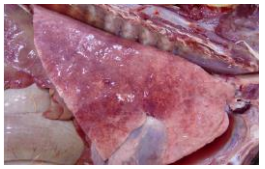

RESPIRATORY SYSTEM L - 4

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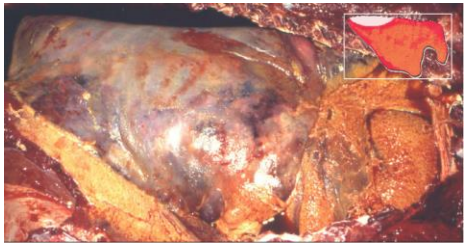
1

ASPIRATION PNEUMONIA



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ASPIRATION PNEUMONIA

(Foreign-body pneumonia, Inhalation pneumonia, Gangrenous pneumonia)

- Aspiration pneumonia is characterized by pulmonary necrosis due to inhalation of foreign material.

Aetiology and Epidemiology

- Faulty **administration** of medicines
- **Drenching** — animal's tongue is drawn out, head is held high, or when the animal is coughing or bellowing.
- In sheep, poor **dipping** technique may cause aspiration of fluid.
- **Inhalation** of irritant gases or smoke is an infrequent cause.
- Aspiration of vomitus
- **Disturbances** to **swallow,**

anesthetized or insensible animals,
acute pharyngitis,
cleft palate,
abscesses or tumours in pharynx,
vagal paralysis,
oesophageal diverticulum,
megaoesophagus or encephalitis,

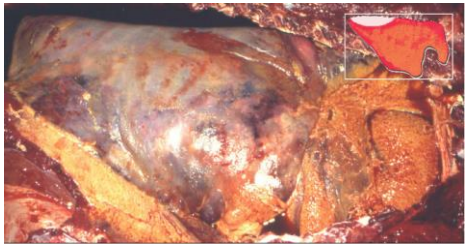
3

Aspiration pneumonia / Acute bronchopneumonia

- **Cranio-ventral** consolidation.
- Monogastric animals die more acutely (shock) than polygastric animals.
- Aspiration of gastric contents or drugs typically results in a **bronchopneumonia** and not always is symmetrical, particularly if aspiration occurs during recumbency or surgery.
- The lesions, depending on severity and composition of aspirated material, vary from **suppurative** to **fibrinous** to **gangrenous**.
- Sometimes, histology is required to confirm food particles in the lung.

4

ASPIRATION PNEUMONIA



Aspiration pneumonia / Horse

- These lungs are from a horse that died 24 hours after a "milk-shake" was given by the owner prior to a race.
- **Note:** large cranioventral consolidation involving **60-70%** of the pulmonary parenchyma. A large portion of the cranial lobes are covered with a thick layer of fibrin. The entire lungs were also edematous hence the rib imprints on the pleural surface.

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ASPIRATION PNEUMONIA

PATHO-PHYSIOLOGY

- History
- Horses may develop **fever of 104-105°F** (40-40.5°C)
- Pyrexia is also seen in cats, dogs, and **less commonly in cattle.**
- The **pulse is accelerated, and respiration is rapid and labored.**
- **Sweetish, fetid breath** characteristic of gangrene.
- **purulent nasal discharge** that sometimes is tinged reddish brown or green.
- May be oil droplets (aspirated contents), can be seen in the nasal discharge or expectorated material.
- On auscultation, fluid sounds
- In cows - **toxemia** is usually **fatal within 1-2 days.**
- **Mortality is high** in all species.
- Recovered animals often develop **pulmonary abscesses**.

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ASPIRATION PNEUMONIA

LESIONS:

- The pneumonia — **anterio-ventral** – unilateral or bilateral
- Marked congestion and interlobular **oedema**.
- **Bronchi** are hyperemic and full of **froth**.
- The pneumonic areas – cone-shaped with the base toward the pleura.
- **Suppuration and necrosis** ---- foci – soft or liquefied, reddish brown, and foul smelling.
- There usually is an acute **fibrinous pleuritis**, often with pleural exudate.

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HAEMORRHAGIC SEPTICAEMIA

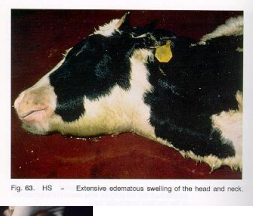

Fig. 63. HS - Extensive edematous swelling of the head and neck.

