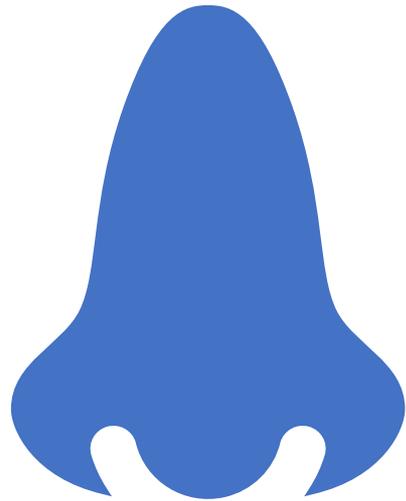
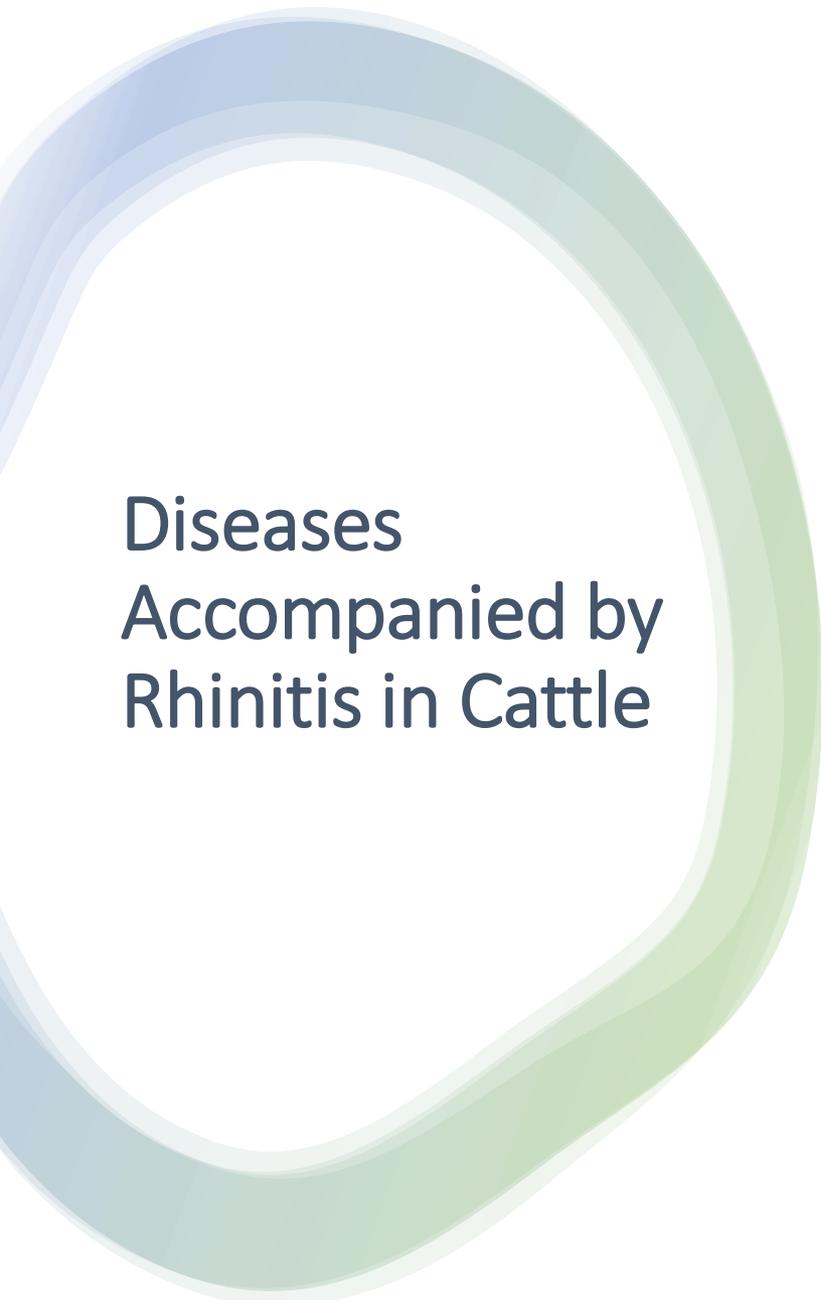


Respiratory System Pathology

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Specific Diseases of the Nasal Cavity and Sinuses



Diseases Accompanied by Rhinitis in Cattle

- Infectious Bovine Rhinotracheitis (IBR) /
Red Nose

Infectious
Bovine
Rhinothacheitis
(IBR) / Red
Nose

Acute, contagious: Inflammatory lesions in the upper respiratory tract, trachea, and conjunctiva

