RESPIRATORY SYSTEM

Coexisting Diseases with Rhinitis in Ruminants

Infectious Bovine Rhinotracheitis (IBR)

- Bovine herpesvirus-1 (BHV1) is an acute, infectious disease characterized by inflammatory lesions of trachea and conjunctiva.
- In addition, a serological virus can also cause infectious pustular vulvovaginitis (IPV) and ballonoposthitis infection.
- BHV-1.1 and BHV-1.2a together make abortion.
- BHV-1 was isolated from the more generalized infections of meningoencephalitis in young calves.
- It was also reported that BHV-1 performed vaginitis and balanitis in pigs.

- It is known that **IBR is widespread all over the world.**
- Serological findings indicate that infection is more common than disease.
- The disease is seen in places where cattle are crowded and is usually shaped more in fattening animals or cattles.

- Dairy cows are usually mild.
- In fattening cattle, the disease starts with the removal of new animals from outside and gains epidemic characteristics with the movements of the inside and outside animals.
- Morbidity is high, but many cases are very mild and not noticeable.
- Mortality is generally low, but it may reach 30% in exceptional outbreaks. Chronic cases are around 10% and the mortality rate is 3%.

Clinical Findings:

- The disease is clinically fever, increased breathing rate, cough and serous nasal discharge.
- The nostrils are enlarged, oral breathing and lacrimation are seen.
- In long-lasting cases, the nasal discharge is mucopurulent and dyspnoe is formed in inspiration.

Macroscopic Findings:

- In typical, uncomplicated cases, seromucinous rhinotracheitis and possibly conjunctivitis are seen.
- In more severe cases, focal hemorrhage, erosion and ulcers are formed by mucopurulent exudate on the mucosa with acute diffuse inflammation, especially in bacterial complications.

- In very severe cases, especially fatal, there are diffuse fibrinopurulent or fibrinonecrotic membranes on nasopharyngeal, laryngeal and tracheal.
- The severity of the lesions is determined by the bacterial complication (especially pasteurella spp. Hyoplasma spp. And F. necrophorum).
- In mild cases there is very little epithelial necrosis with a inflammation ranging from serous to mucopurulent.
- Epithelial necrosis is more severe in lethal cases and a fibrino-necrotic layer is formed on the surface.

- Homogeneous pink colored inclusions are formed.
- These inclusions are light around and the chromatin is completely pushed into the nucleus membrane.
- After 2-3 days (usually 72 hours) is not observed. Since the inclusion bodies are usually seen in experimental cases, they do not have diagnostic values.
- Nonspecific ulcers are formed.

- From the secondary bacteria pasteurella spp. the majority
- Pneumonia with pleuritis in the lungs or with severe fibrin without the formation of pleuritis.
- In addition, respiratory distress, which frequently results from upper and lower airway obstructions, causes interstitial emphysema.
- Severe viral lesions, which do not have an important role in the sequelae, may also occur.
- In these cases, severe necrotic bronchitis and bronchiolitis and serofibrinous exudate are seen in the alveoli.

- Necrotic placentitis and edema are common in patients with abortus. Abortus 5-6 of pregnancy. It is shaped in months.

(Because the fetus died 2 days before the abortion, it is autolytic and no characteristic macroscopic lesion is seen.)

- Microscopic findings can be seen in many parenchymal organs, lymph nodes and plescenta. Severe necrosis, leucocyte infiltration is seen in the liver and mixed with listeriosis.
- Necrosis is sometimes seen in other organs. There are no specific inclusion bodies in autolysed fetuses.

Malignant catarrhal fever *(Coryza gangrenosa bovum)*

- Caused by Herpes virus.
- In experimental cases, the incubation period is 14-60 days.
- High fever, catarrhal conjunctivitis and rhinitis are formed.
- In addition, **mucopurulent discharge** through the nose and nose.
- Erosions of the nasal mucosa are well-circumscribed and non-uniform.
- They are covered with mucopurulent exudate.
- Microscopic epithelial necrosis, rarely small vesicle formation, stromal cellular infiltration.
- Erosion covers the cellular exudate. At oral mucosa, esophagus, rumen, reticulum, omasum; congestion, edema and erosion are found.

- In the determination of the disease, vasculitis (fibrinoid-necrotic) is the specific feature in the lower regions of the lesions.
- Endothelial swelling, hyperplasia, medial necrosis, and macrophage in the veins, plasma cells and eosinophils infiltrates in veins.
- Vasculitis can be seen in every organ and tissue.

Tuberculosis

- Early generalization and tuberculosis of the chronic organ can be shaped like cauliflower-like or polypous nodules and ulcers.

Actinomycosis and Actinobasillosis

- Actinomycotic lesions that are common in sheep or only nasal cavity may be encountered in sheep.
- Nasal or facial actinobacillosis in sheep is usually shaped in relation to lip injury, usually in dry season.

