HISTOLOGY AND FUNCTIONS OF THE RESPIRATORY TRACT

The respiratory tract is arbitrarily divided into three continuous systems

- **Conducting system:** The conducting system includes the nasal cavity, sinuses, larynx, trachea and bronchi. The mucosa of the conducting system is lined primarily by ciliated epithelium and goblet cells.

- **Transitional system:** The transitional system is formed by the bronchioles that are lined by a specialized mucosa containing several types of ciliated and secretory cells such as Clara cells. Unlike the conducting system, the normal bronchiolar mucosa contains no goblet cells.

- **Exchange system:** This system is composed of the alveoli that are lined externally by epithelial cells called pneumonocytes. The type I (membranous) pneumonocytes are thin cells and together with the capillary endothelium and basement membrane constitute the air-blood barrier. Type II pneumonocytes are cuboidal and produce surfactant.

In addition to gas exchange, the respiratory system is involved in:

<table>
<thead>
<tr>
<th>Function</th>
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<tbody>
<tr>
<td>Phonation</td>
<td>Temperature regulation</td>
<td>Blood pressure regulation</td>
</tr>
<tr>
<td>Olfaction</td>
<td>Acid-base balance</td>
<td>Detoxification (Clara cells)</td>
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<tr>
<td>Hormone and enzyme synthesis</td>
<td>Uptake of epinephrine, vasoactive amines</td>
<td>Leukotriene metabolism</td>
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</table>

One or more of these functions can be affected as result of respiratory disease

NORMAL FLORA OF THE RESPIRATORY TRACT

The respiratory tract has a normal bacterial flora as any other body system that is in contact with the external environment. Various species of bacteria will be grown if a sterile swab is passed deep into the nasal cavity of any healthy animal and it is sent for microbiologic culture. These
organisms constitute the normal flora of the respiratory tract. The nasal flora is restricted to the most proximal region of the conducting system (nasal cavity, the pharynx, larynx and trachea). The distal portions of the respiratory tract, bronchioles and alveoli are considered to be essentially sterile.

The types of bacteria present in the nasal flora vary considerably among animal species. Some types of bacteria in the nasal flora are the same pathogens associated with respiratory infections. For instance, *Mannheimia (Pasteurella) haemolytica* is part of the bovine nasal flora, yet this bacterium causes a devastating disease in cattle known as Shipping Fever.

Experimental studies have demonstrated that microorganisms from the nasal flora are continuously carried into the lungs via the tracheal air. In spite of this constant bacterial bombardment from contaminated air, normal lungs remain sterile due to its remarkably effective defense mechanisms.

**DEFENSE MECHANISMS OF THE RESPIRATORY TRACT**

<table>
<thead>
<tr>
<th>The respiratory tract is continuously exposed by inhaled air to:</th>
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<tbody>
<tr>
<td>Microorganisms (virus, bacteria, fungi)</td>
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<tr>
<td>Particles (dust, fibers)</td>
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<tr>
<td>Gases (SO₂, NO₂, H₂S, ozone)</td>
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<td>Vapors (formaldehyde).</td>
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</table>

Under normal conditions, toxicants are detoxified; toxins are neutralized; particles are trapped and removed; and microorganisms are trapped, destroyed and removed from the respiratory tract.

**Defense mechanisms:**

Overall, the most important defense mechanisms against inhaled particles and particularly for bacteria are:

- **Air Filtration** (conchae, bronchial bifurcation, turbulences, attachment to mucus, coughing)

- **Mucociliary clearance** for the conducting system (nasal, tracheal and bronchial mucosa)

- **Phagocytosis** (alveolar macrophages for the exchange system alveoli)

- **Innate and acquired immunity** (Antibodies and cell-mediated immunity)

Defense mechanisms are rather effective destroying inhaled bacteria. If these defense mechanisms fail, inhaled bacteria can colonize the lung and cause bacterial pneumonia. There are many factors known to impair defense mechanisms. The most common are viral infections, immunodeficiency, stress, dehydration, pulmonary edema, uremia, ammonia, etc. Note in this graph the rapid elimination of inhaled bacteria given by aerosol to a normal animal (dotted line). In contrast note in viral infected animals (squared line) that the number of inhaled bacteria increases with time. This experimental model has shown that for instance, PI-3, IBR, BRSV viruses inhibit the pulmonary defense mechanisms of calves predisposing these animals to pulmonary bacterial infections. Stress and other factors cause similar impairment of the defense system in cats, dogs, and farm animals.
POSTMORTEM EXAMINATION OF THE RESPIRATORY TRACT

Nasal Cavity: Make transverse sections of the nasal cavity. Check conchae, meatuses and sinuses and investigate the presence of exudates, parasites, erosions, ulcers, edematous fluid, nodules, polyps or tumors.

Larynx, Trachea and Bronchi: Open all these structures with scissors and check the mucosa. Investigate the presence of exudates, erosions, ulcers, parasites, foam, nodules or tumors.

Thoracic Cavity: Check negative pressure by puncturing the diaphragm and observing the retraction of the diaphragmatic muscle. Failure to retract suggests pneumothorax, pulmonary inflammation, edema or emphysema. Check for fluids or exudates as well as for fractured ribs.

Lungs: Check if the lungs collapse when the thorax is opened. Uncollapased lung in a fresh animal suggest inflammation or emphysema. Check for rib imprints on the pleural surface. Palpate the lungs for texture (normal vs. firm, hard, elastic or nodular). Also examine the lungs on cut surface and compress the parenchyma looking for exudates or parasites. Record the severity and extension of lung involvement expressed as percentage of lung involvement.

PATHOLOGY OF THE CONDUCTING SYSTEM

NASAL CAVITY AND SINUSES

Anatomy of the Conchae (turbinates):

1. Nasal (dorsal)
2. Maxillary (ventral)
3. Ethmoidal
Histology of Nasal cavity and Sinuses:

- Pseudostratified ciliated epithelium with abundant goblet (mucus) +++
- Olfactory epithelium ++
- Squamous epithelium +

The nasal cavity has abundant submucosal mucus glands and blood vessels (highly vascularized tissue).

Meatuses are the narrow spaces between conchae where air circulates.

Circulatory Disturbances of the Nasal Cavity: Congestion and Hyperemia:

The nasal mucosa is highly vascularized and therefore congestion and hyperemia are commonly seen in domestic animals. These changes are most frequently seen in bloated ruminants, toxemia, acute rhinitis, trauma, agonal (circulatory failure), inhalation of irritant gases, etc.

Nasal hemorrhage (Epistaxis and Hemoptysis):

Epistaxis (nose bleeding) and hemoptysis (blood in mouth, saliva or sputum) are common findings in all species. Unlike intestine, the color of the blood does not change between proximal (nasal) and distal (lung) hemorrhage. Epistaxis or hemoptysis do not necessary imply that the hemorrhage has occurred in the nasal or oral mucosa since both conditions can also be caused by pulmonary hemorrhage.

Epistaxis is a common problem in all animal species. It can be the result of trauma, foreign body, nasal neoplasia, pulmonary hemorrhage (aneurism).