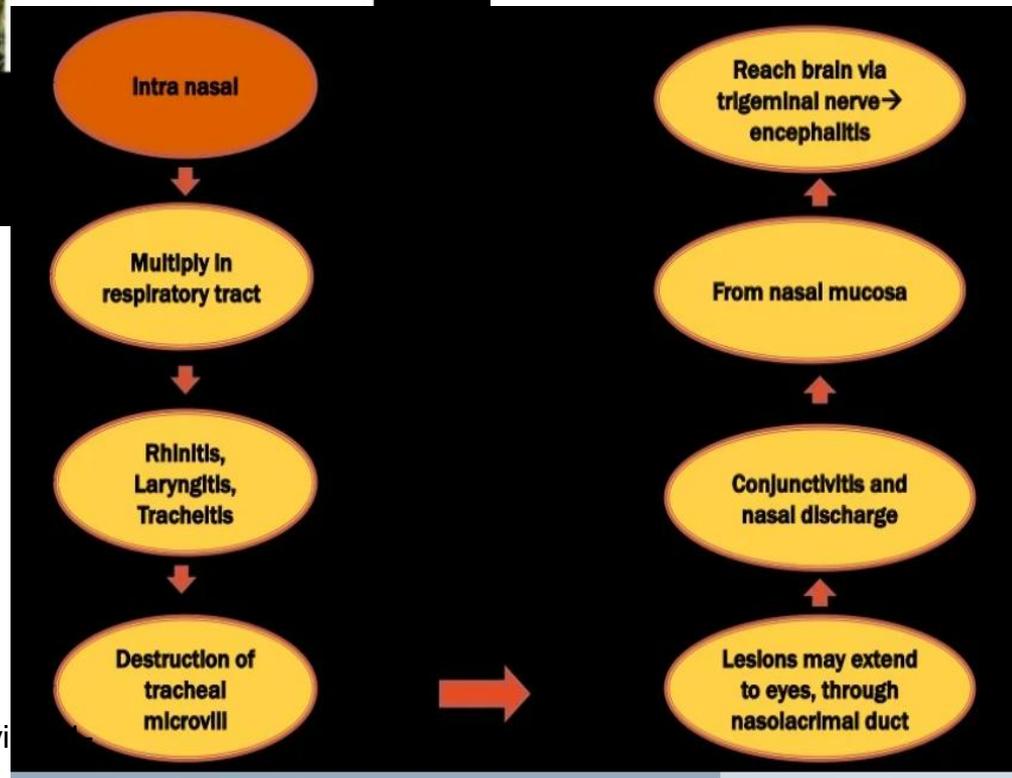
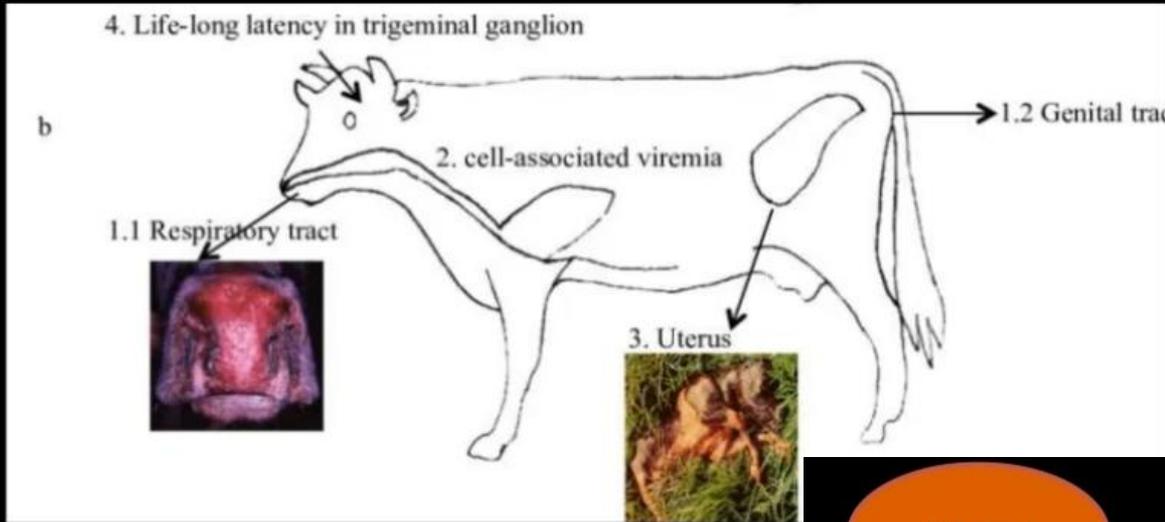
BHV-1: Infectious pustular vulvovaginitis (IPV) and balanoposthitis

BHV-1.1 & BHV-1.2a → abortus

Meningoencephalitis in young calves (in generalized form)

In pigs: vaginitis and balanitis

- Widespread globally
- Serological findings indicate that infection is more prevalent than the disease itself
- More common in crowded and enclosed feedlots
- Mild course in dairy cows
- In feedlot cattle → purchase of new animals + animal movements → epidemic spread
- High morbidity, many cases are mild and go unnoticed
- Low mortality; may reach up to 30% during outbreaks
- Chronic cases ~10%, mortality rate 3%



- **FINDINGS:**

- **Respiratory form**

- Fever
- Increased and rapid respiration / Mouth breathing
- Cough
- Serous to mucopurulent nasal discharge
- Dyspnea
- Hyperemia / hemorrhage in the nasal mucosa

- **Eye Form**

- Konjunctivitis / lacrimation

- **Digestive system form**

- Diarrhea
- Erosion / Ulcer

- **Genital system form**

- Abortion / IPV / IBP

- **Nervous system form**

- Encephalitis
- Incoordination
- Blindness
- Convulsion

- **Clinical Findings**

- Fever, increased respiratory rate, cough, serous nasal discharge
- Dilated nostrils, mouth breathing, and lacrimation
- In prolonged cases: mucopurulent discharge + inspiratory dyspnea

- **Gross Findings**

- In mild cases: seromucinous rhinotracheitis, conjunctivitis
- In severe cases (especially with secondary bacterial infections):
 - Acute diffuse inflammation
 - Mucopurulent exudate
 - Focal hemorrhages, erosions, and ulcers

In Severe Cases

- Widespread fibrinopurulent / fibrinonecrotic membranes in the nasopharynx, larynx, and trachea

The affected region is variable

- During outbreaks, the larynx, pharynx, and trachea are most commonly affected
- **The severity of lesion** is generally dependent on secondary bacterial infections
 - *Pasteurella spp.*, *Mycoplasma spp.*, *Fusobacterium necrophorum*

- **Mild cases:** Serous → mucopurulent inflammation, with minimal epithelial necrosis
- **Severe (fatal) cases:** Marked epithelial necrosis, fibrinonecrotic layer on the surface
- Severe vascular reaction in the underlying tissue, with infiltration of neutrophils and mononuclear cells
- **(12 hours)** Pale, vacuolated/granular cytoplasm in cells of the nasal septum and conchae
- **(24 hours)** The same cells are observed in the pharynx
- **(36 hours)** Intranuclear inclusions → pale eosinophilic foci with chromatin margination against the nuclear membrane

- Later, **homogeneous pink inclusions** form.
- **A slight halo** is seen around them, and the chromatin is completely marginated to the nuclear membrane.
- **After 2–3 days (≈72 hours)**, the inclusions disappear → usually observed in experimental cases, with **no diagnostic value**.
- Subsequently, **nonspecific ulcers** develop.

- The role of BHV-1 in pneumonia is **controversial**.
- The effect of the virus is **difficult to demonstrate** in experimental and natural cases.
- Its primary role → **predisposition to bacterial pneumonia**.
- In mild infections → **the lungs are not affected**.
- In severe infections → **pulmonary immunity is impaired**, secondary bacterial pneumonias develop, and may result in death.

Secondary Infections and Lesions

- Most commonly **Pasteurella spp.** → fibrinous pneumonia ± pleuritis
- Upper/lower respiratory tract obstruction → **interstitial emphysema**
- Occasionally, **severe viral lesions** (without secondary agents)
- In severe cases → **necrotic bronchitis, bronchiolitis, and serofibrinous exudate** in the alveoli

Clinical Findings in Neonatal Calves

- **Typically in calves <1 month old** → generalized disease
- Clinical signs: **Fever, serous ocular and nasal discharge, respiratory distress, anorexia, depression, necrobacillosis-like lesions in the larynx**

Gross Findings in Neonatal Calves:

- **Acute rhinitis, erosive pharyngitis, marked hyperemia, yellow crusts**
- **Conjunctiva:** Hemorrhages, ophthalmia (pink eye)
- Epiglottis: Similar findings
- **The lower respiratory tract is generally not affected**

Gross Findings

- Sometimes **diffuse hemorrhagic bronchopneumonia** is observed.
- The most prominent changes are in **the mucosa of the esophagus and forestomachs**; caseous material covered with clotted milk residues adheres to necrotic epithelium.
- **Necrosis involves the full thickness of the epithelium**, accompanied by dense neutrophilic infiltration.
- **Inclusion bodies are present in the vesicular nuclei** of viable epithelial cells.
- In systemic infection, **acute lymphadenitis with cortical necrosis** develops (especially in lymph nodes of the upper respiratory tract).
- **In the liver**, especially in the right lobe, 1–2 mm miliary white necrotic foci are frequently observed; rarely found in the kidneys and spleen as well.

