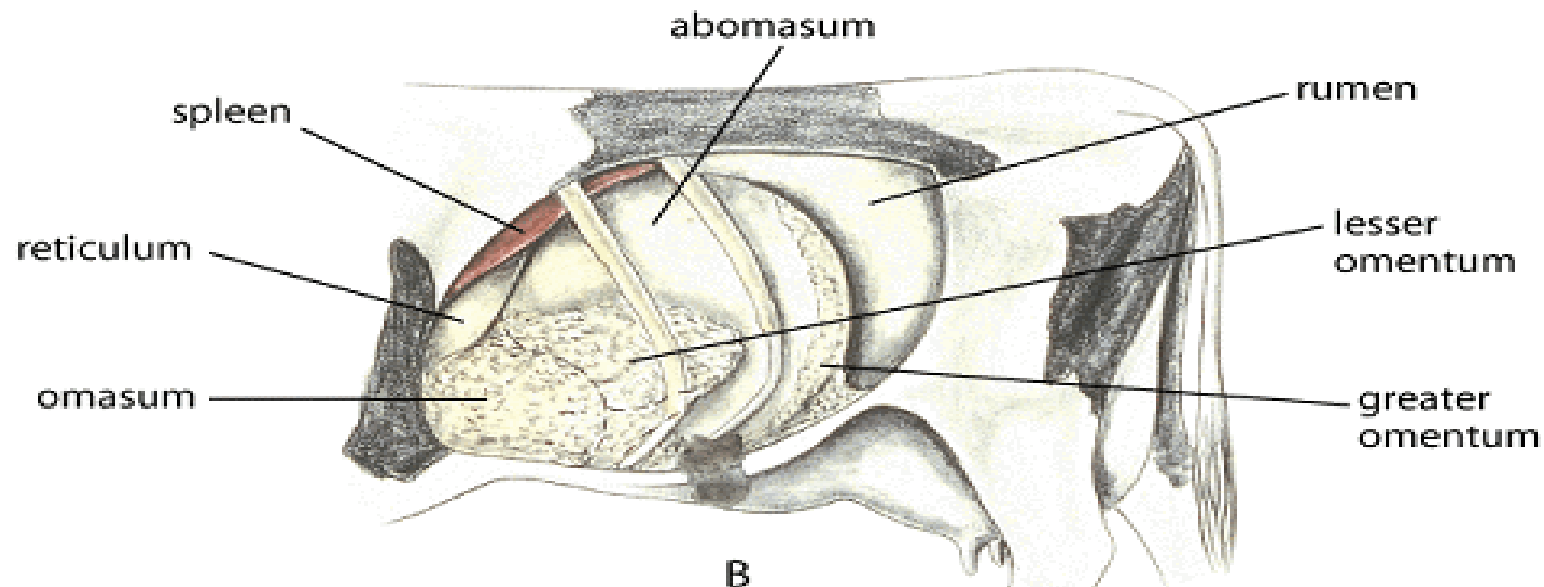
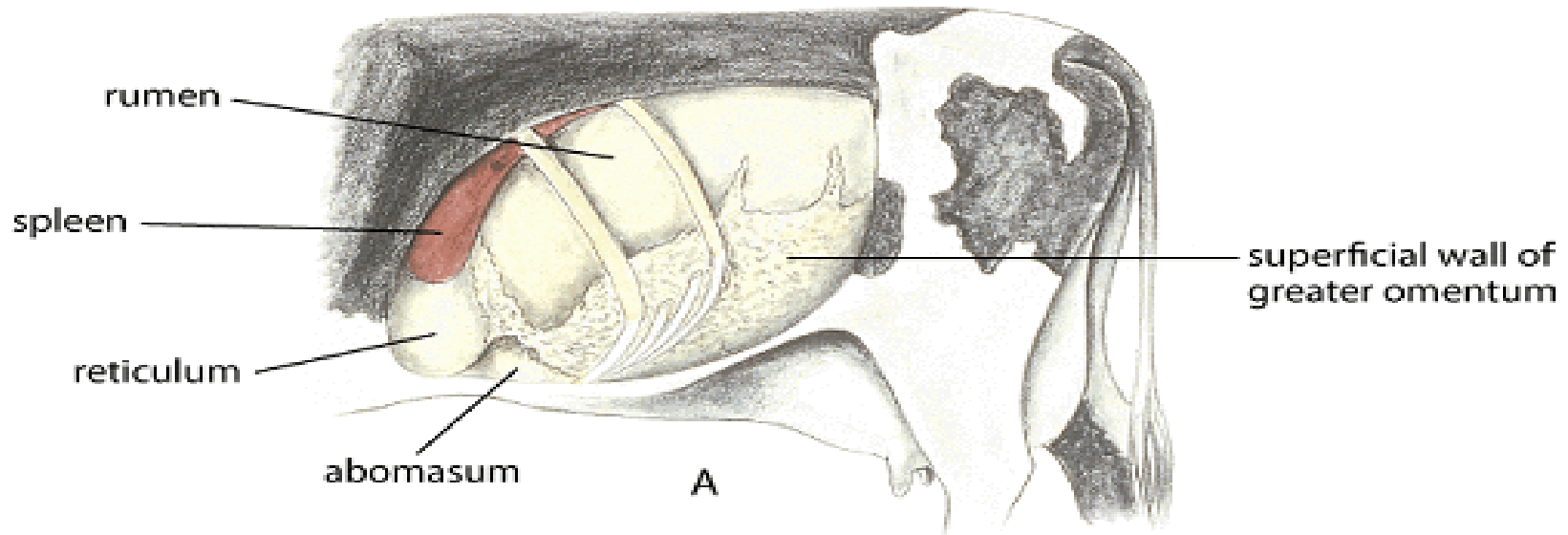
- But if a sharp – pointed objects are found in honey comb structure of the mucosa of reticulum, its contraction push the metallic material to perforate the mucosa of reticulum.
- And cause **traumatic reticulitis**.
- If the reticular wall is injured without penetration to the serous surface,
  - No detectable illness occurs
  - And the foreign body may remain fixed in the site for long periods and gradually be corroded away.
- But in many causes the sharp – pointed metallic materials may perforate the myo and serous membranes of reticulum.
- And most perforations occur in the lower part of the cranial wall of the reticulum.