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HAEMORRHAGIC SEPTICAEMIA

Acute, highly fatal septicaemic disease of cattle and buffaloes caused by certain serotypes of ***Pasteurella multocida multocida*** characterized by oedema in the head-throat-brisket region.

AETIOLOGY AND EPIDEMIOLOGY

- The disease is caused by **two** serotypes B_2 and E_2, type **B** -- in **buffalo**, while type **E** -- in **African cattle**
- HS and **pneumonic pasteurellosis** are two different entities
- The lipopolysaccharide produced is **endotoxin**

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- Three main antigenic component
  - β-antigen: Type specific **polysaccharide**, adsorbed to red cells in indirect haemagglutination procedure,
  - α-complex: Probably a **polysaccharide protein** complex, closely adherent to the cell wall, immunogenic, probably somewhat labile
  - γ-antigen: **LPS** found in organisms from all variants, **derived from cell wall**, each with one or more antigenic determinants responsible for different **O or somatic** serological varieties

Bovine, submandibular region. There is severe subcutaneous/ fascial edema and multifocal hemorrhages. The parotid gland exhibits edema.

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HAEMORRHAGIC SEPTICAEMIA

- Organism also produces **neuraminidase** and **B2 strain produce hyaluronidase** as well
- Disease occur in Southern and Southeast Asia and middle east as well as in Africa, Australia, Canada, Western Europe, South America
- The disease occur in **monsoon season** (wet season)
- In Europe, the disease is reported more in **Italy**
- Disease occurred in **Japan** in 1923 but no epidemic since 1954
- HS is known to occur occasionally in **American bison**
- Disease in **Africa** is caused by a new capsular type, viz., type **E** which infect cattle more frequently than buffalo
- The disease in **Asia** has emerged as of great economic importance
- **In Sri Lanka** mortality range from **20 to as high as 98 %**.
- **In Thailand, 4000 to 10000 deaths** per year are caused by this disease.

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HAEMORRHAGIC SEPTICAEMIA

- **Buffaloes** are more susceptible than **cattle**
- Disease in **goats** is low
- The **carrier** animals harbor the infection for long time
- Infection occur by **direct contact**
- The **organism die soon in dry soil**
- **moist conditions and the presence of blood & tissue fluids may prolong the survival for few days**
- In virgin areas, the disease cause very high mortality in all age groups
- It is generally accepted that this disease causes more losses in **young animals** under two years of age than adults
- Incidence of disease is found to be **4-5 times greater in large herds (over 50 animals)**
- Some animals are naturally immuned to the infection and it was assumed that presence of strains of ***P. multocida*** *antigenically related to type* ***B2*** *or other bacteria with common antigenic components may account for this natural immunity*

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HAEMORRHAGIC SEPTICAEMIA

- Majority of animals harbor the organism in their **nasopharynx** (carrier), or **tonsils**
- After exposure to infection, practically all animals develop high level of **antibody** and harbor the organism in their tonsils and act as latent carrier,
- these animals shed virulent organisms intermittently (**active carrier**)
- This change from latent to active state (when shed virulent organism) is stress related

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HAEMORRHAGIC SEPTICAEMIA

PATHOPHYSIOLOGY

- Infection occur by **inhalation or ingestion**
- Initial site of replication is proposed to be **tonsillar** region
- **Endotoxins** (LPS – a cell wall complex) are the main cause of pathological changes and death
- Endotoxins not only are antigenic but also stimulate the production of **IL-1** which is responsible for **malaise** (unease), **temperature** changes and **weakness**
- These endotoxins also triggers **arachidonic acid metabolite** production such as **prostaglandins** and **leukotrienes** those causes vasodilation, hypotension and other circulatory problems
- Endotoxins also activate the **complement pathway** resulting in **platelet aggregation** and release of **pro-coagulants** result in **thrombus** formation
- Being **extracellular** organism, the immunity produced is **humoral** with polymorphonuclear response and little monocytic response

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HAEMORRHAGIC SEPTICAEMIA

CLINICAL COURSE

- The incubation period of the disease is short (30 hours to **2-3 days**)
- The course of disease is also short especially in buffaloes than cattle
- Death occur from **6 to 24 hours** after the first recognized signs.
- The typical clinical syndrome of HS is characterized by three phases
  - The **initial phase** is temperature elevation with inappetance and sometimes salivation
  - **Second phase** is **respiratory distress** with profuse salivation and nasal discharge
  - In the **third phase** **recumbency** sets in leading to terminal septicemia
- Under field conditions, often the initial phase is not observed
- In many cases **submandibular edema** becomes evident and may spread to the brisket region and occasionally down the forelimbs