Abortion Findings

- In abortion cases, **necrotic placentitis** and fetal edema are observed.
- It usually occurs during the **5th–6th month** of gestation.
- The fetus is in an **autolytic** state due to death occurring approximately 2 days before abortion; no distinct macroscopic lesions are seen.
- **Microscopic findings:** Necrosis and leukocyte infiltration in many parenchymal organs—primarily the liver—as well as in lymph nodes and placenta.
- The most characteristic finding: Severe hepatic necrosis, which **can sometimes be confused with listeriosis**.
- Necrosis may also be present in other organs.
- **Inclusion bodies are not detected in autolytic fetuses.**

Malignant catarrhal fever

(Coryza gangrenosa bovum)

- It is caused by a **herpesvirus**.
- In experimental cases, the incubation period is 14–60 days.
- This is followed by high fever, catarrhal conjunctivitis, and rhinitis.
- Additionally, mucopurulent discharge is observed from the eyes and nose.
- Well-demarcated, irregularly shaped erosions appear on the nasal mucosa.
- These erosions are covered with mucopurulent exudate.
- **Microscopically**, epithelial necrosis, occasionally small vesicle formation, and cellular infiltration in the stroma are observed.
- The erosions are covered by cellular exudate.
- Involvement of the oral mucosa, esophagus, rumen, reticulum, and omasum is also seen with congestion, edema, and erosions.

- In the diagnosis of the disease, the presence of **vasculitis** (fibrinoid-necrotic) in the sublesional areas is a specific feature.
- In the vessels, endothelial swelling, hyperplasia, medial necrosis, and infiltration of macrophages, plasma cells, and eosinophils in the adventitia are observed.
- Vasculitis can be seen in all organs and tissues.

Tuberculosis

- In early generalization and chronic organ tuberculosis, cauliflower-like or polypoid nodules and ulcers may develop.
- **Actinomycosis and Actinobacillosis**
- In sheep, actinomycotic lesions may be widespread or localized only in the nasal cavity.
- In sheep, nasal or facial actinobacillosis usually occurs during dry seasons, possibly related to lip injuries.
- Caseous, fistulous tracts frequently observed in the subcutaneous tissue and nasal submucosa should raise suspicion of this infection.

Necrobacillosis

- The causative agent is *Fusobacterium necrophorum*.
- It typically localizes in the pharynx, larynx, trachea, mouth, esophagus, lungs, and other parts of the digestive system.
- In calves, lambs, and piglets, it is more commonly seen in the nasal region.
- The disease occurs under conditions of general health deterioration, mucosal injury, presence of other diseases, and poor hygiene.
- As in other regions, focal coagulative necrosis lesions form in the nose; dry, grayish-red in color, surrounded by a hyperemic ring.



**Diseases
Accompanied by
Rhinitis in Pigs**

Inclusion Body Rhinitis

General Information

- It is mostly seen in **1–5 week-old suckling piglets**.
- It follows an **acute** or **subacute** course. Temel özellikleri:
 - Seromucous rhinitis
 - Hypertrophy of glandular epithelium (**cytomegaly**)
 - **Syncytium formation**
 - **Intranuclear inclusion bodies**
- Causative agent: ***Cytomegalovirus (Herpesviridae, Beta subfamily)***
- Widespread in Europe; observed in all pig-rearing regions.
- Transmission: **via nasal discharge, urine, and pharyngeal secretions** of infected animals.

- **Clinical Findings**
- **Incubation period:** ~10 days
- **Symptoms:**
 - Mild fever
 - Rhinitis (initially serous, later catarrhal/purulent)
 - Lacrimation
- Environmental stress (cold, overcrowding) increases morbidity (up to 100%)
- Mortality is low, but **increases with secondary complications** (sinusitis, otitis media, pneumonia)

Pathogenesis

- The virus **replicates in the glandular epithelium of the nasal mucosa** → viremia.
- Viremia phase: 2–3 weeks.
- It then persists in pulmonary macrophages.
- In piglets, **death usually occurs during the generalization phase.**
- Transplacental transmission is questionable.

Gross Findings

- Serous–catarrhal rhinitis
- If there is a bacterial complication → purulent rhinitis
- Swelling and hyperemia of the mucosa, with mucous/purulent exudate on the surface

Histopathological Findings

- **Specific diagnostic findings:**
 - Basophilic intranuclear inclusions in glandular and ductal epithelium
 - **Cytomegaly** and **syncytium formation**
- **Squamous metaplasia** in the mucosa
- Necrotic glands → lymphocyte infiltration and mucosal collapse
- Mild vasculitis and lymphocyte infiltration
- Glandular **regeneration occurs** (surface epithelium grows inward)

Systemic Dissemination

- Typical inclusions:
 - Lacrimal gland
 - Harderian gland
 - Renal tubules and glomeruli
 - Hepatocytes
 - Adrenal gland, lymph nodes
- **Focal necrosis** in parenchymal tissues (may be massive in the liver)
- Petechial hemorrhages, subcutaneous edema, anemia
- In the CNS: inclusions in glial cells and focal gliosis
- In pregnant animals: mummification, premature birth, neonatal deaths

Differential Diagnosis

- Atrophic rhinitis (early stage)
- Aujeszky's disease
- Classical swine fever
- Other bacterial rhinitides (in case of complications)

Atrophic Rhinitis in Pigs

(Rhinitis atrophicans contagiosa suum, Dystrophic rhinitis)

- **General Characteristics**
- Characterized by atrophy of the turbinates in young pigs
- Nasal deviation and shortening are observed in advanced cases
- Chronic in course, endemic in nature
- Not fatal but causes growth retardation and decreased productivity
- Leads to **significant economic losses**

- **Etiology**
- **Primary agent:** Toxigenic isolate of *Pasteurella multocida* → protein toxin
- **Predisposing factors for colonization:**
 - *Bordetella bronchiseptica* (cytotoxic, synergistic effect)
 - *Haemophilus parasuis* (likely contributes)
- **Predisposing factors:**
 - Genetic susceptibility
 - Environmental conditions
 - Nutritional deficiencies (especially Ca/P imbalance)
 - Viral infections (e.g., Cytomegalovirus)