- But some occur laterally in the direction of the spleen and medially towards the liver.
- Perforation may proceed beyond the reticulum to the peritoneum and cause involvement inflammation of peritoneum and cause **peritonitis (local or diffuse)**
- Further the metallic foreign body may injure different organs and membrane of the cavities.
- If the metallic material perforate the diaphragm and cause **diaphragmatic abscess or diaphragmatic hernia.**
- And further may perforate the lung and cause **pneumonia.**
- It may affect the pleura and mediastinal cause **pleurisy and mediastinitis** respectively.
- Perforation of the pericardium of the heart result in **pericarditis.**





# Topography of organs of the digestive system of cattle





# Clinical signs

- The disease appears suddenly with complete anorexia and a marked drop in milk yield in lactating cows.
- The animal is reluctant to move and does so slowly.
- Walking, particularly downhill, is often accompanied by grunting.
- Most animals prefer to remain standing for long periods and lie down with great care.
- Defecation and urination cause pain and the acts are performed infrequently and usually with grunting. This results in constipation, scant feces and in some cases retention of urine.

- Elevated temperature (39 0C - 410C)
- Abduction of the elbows
- Arching of the back
- Shallow abdominal respiration



- Rumination is absent and reticulo-rumen movements are markedly depressed and usually absent.
- The rumen may appear to be full because of the presence of a free-gas bloat with moderate distension of the left paralumbar fossa.
- Involvement of the right part of myocardium and endocardium of the heart (Traumatic pericarditis) is characterized by;
  - Highly pulsating jugular vein which results in **JVD (jugular vein distension)** and
  - **Brisket edema**
  - Pericardial friction sound detectable on auscultation



**JVD**

**BRISKET EDEMA**

## Diagnosis

- Diagnose of TR is made based on clinical signs like
  - Sudden onset of the disease with clinical signs like inappetance, absence of rumination, hypotony of rumen and pain
- Diagnosis can be made by;

### ❑ Eliciting pain

- ❖ Deep palpation of the abdominal wall just caudal to the xiphisternum.
- ❖ Pinching the withers to cause depression of the back .
- ❖ Allow the animal to move and walking downhill.



- Rectal palpation of abdomen cause pain
- Laparoscopy during traumatic reticulo peritonitis
- Radiography of cranial abdomen and reticulum
- Ultrasonography of reticulum

## **Differential diagnosis**

- Pericarditis
- Acute pleuritis
- Postpartum septic metritis
- Acute local peritonitis
- Other disease that will cause brisket edema and JVD

# Treatment

Two methods of treatment are in general used

1. Conservative treatment with or without the use of a magnet
2. Ruminotomy

## **1. Conservative treatment with or without the use of a magnet**

1. Immobilization of the animal, this help
  - Facilitates for the formation of adhesion and healing
  - Reduce the pressure of abdominal organs to diaphragm
  - Lastly reduce pain
2. Parenteral administration of broad spectrum antimicrobials to control infection

3. Oral administration of a magnet to immobilize the foreign body
  - Oral administration of a magnet to immobilize the foreign body is useful if
    - ✓ The material is made up of iron
    - ✓ It is found freely in the rumen

## 2. Ruminotomy

- Surgical removal of the foreign body through a ruminotomy incision is widely used as a primary treatment.
- It is used if the foreign body is found in rumen and in reticulum.

**NB.** If the above measures do not help the animal, slaughter the animal.

## Prevention measures

- Use well formulated ration
- Clean the hay, silage and straw from metallic materials using hands

of electromagnetic instruments

- Using magnetic ring as prophylaxis to traumatic reticulitis.

## **II. Diseases of the blood vessels**

### **1. Arterial thrombosis**

- Thrombosis is the partial or total blockage of arterial blood vessels by thrombus.
- Thrombus is a mass of platelets, red and white blood cells and fibrin.
- It develops and maintains a point of attachment to the blood vessels' wall and it never forms outside blood vessels.

## Predisposing factors of arterial thrombosis

- Three factors predispose to thrombus formation.
  1. Endothelial injury/damage
  2. Altered blood flow
  3. Blood hyper coagulation
- When a vessel's lining is damaged, endothelial cells may be stripped away or draw away from adjacent cells and expose the sub-endothelial collagen.
- This condition favors platelet adherence, activation, aggregation and initiation of thrombosis.
- Endothelial injury is particularly important in thrombus formation in the heart and arterial circulation (Eg. myocardial infarction or valvulitis).

# DISEASES OF THE NERVOUS SYSTEM

## Introduction

The functions of the nervous system are directed at the maintenance of the body's spatial relation with its environment. These functions are performed by the several divisions of the nervous system including:

- ✦ The *sensorimotor system*, responsible for the maintenance of normal posture and gait
- ✦ The *autonomic nervous system*, controlling the activity of smooth muscle and endocrine glands, and thereby the internal environment of the body
- ✦ The *largely sensory system of special senses*
- ✦ The *psychic system*, which control the animal's mental state

It is important to distinguish between primary and secondary diseases of the nervous system since both the prognosis and the treatment will differ with the cause.