Epistaxis is commonly seen in horses with:

- Exercise induced pulmonary hemorrhage
- Guttural pouch mycosis
- Ethmoidal hematoma
- Nasal cysts

In all domestic species, nasal hemorrhage is a frequent indicator of trauma or neoplasia. In cattle hemoptysis-epistaxis is also associated with rupture pulmonary vessels (aneurism). Careful examination is required to localize the source of blood.

INJURY AND REPAIR OF THE NASAL CAVITY AND SINUSES

Injury and repair

The nasal mucosa has a remarkable capacity to repair following injury and necrosis. The type of injury and repair seen in the nasal cavity, trachea and bronchi are all morphologically similar since all these structures are lined by the same type of epithelium (ciliated with goblet cells).

The lesions and stages of repair are also similar in viral infections, toxic, or traumatic injury.
Pathogenesis of viral rhinitis / tracheitis / bronchitis

Virus in air or saliva ~> virus replication in epithelial cells ~> cell degeneration ~> loss cellular attachment ~> cell exfoliation ~> ulceration ~> exudation (fluid and cellular) ~> cell mitosis ~> repair. Complete repair occurs in approximately 14 days.

Note: Although respiratory viral infections are generally transient, impairment of defense mechanism can result in severe bacterial rhinitis and pneumonia.

Chronic injury to the nasal (also tracheal and bronchial) epithelium results in **goblet cell hyperplasia** with abundant production of mucus, or squamous metaplasia in which ciliary epithelium is replaced by squamous epithelium. Chronic changes cause also fibrosis and extensive loss of ciliary function.

**INFLAMMATION OF THE NASAL CAVITY / SINUSES (RHINITIS AND SINUSITIS)**

Rhinitis and Sinusitis are clinically characterized by nasal discharge (unilateral or bilateral)

According to exudate rhinitis and sinusitis can be classified as;

- **Serous**: Mild irritants i.e., low levels of ammonia or chlorine
- **Catarrhal**: Mucous i.e., viral infections
- **Purulent**: Pyogenic bacteria i.e., *Streptococcus equi*
- **Fibrinous (Diphtheritic)**: Bacterial + potent bacterial toxins
- **Granulomatous**: Foreign body, allergy, fungi

Viral rhinitis is commonly seen in domestic animals but infections generally are self-limiting unless complicated with bacteria or mycoplasmas. Gross lesions in viral rhinitis are often minimal with only noticeable hyperemia of mucosa unless complicated with a bacterial infection. Laboratory tests such as cultures, virology are often required for etiological diagnosis.

<table>
<thead>
<tr>
<th>Examples of viral rhinitis in domestic animals:</th>
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<tbody>
<tr>
<td>Bovine Rhinotracheitis (IBR/BHV-1)</td>
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<tr>
<td>Feline Rhinotracheitis (FHV-1) and Feline Calicivirus</td>
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</table>

**Examples of Rhinitis in Domestic Animals**
**Horses:**

**Strangles** (*Streptococcus equi*) is an important equine disease that frequently occurs as an outbreak with high morbidity (90%) but low mortality (5% > foals). Lesions: Typically purulent (suppurative) rhinitis and regional lymphadenitis with formation of abscesses. Some horses develop purpura hemorrhagica (vasculitis). Bacterial metastasis to internal organs such as spleen and liver referred to as "bastard strangles" occurs sporadically. Involvement of retropharyngeal lymph nodes can compress laryngeal nerves and cause secondary laryngeal hemiplegia. **Strangles is only seen sporadically in North America.**

**Pigs:**

**Inclusion Body Rhinitis** is a viral disease of piglets caused by a Herpes virus (Cytomegalovirus). In piglets less than 2 weeks old, the infection generally goes unnoticed causing only transient acute rhinitis and epiphora (overflow of tears). Gross lesions are not striking. It only causes nasal hyperemia. Microscopically there is epithelial necrosis, inflammation but most remarkable is the enlargement of epithelial cells (megalocytosis) with large intranuclear inclusion bodies. There is no mortality except in immune-suppressed pigs that can develop a disseminated and often fatal infection.

**Atrophic Rhinitis** is an important and widely distributed disease of pigs. **The etiology is still controversial:** Bacterial? (*Pasteurella multocida*, *Bordetella bronchiseptica*, *Mycoplasma spp.*), Viral? (Inclusion body rhinitis), nutritional (Vitamin D, Ca, P), Genetic?, Environmental? (Humidity, temperature, etc.). Current literature strongly suggests a combined (co-infection) with toxigenic strains of *P. multocida* and *B. bronchiseptica*. The effect of atrophic rhinitis on growth rate and pneumonia is also controversial. Postmortem examination is best achieved by making a transverse cross section of the snout between the 1<sup>st</sup> and 2<sup>nd</sup> premolar teeth. Gross Lesions: Various degrees facial deformity and conchal atrophy (progressive) resulting in widening of the nasal meatuses. Exudate is generally absent but, in advance cases, facial deformity (deviation of the snout) is evident on clinical examination.

**Dogs:**

Viruses involved in upper respiratory infection of dogs include **Canine Distemper** (*Morbillivirus*), **Canine Adenovirus** (CAV-2), **Canine Parainfluenza virus** (CPI-1). CAV-2 and CPI-1 are generally acute and transient a can cause a highly contagious condition referred to as **Kennel Cough**. **Canine Flu (canine influenza)** is a novel disease recently reported in dogs in the United States that has acute and severe respiratory signs similar to those seen with kennel cough. These viral infections can also predispose dogs to secondary **bacterial rhinitis** (*Bordetella bronchiseptica*, *Escherichia coli*, *Streptococci*, *Staphylococci*). Lesions: In pure viral infections changes are minimal except for hyperemia; when viral infections get complicated with bacterial infections; mild hyperemic lesions turn into mucopurulent rhinitis and/or chronic sinusitis. In Canine Distemper, non-suppurative encephalitis and enteritis have more clinical significance.

**Cats**

**Feline Rhinotracheitis** (FHV-1) and **Feline Calicivirus**. As in other species, respiratory viral infections in cats are generally acute and transient, but can also predispose secondary bacterial rhinitis (*Bordetella bronchiseptica*, *Pasteurella multocida* and *Pseudomonas aeruginosa*). Lesions: Mucopurulent rhinitis chronic sinusitis. **Chlamydial** infections (*Chlamydophila psittaci*) have also been associated to rhinitis and conjunctivitis in cats. Rhinitis and sinusitis in cats are frequently associated with the fungus **Cryptococcus neoformans**.
SINUSITIS

The types of injury and host response in the paranasal sinuses are similar to those in the nasal cavity. According to exudate, sinusitis are most commonly serous, catarrhal, purulent, or granulamatous. Unlike nasal cavity, paranasal sinuses have poor drainage and exudate tends to accumulate and cause "sinus mucocele" or "sinus empyema."

In sheep parasitic rhinitis/sinusitis is commonly caused by *Oestrus ovis*.

Allergic Rhinitis is sporadically seen in dogs, cats, cattle and horses.

TUMORS OF NASAL CAVITY AND SINUSES

**Epidemiology:** The incidence of nasal tumors in animals is overall low and most commonly seen in *dogs and cats* and to a lesser extent horses. The concept that long-nose breeds are most frequently affected has been recently challenged. In ruminants, nasal carcinoma may be enzootic (high prevalence) in some geographical areas.