Transmission

- By contact and aerosol-droplet route
- Older pigs are the source of infection
- It can also be transmitted via animals such as mice, dogs, and cats

Clinical Findings

- Progresses slowly
- In acute cases: sneezing, coughing, serous/mucopurulent nasal discharge
- Erosions on the mucosa → purulent/bloody discharge
- **Blood clots** may be expelled during sneezing
- Shortening, deviation, and asymmetry of nasal and facial bones
- **!!**: black material adhering under the eyes (nasolacrimal duct obstruction)
- In severe cases → facial deformity, respiratory distress

Pathogenesis

- Toxins impair osteoblast function and increase osteoclast activity
- Calcium cannot be retained in bones, and osteogenesis is reduced
- **Result:** bone destruction, turbinate atrophy, nasal deformity
- **The ventral turbinates** are most affected (the most active regions in young pigs)

Gross Findings

- Initially serous → catarrhal → purulent rhinitis
- Edema and exudate in the mucosa, sometimes a pale/dry appearance
- Most prominent change: atrophy of the ventral turbinates
- Turbinates → soft, asymmetric, sometimes completely absent
- **Facial deformity:**
 - Unilateral → deviation
 - Symmetrical → upward turning of the nose
- A cross-section between the **first and second premolars** is very useful in diagnosis.

Histopathological Findings:

- In the early stage: increased osteoclast activity, insufficient osteoblast proliferation
- Later: development of fibrous tissue and gelatinous matrix
- Vascular changes such as arteriolar hypertrophy

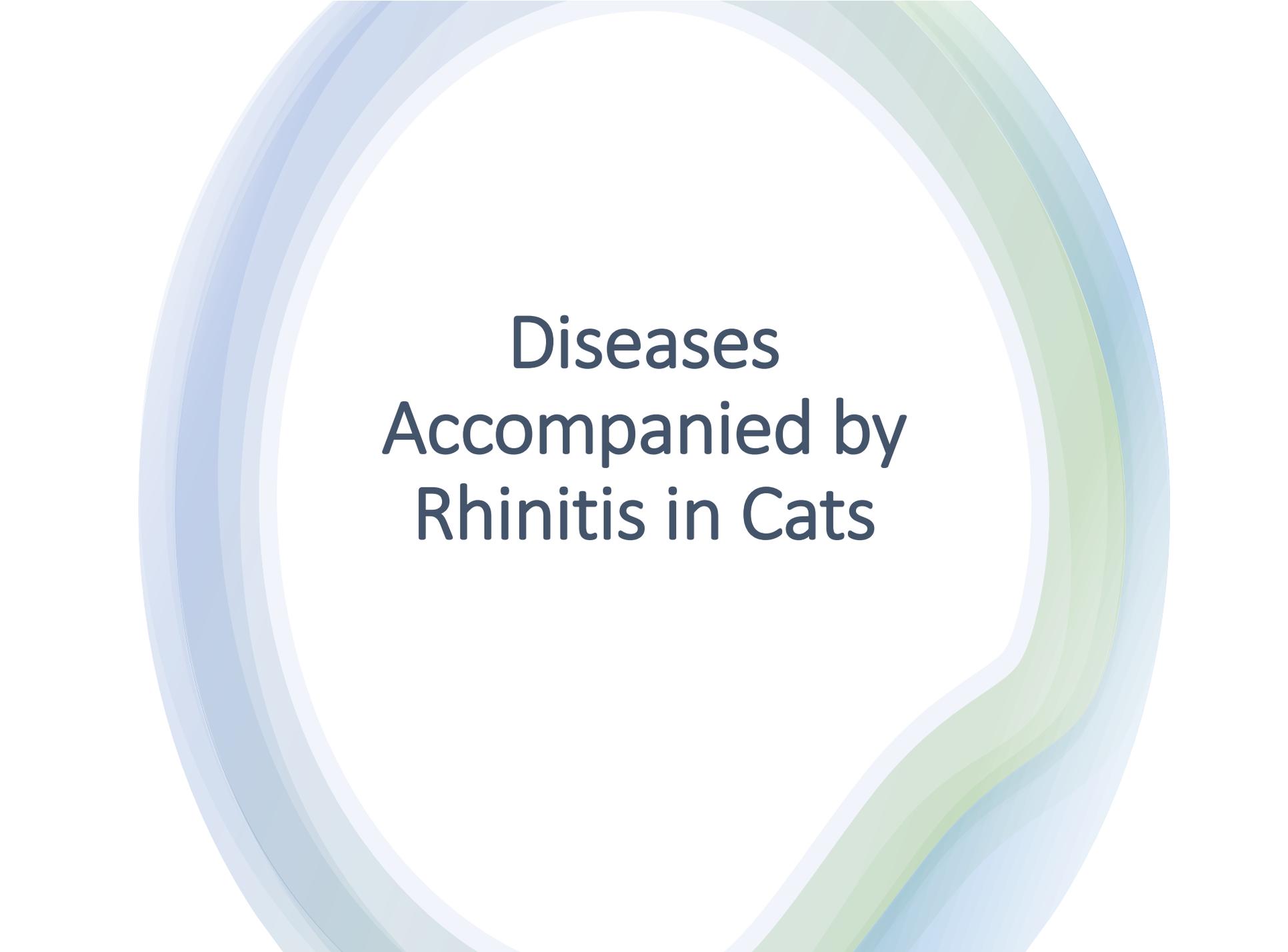
Differential Diagnosis

- If facial deformity is present, diagnosis is easy.
- If deformity is absent: it may be confused with inclusion body rhinitis and other types of rhinitis in pigs.



**Diseases
Accompanied by
Rhinitis in Dogs**

- There is no specific infection in dogs that affects only the nasal cavity and sinuses.
- Acute rhinitis usually occurs as part of general respiratory diseases caused by various viruses.
- These viruses include *canine distemper virus*, *canine adenovirus types 1 and 2*, *canine parainfluenza virus*, *reovirus*, and *canine herpesviruses*.
- Viral lesions in the respiratory tract are usually transient; however, the effects of these viruses on other tissues—such as encephalitis in distemper can be fatal.
- As in other species, viral infections of the respiratory tract in dogs may also result in secondary bacterial rhinitis and sinusitis.
- The most commonly encountered agents in bacterial rhinitis in dogs are *B. bronchiseptica*, *E. coli*, and *P. multocida*.



**Diseases
Accompanied by
Rhinitis in Cats**

Feline Respiratory Disease Complex

- **Feline viral rhinotracheitis**, characterized by inflammation of the upper respiratory tract, holds the most important place within this complex.
- Another significant disease in this group is *feline calicivirus* infection.
- *Feline reovirus* and *Chlamydia psittaci* adapted to cats (the agent of feline pneumonitis) are of lesser importance.
- *Mycoplasma felis* acts as an opportunistic pathogen, combining with viral and chlamydial infections and causing mucopurulent conjunctivitis.

