RESPIRATORY SYSTEM

Structure and Defense Mechanisms

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Morphology

The respiratory tract is divided into three independent but continuous systems

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

2.- **Transitional system** consists of bronchioles which are primarily lined by non-ciliated cells, Clara cells and a few ciliated cells. Healthy bronchioles do not have goblet cells.

3. **Exchange system** consists of alveoli lined by epithelial type I (membranous) and type II pneumonocytes.
• 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 into the nasal cavity of any healthy animal and sent for microbiologic culture.

• These organisms constitute the normal flora of the respiratory tract.
This blood-agar plate shows different bacterial colonies that grew from a nasal swab taken from a normal animal.

Although most organisms of the nasal flora are harmless, some others are potentially pathogenic. For example, in cattle *Mannheimia haemolytica* and in swine *Bordetella bronchiseptica* are normal nasal flora, yet both organisms are involved with "Shipping Fever" and "Atrophic Rhinitis" respectively. This is an important fact to remember since isolation of bacteria belonging to nasal flora is sometimes erroneously interpreted by veterinarians.

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.
Experimental studies have conclusively demonstrated that basal bacterial flora, including some potentially important pathogens, are constantly being carried into the lung by inspired air. In spite of this constant bacterial bombardment, the lower respiratory tract remains essentially sterile due to the extraordinary function of the respiratory defense mechanisms.
The respiratory system is constantly exposed to particles, bacteria, viruses, spores, gases, vapors, etc.

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

- **None specific**
  - Air turbulences
  - Mucus Trapping
  - Mucociliary clearance
  - Phagocytosis
  - Coughing
  - Sneezing

- **Specific**
  - Antibodies
  - Cell mediated immunity
  - Secretions
  - Phagocytosis
• If a healthy animal is exposed to an aerosol of bacteria, the pulmonary defense mechanisms rapidly eliminate these organisms from the lung (bacterial clearance).

• If these defense mechanisms fail, inhaled bacteria colonize the lung and cause bacterial pneumonia.

Note in this graph the rapid elimination of inhaled bacteria given by aerosol to a normal animal (---). In contrast note that the number of inhaled bacteria increases with time in viral infected animals (---).

This experimental model demonstrated two things:

1. Healthy lung rapidly eliminates inhaled bacteria

2. Viruses inhibit the pulmonary defense mechanisms and predispose lungs to secondary bacterial pneumonia.

Other known factors that impair pulmonary defense mechanisms are:

• Stress
• Dehydration,
• Uremia
• Lung edema,
• Immunodeficiency
• Ammonia, etc.
The **dorsal concha** (D) extends from just behind the nares to the dorsal aspect of the **ethmoidal conchae** (E); the **ventral concha** (V) is located in the ventral aspect of the nasal cavity; the **ethmoidal concha** (E) is the smallest of all three and is located in the caudal part of the nasal cavity.

The narrow spaces (arrows) located between conchae are the dorsal, middle and ventral **meatuses** where inspired and expired air travels within the nasal cavity. These narrow spaces act as filters for large particles during inspiration. **LT** = lymphoid tissue
Nasal Cavity

Note the characteristic coiled appearance of conchae formerly called turbinates.

Nasal septum (cartilage) dividing left and right sides of nasal cavities (*).

Large sized particles (>10μm) are trapped in the nasal cavity due to centrifugal forces resulting from air turbulence in the nasal cavity. Large particles are impacted against the wall and trapped by the mucus that covers the ciliated epithelium.

Only particles smaller than 10μm may pass the nasal cavity into the trachea and bronchi.
Histology of the conducting system (nose to bronchi):

- Pseudo-stratified ciliate epithelium with numerous goblet cells (arrows)
- The submucosa contains many blood vessels (bv) and glands (arrowheads)
MUCOCILIARY CLEARANCE

Note numerous ciliated cells with approximately 200 cilia/cell and a layer of mucus on the surface. Particles impacted in the upper respiratory tract (see ●) are removed via the mucociliary escalator (apparatus). This escalator is composed by ciliated cells (beating 25 times per second) and a thin coat of mucus.