**Pathology and Clinical Signs:** Nasal tumors could arise from epithelial or mesenchymal cells. Epithelial tumors of the nasal passages include adenomas (benign and rare) and carcinomas (malignant and common). Tumors of stromal origin include fibrosarcoma, osteosarcoma and chondrosarcoma. Malignant tumors often metastasize. Nasal tumors often bleed causing epistaxis or become infected causing a nasal discharge (exudate) that may be mistaken for a simple bacterial or mycotic rhinitis. Some locally invasive tumors may cause severe damage to nerves and brain and cause a variety of neurological signs.

**Endemic Nasal Carcinoma (Endemic Ethmoidal Tumor)** is a retroviral-induced neoplasia of sheep, goats and cattle and in some endemic regions the incidence may be considerable. Clinical signs are nonspecific (hemorrhage) and the tumor often becomes infected causing mucopurulent nasal discharge. Diagnosis: Biopsies are required for confirmation of nasal tumors. In general, 80% of nasal tumors are malignant. Clinical signs are variable but in severe cases, there may be craniofacial deformation, exophthalmia, and metastasis to brain causing nervous signs.

**GUTTURAL POUCHES**

**General:**
There are some important anatomical differences in domestic animals (i.e., syrinx in birds, guttural pouches in horses).

**Guttural pouches** are ventral diverticula of Eustachian tubes in horses (pharynx to middle ear).

**Guttural Pouch Empyema:** accumulation of pus in the equine guttural pouches is a common and important disease. It is caused by pyogenic bacteria such as *Streptococci spp.* Lesions: accumulation of purulent exudates in guttural pouches.

**Guttural pouch mycosis** (*Aspergillus spp.*). It is an important and occasionally fatal disease of horses. Sequel includes erosion of carotid artery, massive nasal bleeding and or cerebral infarcts.
Lesions: Plaques of fibrinonecrotic exudate in guttural pouches.

Laryngeal entrapment. Horses are also susceptible to entrapment of the epiglottis under soft palate. The pathogenesis is unclear but hypoplasia of the larynx has been incriminated.

**LARYNX**

**Necrotic Laryngitis (Calf diphtheria).**
Important secondary infection caused by *Fusobacterium necrophorum* that typically occurs following trauma or viral infection (*i.e.*, IBR). Pathogenesis is similar to oral or nasal necrobacillosis. Lesions: Exuberant plaques of fibrinonecrotic exudate on top of deep ulcers. Exudates can be aspirated and cause aspiration pneumonia, or in severe cases can also result in toxemia or fusobacteremia.

**TRACHEA AND BRONCHI**

General:
The trachea and bronchi are formed by cartilage lined by a mucosal membrane. This membrane is primarily formed by respiratory ciliated epithelium (ciliated pseudostratified + goblet cells and serous glands) with a thin layer of mucus on the surface. The mucus and the cilia constitute the so-called mucociliary escalator. The tracheal and bronchial mucosas have numerous bronchial glands which become enlarged in chronic irritation.

Examples of tracheobronchitis:
- **Canine Infectious Tracheobronchitis (Kennel Cough):** Acute, self-limiting tracheobronchitis (cough) of complex etiology (Canine Adenovirus-2, Canine Parainfluenza virus, *Bordetella bronchiseptica*, Mycoplasmas). Lesions: rarely seen at the postmortem since it is acute and transient. Necrosis, inflammation and repair are the same as for rhinitis.

- **Parasitic Tracheobronchitis (*Oslerus <Filaroides> osleri):** Important parasitic disease of Canidae. LESIONS: typical large parasitic nodules (1 cm) on the mucosal surface of distal trachea and/or proximal bronchi. There is mild inflammatory reaction around the parasite (histology) and minimal or no clinical signs.

**PATHOLOGY OF THE TRANSITIIONAL SYSTEMS (BRONCHIOLES)**

**BRONCHIOLES (Transitional System)**

Unlike bronchi, the walls of the bronchioles do NOT contain cartilage and the mucosa does not normally have goblet cells. The pseudo-stratified epithelium in bronchi gradually flattens and loses their cilia in bronchioles.

Primary diseases of the bronchioles are rare in domestic animals except for Equine Heaves and some parasitic diseases (lungworms) that cause chronic bronchiolitis. In respiratory viral infections and some toxic conditions there is bronchiolar necrosis and inflammation. However, in these conditions bronchioles are affected along with the alveolar walls and other structures of the lung.
"Heaves" also referred as Chronic Obstructive Pulmonary Disease (COPD) or Recurrent Airway Obstruction (RAO) is an important equine disease where the pulmonary lesions are centred in the bronchioles. Mild injury and recurrent inflammation induces goblet cell metaplasia in bronchioles. There are no goblet cells in the healthy bronchiole hence the term metaplasia rather than hyperplasia. Goblet cell metaplasia causes accumulation of mucus in the bronchioles that cannot be cleared by mucociliary movement. Bronchioles plugged with mucus have airflow impairment and eventually leads to alveolar emphysema (discussed under inflation disturbances of the lung. Abnormal respiration in affected horses causes hypertrophy of the abdominal muscles which is clinically referred to as "heave line." Gross pulmonary lesions are not remarkable except perhaps for some degree of emphysema. However, microscopically, there is bronchiolar goblet cell metaplasia and extensive mucus obstruction of small airways (bronchioles). COPD with similar bronchiolar changes are typically found in chronic smokers.

Note: If you are really interested about equine diseases and would like to read more about RAO/COPD click here and enjoy this excellent paper.

LUNGS

General:

- There are notable differences in lung morphology among animal species; i.e., equine poorly defined pulmonary lobes; bovine and canine well defined lobes.
- Pulmonary lobes are classified into: cranial lobes (formerly apical), middle lobes (formerly cardiac/intermediate) and caudal lobes (formerly diaphragmatic).
- Pulmonary lobes are subdivided into lobules by interlobular septa.
- Lobules are prominent in bovines and pigs, poorly defined in horses and man, and absent in dogs and cats.

Alveoli are covered by thin type I pneumonocytes (membranous); interposed occasionally with type II pneumonocytes (granular pneumonocytes). Type II pneumonocytes produce important phospholipids known as pulmonary surfactant. This surfactant prevents alveolar collapse during respiration. Type I (membranous) pneumonocytes are notably slender and particularly susceptible to injury. Necrotic pneumonocytes type I are replaced by type II which eventually differentiate into type I.

Lymphatic vessels are abundant in the lung and are constantly removing fluids from this organ.

Bronchial associated lymphoid tissue (BALT) is involved in lung defenses particularly in local immunity of the lung.

Bronchoalveolar cells: The bronchoalveolar spaces contain numerous free cells that can be easily collected by bronchoalveolar lavage (transtracheal washing). The normal cellular composition is approximately 90% macrophages, 5% lymphocytes, 2% neutrophils and 3% other cells such as eosinophils, basophils and epithelial cells. Bronchoalveolar cells are extremely valuable for the diagnosis of some pulmonary diseases.