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HAEMORRHAGIC SEPTICAEMIA

CLINICAL COURSE

- The **pneumonic** syndrome in **young** buffaloes has also been recorded with Asian HS serotype
- The three forms described in the literature are
  - The **septicaemic** form
  - The **respiratory** form and
  - The **cutaneous** or edematous form
- Their identity is based on which clinical signs are dominant

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HAEMORRHAGIC SEPTICAEMIA

AT POSTMORTEM

- On opening the first lesion is the subcutaneous infiltration of a yellow **sero-gelatinous** (or rarely sero-sanguineous) fluid in the submandibular, throat, pharyngeal and brisket region.
- There may be additional fluid in the pericardial sac and the pleural cavity.
- Petechial and **ecchymotic haemorrhages** are seen to a varying degree on the epicardium of the heart, particularly involving the auricular region and base of the ventricles.
- In some cases, there is marked serofibrinous pericarditis, pleuritis and peritonitis with adhesions.
- The principal change in the **lungs** is **congestion** but there may also be some consolidation with thickening of the interlobular septa.

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HAEMORRHAGIC SEPTICAEMIA

AT POSTMORTEM

- The **lymph nodes** are generally enlarged and when cut reveal red to dark surfaces with many small haemorrhages
- Spleen is usually normal or mildly congested with varying degrees of petechial or ecchymotic haemorrhages
- Large haemorrhagic patches may be seen on the **abomasum**

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HAEMORRHAGIC SEPTICAEMIA

AT POSTMORTEM

- If animal die within 24 to 36 hours,
  - only **petechial haemorrhages** on the **heart** and
  - generalized **congestion** of the **lungs** will all be seen.
- In cases survived for more than 72 hours,
  - petechial and ecchymotic **haemorrhages**,
  - signs of **pericarditis,** generalized congestion of **lung** with consolidation and lobulation prominent.

MORBIDITY AND MORTALITY

- **Husbandry**, **weather** and **immunity** affect morbidity.
- In endemic areas, from **10 to 50 %** of the cattle or buffalo populations acquire solid immunity through exposure or subclinical infection.
- Close herding and wetness predispose to an increased morbidity.
- Most animals that develop clinical signs die – case fatality rate is high.

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HAEMORRHAGIC SEPTICAEMIA

DIAGNOSIS

- History, clinical signs and gross pathological lesions
- **Isolation** of the organism from **blood**, **bone marrow**
- Serological typing
- **Animal inoculation tests** (mouse)

DIFFERENTIAL DIAGNOSIS

- Must be differentiated from causes of sudden death including
  - Snake bite,
  - Lightening,
  - Anthrax,
  - Rinderpest
  - Blackleg

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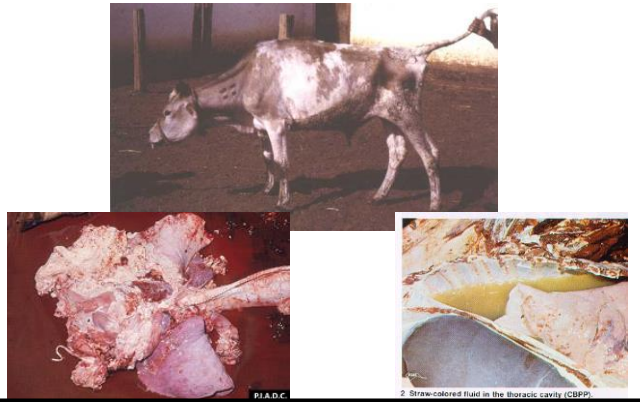
CONTAGIOUS BOVINE PLEUROPNEUMONIA



2 Straw-colored fluid in the thoracic cavity (CBPP).

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Contagious bovine pleuropneumonia (CBPP) is a **highly infectious acute**, **subacute**, or **chronic** disease, primarily of cattle, affecting the lungs and occasionally the joints, caused by *Mycoplasma mycoides mycoides*.

Etiology

- Contagious bovine pleuropneumonia is caused by *M. mycoides mycoides* small-colony type (**SC type**).
- *M. mycoides mycoides* large-colony type is pathogenic for sheep and goats but not for cattle.
- **Does not survive** in meat or meat products and does not survive outside the animal in nature for more than a few days.
- Many of the routinely used disinfectants effectively inactivate the organism.