Feline Viral Rhinotracheitis

Causative agent: Feline herpesvirus-1 (FHV-1)

- Responsible for approximately 80% of feline respiratory disease complex cases

Clinical signs: Fever, sneezing, salivation, mouth breathing, cough, serous → mucopurulent nasal and ocular discharge

! In young kittens or debilitated animals—especially those immunosuppressed due to feline leukemia or feline immunodeficiency virus infections—mortality is high, whereas many cats recover within 7–14 days.



Gross Findings

- The distribution of macroscopic lesions varies according to the site of viral replication.
- The virus primarily replicates in the **epithelium of the nasal cavity, pharynx, soft palate, conjunctiva, and tonsils.**
- The trachea is less affected.
- Initial stage: **Serous inflammation**, progressing within a few days to mucopurulent/fibrinous.
- Severe cases: **Fibrinous rhinotracheitis**, sometimes acute pneumonia.
- Tonsils: Enlarged with petechial hemorrhages.
- Regional lymph nodes: Swollen, edematous, and red.
- Tongue: Rare ulcers (in severe cases).
- Eyes: Purulent conjunctivitis may progress to ulcerative keratitis.

- Ulcers on the tongue are rarely observed and occur only in severe cases.
- In contrast, **calicivirus infections** frequently cause **vesicular and ulcerative lesions** on the tongue, hard palate, or nose.
- Ocular lesions are mostly in the form of **purulent conjunctivitis**, but may progress to **ulcerative keratitis**.

Larinks-Trakea

Histopathological Findings

- Multifocal necrosis and **intranuclear inclusions (Cowdry type A) in the epithelium**
- In necrotic foci: erosion, ulceration, fibrin deposition, and neutrophilic infiltration
- **Tonsils and lymph nodes:** acute inflammation with focal necrosis
- **Severe cases:** necrotizing bronchitis/bronchiolitis, interstitial pneumonia, serofibrinous exudate in the airways
- Secondary bacterial pneumonia is commonly observed

- The virus is virulent enough to cause severe lesions; however, in many cases, it plays a role in the formation of **purulent lesions** when combined with secondary bacterial infections involving *P. multocida*, *B. bronchiseptica*, *Streptococcus spp.*, and *Mycoplasma felis*.

- Its association with abortion is questionable (not proven in natural cases).
- Experimental inoculation in pregnant cats has resulted in reports of abortion and neonatal infections.
- In experimental cases: necrosis in osteogenesis regions of bones along with inclusion bodies.
- Degeneration may be observed in olfactory nerves, but no brain lesions are present.

Calicivirus infection (*Feline infectious coryza*, *feline cold*, *feline influenza*)

- In recent years, **calicivirus infection** has become one of the two major respiratory diseases in cats.
- The other is **feline viral rhinotracheitis**.
- Although clinical signs often overlap with herpesvirus infection, both viral diseases may also occur simultaneously.
- The most important feature of calicivirus is its **affinity for oral and pulmonary epithelium**.
- The most significant findings of the disease are **oral ulceration** and **primary pneumonia**.
- It causes milder inflammatory changes in the **upper respiratory tract and conjunctiva**.

Clinical Findings:

Fever, rhinitis, conjunctivitis followed by nasal and ocular discharge, vesicles and ulcerations in the oral cavity, and occasionally pneumonia are observed.

As a result of bacterial complications, purulent rhinitis develops and tearing increases.

These lesions also occur in **rhinopneumonitis**.

However, in calicivirus infection, oral ulceration is very pronounced, whereas, unlike the other disease, **keratitis or corneal ulcers** are absent.

Macroscopically, **ulcers** are found in the mouth, on the tongue, the hard palate, and along the nasal margins.

The severity and distribution of the lesions depend on the virulence and tissue tropism of the calicivirus strain, as well as the intensity of the infection.

- Ulcers are initially preceded by a transient vesicular stage.
- They are commonly found on the **dorsal and lateral surfaces of the tongue**, and less frequently on the **hard palate and nose**.
- **Serous or mucoid rhinitis and conjunctivitis** are less commonly observed signs.
- These signs are frequently seen in natural infections, while in experimental cases they occur following **intranasal inoculation**.
- However, in the absence of bacterial complications, infected cats generally recover within **7–10 days**.

An interstitial type of pneumonia develops, which distinguishes it from feline viral rhinotracheitis, where bronchointerstitial pneumonia is observed.

Lesions are generally observed in the anteroventral regions of the lungs, although irregular focal areas can also be present in other parts.

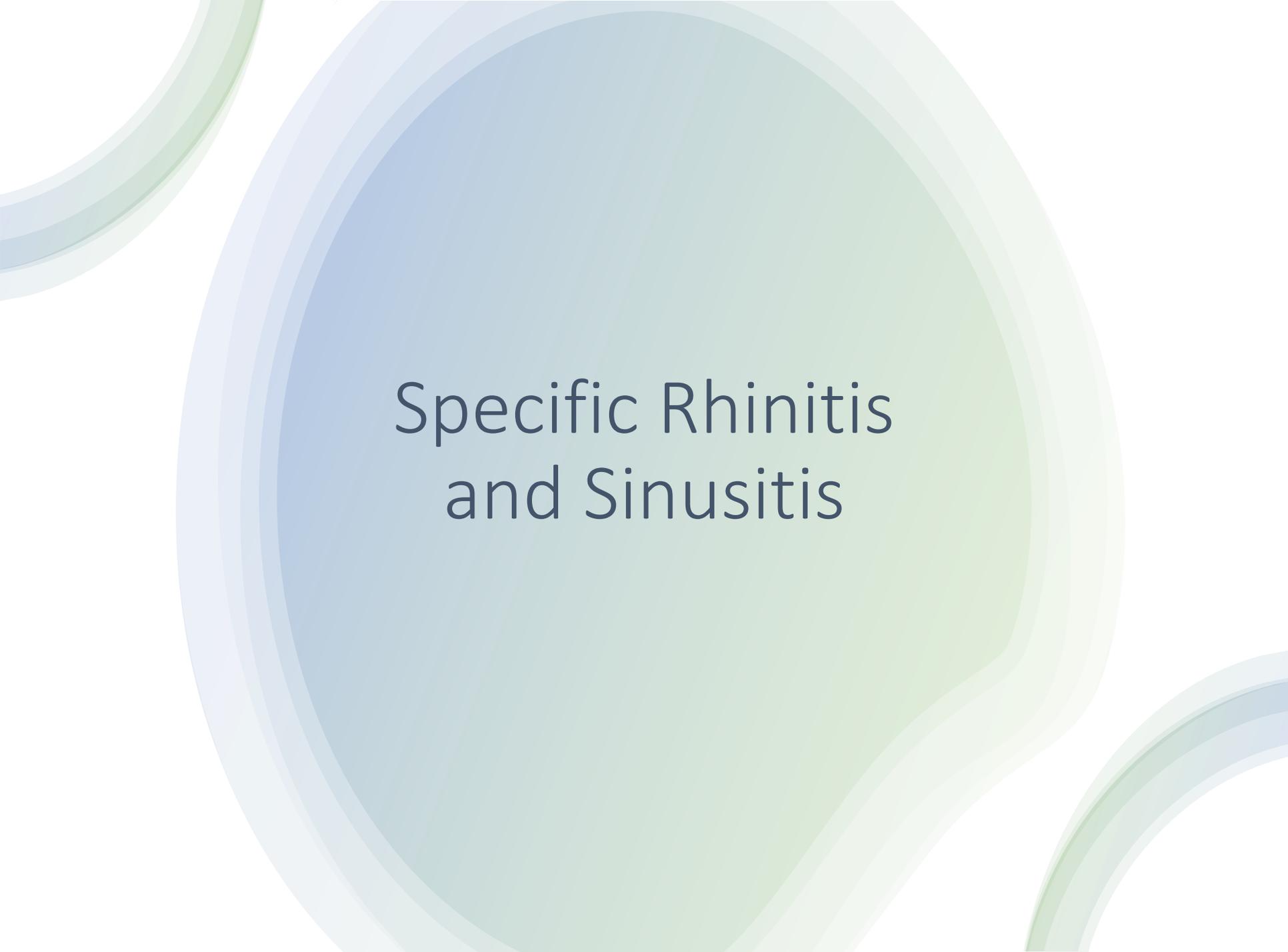
Initial lesions appear bright red to grayish-red in color, later becoming gray-tan as resolution occurs.