The Blood-Air Barrier:
Congenital anomalies of the Lung

Congenital lung anomalies are relatively rare in animals. Most common ones are: lung or bronchial hypoplasia and pulmonary hamartoma.

Abnormal Pigmentations of the Lung

- **Pneumoconiosis** is a general term used to describe pulmonary diseases characterized by deposition of inhaled particles in the lung. The most common particles are such as carbon (anthracosis), silica (silicosis) and asbestos (asbestosis).

- **Anthracosis** is sporadically seen in domestic animals, particularly dogs, exposed to carbon particles suspended in the air. Lungs have focal or confluent areas of black discoloration. It is considered an incidental finding.

**Important**: Pulmonary melanosis is a common incidental pleural finding, particularly in sheep. There is change in the texture of the lung.

CIRCULATORY DISTURBANCES OF THE LUNG

**Hyperemia (active)** is usually seen in acute pulmonary inflammation when the release of inflammatory mediators causes vasodilation and exudation. Hyperemic lungs are heavy with a deep red color. Blood oozes on cut surface.

**Congestion (passive accumulation of blood)** is commonly associated with congestive heart failure (left, uncompensated). It can progress to lung edema and intra-alveolar hemorrhage with erythrophagocytosis ("heart failure cells"). "Wet and heavy lungs" with red patchy discoloration.

**Pulmonary hemorrhages** are commonly seen in postmortem specimens. The most common causes of pulmonary hemorrhages are:

- Heart failure (see congestion).
- Trauma: punctured lungs by fractured ribs or penetrating wound, etc.
- Coagulopathies: dicumarol toxicity, immune-mediated thrombocytopenia, etc.
- Embolisms (Thromboembolism): septicemia, DIC, blood parasites, etc.
- Rupture of vessels: pulmonary aneurysm, erosion of vascular wall by abscesses, etc.
- Exercise Induced Pulmonary Hemorrhages (EIPH) in horses
- **Gross:** Depending on magnitude, hemorrhages vary from petechial to patchy or locally extensive areas of red discoloration. A special form of pulmonary hemorrhage in horses is called "Exercise induced pulmonary hemorrhage. Pulmonary hemorrhage + mucociliary clearance or cough causes epistaxis and hemoptysis.

- **Microscopically:** pulmonary hemorrhage typically exhibit siderophages (macrophages filled with hemosiderin).

"Exercise Induced Pulmonary Hemorrhage" (EIPH) has been well described in race horses. Clinically, affected horses have nose bleeding following exercise. Some horses have a reduced athletic performance. Only exceptional cases result in acute death. Postmortem findings are minimal and characterized by dark-yellow (hemosiderin pigment) discoloration of the dorsocaudal aspects of the lung. Pathogenesis of EIPH is unclear but mechanical stress with rupture of alveolar walls has been proposed. Histopathology: Hemorrhage, siderophages, and moderate fibrosis.

### Pulmonary Thrombosis and Infarcts

**Lung thromboembolisms** are only occasionally seen in domestic animals and generally have little clinical significance.

**Common causes of pulmonary thromboembolisms**

<table>
<thead>
<tr>
<th>Endocarditis (right heart)</th>
<th><em>Dirofilaria immitis</em> in dogs</th>
<th>Vena cava thrombosis in cattle (hepatic abscesses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jugular vein thrombosis (IV injection/catheter)</td>
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**Pulmonary infarcts:** Pulmonary infarcts are rarely seen due to the double circulation of the lungs (pulmonary and bronchial). Generally lung infarcts are non-fatal but can evolve into embolic pneumonia. Lesions: Although rarely seen at necropsy, lung infarcts appear as focal discoloration / hemorrhage generally in margins of the lung.

### PULMONARY EDEMA

Pulmonary edema is a very common and sometimes important pulmonary lesion characterized by accumulation of fluid in interstitium and alveoli. Generally a nonspecific lesion seen in many pulmonary, cardiac and neurological diseases and it is the terminal cause of death for many illnesses.

**Pathogenesis of lung edema**

Normal lung produces fluid (transudate) that is rapidly removed by the lymphatic system. When fluid production exceeds lymphatic removal, pulmonary edema may rapidly follow.

- **Increased hydrostatic pressure (cardiogenic edema)** is commonly seen in animals with left heart failure or with fluid overload (iatrogenic). Left (uncompensated) heart
failure causes first pulmonary congestion and then intra-alveolar hemorrhages that result in the formation of "heart failure cells" in the lung. Lung edema also develops after severe brain injury which is referred to as "Neurogenic lung edema." The edematous fluid in a cardiogenic pulmonary edema usually has low protein content.

- **Increase vascular permeability (permeability lung edema)** is seen when there is injury to the air-blood barrier i.e., toxic gases, inflammation, allergies and pancreatitis. The edematous fluid in a permeability edema has usually high protein content.

- **Obstruction to lymphatic drainage (obstructive lung edema)**, i.e., Neoplasia involving thoracic lymphatic nodes or vessels. Edematous fluid has usually low protein content.

**Gross lesions of lung edema:** Foamy fluid in conducting system. If there is no foam it is not possible to diagnose pulmonary edema as the terminal cause of death. The lungs appear wet, heavy and fail to collapse when the thorax is opened. There is distention of the interstitial septa which causes accentuation of the **lobular pattern**, particularly in cattle and pigs. These two species have well developed pulmonary interlobular septa.

**CAUTION:** an acute pulmonary edema may resemble pneumonia.

**Histopathology of lung edema:** Alveoli are flooded with fluid. In permeability edema the fluid is generally rich in protein (eosinophilic) while in cardiogenic edema the fluid is pale and difficult to see microscopically.

**Distention of the interlobular septa.**

**Lymphatic vessels are notably distended** which reflects the attempts by the lung to clear the excess fluid.

**INFLATION DISTURBANCES (ATELECTASIS AND EMPHYSEMA)**
ATELECTASIS

Atelectasis (Greek \textit{ateles}-incomplete; \textit{ectasis}-expansion) refers to an incomplete expansion of the lung or portion of a lung. Fetal lungs have no air and therefore sink when placed in water.

**Neonatal Atelectasis:**

- Airway obstruction at birth. Aspiration of meconium due to the so-called "valve effect" in which the meconium plug allows the exit but no entrance of air into the lung.
- Lack of pulmonary surfactant (\textit{Respiratory Distress Syndrome / Hyaline membrane disease} > humans > foals, piglets > other species).

**Acquired Atelectasis:**

- **A. Normal alveolus**
- **B. Obstructive atelectasis:** Airway obstruction such as exudate, parasites, food particles.
- **C. Compressive atelectasis:** Large intra-thoracic masses such as abscesses, tumors, pericarditis, and external trauma.

**Gross lesions in atelectasis:** Collapsed lungs, dark, firmer texture (resembles pneumonia). Collapsed alveoli, loss of alveolar spaces.