- ✦ In **primary disease** of the nervous system the lesion is usually an anatomical one with serious, long-range consequences.
- ✦ In **secondary disease** the lesion, at least in its early stages, is more likely to be functional and therefore more responsive to treatment, provided the defect in the primary organ can be corrected.

The clinical findings that should arouse suspicion of neurological disturbance include abnormalities in the three main functions of the system.

### 1. Posture and gait

An animal's ability to maintain a normal posture and to proceed with a normal gait depends largely upon the tone of skeletal muscle but also upon the efficiency of the postural reflexes. Abnormalities of posture and gait are among the best indications of nervous system disease because these functions are governed largely by the coordination of nervous activity.

### 2. Sensory perceptivity

Tests of sensory perception in animals can only be objective, and never subjective as they can be in humans, and any test used in animals is based heavily on the integrity of the motor system.

### 3. Mental state

Depression or enhancement of the psychic state is not difficult to judge, particularly if the animal's owner is observant and accurate. The difficulty usually lies in deciding whether the abnormality is due to primary or secondary changes in the brain.

## MODES OF NERVOUS DYSFUNCTION

Nervous dysfunction can thus be broadly divided into two forms, depressed activity and exaggerated activity. These can be further subdivided into four common modes of nervous dysfunction; **excitation (irritation) signs, release of inhibition signs, paresis or paralysis due to tissue damage, and nervous shock.**

### **Excitation (irritation) signs**

Increased activity of the reactor organ occurs when there is an increase in the number of nerve impulses received either because of excitation of neurons or because of facilitation of passage of stimuli.

The excitability of nerve cells can be increased by many factors, including stimulant drugs, inflammation and mild degrees of those influences that in a more severe form may cause depression of excitability. Thus early or mild hypoxia may result in increased excitability while sustained or severe hypoxia will cause depression of function or even death of the nerve cell.

Irritation phenomena may result from many causes, including inflammation of nervous tissue associated with bacteria or viruses, certain nerve poisons, hypoxia and edema. In those diseases that cause an increase in intracranial pressure, irritation phenomena result from interference with circulation and the development of local anemic hypoxia.

### **Release of inhibition signs**

Exaggeration of normal nervous system activity occurs when lower nervous centers are released from the inhibitory effects of higher centers.

### **Paresis or paralysis due to tissue damage**

Depression of activity can result from depression of metabolic activity of nerve cells, the terminal stage being complete paralysis when nervous tissue is destroyed. Such depression of activity may result from failure of supply of oxygen and other essential nutrients, either directly from their general absence or indirectly because of failure of the local circulation. Infection of the nerve cell itself may cause initial excitation, then depression of function and finally complete paralysis when the nerve cell dies.

### **Nervous shock**

An acute lesion of the nervous system causes damage to nerve cells in the immediate vicinity of the lesion but there may be, in addition, a temporary cessation of function in parts of the nervous system not directly affected. The loss of function in these areas is temporary and usually persists for only a few hours. Stunning is the obvious example. Recovery from the flaccid unconsciousness of nervous shock may reveal the presence of permanent residual signs caused by the destruction of nervous tissue

### **Clinical manifestations of disease of the nervous system**

The major clinical signs of nervous system dysfunction include:

- ⊕ Altered mentation
- ⊕ Involuntary movements
- ⊕ Abnormal posture and gait
- ⊕ Paresis or paralysis
- ⊕ Altered sensation
- ⊕ Blindness
- ⊕ Abnormalities of the autonomic nervous system.

## I. ENCEPHALITIS

*Encephalitis* refers to inflammation of the brain. *Meningitis* is inflammation of the meninges. *Encephalomyelitis* refers to inflammation of the brain and spinal cord. *Meningoencephalomyelitis* refers to inflammation of the meninges, brain and spinal cord.

**Etiology and pathogenesis** Compared to other extraneural tissues, the inflammatory response mounted by the nervous system is unique.

- ✦ The CNS is immunologically dormant state within the body
- ✦ The capillary endothelial blood-brain-barrier (BBB) restricts free access by the blood constituents; but when this endothelium is damaged, infection gets access to the brain
- ✦ The CNS also lacks specialized dendritic antigen-presenting cells
- ✦ There is no lymphatic system within the nervous tissue, but cells and antigens within the CNS drain into the circulation and to the cervical lymph nodes.

Infection may invade the CNS through various routes. These include the following:

- ✦ Hematogenous route
- ✦ Extension from the nasal or olfactory tissues to the brain
- ✦ Through the peripheral nerve trunk
- ✦ Direct contact with infection after fracture or penetration of the skull

A specific infection may have a predilection site for particular parts of the nervous tissue. Many infectious diseases are characterized by encephalitis either as a primary manifestation or as part of a generalized disease. These include inflammation caused by *viruses, bacteria, chlamidia, fungi, protozoan parasite*.

Infectious thromboembolic meningoencephalitis of cattle caused by *Haemophilus somnus* is characterized by purulent inflammation of the brain and meninges, principally within and surrounding vascular walls. Diffuse suppurative or fibrinopurulent meningitis occurs as a wound infection during trauma of the brain. Some bacteria cause diffuse encephalitis and exert their effects primarily on vascular epithelium. Example – *L. monocytogenes* causes microabscesses.

Infiltration of lymphocytes, which may be only perivascular, constitute the most characteristic lesion in *viral encephalitis*. The primary lesion in most viral encephalitis is *necrosis of neurons*. But in the early stages of some viral diseases such as rabies, no particular visible lesions exist. Some viruses may cause demyelination where the demyelinating process may be initiated directly by the infectious agent alone or by an immunological response initiated by the agent. Examples include *encephalitis*.