Histopathologically:

- **Interstitial pneumonia** begins with **necrosis of type I alveolar cells**.
- This is related to the virus's tropism for **type I alveolar epithelial cells**.
- During the first few days, necrosis occurs in these cells, accompanied by **serofibrinous and neutrophilic exudation** and sometimes the formation of **hyaline membranes**.
- At the beginning of the second week, **hyperplasia of type II alveolar epithelial cells** begins (fetalization, epithelialization).
- **Hyaline membranes** form in the alveolar lumens.
- Approximately **30 days after infection**, **fibrosis** develops as connective tissue cells progressively increase in the alveolar walls.
- Due to **secondary bacterial infections**, the condition may transform into other types of pneumonia.
- **Differential diagnosis** – In addition to **feline rhinotracheitis**, it may be confused with complicated cases involving **chlamydia, mycoplasma**, and other **bacterial agents**.

Feline Chlamydiosis

- This is a **persistent respiratory tract infection** in cats caused by **Chlamydia psittaci**.
- The infection results in **mild conjunctivitis** (similar to human trachoma) and **rhinitis**.
- However, in severe cases, a **mild and transient bronchointerstitial pneumonia** develops.
- This pneumonia was formerly referred to as "**feline pneumonia**."
- **Feline reovirus** and **Mycoplasma** species can also cause **mild upper respiratory tract infections**.
- The clinical signs and lesions overlap with those seen in **viral rhinotracheitis, calicivirus infection, and chlamydiosis**.
- Many respiratory tract infections in cats are compounded by the **immunosuppressive effect of feline leukemia virus**.



Specific Rhinitis and Sinusitis

Mycotic Rhinitides

Aspergillosis:

- Most common in dogs
- Chronic, necrotic, granulomatous foci
- Destruction of turbinates, sometimes with bone invasion

Cryptococcosis:

- More common in cats
- Gelatinous lesions, encapsulated fungi → weak inflammation
- Facial swelling, spread to bone and nervous system

Zygomycosis:

- In horses and sheep
- Ulcerative, granulomatous masses in the nasal and facial regions

Rhinosporidiosis:

- In horses, cattle, goats, and birds
- Chronic, polypoid rhinitis (polyps bleed easily)
- Causative agent: *Rhinosporidium seeberi*

Parasitic Rhinitides

Oestrus ovis (sheep):

- Larvae in the nasal cavity and sinuses
- Sneezing, discharge, wheezing, and sometimes meningitis

Linguatula serrata:

- Arthropod residing in the nasal cavity of carnivores
- Causes catarrhal inflammation

Schistosoma nasalis:

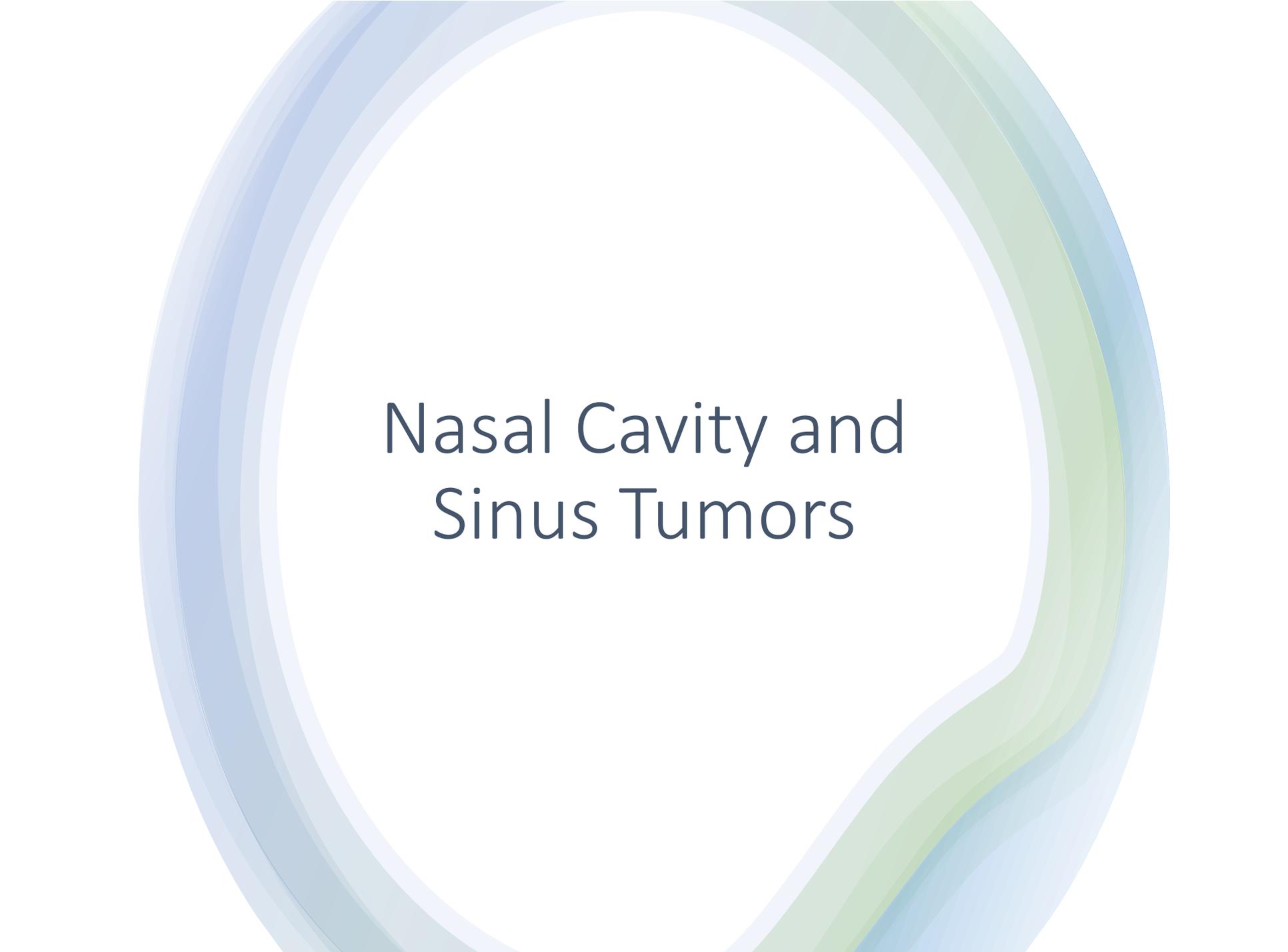
- In goats, horses, and cattle
- Lesion resembling granulomatous rhinitis