EMPHYSEMA

**Definition:** Emphysema is an abnormal and permanent enlargement of airspaces distal to terminal bronchioles with destruction of their alveolar walls. Primary lung emphysema is rare in animals but extremely important in human beings. Most common types in domestic animals are:

- **Alveolar emphysema:** Alveolar emphysema is difficult to evaluate in mild cases. It may require inserting a plug in the trachea during the necropsy and rapidly perfusing the lungs with fixative. Equine Heaves is an important disease causing alveolar emphysema in horses (See Pathology of the Transitional System).
- **Interstial emphysema:** Interstitial emphysema is rather common in bovines and characterized by distention of interlobular septa and pleura with gas. It is presumably caused by violent respiratory movements. Bovines have poor collateral ventilation (communication between lobules).
- **Bullous emphysema:** Bullous emphysema is a large focal accumulation (pocket) of air.
(bulla) in the lung. Large bullas form when there is rupture of pulmonary tissue.

Pathogenesis of Pulmonary Emphysema:

For many years it was thought to be the result of chronic obstruction of airways. Numerous investigations have revealed that alveolar emphysema in human beings results from the effects of elastases and other proteolytic enzymes released by phagocytic cells in the lung. Whether the same mechanism occurs in animal diseases such as Heaves in horses (COPD) remains to be elucidated. Be also aware that pulmonary emphysema commonly develops in agonic states (violent gasping air), particularly in cattle.

Besides lung cancer and cardiovascular diseases, there is also unequivocal scientific association between cigarette smoking and pulmonary emphysema (COPD). Click here for more information about the health effects of cigarette smoking.

PNEUMONIA

General: There is no universal classification of pneumonias in veterinary medicine.

There are numerous classifications such as:

- **Etiological:** Viral pneumonia, Mannheimiosis, Histophilosis pneumonia, distemper pneumonia, allergic pneumonia, etc.

- **Epidemiological:** Enzootic pneumonia, contagious bovine pleuropneumonia, etc.

- **Exudate:** Suppurative, fibrinous, or granulomatous pneumonias.

- **Topographical (distribution):** Lobar, lobular, diffuse, interstitial, focal, etc.

- **Miscellaneous:** Progressive pneumonia, proliferative pneumonia, atypical pneumonia, pneumonitis, etc.

**Important Note:** Confusion in veterinary medicine is rampant i.e. viral pneumonia of pigs, enzootic pneumonia of pigs, mycoplasma pneumonia of pigs, and chronic bronchopneumonia may be the same disease caused by *Mycoplasma hyopneumoniae*. 

**CLASSIFICATION ACCORDING TO EXUDATE, TEXTURE AND PORT OF ENTRY**

1. Suppurative Bronchopneumonia (Lobular pneumonia)

   - **Distribution:** Cranioventral consolidation of lungs.
   
   - **Lung texture:** Firm
   
   - **Port of entry:** Aerogenous
• **Etiology:** Generally caused by **bacteria or mycoplasma** which produce **mild to moderate injury** to the lung (i.e. *P. multocida, A. pyogenes, Bordetella bronchiseptica*).

• **Death** due to bronchopneumonia occurs when there is more than 60% of lung involvement in uncomplicated cases.

• **Gross lesions:** Affected lung is consolidated and the lobular pattern is accentuated. Color varies from red (acute, hyperaemia) to grey (chronic inflammation, atelectasis, fibrosis). Typically, purulent/pus exudate can be expressed from airways; in chronic bronchopneumonia the exudate takes a mucoid appearance.

• **Histopathology:** Large number of polymorphonuclear leukocytes in bronchoalveolar space in acute cases and a mixture of PMN,PAM and mucus (goblet cell hyperplasia) in the more chronic cases.

• **Examples:** Enzootic pneumonia of pigs, calves and lambs.

• **Common sequels:**
  - Abscesses (cranioventral lobes)
  - Bronchiecatasis (cranioventral lobes)
  - Fibrosis and fibrous pleural adhesions (cranioventral lobes)

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2. **Fibrinous Bronchopneumonia (Fibrinous pneumonia or Lobar pneumonia)**

Some authors consider fibrinous pneumonia a "peracute and generally fatal form of bronchopneumonia."

• **Distribution:** Cranioventral consolidation (except Porcine Pleuropneumonia *(Actinobacillus pleuropneumonia)*

• **Lung texture:** Hard

• **Color of affected lung:** Red -> yellow -> grey / Fibrin on pleura / necrosis of cut surface

• **Port of entry:** Aerogenous

• **Etiology:** Caused by agents that produce **severe injury to the lung** (i.e., *Mannheimia haemolytica, Actinobacillus pleuropneumonia*, etc). There is severe toxemia due to bacterial toxins and tissue necrosis. Also aspiration of harsh material.

• **Sequels:** Survivors can develop large pulmonary sequestra (pieces of necrotic lung surrounded by connective tissue).

• **Histopathology:** Notable dilation and thrombosis of lymphatic vessels, massive exudation of fibrin and polymorphonuclear leukocytes into the bronchoalveolar space and pleural, areas of coagulative necrosis.
- **Common sequela**: Pulmonary sequestra, pleural adhesions, fibrosis.

- **Examples of disease causing fibrinous bronchopneumonia**: Pneumonic Mannheimiosis (shipping fever), Porcine Pleuropneumonia, Contagious Bovine Pleuropneumonia (does not occur in the American continent).

3. **Interstitial Pneumonia (Pneumonitis):**

- **Distribution**: Diffuse, lungs fail to collapse

- **Texture**: Elastic with rib imprints. Cut surface has a meaty and often edematous appearance

- **Port of Entry**: Aerogenous or hematoagenous

- **Etiology**: viremia, airborne-viruses, septicemia, blood-borne toxins, systemic toxicants, allergy, inhaled gases.

- **Gross lesions**: The lungs fail to collapse when the thorax is opened; occasional costal imprints are visible on the pleural surface. The color depends on blood: tissue ratio and type of exudate or fibrous scarring. Histopathology: Changes are often subtle and difficult to diagnose grossly generally requiring histopathologic confirmation. Interstitial pneumonia may coexist with edema, emphysema or bronchopneumonia (*Broncho-interstitial pneumonia*).

- **Histopathology**: The primary lesion is centered in the alveolar wall. Thickening of alveolar walls. Interstitial exudation or proliferation of type II pneumonocytes. In chronic interstitial pneumonia there is alveolar fibrosis.

- **Examples**: Porcine and equine influenza, Bovine Pulmonary Edema and Emphysema, Canine Distemper (without secondary bacterial infection), Porcine Respiratory and Reproductive Syndrome (PRRS) etc. Examples caused by pneumotoxicants: “fog fever in cattle,” paraquat, etc. Examples caused by allergens: "Extrinsic Allergic Alveolitis (Framer’s lung), "Reinfection Syndrome."

- **Bronchointerstitial pneumonia** is a term currently used in veterinary pathology to describe the microscopic lesions caused by viral infections. Some respiratory viruses cause necrosis and inflammation of the bronchial/bronchiolar epithelium (bronchopneumonia) and also interstitial necrosis and proliferation of pneumonocytes (interstitial pneumonia). Most viral pneumonias are transient unless complicated with bacteria.

- **Common sequels**: Pulmonary fibrosis, interstitial emphysema.

4. **Embolic Pneumonia:**

- **Distribution**: Multiple foci or small nodules randomly distributed in all pulmonary lobes. Color varies depending on the amount of connective tissue and blood vessels
• **Texture**: Nodular. Variable size granulomas on cut surface

• **Port of entry**: Always Hematogenous.