In verminous encephalomyelitis, destruction of the nervous tissue may occur in many parts of the brain and in general the severity of the signs depends up on the size and mobility of the parasites and the route of entry.

### **Clinical findings**

Clinical signs of encephalitis are usually referable to a general stimulatory or lethal effect on neurons in the brain.



This may be in part due to the general effect of inflammatory partly due to the direct effects of the agent on nerve cells. The spectrum of clinical signs observed in cases of encephalitis includes the following:

- ✦ **Signs of toxemia** – because encephalitis is caused by infectious agents, it is accompanied by fever, anorexia, depression and increased heart rates. But this is not the case in more chronic diseases such as scrapie.
- ✦ **Behavioural changes:**
  - ✓ in the early stages, the animal responds excessively to normal stimuli (excitement or mania)
  - ✓ it may exhibit viciousness and uncontrolled activity including blind charging, bellowing, kicking and pawing
  - ✓ self-mutilation may occur in diseases such as pseudorabies
- ✦ **Involuntary movements**- involuntary movements are variable in their occurrence or may not appear at all. When they occur they include convulsions, usually clonic and may be accompanied by nystagmus, champing of the jaws, excessive frothy salivation, and muscle tremor especially in the faces and limbs.
- ✦ **Signs of loss of nervous function** – these may be the only signs in some instances
  - ✓ Excessive drooling of saliva and pharyngeal paralysis is common in rabies
  - ✓ In horses with equine encephalomyelitis, feed may be left hanging from the mouth
  - ✓ Recumbency and inability to rise may be the first clinical finding in many cases of meningoencephalitis caused by *Haemophilus somnus*.
  - ✓ Staggering gait may be observed in *bovine spongiform encephalopathy* (BSE)
  - ✓ Some diseases causing encephalitis may be characterized by deviation of the head, walking in circles, abnormalities of postures, ataxia and incoordination especially in their recovery stages.

### Clinical pathology

Clinical pathology may be of considerable assistance in the diagnosis of encephalitis but the techniques used are specific to individual diseases. The general parameters used in examination include:

- ✦ **Hematology** – complete and differential blood counts and serum chemistry profiles are recommended in most neurological cases in horses.
- ✦ **Serology** – sera may be taken for serological examination of some diseases
- ✦ **Cerebrospinal fluid**- laboratory examination of CSF for cellular content and pathogens may be indicated.

### Diagnosis

The diagnosis of encephalitis cannot depend entirely on the recognition of the typical syndrome because similar syndromes may be caused by many other brain diseases. Acute cerebral edema and space-occupying lesions of the cranial cavity, a number of poisonings, including salt, lead, arsenic, mercury, rotenone and chlorinated hydrocarbons, all cause similar syndromes as do hypovitaminosis-A, hypoglycemia, encephalomalacia and meningitis.

Fever is common in encephalitis but may not be present for example in rabies and scrapie, but it may occur in the non-inflammatory diseases if convulsions are severe.

Generally, the clinical diagnosis rests upon recognition of the specific cause and elimination of the other possible causes on the basis of:

- History
- Clinical pathology especially in poisonings
- Clinical findings characteristic of that particular disease
- In many cases a definite diagnosis can only be made on necropsy

#### Treatment

- Specific treatment for specific diseases diagnosed
- Antimicrobials are indicated for bacterial meningoencephalitis
- Supportive treatment- IV fluids should be given for electrolyte therapy including stomach feeding during the acute phase
- Sedation during the excitement stage may prevent the animal from injuring itself
- Nervous system stimulants during the period of depression may maintain life through the critical phase

## II. MENINGITIS

Inflammation of the meninges occurs most commonly as a complication of a pre-existing disease. It is usually caused by a bacterial infection and it is clinically manifested by fever, cutaneous hyperesthesia and rigidity of the muscles. It can affect both brain and the spinal cord.

### Etiology

Most cases of meningitis are bacterial, but most viral cases of encephalitis have some meningitic component. Some examples include the following:

#### Cattle

- Viral disease- bovine malignant catarrhal fever, sporadic bovine encephalomyelitis
- Bacterial diseases- Listeriosis, *Haemophilus somnus*

**Horses-** Strangles, *Pasteurella haemolytica*

**Sheep (lambs)-** *Staphylococcus aureus*, *Pasteurella multocida*, *Pasteurella haemolytica*

**Pigs-** Swine erysipelas, Salmonellosis, *Streptococcus suis* Type 2

#### Young animals

Streptococcal and coliform septicemia are probably the most common causes of meningitis in neonatal farm animals. The infection may originate from an omphalophlebitis or from a bacteremia. Some of the common infections include: *E. coli* in calves, *Streptococcus zooepidermicus* and *Streptococcus suis* Type I in piglets and *Str. Zooepidermicus* in lambs.

### Pathogenesis

Inflammation of the meninges causes local swelling and interference with blood supply to the brain and spinal cord. Failure to treat meningitis caused by pyogenic bacteria often permits the

development of a fatal choroiditis, with exudation into CSF, and ependimitis. There is also inflammation around the nerve trunks as they pass across the subarachnoid space. The signs produced by meningitis are thus a combination of those resulting from irritation of both central and peripheral nervous systems.

In spinal meningitis, there is

- ✓ *Muscular spasm with rigidity of the limbs and neck*
- ✓ *Arching of the back, and*
- ✓ *Hyperesthesia with pain on light touching of the skin.*

***In cerebral meningitis, there are signs of irritation including muscle tremors and convulsions.*** Since meningitis is usually bacterial in origin, fever and toxemia can be expected if the lesion is sufficiently extensive. Defects of drainage of CSF occur in both acute and chronic meningitis, and produce intracranial pressure.