It is estimated that mucociliary escalators of trachea moves deposited particles at a rate of 1.5 cm/minute.

Mucociliary Escalator

Electron micrograph of bovine trachea showing ciliated cells.

Note the abundance of cilia and a layer of mucus on top which contains trapped particles. Mucus is propelled towards pharynx and swallowed.

Courtesy of Dr. David Sims
Some particles that bypass the nasal cavity are trapped at bronchial bifurcations due to sudden change in direction (centrifugal force) of inhaled air (Circled in red). Strategically situated in these bifurcation there are specialized structures known as bronchial-associated lymphoid tissue (BALT) (Circled in red).

Particles trapped in mucus are expelled from the conducting system by the mucociliary movement. Only particles <2 μm in diameter can reach the transitional and exchange systems. Here particles are phagocytized by pulmonary alveolar macrophages which move toward the bronchioles until they reach the mucociliary escalator. Once there, macrophages are moved out of the lung by the mucociliary clearance as any other particle (see arrows).

Green et al; Am Rev Respir Dis 115: 479, 1977}
Pulmonary Alveolar Macrophages

- Alveoli do not have cilia. The main defense mechanism the alveoli is the pulmonary alveolar macrophage (PAM).

- Bacteria are all in the size range (2μm) that reach the alveolar region.

- PAMs are derived from blood monocytes. Phagocytosis is nonspecific and thus antibodies are not required. Once phagocytosis has taken place, alveolar macrophages move to bronchioles and from here the phagocytic cells are removed via the mucociliary escalator.

- PAM is the main effector cell in the inflammatory process of the lung through production of cytokines.
PATHOLOGY OF THE NASAL CAVITY
EPISTAXIS

- The nasal mucosa is highly vascularized and prone to hemorrhage.

- **Epistaxis** (nose bleeding) and **hemoptysis** (blood in mouth, saliva or sputum) are common findings in all species.

- Epistaxis or hemoptysis do not necessarily imply that the hemorrhage has occurred in the nasal or oral mucosa. Both conditions can be caused by pulmonary hemorrhage too.

- Epistaxis is a frequent indicator of nasal trauma or neoplasia.

- Hemoptysis-epistaxis in cattle is associated with ruptured pulmonary vessels (aneurysm).

- In a horse, epistaxis is also seen in the condition called “**Exercise-induced pulmonary hemorrhage.**”

- Careful examination is required to localize the source of blood.

**Note blood coming out of the nostrils of a dead cat and a live horse**

*Courtesy of Dr. Jeanne Lofstedt*
Ethmoidal hematoma is important in older horses and is characterized clinically by chronic, progressive and generally unilateral nasal bleeding.

Grossly, an ethmoidal hematoma appears as a single, tumor-like, pedunculated, soft mass (arrows) arising from the ethmoidal mucosa and readily seen by endoscopy.
Nasal Congestion / Hyperemia

- Remember that nasal mucosa is very well vascularized and therefore submucosal vessels easily become engorged with blood following irritation (hyperemia) or circulatory failure (congestion).

- Nasal congestion and hemorrhage are commonly seen in animals exposed to irritant gases (ammonia, $\text{H}_2\text{S}$, $\text{NO}_2$, etc.), shock, bloat, and of course in rhinitis.

**Head hemisection; cow; rhinitis.**

Note the markedly congested mucosa with excessive exudate on the surface of conchae. Ian example of inflammatory hyperemia.
Lesions and stages of repair are similar:

- All levels of the conducting system.
- Viral, toxic and traumatic injury
- The diagram on the left shows the stages of degeneration and repair that take place in the respiratory epithelium lining the conducting system (nose to bronchi)

The next four slides provide a histological view of the sequence of injury, necrosis and repair that occurs in the respiratory epithelium following viral injury.
Normal ciliated epithelial cells with abundant cilia. Now assume that a virus such as influenza replicate in these cells and because of viral-induced cell injury, ciliated cells begin to degenerate and exfoliate (next slide).