• **Etiology**: Endocarditis, ruptured hepatic abscess (vena cava thrombosis in cattle), omphalophlebitis.

• **Gross lesions**: Variable number of foci, often with a white center and red hemorrhagic margins. Eventually embolic lesions may progress to abscesses.

• **Histopathology**: Septic emboli attached to pulmonary capillaries, pulmonary edema, microabscesses.

• **Common sequels**: Abscesses in all pulmonary lobes.

5. **Granulomatous Pneumonia**:

• **Distribution**: Randomly distributed nodules.

• **Texture**: Nodular. **Cut surface**: granulomas

• **Port of entry**: Aerogenous or hematogenous.

• **Etiology**: Tuberculosis, systemic mycosis, some parasites (*Muellerius capillaris*; larva migrans). Usually caused by microorganisms, parasites (ova, larvae) or foreign material (inhaled food particles) difficult to eliminate by phagocytosis.

• **Gross lesions**: Granulomas in the lung and sometimes in other organs too. Be aware that granulomatous pneumonia can resemble lung cancer and may require histopathological confirmation.

• **Histopathology**: Variable size nodules with a necrotic center infiltrated by macrophages and giant cells and surrounded by connective tissue mixed with lymphocytes and plasma cells.

• **Common sequels**: Cachexia (wasting) in chronic cases

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**EXAMPLES OF BRONCHOPNEUMONIAS IN DOMESTIC ANIMALS**

**Cattle**.-

**Enzootic Pneumonia of calves** is a clinico-epidemiological term / Chronic suppurative bronchopneumonia (a pathologic term): Multifactorial Disease Complex of young calves (Calf pneumonia). Environmental factors (temperature, humidity, poor air circulation, crowding, stress), immune status, viral infections (PI-3, Adenovirus, BRSV, etc.), bacterial (*Pasteurella multocida* (A, D *Hemophilus somnus*, *Arcanobacterium pyogenes*), Mycoplasmal infection (*M. bovis*, *M. dispers*, *Ureaplasma*, etc.). It has high morbidity, low mortality and multifactorial pathogenesis. Gross lesions
UK), mucopurulent exudate in airways.

**Bovine Pneumonic Mannheimiosis (Shipping fever)** is the number one cause of feedlot mortality in North America. It is caused by *Mannheimia (Pasteurella) haemolytica A1* which is not considered a true primary pathogen since it is often present as normal flora and effectively destroyed and cleared by the normal bovine lung. When the defense mechanisms are compromised by viral infections (PI-3, BHV-1, BRSV viruses) or stress, inhaled *M. haemolytica* colonizes the lung and causes a severe **fibrinous bronchopneumonia** and toxaemia. A powerful cytotoxin for ruminant leukocytes is produced by *M. haemolytica A1* which further compromise pulmonary defense mechanisms. Gross lesions: Fibrinous bronchopneumonia, fibrin on pleura, pleural effusions. On cut surface, there are typical irregular areas of necrosis. Histopathology: Distended interlobular septa with thrombosis of lymphatic vessels, irregular areas of coagulative necrosis which are surrounded by abnormal leukocytes (oat-shaped cells), abundant fibrin and PMN in bronchioles and alveoli. **Bacteriology**: Consolidated lung typically yield pure cultures of *M. haemolytica*. Determining the primary cause is required (virus, stress, management) to reduce the incidence of this disease. *Mannheimia (Pasteurellae)* vaccines are of questionable value in field conditions. Viral vaccines and management practices reducing stress have a beneficial effect in the incidence of shipping fever. Shipping fever should not be confused with **Hemorrhagic Septicemia** caused by *Pasteurella multocida* (Serotypes BE) which is an important acute septicemia in Asia and Africa but not seen in the American continent.

**Histophilus somni (Haemophilus somnus) / Histophilosis Complex**: *Histophilus somni* is an important bovine pathogen but it can be found as normal vaginal flora. This "Complex" includes several forms:

- **Encephalitic** (TEM)
- **Reproductive** (infertility, endometritis and abortion)
- **Myocardial** (myocarditis)
- **Otic** (otitis)
- **Respiratory**: Respiratory Histophilosis can result either in fibrinous bronchopneumonia identical to shipping fever, or in chronic suppurative bronchopneumonia in enzootic pneumonia.

*Mycoplasma bovis* has been incriminated with increase frequency as a cause of bovine bronchopneumonia in young cattle. It causes a necrotizing bronchopneumonia with large caseated or non-caseated nodules in the cranioventral lung.

**Verminous Pneumonia** (*Dictyocaulus viviparous*; Lungworm). This is a parasitic bronchitis rather than pneumonia. The larval stages cause a transitory interstitial pneumonia and edema. The adult nematodes live inside the large airways causing chronic bronchitis and atelectasis, particularly in the dorsal aspect of the caudal lobes. Adult parasites *D. viviparous* is also associated to hypersensitivity reaction (see reinfection syndrome).

**Sheep-**

**Ovine Enzootic Pneumonia**: (clinico-epidemiologic term) / Chronic Bronchopneumonia (pathologic term): Multifactorial Disease of lambs (less than 1 year old). Environmental stressors,
viral infections (PI-3, Adenovirus, RSV, etc), bacterial (*M. haemolytica*), Mycoplasmal infection (*Mycoplasma ovipneumoniae*). High morbidity, low mortality. Gross lesions: Chronic bronchopneumonia / bronchointerstitial with mucopurulent exudate in airways.

**Ovine Mannheimiosis** occurs in two distinct types:

- **Pneumonic Mannheimiosis** (*Mannheimia haemolytica*); Lesions and pathogenesis are similar to shipping fever of cattle: acute fibrinous pneumonia predisposed by stress, viral infections (PI-3, Adenovirus), chlamydial infections.

- **Septicemic Mannheimiosis** (*M. haemolytica*) in stressed sheep which is characterized by a fulminating septicemia with DIC, petechial hemorrhages, lung edema, (sometime necrotizing pharyngitis). *M. haemolytica* is present in the tonsils of 95% of normal sheep.

**Ovine Verminous Pneumonia** (*Dictyocaulus filariae*; Lungworm). As in calves, this is a parasitic bronchitis rather than pneumonia. The larval stage causes a transitory interstitial pneumonia and edema. The adult nematodes live inside the large airways causing chronic bronchitis and atelectasis, particularly in the dorsal aspect of the caudal lobes.

**Pigs.**

**Porcine Enzootic Pneumonia** (clinico-epidemiological term) / Chronic Bronchopneumonia (pathologic term): Multifactorial and generally nonfatal disease of young pigs produced by *Mycoplasma hyopneumoniae*. Predisposing factors include temperature, humidity, ammonia, crowding, and stress. Secondary infections with *P. multocida*, *Haemophilus spp.*, *A. pyogenes*, and *Mycoplasma hyorhinis* are common. It has high morbidity but low mortality. Gross lesions: Chronic suppurative bronchopneumonia (BALT hyperplasia) = mucopurulent exudate in airways. *Mycoplasma hyopneumoniae* is a rather fastidious organism to grow and only a few laboratories in the world are capable of isolations.

