PASTEURELLOSIS:

It is include:

1- Septicemic pasteurellosis of cattle (hemorrhagic septicemia or barbone), commonly associated with infection by \textit{P. multocida} type 1 or B, is the classical disease of southern Asia characterized by a peracute septicemia and a high mortality rate.

2- Pneumonic pasteurellosis of cattle, commonly associated with infection by \textit{Mannheimia} (formerly \textit{Pasteurella}) \textit{haemolytica} biotype A serotype 1, and \textit{P multocida} biotype A, is a common disease in Europe and the western hemisphere.

3- Pasteurellosis of sheep, and goats, usually associated with infection by \textit{M. haemolytica}.

Septicemic Pasteurelosis of Cattle (Hemorrhagic Septicemia, Barbone):

ETIOLOGY:

1- Hemorrhagic septicemia is associated with two specific serotypes of \textit{P. multocida}. The Asian serotype is designated B:2 and the African serotype is E:2

2- occurs in cattle, yaks, camels, and water buffalo.

3- the most susceptible age group is 6 months to 2 years of age.

4- morbidity and case-fatality rates vary between 50\% and 100\%.

5- In endemic areas, adult animals develop a naturally acquired immunity.

6- Outbreaks of the disease are often associated with wet humid weather during the rainy season.
7- the causative organism persists on the tonsillar and nasopharyngeal mucosae of carrier animals.
8- Spread occurs by the ingestion of contaminated foodstuffs.
9- the organism does not survive on pasture for more than 24 hours.

PATHOGENESIS:

1- The portal of entry of infection is thought to be the tonsils.
2- A fulminating septicemia occurs, which is associated with the capsular material of the organism.
3- The effects of the septicemia are most severe in the respiratory tract, heart, and gastrointestinal tract.
4- In cattle and buffalo there is rapid translocation of bacteria from the respiratory tract to the blood, liver, and spleen, suggesting that the bacteria are able to invade via the mucosal epithelial layers.

CLINICAL FINDINGS:

1- acute septicemia clinically characterized by:
   a- a sudden onset of fever (41-42°C, 106-107°F)
   b- profuse salivation
   c- submucosal petechiation
   d- severe depression
   e- death in about 24 hours.
   f- on range lands, animals may be found dead without any clinical signs having been observed.
2- Localization may occur in subcutaneous tissue, resulting in the development of warm, painful swellings about the throat, dewlap, brisket or perineum
3- severe dyspnea may occur if the respiration is obstructed. In the later stages of an outbreak.

CLINICAL PATHOLOGY:

1- Culture and detection of bacteria.
2- Serology.

NECROPSY FINDINGS:

1- generalized petechial hemorrhages, particularly under the serosae.
2- edema of the lungs and lymph nodes.

TREATMENT:

1- sulfadimidine
2- The NSAID flunixin meglumine can ameliorate the inflammatory response to endotoxin, and treatment with flunixin meglumine has been shown to improve outcome in individual animals.

CONTROL:

1- vaccine composed of killed organisms in an adjuvant base containing paraffin and lanolin.
2- Immunity after vaccination appears to be solid for at least 12 months.
PNEUMONIC PASTEURELLOSIS OF CATTLE (SHIPPING FEVER PNEUMONIA):

is an entity within the bovine respiratory disease complex, characterized clinically by acute bronchopneumonia with toxemia and pathologically by lobar, anteroventrally distributed, exudative pneumonia in which fibrin is usually a prominent part of the exudate and fibrinous pleuritis is common

ETIOLOGY:

1- *Mannheimia (Pasteurella) haemolytica* biotype A serotype 1.
2- Eleven serotypes have been demonstrated.
3- Pneumonic pasteurellosis is a common disease of young growing cattle.
4- The morbidity may reach 35%, the case fatality rate may range from 5-10%.
5- occurs most commonly in young growing cattle from 6 months to 2 years of age.
6- Transmission occurs by the inhalation of infected drop lets coughed up or exhaled by infected animals.