### **Clinical findings**

- ✓ Acute meningitis usually develops suddenly and is accompanied by fever and toxemia in addition to nervous signs.
- ✓ Vomiting is common in the early stages in pigs
- ✓ There is trismus (locked jaw), opisthotonus and rigidity of the neck and back
- ✓ Motor irritation signs include, tonic spasms of the muscles of the neck causing retraction of the head, muscle tremor and padding movements
- ✓ Cutaneous hyperesthesia is present in varying degrees, even light touching of the skin causing severe pain in some cases
- ✓ There may be disturbance of consciousness manifested by excitement or mania in the early stages, followed by drowsiness and eventual coma
- ✓ Blindness is common in cerebral meningitis but it is not a constant clinical finding

### **Clinical pathology**

- ✓ Examination of CSF from lumbosacral
  - Elevated total protein levels
  - High cell count
  - Usually contains bacteria
- ✓ Hematology – reveals marked leukocytosis reflecting the severity of the systemic illness secondary to septicemia

### **Diagnosis**

Hyperesthesia, severe depression, muscle rigidity and blindness are common clinical findings in cerebral meningitis but it is often difficult to differentiate it from encephalitis and acute cerebral edema. Examination of the CSF is the only means of confirming the diagnosis before death. Sub-acute or chronic meningitis is difficult to recognize clinically. The clinical findings may be restricted to recumbency, apathy, anorexia, slight incoordination and some impairment of eyesight.

### **Treatment**

***Antimicrobials*** Meningitis is usually bacterial and parenteral treatment with antimicrobials is necessary. Large doses daily for several days are required. The levels of antimicrobials which are achieved in the meninges and CSF following parenteral administration to farm animals is not known. Presumably the blood-brain and blood-CSF barriers are not intact in meningitis and minimum inhibitory concentrations of some drugs may be achieved.

The choice of antimicrobials depends on the suspected cause of meningitis. The common antimicrobials such as penicillin and oxytetracycline are effective for the treatment of meningoencephalitis in cattle due to *Haemophilus somnus* when treatment is started early. Neonatal

Streptococcal infections also respond to penicillin when treated before irreversible injury has occurred.

The most promising antimicrobials for the treatment of meningitis in farm animals, particularly the neonates, may be the new third generation cephalosporins:

- ✚ Which resist hydrolysis by beta-lactamases,
- ✚ Have enhanced penetration to the CSF and
- ✚ Are bactericidal at very low concentrations.

The response depends on the causative agent, the severity of inflammation and the time treatment is started. For example some cases of swine meningitis caused by *Streptococcus suis* Type 2 commonly do not respond to treatment when clinical signs are obvious. On the contrary, the meningoencephalitis in cattle caused by *Haemophilus somnus* will respond dramatically if treatment is begun as soon as clinical signs are apparent. The prognosis in meningitis caused by infection with *E. coli* is unfavorable.

### III. ENCEPHALOMALACIA

The degenerative diseases of the brain are grouped under the name encephalomalacia. By definition, encephalomalacia, Leukoencephalomalacia and polioencephalomalacia refer to softening of the white and gray matter respectively.

#### Etiology

The causes of encephalomalacia (degenerative diseases of the brain) can be categorized as:

- ✚ Metabolic and circulatory disorders
  - ✓ Hepatic encephalopathy associated with liver disease and the resultant hyperammonia and other toxic factors which are neurotoxic
  - ✓ Hypoxia is lethal to neurons and upon recovery from the anesthesia, affected animals may temporarily blind and seizures may occur
- ✚ Intoxication and toxico-infectious disorders
  - ✓ Poisonous plants
  - ✓ Heavy metals (lead, arsenic, mercury)
  - ✓ Salt poisoning
  - ✓ Herbicides
  - ✓ Insecticides can affect the nervous system when ingested by animals
  - ✓ Endotoxin and exotoxins produced by bacteria
- ✚ **Nutritional diseases**
  - ✓ Vitamin A deficiency
  - ✓ Copper deficiency
  - ✓ Thiamine deficiency
- ✚ **Viral infections**
  - ✓ Acquired hypomyelination due to equine herpesvirus 1 infection
- ✚ **Hereditary diseases**

#### Pathogenesis

#### Clinical findings

Weakness in all four limbs is accompanied by:

- ✓ Dullness
- ✓ Blindness
- ✓ Head-pressing
- ✓ Ataxia
- ✓ Circling
- ✓ Terminal coma

In the early stages, particularly in ruminant polioencephalomalacia, there are involuntary signs including muscle tremor, opisthotonus, nystagmus and convulsion.

### **Diagnosis**

Diagnosis depends on clinical signs and diagnosis of some of the specific diseases mentioned under etiology.

### **Treatment**

The prognosis depends on the nature of the lesion. Early cases of polioencephalomalacia may recover completely if treated with adequate levels of thiamine. Encephalomalacia due to lead poisoning is incurable. Generally, treatment depends on the diagnosis of specific diseases causing encephalomalacia.

# DISEASES OF THE MUSCULO-SKELETAL SYSTEM

Diseases of the organs of support include muscle, bone and joints. Due to enormous functional relationship disorders involving anyone of them come up with more or less similar clinical manifestations. These are lameness, abnormal posture and movement, deformity (contracture or hyperextension of joints).

## I. MYOPATHY

Myopathy describes the non-inflammatory degeneration of the skeletal muscle which is clinically characterized by muscle weakness and pathologically by hyaline degeneration of muscle fibers. The serum levels of some muscle enzymes are elevated and myoglobinuria is a common accompaniment.