Exfoliation of Ciliated Epithelium a few hours after injury H&E. Note ciliated cells detaching from basement membrane. This change is a first sign of cell injury. Affected cells lose their normal attachment to the basement membrane and large portions of the mucosa may become ulcerated (arrows). Since the basement membrane remains intact, repair by mitosis and maturation of new cells is remarkable (see next slide).
Nasal cavity 72 hours after mucosal injury

This slide illustrates a common inflammatory change which occurs in the nasal mucosa during inflammation. After injury, chemotactic factors (i.e., leukotrienes, C5a, factor, Interleukins, etc.) are locally released in the affected tissue producing exudation of leukocytes into the surface of respiratory mucosa. Note numerous leukocytes forming a plaque of exudate on mucosal surface.
Ciliated Epithelium 14 days after injury H&E.

The sequence of injury, exfoliation and repair is exactly what occurs when you get a common cold.

The virus replicates in your cells and your nose and throat feel dry and painful (sore throat). Large numbers of your ciliated cells exfoliate leaving your nasal basement membranes denuded. This change is chemotactic to leukocytes which in turn release interleukins that makes you feel really bad (you feel like staying home but you don’t want to miss your pathology lecture <fat chance>).

Secretion of sub-mucosal glands and goblet cells are increased thus you have a runny nose. Three days later, you start feeling better when your basement membranes are fully covered by new ciliated cells, that is, until you catch your next cold from the sneezing classmate sitting next to you.
Summary
Injury and Repair of the Nasal Mucosa

- Ciliated epithelium
- ~250 cilia/cell
- Highly vascularized
- Abundant glands
- Degeneration
- Loss of attachment
- Necrosis
- Exfoliation
- Repair
- Pre-ciliated cells
- Mitosis
- Cell differentiation
- Healed epithelium
- Normal function
Rhinitis

According to the type of exudate, rhinitis (also tracheitis and bronchitis) can be classified into:

- **Serous**  
  Increased secretion of submucosal glands.

- **Catarrhal**  
  Goblet cell hyperplasia and excessive production of mucus.

- **Fibrinous**  
  Altered vascular permeability and exudation (escape) of fibrin.

- **Purulent**  
  Chemotaxis for polymorphonuclear leukocytes / pus.

- **Granulomatous**  
  Granuloma / fibrosis / polyps.

In some instances, there is combined exudation and terms such as *mucopurulent*, *fibrinopurulent*, *fibrinonecrotic*, etc are used.

**Clinical signs:**
- Nasal discharge
- Unilateral or bilateral

**Laboratory tests:**
- Cultures, virology
- Cytology
**Horse with purulent rhinitis.** Note the purulent discharge in the left nostril.

**Strangles (*Streptococcus equi*)** is an important equine disease that may occur as an outbreak with high morbidity (90%) but low mortality (5% > foals).

Strangles is characterized by purulent (suppurative) rhinitis and regional lymphadenitis with formation of abscesses.

Some horses with strangles develop **purpura hemorrhagica** (vasculitis). Bacterial metastasis to the internal organs is referred to as "**bastard strangles.**"

Involvement of retro pharyngeal lymph nodes can compress laryngeal nerves and cause secondary **laryngeal hemiplegia**.

Strangles is only seen sporadically in North America.
Example of mucopurulent rhinitis in a dog

Note mucopurulent discharge from nostrils.

Viral infections can also predispose dogs to secondary bacterial rhinitis (*Bordetella bronchiseptica*, *E. coli*, *Streptococci*, *Staphylococci*).

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 and can cause a highly contagious condition referred to as **Kennel Cough**.

**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.
Mucopurulent rhinitis and Sinusitis / Dog / Necropsy.

- Large amounts of mucus mixed with pus in the nasal cavity. In this photo, note that the palate has been removed to better visualize the nasal cavity.

- Remember that the mucus production goes along with chronicity since in chronic inflammation there is severe goblet cell hyperplasia with a concurrent increase in the production of mucus.