**Porcine Pasteurellosis**: Secondary infection by *P. multocida*. Lesions: Sometimes produce a fulminating fibrinous bronchopneumonia. In others, it produces chronic suppurative bronchopneumonia. *P. multocida* is isolated from the lungs of most pigs with chronic bronchopneumonia.

**Porcine Pleuropneumonia**: *Actinobacillus (Haemophilus) pleuropneumonia*. A primary pathogen. Aerogenous infection. Gross lesions: Cranioventral or dorsocaudal (often unilateral) swollen areas (lumps) of consolidation covered with fibrin. On cut surface, areas of coagulative necrosis similar to pneumonic pasteurellosis. Survivors can develop large pulmonary sequestra.

**Streptococcus suis type II**: Important zoonosis. Various presentations: neonatal septicemia, meningitis, arthritis, polyserositis, myocardial necrosis, endocarditis, abortion and bronchopneumonia. Gross and histological lesions: Nonspecific (often combined with other pathogens): Fibrinous or suppurative bronchopneumonia. Myocardial lesions are often indistinguishable from Mulberry heart, a disease attributed to selenium and vitamin E deficiencies.

**Dogs and Cats** (all species).-

**Aspiration Pneumonia** is seen in all species and it is caused by the aspiration of gastric contents following vomit (i.e., canine parvovirus, feline panleukopenia, following anesthesia) or neurological signs (i.e. rabies, listeriosis), improper intubation or inappropriate oral administration of medicines (iatrogenic).
EXAMPLES OF INTERSTITIAL PNEUMONIAS IN DOMESTIC ANIMALS

Cattle.-

Viral Pneumonias: Infectious Bovine Rhinotracheitis (IBR): Bovine Herpesvirus-1 (respiratory, genital and abortion). Respiratory form is generally an acute and transient rhinotracheitis Parainfluenza-3 Virus (PI-3 virus) and Bovine Respiratory Syncytial Virus (BRSV) transient rhinitis and tracheitis and bronchointerstitial pneumonia with intracytoplasmic inclusions and formation of syncytial (epithelial) cells. All these bovine respiratory viruses can cause severe impairment of defense mechanisms and predispose animals to secondary bacterial pneumonia such as Shipping Fever (Mannheimia haemolytica).

Atypical Interstitial Pneumonia: This is an archaic term that must be abandoned. What use to be atypical in now typical? The so-called "atypical interstitial pneumonia" of cattle comprises several distinct conditions characterized grossly by diffuse interstitial pneumonia:

- Extrinsic allergic alveolitis (also known as hypersensitivity pneumonitis or farmer's lung). Caused by inhalation of fungal spores of Saccharopolyspora rectivirgula (Micropolyspora faeni) from moldy hay -> Antibody response -> deposition of antigen/antibody complexes in the blood air barrier (a type III hypersensitivity) -> C'PMN mediated injury to pneumonocytes type-I -> interstitial pneumonia. Extrinsic allergic alveolitis is most commonly seen in cattle fed silage.

- Bovine Pulmonary Edema and Emphysema (BPEE; fog fever). Ingestion of pasture (foggage) containing large amounts of L-tryptophan metabolized 3-methylindole (3-MI) -> toxic injury to pneumonocytes type 1 -> interstitial pneumonia with severe edema and emphysema. BPEE is most commonly seen in grazing cattle. Experimental injections of 3-MI consistently reproduces the pulmonary lesions.

- Reinfection syndrome. It is a hypersensitivity reaction to re-infection with larvae of Dictyocaulus viviparous. Pathogenesis of the lesions similar to extrinsic allergic alveolitis. Most commonly seen in calves recently moved to pasture.

- Bovine Respiratory Syncytial Virus. It has only been recently described. It is an acute fatal pneumonitis in feedlot cattle due to BRSV and presumably to hypersensitivity reaction against this virus. A similar condition occurs in children with the human strain of RSV.

- Other respiratory syndromes with interstitial pneumonia. Milk allergy (type I hypersensitivity in dairy cows), pit (manure) gases (H2S); "silo filler disease/silage" (NO2), etc.

Pigs.- (Porcine Viral Pneumonias):

- Swine Influenza: Transient tracheitis, Bronchointerstitial pneumonia. This viral infection predisposes pigs to secondary bacterial pneumonia (Pasteurella multocida, Haemophilus suis). Gross lesions are minimal and consist of a mild hyperemia of the tracheobronchial mucosa. Secondary bacterial infection leading to bronchopneumonia is commonly seen.

- Porcine Reproductive and Respiratory Syndrome (PRRS); Caused by a virus
classified as "arterivirus." Lesions: Proliferative interstitial pneumonia. This syndrome has been also associated with abortion and reproductive failure in sows. Although the role of this virus causing immunosuppression has been controversial, it is well known that pigs with PRRS are susceptible to other infections including *Pneumocystis carinii*.

- **Postweaning Multisystemic Wasting Syndrome:** This is a relatively new viral disease of pigs caused by Porcine Circovirus-2 and characterized clinically by progressing emaciation. Grossly there interstitial pneumonia and generalized lymphadenopathy. Laboratory tests are required for the diagnosis and differentiation from PRRS.

### Horses- (Viral pneumonias):

- Equine Viral Rhinopneumonitis (EVR/EHV-4)
- Equine Influenza; Equine Viral Arteritis (EVA)
- Equine Adenovirus

**Lesions:** Minimal, only a transient tracheitis, or bronchointerstitial pneumonia. Virus impairment of lung defenses causes secondary bacterial pneumonia (*P. multocida, Streptococcus spp; E. coli; Rhodococcus equi*). Fatal Adenovirus infection is commonly seen in Arabian foals with Combined Immunodeficiency (CID).

### Dogs.- (Canine Viral pneumonias):

- Canine Distemper
- Canine Adenovirus
- Canine Parainfluenza virus
- Canine Influenza or Canine Flu: This disease was recently reported in the United States and appears to be caused by an equine strain of influenza virus. It has high morbidity and clinical sings similar to kennel cough. The mortality is low. Histopathology and histochemistry are required for confirmatory diagnosis.

**Lesions:** Transient tracheobronchitis, bronchointerstitial pneumonia. Virus impairment of lung defense causes secondary bacterial pneumonia (*Bordetella bronchiseptica; E. coli, Streptococcus spp*).

### Cats.- Feline Interstitial Pneumonias

- *Feline Pneumonia:
• Feline Viral Rhinotracheitis
• Feline Calicivirus.
• *Chlamidophila (Chlamydia) psittaci*

**Gross lesions:** Generally minimal, hyperemia of nasal and tracheobronchial mucosa. Secondary bacterial infections by *P. multocida, B. bronchiseptica, Streptococcus spp.* can result in bronchopneumonia. Calicivirus also causes ulcerative lesions in oral cavity of cats.

**Sheep and goats.-**

*Ovine Progressive Pneumonia (Maedi-Visna / Lymphoid Interstitial Pneumonia).* In Iceland, "Maedi" means shortness of breath. Clinical disease only in sheep older than 2 years; it is caused by a retrovirus. Gross lesions: Lungs fail to collapse, heavy, elastic texture and prominent rib imprints typical of a severe interstitial pneumonia. Histopathology: Interstitial lymphocytic infiltrates with notable hyperplasia of bronchial associated lymphoid tissue (BALT) and smooth muscle.