### Etiology

1. **Enzootic nutritional dystrophy**
  - ✦ One of the most important myopathies of farm animals
  - ✦ A nutritional deficiency of Vitamin E and/or selenium
  - ✦ Common in young calves, lambs, foals and piglets
2. **Exertional or post-exercise rhabdomyolysis**
  - ✦ Occurs as equine paralytic myoglobinuria (azoturia) in horses due to unaccustomed exercise or insufficient exercise
  - ✦ It also occurs in sheep chased by dogs or foxes
  - ✦ In cattle after running for few hours
  - ✦ In wildlife, during capture
3. **Degenerative congenital myopathy**
  - ✦ This occurs in newborn calves, sheep and goats affected by Acabane virus infected *in utero*
4. **Toxic agents**
  - ✦ Caused by some poisonous plants
5. **Ischemia**
  - ✦ Ischemic myonecrosis occurs in the thigh muscles of cattle recumbent for about 48 hours or more (downer cow syndrome)
6. **Neurogenic**
  - ✦ Neurogenic muscular atrophy occurs sporadically due to traumatic injury and subsequent degeneration or complete blockage of nerve supply to skeletal muscle
7. **Neoplasms**
  - ✦ Rhabdomyosarcomas are reported in the horse

### Pathogenesis

1. **Muscular dystrophy due to Vitamin E and /or selenium**
  - ✦ **Lipoperoxidation**- Selenium is an essential component of glutathione peroxidase. This enzyme, by destroying hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) and organic hydroperoxidase prevents damage of the cell membranes. Vitamin E is similarly an integral part of the cell membrane and is an anti-oxidant. Hence, deficiency of selenium and Vitamin E results **lipoperoxidation** of the cellular membranes of muscle fibers resulting in degeneration and necrosis.

- ⌘ **Myoglobinuria**- because of the necrosis of muscle, myoglobin is excreted in the urine and myoglobinuricnephrosis is an important complication, particularly of acute primary myopathy. The degree of myoglobinuriadepends on the severity of the lesion, acute cases resulting in marked myoglobinuria.
- ⌘ **Muscle enzymes** – an important biochemical manifestation of myopathy is the increased release of muscle cell enzymes which occurs during muscle cell destruction. *Creatinine phosphokinase (CPK)* and **serum glutamic oxaloacetate transaminase (SGOT)** are both elevated in myopathy and CPK especially is a more reliable and specific indication of acute muscle damage.

## 2. Exertionalrhabdomyolysis

- ⌘ In exertionalrhabdomyolysis in horses, there is
  - ✓ Enhanced glycolysis with depletion of muscle glycogen
  - ✓ Accumulation of large amounts of lactate, in muscle and blood
  - ✓ The development of hyaline degeneration of muscle fibers
- ⌘ *During enforced exercise, there is local muscle hypoxia and anaerobic oxidation resulting in the accumulation of lactate and myofibrillar degeneration.*

3. **Myopathy due to ischemia** - in this case, there may be multiple focal areas of necrosis which causes muscle weakness and results in an increase of muscle enzymes in the serum.
4. **Neurogenic atrophy of muscles**- in this case there is flaccid paralysis, a marked decrease in total muscle mass and degeneration of myofibers with failure to regenerate unless the nerve supply is at least partially restored.

## Clinical findings

- ✓ Sudden onset of weakness
- ✓ Stiff gait, inability to move the limbs and recumbency
- ✓ Respiratory and circulatory insufficiency
- ✓ Affected muscles become, swollen, hard and painful
- ✓ Myoglobinuria
- ✓ Acute cases of primary myopathy die within 24 hours after the onset of signs

## Clinical pathology

- ✓ **Muscle derived serum enzymes** – the levels of muscle enzymes are characteristically elevated following myopathy due to release of the enzymes from altered muscle cell membranes. These include the following:
  - ✚ *Creatinine kinase (CK)* – highly specific indication of both myocardial and skeletal muscle degeneration
  - ✚ *Serum glutamic oxaloacetate transaminase (SGOT)*
- ✓ **Myoglobinuria**- this is a common finding in adult horses in acute paralytic myoglobinuria. The urine becomes red or chocolate brown. Urine becomes dark when the myoglobin levels exceed 40mg/dl of urine.

## Diagnosis

- ✚ Most myopathies in farm animals occur in rapidly growing young animals and are characterized clinically by a sudden onset of acute muscular weakness, and pain often precipitated by unaccustomed exercise.

- ✚ There may be evidence of dietary deficiency of vitamin E and selenium in the case of nutritional muscular dystrophy.
- ✚ A sudden onset of recumbency or stiffness in young farm animals, which are bright and alert, should arouse suspicion of acute muscular dystrophy.
- ✚ The clinical sign of exertional myopathies are obvious and combined with history give a good diagnosis.
- ✚ **Myositis** may present a similar syndrome but it is usually present as a secondary lesion in clinically distinguishable primary diseases or is accompanied by obvious trauma or toxemia.

## Treatment

- ✚ Vitamin E and selenium are indicated for the treatment of nutritional muscular dystrophy.
- ✚ The treatment of exertional rhabdomyolysis is not well defined but enforced rest and relief of pain is recommended. Reduction of concentrate food and administration of anti-inflammatory drugs is also recommended.
- ✚ Supportive therapy for all myopathies
  - ✓ Sodium bicarbonate solution parentally to counteract systemic acidosis
  - ✓ Provide comfortable bedding in softer ground
  - ✓ Frequent turning from side to side to minimize secondary myopathy
  - ✓ Provision of balanced fluid therapy to prevent myoglobinuric nephrosis
  - ✓ Provision of palatable nutritional diet

## II. MYOSITIS

Myositis is an inflammation of muscle. It may arise from direct or indirect trauma to muscle and occur as a part of syndrome in a number of specific infectious diseases (blackleg, FMD, blue tongue, Sarcosporidiosis). On the other hand a sporadic case of localized infectious myositis of skeletal muscle is not uncommon. The disease has significant place in horses. (*horsei.m. injection is done on the neck to prevent iatrogenic myositis*).