- This dog was euthanized because recurrent and poorly responsive rhinitis and sinusitis.
Rhinitis and Conjunctivitis in cats

Viral:
- Feline Calicivirus
- Feline Infectious Rhinotracheitis

Chlamydial:
- Chlamydiophila psittaci

Bacterial:
- Bordetella bronchiseptica
- Escherichia coli
- Streptococcus
- Staphylococcus

Mycotic:
- Cryptococcus neoformans

Viral rhinitis in cats often together with conjunctivitis. Note crusts of exudate in nostrils and conjunctiva.

(Courtesy of Dr. Cheryl Cullen)
Mucopurulent Rhinitis

Note mucopurulent discharge from nostrils.

Feline Rhinotracheitis (FHV-1) and Feline Calicivirus are common agents involved in feline rhinitis-sinusitis.

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 have also been associated to rhinitis and conjunctivitis in cats. Rhinitis and sinusitis in cats are frequently associated with the fungus *Cryptococcus neoformans*. 
Note a purulent exudate on the surface of dorsal and ventral conchae and meatuses. Mucosal surfaces are not hyperemic and this could raise the possibility that the exudate was produced elsewhere such as in the trachea and lung and coughed into the nasal cavity. Histology would be required here to confirm that exudation of polymorphonuclear leukocytes is taking place in nasal mucosa.
Note the thick plaques of fibrin on the nasal mucosa. Remember that fibrinous exudation implies severe damage to the vasculature and escape of fibrinogen from blood that subsequently is polymerized into fibrin.

This is an example of bacterial rhinitis superimposed to a viral lesion (IBR). Remember that some bacteria causing this type of infection are present in normal flora. Tissues submitted from the nasal cavity tested positive for IBR virus.
Note the multiple small nodules on nasal mucosa. Granulomatous inflammation always indicates chronic inflammation. Lesions like these may be caused by hypersensitivity reactions or mycotic infections.

Granulomatous rhinitis is rare and most likely of little clinical significance. If found during a physical examination, a biopsy is recommended.

Examples of granulomatous rhinitis in a Cow and a Mule

Note polypoid nodule in the nasal cavity of this mule. Although similar to a tumoral growth, microscopic examination revealed a multilobulated granoloma containing numerous round bodies with a thick capsule (see asterisks in the insert).

The microorganisms were identified as *Rhinoporida seeberi*, a human and animal pathogen currently classified as a protist and not as a true fungus.

Courtesy of Dr. Alexis Berrocal, Costa Rica
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).

There is no mortality except in immune-suppressed pigs that develop a disseminated fatal infection.

Note epiphora (tears) which result in a dark discoloration around the eyes.

Irritation of nasal mucosa and obstruction of lachrymal ducts results in epiphora which often mixes with dust and produces localized skin irritation.
Inclusion Body Rhinitis / Piglet

Note severe hyperemia of nasal mucosa. Except for rare cases in which disseminated infection occurs in immunosuppressed piglets, inclusion body rhinitis is a transient, self-limited rhinitis.

**Histological section H&E.** Note the enormously large cells with large viral intranuclear inclusion bodies (arrows), hence the term “Cytomegalovirus.” As mentioned before, cases like this one are rarely seen in the postmortem room since infection tends to be mild and transient. See Thomson’s Book Figure 3-8
Atrophic Rhinitis

Note facial deformity in a pig with severe atrophic rhinitis.

Atrophic Rhinitis is an important and widely distributed disease of pigs. The etiology is still controversial: Bacterial? (*Pasteurella multocida, Bordetella bronchiseptica, Mycoplasma spp.*), Viral?. nutritional (Vitamin D, Ca, P), Genetic?, Environmental? (Humidity, temperature, etc.).

Current literature strongly suggests a combined (co-infection) with toxigenic strains of *Pasteurella multocida* and *Bordetella bronchiseptica*. 
Note complete absence (atrophy) of left conchae, deviation of septum, and moderate atrophy of right conchae.