### EXAMPLES OF GRANULOMATOUS PNEUMONIAS IN DOMESTIC ANIMALS

**Tuberculosis:** Important disease of human beings, ruminants, pigs, poultry and horses. Not a problem in dogs and cats. Lesions may be found in any organ or tissue. Lungs may be the main primary site or only one of many organs involved in the disseminated infection. Gross lesions: Multiple, random, nodules with caseous centers (Granulomatous pneumonia). Histopathology: granulomas containing acid fast organisms (necrotic center, macrophages, and giant cells, surrounded by connective tissue and lymphocytes). Tuberculosis once thought eradicated, is rapidly reappearing in western countries due to AIDS and antibiotic resistance.

**Horses.-** *Rhodococcus equi Pneumonia:* Disease of world wide importance caused by a telluric organism. Infection in foals has three important forms: Respiratory, Intestinal and Skeletal. The respiratory form is characterized by chronic cough and weight loss. Gross lesions: Bronchopneumonia, abscesses and pyogranulomas with necrotic centers. Histopathology: Pyogranulomas formed by macrophages, neutrophils, giant cells and fibrous tissue.

**Systemic Mycosis (Blastomyces dermatitides; Coccidiodes immitis; Cryptococcus neoformans, Histoplasma capsulatum).** Gross lesions: Granulomatous Pneumonia. Histopathology: Granulomas + fungi, giant cells. Typically, there is involvement of other organs and particularly lymph nodes.

**Feline Infectious Peritonitis (FIP):** For details on the pathogenesis of FIP in cats see the section of digestive system. In the respiratory system, FIP virus causes a conspicuous pyogranulomatous pneumonia.

**Muellerius capillaris (Nodular lung worm):** Some parasites induced granulomatous reaction in the lungs. *Muellerius capillaria* is an Important parasitic disease of sheep and goats that induces multifocal sub-pleural calcified granulomas mainly in the dorsal caudal lobes. It is generally an incidental finding except for severe parasitic infections. The granulomas contain dead larvae,
parasitic eggs and many eosinophils.

**THORACIC CAVITY**

**The thoracic (pleural cavity)** is lined by the parietal pleura (ribs, diaphragm and mediastinum) and the visceral pleura (lungs). There is negative pressure in the thoracic cavity and the lungs collapse when the negative pressure is lost by opening the thorax. Under normal conditions once the cavity has been opened, the lungs appear smaller in comparison to the rest of the cavity.

**Pneumothorax**

Pneumothorax is the presence of air in the thoracic cavity (loss of negative pressure). It is caused by trauma, rupture of lung or emphysematous bulla, rupture of esophagus (gas in mediastinum), iatrogenic (i.e. biopsy). Gross lesions: Failure of diaphragm to retract, atelectasis.

**Hydrothorax**

Hydrothorax is the accumulation of abnormal quantities of transudate (serous fluid) in thoracic cavity. Common causes of hydrothorax include congestive heart failure, hypoproteinemia (liver, renal, intestinal disease, starvation), and lymphatic obstruction. Lesions: Fluid in cavity. Transudation must be differentiated from exudation. Chronic transudation of fluid into thoracic cavity (irritation) may result in mild pleural inflammation (modified transudate) and fibrosis.

**Hemothorax**

Hemothorax is the presence of free blood in thoracic cavity. It is caused by severe trauma or penetrating wound into the lungs, rupture of major blood vessels (aneurism), coagulopathies, warfarin poisoning, etc.

**Chylothorax**

Chylothorax is the presence of free lymph (chyle) in the thorax and it is caused by rupture of a major lymphatic duct. Most common causes are thoracic trauma, iatrogenic (surgery) and neoplasia. There is also an idiopathic form of chylothorax in dogs. Lesions: Milky fluid in cavity. For diagnosis in live animals submit fluid to clinical pathology, it should be high in triglycerides and lymphocytes. Click here for an excellent review on chylothorax by the University of Georgia

**Pleuritis (pleurisy):**

Pleuritis or pleurisy is the inflammation of pleura and it can occur alone or in combination with pneumonia (i.e. fibrinous pneumonia/pleuropneumonia). Depending on exudate, pleuritis could be purulent, fibrinous, granulomatous (sulfur granules), etc. Chronic pleuritis results in fibrous adhesions between visceral and parietal pleura. Etiologic diagnosis requires microbiology.

**Pyothorax (Empyema)**

Pyothorax (pleural empyema) is the accumulation of purulent exudate (pus) in the thoracic cavity. In cats, it is commonly produced by *P. multocida*, in dogs by *Nocardia asteroides* and in horses by *Streptococcus* or *Mycoplasma*.
Primary lung tumors

Tumors arising from the lung are relatively rare in animals (dogs and cats, > other domestic species) but very common in human beings; tumors originate from any cell line of the lung (carcinoma > sarcoma); generally seen as a single nodule or mass in animals with respiratory disorders. Signs could also be related to distant metastasis.

Primary epithelial lung tumors:

- Bronchial / alveolar adenoma / papilloma (benign)
- Bronchogenic, squamous, adenocarcinoma (malignant).

Primary mesenchymal lung tumors:

- Hemangioma or hemangiosarcoma
- Fibroma or fibrosarcoma, etc.

Pulmonary Adenomatosis (Pulmonary ovine carcinoma): Retrovirus infection. Most commonly reported from Scotland and North America; none yet seen in Australia or New Zealand. Chronic respiratory signs and weight loss. Gross lesions: Lungs fail to collapse, heavy, wet, with consolidation and nodules in cranioventral regions (looks like bronchopneumonia). In more chronic cases the lesions extends to all pulmonary lobes. Histopathology: Nodules are formed by papillary growth of epithelial cells. Metastasis to lymph nodes occurs in some cases.

Mesothelioma is a rare tumor of serosal membranes (mesothelium) which readily disseminates by implantation. Lesions: multiple growths involving most of the serosal surface. Histopathology or cytology required for confirmation. In humans there is strong correlation between exposure to asbestos and mesotheloma.

Secondary (Metastatic) Tumors in lungs are more common than primary lung tumors. Lungs are highly vulnerable to metastasis because of circulation and density of capillaries. Metastatic tumors are seen as multiple nodules or masses with random distribution. Differential diagnosis requires clinical, radiographic, and histopathological examination. Tumors in the thorax (space occupying lesions) are known to predispose to Hypertrophic Pulmonary Osteoarthropathy.

Table 1. Common tumors that often metastasize to the lung

<table>
<thead>
<tr>
<th>Osteosarcoma</th>
<th>Chondrosarcoma</th>
<th>Fibrosarcoma</th>
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<tbody>
<tr>
<td>Hemangiosarcoma</td>
<td>Lymphosarcoma</td>
<td>Malignant Melanomas</td>
</tr>
<tr>
<td>Mammary carcinoma</td>
<td>Uterine carcinoma</td>
<td>Adrenal carcinoma</td>
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THE END