Limnatis spp. (sülükler):

- Transmitted through drinking water
- Anemia, edema, respiratory distress, and bloody discharge from the nose and mouth

Habronema:

- Granuloma in the nasal cavity (similar to a “summer sore” on the skin)

A decorative graphic consisting of several concentric, overlapping circular bands. The bands are colored in shades of light blue and light green, creating a soft, ethereal effect. The center of the graphic is white, where the text is located.

Nasal Cavity and Sinus Tumors

- Rare but serious tumors, usually primary in origin
- Most common in dogs (especially long-nosed breeds such as Collies and German Shepherds)
- In cats: typically located in the vestibule; in horses: in the maxillary sinus

Clinical findings:

- Catarrhal/mucopurulent discharge, epistaxis, ocular discharge
- Sneezing, snoring, dysphagia
- Facial swelling, exophthalmos, neurological symptoms
- **Benign tumors:** Nodular and well-circumscribed
- **Malignant tumors:** Soft, friable, invasive, and destructive to bone

Histomorphology:

- **Epithelial:** Papilloma, squamous cell carcinoma, adenoma, adenocarcinoma, anaplastic carcinoma
- **Mesenchymal:** Fibrosarcoma, chondrosarcoma, osteosarcoma, myxoma, fibroma
- **Species-specific prevalence:**
- **Dog:** Adenocarcinoma, anaplastic carcinoma, fibroma
- **Cat / Horse:** Squamous cell carcinoma
- **Horse / Cattle:** Osteoma
 - Metastasis is rare; local invasion is more common.

Endemic Ethmoidal Tumor

- **Originates from the ethmoturbinate region; carcinoma type**
- **Occurs in various animal species (sheep, goats, cattle, horses)**
- **Endemic distribution by country:**
 - **Sheep:** Germany, Spain, USA, France, Canada
 - **Goats:** India, Spain, France
 - **Cattle:** Scandinavian countries, Brazil, India, South Africa
 - **Horses:** Scandinavian countries
- **Characteristics:**
 - Affects many animals within a flock; may persist for years
 - Different species on the same farm may be affected
- **Etiology:**
 - Ultrastructurally detected **retrovirus-like (Type C) particles**
 - Though not definitively proven, a **viral origin** is suspected (**similar to ovine pulmonary adenomatosis**)
- **In sheep:** Known as "**contagious papillary adenocarcinoma**"
- Local, progressive, and destructive
 - May present as **adenopapillomatosis** or **adenocarcinoma**
- **In cattle:** Regional lymph node metastasis has been reported



Pharynx and Air Sacs



Anomalies:

- More common in horses and dogs.
- Respiratory difficulty worsens with exercise, stress, or heat.
- **Example:** Brachycephalic Airway Syndrome (elongated soft palate, nasal stenosis, laryngeal edema/collapse).

Subepiglottic / Pharyngeal Cyst:

- Especially in horses, originating from remnants of the thyroglossal duct.
- Filled with mucus and lined by epithelium

Pharyngitis (Inflammation of the Pharynx):

- Nonspecific causes (trauma, tubing, foreign body).
- **Clinical signs:** dysphagia, respiratory distress, abscess formation.

Pharyngeal Diverticulum (pig):

- Located dorsally to the entrance of the esophagus.
- Foreign bodies may cause inflammation, obstruction, and cellulitis.

Pharyngeal Lymphoid Hyperplasia in Horses:

- Seen in 2–3-year-old racehorses.
- Noisy breathing, airway obstruction.
- **Endoscopy:** white plaques or nodules.
- **Histology:** lymphoid hyperplasia.

Guttural Pouch Mycosis (especially *Aspergillus*):

- Usually unilateral, associated with inhalation of moldy hay.
- **Lesion:** fibrinonecrotic exudate, vasculitis, fungal hyphae (mycelia).
- **Clinical signs:** epistaxis, dysphagia, Horner's syndrome, laryngeal paralysis.

Larynx

- **Anomalies:**
 - **Hypoplastic epiglottis** (horse, pig)
 - **Laryngeal paralysis** (especially left side, in horses — "roaring")
 - **Cause:** nerve degeneration, trauma, toxins, infection
 - **Clinical sign:** noisy (stridorous) inspiration
- **Dystrophic Changes:**
 - Calcification/ossification of the cartilages with age.
- **Circulatory Disorders:**
 - Hyperemia, edema, petechiae.
 - Edema in the epiglottis → risk of asphyxiation.
- **Laryngitis (Inflammation):**
 - Catarrhal, purulent, pseudomembranous.
 - In various species: necrobacillosis, diphtheroid membranes, abscesses.
 - In cats with panleukopenia: fibrinonecrotic laryngitis.
- **Laryngeal Contact Ulcer (cattle):**
 - Round ulcer on the arytenoid cartilage.
 - **Cause:** coughing, trauma, irritants, infection.
 - Often found incidentally at the slaughterhouse.

Laryngeal Paralysis (Hemiplegia laryngis, Roaring, Cornage)

Definition

- A common disorder in horses.
- Characterized by neurogenic atrophy of the dorsal and lateral cricoarytenoid muscles.
- Mostly occurs on the left side; rarely bilateral.