### Etiology

- Infectious diseases
- Trauma
- Iatrogenic
- Screw worm infestation

### Pathogenesis

The central focus of tissue reaction involves the interstitial components in contrast to degenerative myopathy where the foci centre on alteration within the muscle fiber. The interstitial reaction in myositis is characterized by influx of inflammatory cells and serous fluid, vascular congestion and proliferation of interstitial connective tissue cells. The type of inflammation observed are generally within hemorrhagic, necrotizing, suppurative or granulomatous.

### Clinical findings

The course could be acute or chronic

- ✓ Severe lameness



- ✓ Heat
- ✓ Toxemia
- ✓ Swelling
- ✓ Pain on palpation
- ✓ Fever

In chronic course

- ✓ Much wasting of the affected muscle

### **Diagnosis**

- ✓ signs
- ✓ Histopathology
- ✓ Confirmatory diagnosis to suspected systemic problem

### **Treatment**

- ✓ Infectious- depending on the etiology of systemic problem
- ✓ Sporadic case- abscess and necrosis- incision and drainage – and antibiotic, anti-pain, rest

## **III. ARTHRITIS AND SYNOVITIS**

Inflammation of the articular surfaces and synovial membrane as a result of infection occurs commonly in farm animals. It is characterized by varying degrees of lameness and a warm and swollen painful joint. The synovial fluid is usually abnormal, containing increased leukocyte count and the pathogens causing the arthritis. The arthritis may be severe enough to cause systemic illness, and in some cases a draining sinus tract may occur.

### **Etiology**

Special bacterial infections of the joints are most common in newborn farm animals in which localization of infection occurs in joints following bacteremia or septicemia. Infection can enter to the joints through the following routes:

- ❖ ***Hematogenous spread*** - from suppurative lesions commonly from udder, uterus, diaphragmatic abscesses or other wounds.
- ❖ ***Spread from surrounding tissues***- such as footrot and interdigital abscesses
- ❖ ***Traumatic perforation of the joint capsule***

### **Pathogenesis**

In infectious arthritis which is hematogenous in origin there is usually a synovitis initially, followed by changes in articular cartilages and sometimes the bones. With localization of the infectious agent in the synovial membrane and joint cavity, there will be inflammation, edema and deposition of fibrin in the synovial membrane. The synovitis causes distension of the joint capsule with fluid and the joint becomes painful and warm. Successful treatment and elimination of the infection at early stage synovitis will minimize changes in articular cartilage and bone healing will result.

A progressive infectious synovitis commonly results in pannus formation between articular surfaces with erosion of articular cartilage, infection of subchondral bone and osteomyelitis. In the chronic stages, there is extensive granulation tissue formation, chronic synovitis and degeneration.

Depending on the organism, the arthritis may be suppurative or sero-fibrinous. Suppurative arthritis is particularly destructive of cartilage and bone and commonly there is rupture of the joint capsule.

### **Clinical findings**

- ❖ ***Inflammation of the synovial membrane causes pain and lameness*** in the affected limb, sometimes to the point that the animal will not put it to the ground
- ❖ ***Pain and heat*** are usually detectable on palpation
- ❖ ***The joint may be swollen*** but the degree will depend on the type of infection – ***pyogenic bacteria*** cause the greatest degree of swelling and may result in rupture of the joint capsule
- ❖ Fever, inappetence to anorexia, endotoxemia, loss of body weight, and discomfort may occur in animals with only one joint severely affected.
- ❖ The joints most commonly affected are the ***hock, stifle and knee*** but infection of the fetlock, interphalangeal and intervertebral joints is not uncommon

### **Clinical pathology**

- ❖ ***Arthrocentesis***- aspiration of joint fluid for culture and analysis is necessary for a definitive diagnosis. Careful disinfection of the skin and the use of sterile equipment is essential to avoid introduction of further infection.
- ❖ ***Analysis of joint fluid*** –
  - ✓ Total and differential cell count
  - ✓ Total protein concentration and
  - ✓ Specific gravity
- ❖ ***Culture of joint fluid*** –joint fluid must be cultured for aerobic and anaerobic bacteria
- ❖ ***Serology of joint fluid*** – serological tests may be of value in determining the presence of specific infections with *Salmonella spp*, *Brucella spp*. and other infections
- ❖ ***Radiography and Ultrasonography***

### **Diagnosis**

- ✓ Clinical findings
- ✓ Clinical pathology
- ✓ Examination of the joint

### **Treatment**

- ❖ Antimicrobial
  - ✓ *Parenteral antimicrobials*
  - ✓ *intra-articular antimicrobials*
- ❖ Lavage (drainage) of the affected joint
- ❖ Physical therapy- local application of heat
- ❖ Anti-inflammatory therapy



## DISEASES OF THE SKIN AND CONJUNCTIVA

### PHOTOSENSITIZATION

Photosensitization, resembling but distinct from sunburn, is a severe dermatitis of animals resulting from a complex reaction induced by plant pigments exposed to ultraviolet (UV) wave length sunlight in the skin of animals that have eaten certain plants. This reaction is most severe in non-pigmented skin where these reactive compounds are most directly exposed to light in the UV spectrum. The precise mechanism of this reaction is unknown, but it is thought to be a light-enhanced oxidation reaction. The amino acids (histidine, tyrosine, tryptophan) are particularly susceptible to oxidation and once oxidized evoke an intense inflammatory response in the blood vessels and surrounding cells that results in tissue necrosis. In addition to plant pigments, fungal toxins, chemicals, and occasionally congenital diseases affecting porphyrin metabolism in the liver may induce photosensitization. Quite frequently horses and cattle develop photosensitization while on pasture with no determinable cause.