PATHOGENESIS:

1- Exposure of healthy cattle to stressors such as viral infection, change in management practices and environmental changes leads to an explosive growth and selective colonization by *M. haemolytica* A 1 in the upper respiratory tract.
2- When the large numbers of organisms enter and colonize the lung they interact with alveolar macrophages with decreasing clearance mechanism.
3- Four virulence factors have been associated with M. haemolytica: Fimbriae, A polysaccharide capsule, Endotoxin (lipopolysaccharide) and Leukotoxin.

CLINICAL FINDINGS:

1- In the feedlot, the disease usually occurs within 10-14 days after the animals have been stressed.
2- Animals found dead without any previous warning signs may be the first indication of an outbreak
3- Affected cattle are depressed and their respirations are rapid and shallow.
4- There may be a weak protective cough, which becomes more pronounced and frequent if they are urged to walk.
5- Those that have been ill for a few days will appear gaunt because of anorexia.
6- A mucopurulent nasal discharge, a crusty nose, and an ocular discharge are common.
7- Outbreaks of the disease in feedlots may last for 2-3 weeks.
8- fever of 40-41 ºC.
9- In the early stages there are loud breath sounds audible over the anterior and ventral parts of the lungs.
10- As the disease progresses these breath sounds become louder and extend over a greater area; crackles become audible, followed by wheezes in a few days, especially in chronic cases.
11- The course of the disease is only 2-4 days.

NECROPSY FINDINGS

1- There is marked pulmonary consolidation, usually involving at least the anteroventral third of the lungs.
2. A catarrhal bronchitis and bronchiolitis, and a fibrinous pleuritis are usually present and may be accompanied by a fibrinous pericarditis.

3. The lung is firm and the cut surface usually reveals an irregular, variegated pattern of red, white, and gray tissue due to hemorrhage, necrosis, and consolidation.

Differential diagnosis:

1. Viral interstitial pneumonia
2. Lungworm pneumonia
3. Epidemic acute interstitial pneumonia (fog fever)
4. Infectious bovine rhinotracheitis
5. Contagious bovine pleuropneumonia

TREATMENT:

1. Antimicrobial therapy:
   a. Oxytetracycline, trimethoprim-sulfadoxine, the sulfonamides, and penicillin, Florfenicol.
   b. Tilmicosin single subcutaneous injection at 10 mg/kg BW
   c. Enrofloxacin 2.5-5.0 mg/kg BW subcutaneously daily for 3-5 days
   d. Ceftiofur 4.4-6.6 mg/kg administered subcutaneously.

2. Anti-inflammatory agents
   A. Corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs)

3. Expectorants
TETANUS:

ETIOLOGY:

1- An exotoxin, tetanospasmin,
2- is produced by *Clostridium tetani* growing under anaerobic conditions.
3- The organism forms spores that can persist in soil for many years.
4- The spores are resistant to many standard disinfection procedures, including steam heat at 100°C (212°F) for 20 minutes
5- but can be destroyed by heating at 115°C (239°F) for 20 minutes.
6- most common in closely settled areas under intensive cultivation.
7- It occurs in all farm animals, mainly as individual, sporadic cases.
8- case fatality rate is over 80% in young ruminants, but the recovery rate is high in adult cattle.
9- *C. tetani* organisms are commonly present in the feces of animals, especially horses.
10- The portal of entry is usually through deep puncture wounds

PATHOGENESIS:

1- The tetanus bacilli remain localized at their site of introduction and do not invade surrounding tissues.
2- They proliferate and produce tetanolysin and tetanospasmin in a lowering of the local tissue oxygen tension.
3- Tetanolysin promotes local tissue necrosis.
4- Tetanospasmin diffuses to the systemic circulation, is bound to motor end-plates and travels up peripheral nerve trunks.
5- Cause disinhibition of gamma motor neurons result in a state of constant muscular spasticity.
6- Death occurs by asphyxiatiion due to fixation of the muscles of respiration.