Causes

- **Primary:** Axonal degeneration (idiopathic)
- **Secondary:** Compression, inflammation, surrounding tissue diseases (e.g., strangles, guttural pouch diseases), vitamin deficiencies, neurotoxins (e.g., organophosphates, Halothane)

Anatomy: The left recurrent laryngeal nerve is longer → more susceptible.

Laryngeal Paralysis (Hemiplegia laryngis, Roaring, Cornage)

Pathogenesis

- Atrophy of the cricoarytenoid muscle → the larynx cannot fully open.
- The left arytenoid cartilage collapses into the laryngeal lumen.
- Reduced vocal cord vibration → noisy respiration (roaring).

Histopathological Findings

- **In the muscles:** Denervation atrophy
- **In the nerve:** Chronic demyelination, remyelination, Wallerian degeneration

Clinical

- Noisy inspiratory sound during exercise.
- Impaired airflow and reduced performance.

Occurrence Among Species

- **Horses:** Common in large-bodied, purebred horses
- **Dogs:** Bilateral; occurs in older and large breeds; may be congenital (e.g., husky, bull terrier). Associated with hypothyroidism or neuromuscular diseases
- **Cats:** Very rare

Trachea

Tracheal Shape Alterations

- May be congenital anomalies or acquired later in life.

Tracheal Collapse:

- Especially in small/miniature dog breeds.
- Dorsoventral flattening of the trachea.
- Cartilage bending and sagging of the dorsal membrane into the lumen.
- Foam and sometimes diphtheritic membranes may be observed.
- Usually affects the entire trachea, but may be limited to the cervical region.

Tracheal Collapse

Pathogenesis

- **The exact cause is unknown.**
- Microscopically: **Erosion/metaplasia** of the epithelium, cystic mucous glands, inflammation in the submucosa.
- **Qualitative and quantitative defects** have been reported in the cartilage.
- **In toy breeds**: chondrodysplasia, focal cellular depletion in cartilage, replacement by fibrous tissue.
- **Decreased chondroitin sulfate and calcium levels**, along with reduced chondrocyte density.
- Most commonly seen in **adult and obese dogs**.
- Sometimes develops following **infectious respiratory diseases**.

Tracheal Anomalies

- **Aggenesis:** Partial or total; extremely rare.
- **Hypoplasia:** Narrow lumen diameter throughout the trachea → sometimes accompanied by bronchial hypoplasia.
 - Most commonly seen in English Bulldogs; hereditary in origin.
- **Other Anomalies:**
 - **Horse** → Scabbard trachea (laterally flattened trachea)
 - **Dog and cattle** → Tracheoesophageal fistula

Acquired Tracheal Shape Alterations

- **External pressure:** Enlarged thyroid gland, lymph nodes, inflammation, or tumors.
- **Aging / Patent ductus arteriosus displacement:** Causes narrowing of the esophagus and trachea, leading to choking → dysphagia and respiratory distress.
- **Scabbard Trachea:**
 - **Horse** → narrowing of the lumen due to folding of the cartilage rings.
 - Scar tissue (e.g., following tracheotomy) may also contribute.

Tracheal Edema Syndrome in Feedlot Cattle (Honker Syndrome)

- Etiology: Unknown
- Occurs in large-framed cattle during summer months
- Clinical findings: Loud inspiratory noise (“honking”) Inspiratory dyspnea. Progresses to mouth breathing, recumbency → asphyxiation and death
- Macroscopic findings: Mucosal and submucosal edema in the distal trachea, with minimal hemorrhage
- Possible contributing factors: Body size, heat, exercise, trauma, pressure, dust, and feed toxins are suspected but not confirmed

Tracheitis (Inflammation of the Trachea)



Canine Infectious Tracheobronchitis (*kennel cough*)

- A highly contagious infection in dogs, characterized by sudden, paroxysmal coughing episodes.
- The term used for the disease is nonspecific similar to "common cold" in humans or "shipping fever" in cattle.
- Infection typically occurs when dogs from different origins are brought together.
- For this reason, it is commonly seen in **animal shelters**, commercial sales areas, and veterinary clinics.

Kennel Cough

- Clinically, the disease is characterized by a harsh, persistent, and often paroxysmal cough.
- Between coughing fits, the animals appear healthy.
- In some cases, there may be no detectable pathology beyond the cough; in others, rhinitis, pharyngitis, tonsillitis, or conjunctivitis may be present. Affected dogs usually recover, but clinical signs can persist for three weeks or longer. In some cases, secondary pneumonia may develop.
- The etiology of infectious tracheobronchitis in dogs is complex.
- Multiple pathogens and environmental factors are involved. The most important agent is *Bordetella bronchiseptica*. Other frequently involved viruses include: *Canine adenovirus type 2* (CAV-2) *Canine parainfluenza virus type 2* (CPIV-2) *Canine distemper virus*
- The severity of the disease is greater when multiple agents are involved or under stressful environmental conditions.
- Although less important, the following agents may also play a role: Infectious canine hepatitis virus
- Reovirus type I Canine herpesvirus Mycoplasma cynos These pathogens may act individually or in combination.
- The role of reoviruses, Mycoplasma, Pasteurella multocida, and other Gram-negative bacteria remains unclear.

Kennel Cough

- Depending on the causative agent, **gross findings may sometimes be absent**, or may range from **catarrhal to mucopurulent tracheobronchitis**.
- In progressive cases, **serous to mucopurulent rhinitis** and **cranioventral bronchopneumonia** may also be observed.
- The **tonsils, retropharyngeal**, and **tracheobronchial lymph nodes** are consistently **swollen and red**.
- **Microscopically**, varying degrees of **tracheobronchitis and bronchiolitis** are present.
- Findings range from **focal superficial necrotic tracheobronchitis and bronchiolitis** to more severe **mucopurulent inflammation**.
- There is typically **epithelial degeneration and necrosis**, with disorganization of the normal pseudostratified structure in necrotic areas.
- There is generally **no significant reaction in the lamina propria**.

Kennel Cough

- *In B. bronchiseptica infections, there is intense neutrophilic infiltration accompanied by mucopurulent rhinitis, tracheobronchitis, and bronchiolitis.*
- *The lesions may then extend both proximally and distally along the respiratory tract, potentially leading to bronchitis and bronchopneumonia.*
- *The bacterium adheres to cilia and subsequently causes ciliostasis. When sufficient bacteria are present on the cilia, Gram-negative rods can be easily stained and observed under the light microscope.*
- *In contrast, if the lesions are due to pure viral infections, the primary microscopic change is focal necrosis in the tracheobronchial epithelium. This is particularly observed in canine parainfluenza virus type 2 infections.*