Photosensitization may be conveniently classified into two basic types - **primary** and **secondary**.

**Primary photosensitization** is associated with photodynamic compounds in certain plants, which once absorbed from the digestive tract, **react in the nonpigmented with UV light to cause a severe dermatitis**. Also in this category are the congenital photosensitivity diseases associated with defective pigment (porphyrins) metabolism in the liver of animals.

**Secondary or hepatogenous photosensitization**, as the name implies, results when an animal's liver is sufficiently diseased to be unable to remove plant by-products that can react with UV light to cause photosensitization. Phylloerythrin, a bacterial breakdown product of chlorophyll, is the photosensitizing compound. Normally phylloerythrin is removed by the liver and excreted in the bile, but if the liver is severely diseased, it accumulates in the blood to cause photosensitization if a white skinned animal is exposed to a UV light.

Hepatogenous photosensitization can be further subdivided into that attributable to liver disease as opposed to that caused by biliary system disease that causes a backup of bile. Secondary photosensitization is much more common in livestock than primary photosensitization, and because of the severity of the underlying liver disease, it always carries a poor prognosis. Occasionally photoreactive pigments (porphyrins), produced in animals as a result of normal hemoglobin breakdown, accumulate and cause photosensitization.

Congenital porphyria is an inherited defect in various breeds of cattle as a result of a specific enzyme deficiency that normally regulates metabolism of porphyrins. Southdown sheep may also develop photosensitivity due to a congenital defect in the liver's ability to excrete the photoreactive compound phylloerythrin. As it accumulates in the skin, phylloerythrin causes photosensitivity when the animal is exposed to sunlight. Chemicals such as phenothiazinesulfoxide, a derivative of the anthelmintic phenothiazine, may also produce photosensitivity in ruminants if they are exposed to sunlight after treatment with phenothiazine.

## **Primary Photosensitization**

Primary photosensitization develops when animals eat plants containing polyphenolic pigments. These compounds are at highest concentration in the green plant and are readily absorbed from the gastrointestinal tract to circulate in the blood. In nonpigmented skin these compounds react with UV light to produce radiant energy that oxidizes essential amino acids in the skin's cells. The cells die in the photosensitization process, and the affected skin eventually sloughs off. Two plants associated historically with primary photosensitization are buckwheat (*Fagopyrum esculentum*), and St. John's wort (*Hypericum perforatum*). Both plants contain polyphenolic pigments capable of causing primary photosensitization. Several plant species including bishop's weed (*Ammimajus*), spring parsley (*Cymopterus watsonii*), and Dutch (*Thamnosmatexana*) contain photodynamic furanocoumarin compounds that have been associated with photosensitivity through ingestion and direct contact with the skin.

The detection of primary photoreactive compounds in plants can be accomplished using a screening test that is based on the sensitivity of the fungus *Candida albicans* to irradiation. The simple procedure involves exposing suspect plant material on agar plates seeded with *C. albicans* to UV light. Photoreactive plants will inhibit the growth of the *C. albicans*.

## **Secondary Photosensitization**

Secondary or hepatogenous photosensitization in animals occurs more commonly than primary photosensitization. Liver disease, the underlying cause of secondary photosensitivity, results from ingestion of plants containing compounds toxic to the liver. A variety of compounds toxic to the liver are found in plants, the most important of which are the pyrrolizidine alkaloids (PAs).

Once 80 percent or more of the liver is destroyed by these alkaloids, it is unable to eliminate phylloerythrin, a bacterial breakdown product of chlorophyll. Phylloerythrin then accumulates in the blood, and as it circulates through the skin and is exposed to UV light, it fluoresces and causes oxidative injury to the blood vessels and tissues of the skin. The resulting intense inflammatory response is most severe in the non-pigmented skin. In severe cases of PA poisoning, acute liver failure and death may result before signs of photosensitization have time to develop. Secondary photosensitization is also caused by a variety of plant toxins other than pyrrolizidine alkaloids.

## **Biliary occlusive photosensitization**

Photosensitivity in livestock has been attributed to ingestion of various plant species other than those containing primary photosensitizing compounds or those causing liver disease as a result of pyrrolizidine alkaloids. The principal toxin(s) responsible for photosensitization in this diverse group of plants are not always known. Some of these photosensitizing plants, saponins, that cause inflammation and obstruction of the biliary system.

When bile cannot be excreted normally by the liver, photosensitizing compounds will accumulate in the animal's bloodstream and will result in photosensitization. A prime example of this is facial eczema, a disease of sheep principally in New Zealand and Australia. It is caused by the mycotoxin sporodesmin produced by the fungus *Pithomyces chartarum* growing on wet rye grass pastures. Sheep and cattle eating the affected rye grass excrete the sporodesmin in the bile, which causes a cholangitis and biliary occlusion. This in turn results in the accumulation of phylloerythrin and subsequently photosensitization.

**Clinical findings**

- Erythema
- Edema,
- Subsequent weeping
- Gangrene, and sloughing of large area of the skin
- The lesion distribution is limited to unpigmented areas of skin
- Intense irritation and animal rubs the affected part against hard substance and become lacerated.

**Treatment**

- Immediate removal from direct sunlight
- Prevention of further ingestion of toxic feeds
- Laxatives to evacuate what has been ingested
- Antihistamines may be helpful in early stages to reduce the extent of skin slough
- Antibiotics to prevent secondary bacterial complication