In spite of the name, inflammation in atrophic rhinitis is minimal. However, as you may expect there is notable resorption of the osseous and cartilaginous structures of the conchae. Different degrees of atrophy are shown in the next slide.
Several sections of nasal bones showing moderate to severe (total) atrophy of conchae. Coiled conchae normally produce air turbulence which, in turn produce impaction of inspired particles into the wall. Pigs with atrophic rhinitis may have a higher incidence of pneumonia. The effect on weight gain of this disease has been a subject of controversy.
There are several nasty sequels to nasal infection and rhinitis. Infection can spread:

- to sinuses causing sinusitis (S).
- through the “lamina cribosa” (small arrow) into the meninges and cause meningitis.
- through the Eustachian tube into the middle ear and cause otitis.
- through the Eustachian tube in guttural pouches (gp) and cause guttural pouch empyema.
- Via lymphatic vessels to retorpharingeal lymph nodes (ln) and cause lymphadenitis.
Sinusitis and Sinus Empyema

Bovine

- Note accumulation of inspissated purulent exudate in paranasal sinuses.

- Remember that drainage in sinuses (and guttural pouches in horses) is poor, therefore mucus and pus can easily accumulate in these structures.

- Accumulation of pus is referred as **empyema**, while accumulation of mucus is known as mucocele.

- Sinusitis in cattle can be caused by improper dehorning.
Guttural Pouches

- Guttural pouches are normal dilations of Eustachian tubes in horses.
- Eustachian tubes communicate the middle ear with the pharynx in all species.
- Note the **forceps** illustrating the communication between pharynx and guttural pouches.

Mucus is constantly produced and removed from guttural pouches into the pharynx where this material is swallowed. Obstruction of flow may result in accumulation of mucus known as "**guttural pouch mucocele.**" Like other parts of the upper respiratory tract, alterations in the defense mechanisms may result in secondary bacterial or fungal infection.

Infections with pyogenic organisms often results in accumulation of pus which is referred as "**guttural pouch empyema.**"

Other abnormalities of guttural pouches include **tympani** (accumulation of gas) and fungal infections (guttural pouch mycosis).
Note swelling and distention of the parotid region and discharge from the nostrils.

Empyema of guttural pouches can be uni- or bilateral and it is often a sequel of upper respiratory infection with *S. equi* (Strangles) or other pyogenic bacteria.

Clinical signs included intermittent nasal discharge, enlargement of retropharyngeal lymph nodes, and parotid swelling. In severe cases guttural pouch empyema can interfere with swallowing (dysphagia).

(Courtesy of Dr. Jeanne Lofstedt)
Guttural Pouch Empyema

• Note thick purulent exudate in a guttural pouch (asterisk).

• Bacteriologic examination is required for identification of an etiologic agent.

• Endoscopic examination reveals purulent exudate in guttural pouches.
Epidemiology
The overall incidence of nasal tumors in animals is low. These tumors are 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. Enzootic nasal carcinoma may be enzootic for ruminants in some geographical areas.

Pathology
In general, 80% of nasal tumors are malignant. 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.

Clinical Signs
Clinical signs are variable but in severe cases, there may be craniofacial deformation, exophthalmia, and metastasis. 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.
• Note invasion of the nasal cavity by a large tumoral mass (asterisks).

• The most common nasal stromal tumors are fibrosarcoma, osteosarcoma, and chondrosarcoma and the most common epithelial tumors are carcinoma, adenocarcinoma and undifferentiated carcinoma.

• Biopsies or cytological examinations of the tumor are required to identify the cell origin and arrive at a final diagnosis in the live animal.
This cat suffered from progressive swelling and deformation of facial bones. The cat also had loose teeth.

Cytological examination of nasal lavage fluid demonstrated malignant epithelial cells and the diagnosis of a nasal carcinoma was made.

Because of the poor prognosis, the cat was euthanized and sent to postmortem examination.
Feline Nasal Carcinoma

Cut surface of the nasal cavity.

Note large mass arising from the nasal epithelium. This tumor was locally invasive, but there was no evidence of metastasis in this cat.