CLINICAL FINDINGS:

1- The incubation period varies between 3 days and 4 weeks
2- Initially, there is an increase in muscle stiffness, accompanied by muscle tremor.
3- There is trismus with restriction of jaw movements.
4- Prolapso of the third eyelid.
5- Stiffness of the hind limbs causing an unsteady, straddling gait.
6- The tail is held out stiffly, especially when backing or turning.
7- Hyperesthesia with exaggerated responses to normal stimuli.
8- Falling occurs with the limbs still in a state of tetany and the animal can cause itself severe injury.

DIFFERENTIAL DIAGNOSIS:

1- All species:
   a- Strychnine poisoning
   b- Meningitis
2- Horses
   a- Hypocalcemic tetany (eclampsia)
   b- Acute laminitis
   c- Hyperkalemic periodic paralysis
   d- Myositis, particularly after injection in the cervical region
3- Ruminants
a- Hypomagnesemia - cows, sheep and calves
b- White muscle disease
c- Polioencephalomalacia
d- Enterotoxemia

TREATMENT:

1- The main principles in the treatment of tetanus are to:
   a- Eliminate the causative bacteria by the parenteral administration of penicillin in large doses.
   b- Neutralize residual toxin using Tetanus antitoxin.
   c- Control muscle spasms until the toxin is eliminated or destroyed by using Chlorpromazine (0.4-0.8 mg/kg body weight (BW) intravenously, 1.0 mg/kg BW intramuscularly, three or four times daily).
   d- Maintain hydration and nutrition.
   e- Provide supportive treatment.

BLACKLEG

ETIOLOGY

1- True blackleg, the clostridial myositis of skeletal muscles.
2- is associated with Clostridium chauvoei.
3- a Gram-positive, spore- forming, rod-shaped bacterium.
4- The spores are highly resistant to environmental changes and disinfectants and persist in soil for many years.
5- The disease is enzootic in particular areas, especially when they are subject to flooding
6- The case fatality rate in blackleg approaches 100%.
7- Source of infection is a soil
8- transmission through Infection of skin wounds at shearing and docking and of the navel at birth
9- Typical blackleg of cattle has a seasonal incidence, with most cases occurring in the warm months of the year.

PATHOGENESIS:

1- Trauma is inciting factor.
2- Toxin formed by the organism produces a severe necrotizing myositis locally in skeletal muscles
3- systemic toxemia that is usually fatal.
4- In cattle and sheep atypical outbreaks of sudden death occur in which the lethal lesion is a clostridial cardiac myositis

CLINICAL FINDINGS:

A- Cattle
1- If the animal is observed before death there is severe lameness
2- pronounced swelling of the upper part of the affected leg.
3- On closer examination the animal will be found to be very depressed
4- complete anorexia and ruminal stasis,
5- high temperature (41°C, 106°F)
6- pulse rate (100- 20/min).
7- Pyrexia is not present in all cases
8- In the early stages the swelling is hot and painful to the touch
9- but soon becomes cold and painless,
10- edema and emphysema can be felt.
11- The skin is discolored and soon becomes dry and cracked.

B- Sheep
1- When blackleg lesions occur in the limb musculature in sheep
2- there is a stiff gait
3- disinclined to move because of severe lameness in one limb
4- The lameness may be severe enough to prevent walking in some animals but be only moderate in others.

ECROPSY FINDINGS:

1- Cattle found dead of blackleg are often in a characteristic position; lying on the side with the affected hind limb stuck out stiffly.
2- Incision of the affected muscle mass reveals dark red to black, swollen tissue with a rancid odor and thin, sanguineous fluid containing bubbles of gas.

DIFFERENTIAL DIAGNOSIS:

1- Malignant edema.
2- Anthrax
3- Lightning strike
4- Bacillary hemoglobinuria
5- Other causes of sudden unexpected death.

TREATMENT

1- Penicillin Large doses (40 000 IU/kg BW) should be administered, commencing with crystalline penicillin intravenously and followed by longer-acting preparations
2- surgical debridement of the lesion