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Host Range

- **Cattle** (*Bos taurus), zebu (Bos indicus) and water **buffalo** (Bubalus bubalis) are susceptible*
- Wild bovids and camels are resistant

Geographic Distribution

- CBPP is endemic in most of **Africa**.
- It is a problem in parts of Asia --- **India, Pakistan** and **China**.
- Periodically, CBPP occurs in Europe, and outbreaks within the last decade have occurred in **Spain**, **Portugal**, and **Italy**.
- **eradicated** from the **United States** in the 19th century.

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Transmission

- Spread by **inhalation** of droplets from an infected animal.
- Relatively **close contact** is required
- It is widely believed that recovered animals harbor infectious organisms within a pulmonary sequestrum, may become active shedders when stressed.
- Transmission up to **several kilometers** has been suspected under favorable climatic conditions

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Incubation Period

- Generally quite **long**.
- It has been shown that healthy animals placed in a CBPP-infected herd may begin showing signs of disease 20 to 123 days later
- During an outbreak of natural disease, only **33**% of animals present symptoms, 46% are infected but have no symptoms (subclinical forms) and 21% seems to be resistant

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Clinical Signs

- **Depression**, **in-appetence** and **fever**
- **Coughing** may be the next sign
- Evidence of thoracic pain and an **increased respiratory rat**e.
- As pneumonia progresses, animals are inclined to stand with **elbows abducted**.
- Auscultation of the lungs reveals variety of sounds, depending
  - Crepitating rales, and pleuritic friction rubs are all possible.
- Percussion over affected areas reveals **dullness**.
- When pulmonary involvement is extensive and severe --- labored respiration, sometimes --open-mouth breathing.

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

- reluctant to move and stand stiffly with arched back.
- Getting up and down may cause discomfort.
- Large joints may be distended and warm on palpation.

Gross Lesions

- The gross pathologic features of CBPP are quite characteristic.
- extensive and marked **inflammation** of the **lung** and associated **pleurae**.
- In severe cases, abundant fluid in the thoracic cavity (**upto 30 liters**).

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Gross Lesions

- The affected pulmonary parenchyma is odorless and often has all stages of lesions with both **acute** and **chronic inflammatory** changes adjacent to one another.
- The predominant gross change is consolidation, or thickening, of individual lobules, widened **interlobular septa**, resulting **marbled** appearance.
- **Interlobular septa** become distended first by **edema**, then by **fibrin**, and finally by **fibrosis**.
- The **pleura** may be very **thickened**, **fibrosed**, often resulting in adhesions between parietal and visceral pleurae.

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Gross Lesions

- within an affected lung will be found a sequestrum – area with coagulative necrosis and sealed off.
- Such sequestra may even be found in recovered animals.
- The organism can survive within these sequestra for months or possibly longer.

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Morbidity and Mortality

- It is **not** thought to be a highly contagious disease.
- With increased **confinement** of animals, morbidity rises.
- The mortality -- **10 to 70 %**

Diagnosis

- Clinical diagnosis of CBPP is difficult.
- At postmortem the gross lesions of CBPP are somewhat distinct.
- Often there is an extensive deposition of fibrin and a large quantity of straw-colored fluid in the thoracic cavity with a prominent marbling of pulmonary parenchyma.
- Generally, all stages of pathologic changes, from acute through to chronic, are present in one animal.
- In some chronic cases the nodules of inflammation may not be readily apparent from the pleural surface but can be palpated within the parenchyma.
- Unlike many other pneumonias, CBPP is often **unilateral**.

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CONTAGIOUS BOVINE PLEUROPNEUMONIA

Specimens for Laboratory

- From a live animal, nasal swabs, transtracheal washes, or pleural fluid obtained by thoracic puncture all provide good samples for isolation attempts.
- From a dead animal that has had severe clinical disease, the best specimens to submit are affected **lung**, swabs of major **bronchi, tracheo-bronchial** or **mediastinal lymph nodes**, and **joint fluid** from those animals with arthritis.

Laboratory Diagnosis

- A definitive diagnosis is made by isolating and identifying the organism.
- Serology is helpful in the diagnosis of CBPP.

Differential Diagnosis

- bovine pasteurellosis: bovine pasteurellosis spread much more rapidly and consequently the epidemiologic picture may be distinct.

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WARNING

Besides lung cancer and cardiovascular diseases….
--there is also unequivocal scientific association between
Cigarette smoking
and
pulmonary emphysema

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