Histological section H&E.

Note sheaths of neoplastic epithelial cells with eosinophilic cytoplasm, large nuclei containing prominent nucleoli.
Equine Nasal Fibrosarcoma

Note large tumoral mass occupying the entire left nasal cavity and pressing on the nasal septum.

Remember that nasal tumors can become rather serious because of their close association to the brain and nerves.
Nasal Carcinoma

Metastasis to the Brain

Nasal tumor eroding the cribiform plate (arrows)

Coronal section
Metastatic nasal carcinoma (arrow)

Tomography
Brain metastasis (arrow)
Endemic Nasal Carcinoma or “Endemic Ethmoidal Tumor” is a retroviral-induced neoplasia of sheep, goats and cattle. In some endemic regions the incidence may be considerable. Clinical signs are nonspecific (hemorrhage) and the tumor often becomes infected causing mucopurulent nasal discharge. Note the large mass occupying the nasal cavity.
• **Ventral view of equine head.** Note the two occipital condyles (C) and the guttural pouch mucosa with mycotic lesions on the surface. Mycotic lesions consist of well-demarcated plaques of exudate (arrows).

• **Guttural pouch mycosis** may result in the erosion and rupture of a carotid artery with profuse hemorrhage through the nasal cavity (epistaxis).

• In other instances, **thrombosis of a carotid artery** due to guttural mycosis may result in brain thromboembolism (ataxia /blindness).
Necrotic Laryngitis (Calf Diphtheria)

Note the plaques of fibrinonecrotic diphtheritic exudate (asterisk) in the larynx of these two calves. This lesion is the result of a secondary infection caused by *Fusobacterium necrophorum* in animals in which the integrity of the normal laryngeal mucosa has been compromised such as in viral infections (i.e. IBR), trauma, etc.

Pieces of exudate may be aspirated into the lung and cause bronchopneumonia.

In pigs the lesions associated to secondary infection (*Fusobacterium necrophorum*) are generally restricted to the oral mucosa and the condition is referred to as oral necrobacillosis (diphtheria).
Viral Tracheitis

Note hyperemic mucosa in the trachea affected by pure viral infection alone. Injury, inflammation and repair for trachea and bronchi are identical to those previously described for nasal cavity. Pure viral tracheitis are rarely seen at necropsy since they are not fatal unless complicated with secondary bacterial infection.

Viral + Bacterial Tracheitis

Note the accumulation of fibrinonecrotic exudate in the trachea (bottom) with secondary bacterial infection.

Tracheitis is generally found along with rhinotracheitis, laryngotracheitis, tracheobronchitis or bronchopneumonia.
Suppurative Tracheitis

Note plaques of purulent exudate on the surface of the tracheal mucosa.

The mucosa is also notably hyperemic due to an underlying viral infection. Affected mucosa was subsequently complicated with a secondary bacterial infection.

In cattle, this lesion is often seen in IBR, BRSV and PI-3 virus infections.

In horses suppurative tracheitis may be a sequel to EVR infection.
Fibrino necrotic (Diphtheritic) Tracheitis

Note the necrotic mucosa detaching from the trachea (diphtheritic membranes).

This cow was accidentally dosed with a disinfectant (ammonium chloride).

Note the fibrino-necrotic exudate on tracheal mucosa (diphtheritic membranes).

This steer died of IBR
Canine lungs with a parasitic bronchitis caused by *Oslerus (Filaroides) osleri*.

Close-up of multiple parasitic nodules in the tracheobronchial bifurcation. Insert: Histological view of the nodules in the bronchial lumen (BL) lined by ciliated epithelium (arrows) and containing coiled parasites (asterisks).

*Oslerus osleri* is a metastrongyle parasite found worldwide that generally affects young dogs.

Parasitic nodules are typically seen at the tracheobronchial bifurcation but occasionally nodules extend deeper into the lung.

Clinical signs with *Filaroides osleri* are only observed (cough, inspiratory wheezing, exercise intolerance, etc) in severe parasitic infections.
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THE END