Necrobacillosis

- The causative agent is Fusobacterium necrophorum.
- It is usually localized in pharynx, larynx, trachea, mouth, esophagus, lung and other digestive system.
- In calves, lambs and piglets, it is mostly seen in the nose.
- The disease is caused by disruption of the general condition, injuries to the mucosa, presence of other diseases, and poor hygienic conditions.
- As in other regions, dry red gray focal

coagulation necrosis of the nose,

circumscribed with a hyperemic ring shaped

lesions

Coexisting Diseases with Rhinitis in Pigs

Inclusion-body rhinitis

- One to five weeks of milk sucking pigs are more common
- It is characterized by sero-mucous rhinitis, growth in gland epithelium (cytomegalia), syncytium formation and intranuclear inclusion bodies.
- An acute or subacute viral disease.
- The disease is common in Europe. However, pig is seen in every country grown.

- An effective cytomegalovirus (from the family Herpesviridae, beta herpeavirinae subfamily) is.
- The disease is transmitted by the nasal discharge of sick animals.
- The virus is also found in urine and pharynx.
- Young pigs are very allergic to patients in the viremia

Clinical Findings :

- The duration of incubation in the disease is about 1 day.
- The disease begins with mild fever and rhinitis.
- Tear is seen.
- The nasal discharge is initially sero-musinous, but in long-lasting cases it is catarrhal or purulent, which is due to a secondary bacterial complication.
- The morbidity is high in the disease and it can be as high as 1% by the effect of environmental conditions (such as cold).
- Mortality is low if there is no serious complications.
- Sinusitis, otitis media and pneumonia may be seen as complications.

- The virus comes from the epithelium of the nasal mucosa to the epithelium.
- Viremic phase may last for 2-3 weeks.
- It then settles in the lung macrophages and persistent infections are formed.
- Piglets usually die during generalization.
- The presence of transplacental infection is questionable.

Macroscopic Findings:

- Serous catarrhal rhinitis are related changes.
- When bacterial complications occur, purulent rhinitis develops.
- The nasal mucosa is bulging, hyperemic and covered with a mucous or purulent exudate.

Microscopic Findings:

- Serous or catarrhal rhinitis or purulent rhinitis at complicated cases is seen
- In uncomplicated nonsupurative rhinitis cases, the presence of specific basophilic inclusion bodies in squamous metaplasia, glands, and epithelial cells of the mucosa is the main finding.
- The glands are infected.
- All cells may have an inclusion body.
- The bodies stay for a month, then they are found in a small number.

- Epithelial cells form syncytium with neighboring cells.
- Melting of epithelium is followed by melting and collection of leukocyte residues.
- The necrotic glands are clogged by lymphocyte infiltration and collapse of L.production.
- There is a slight vascular reaction in this disease.
- Infiltrating cells are usually lymphocytes.
- Although they are commonly found, they settle in the superficial regions of more L.p roam.
- The regeneration of the glands occurs by progression and differentiation from the superficial epithelium.

- Piglets die during generalization.
- Infection of the sinusoidal and endothelial cells creates petechial hemorrhages.
- Subcutis and edema in the thorax are shaped.
- Parenchymal tissues have focal necrosis.
- They may be massive in the liver.
- Piglets are anemic.

- The presence of inclusions in intravascular and spleen mononuclear cells suggests that anemia occurs as a result of bone marrow injury.
- Inclusions and focal gliosis within the glia cells can be found in each region of the central nervous system.
- Mutation, preterm delivery, neonatal death may occur in the infection of sensitive pregnant animals.
- The presence of transplacental infections is doubtful.

Differential diagnosis

- The disease can be diagnosed by cytomegaly and intranuclear inclusion bodies in the cells.
- However, atrophic rhinitis of the pigs (initially), Aujeszky disease, swine fever and bacteria may be mixed with other bacterial rhinitis.

Rhinitis atrophicans contagiosa suum, Distrophic rhinitis

- It is mostly characterized by different degree of atrophy of the concha nasalis in young pigs.
- It is a chronic disease which occurs as bending of nose and shortening in advanced cases.
- Pigs are endemic in the cultivated areas.
- It is not lethal unless it is complicated.

- Generally, it causes stopping of development and low productivity.
- Causes significant economic loss among young pigs.
- Acute epizooties are always seen in a herd in the first infection.
- The primary factor in the disease is a protein toxin, which is mainly made by the toxicogenic isolate of Pasteurella multocida.
- P.multocida toxin is the main cause of atrophy.
- However, additional factors are also needed for colonization, proliferation and effective toxin formation in the respiratory mucosa.

- However, additional factors are also needed for colonization, proliferation and effective toxin formation in the respiratory mucosa.
  - Of these, Bordetella bronchiseptica is considered to be the first one and probably Hemophilus parasuis is involved.
  - B.bronciseptica is cytotoxic and has a synergistic effect with P. multocida.
  - Predisposing factors such as genetic susceptibility of individuals or environmental conditions, malnutrition, cytomegalovirus infection also play a role in the development of the disease.

- These factors facilitate colonization of toxigenic P.multocida.
- Nutritional disorders, especially calcium and phosphorus defect in the bone metabolism in the form of defects, young pigs in the fast-growing and newly shaped cones are susceptible to P.multocida toxins.
- However, it does not cause atrophic rhinitise only in the lack of nutrition.

Contamination:

- The disease is caused by contact and airway droplet infection.
- The patient, especially old pigs, is involved in the transmission.
- It has been reported that the disease will be transmitted through animals such as rats, dogs and cats.

Clinical Findings:

- Clinical symptoms develop slowly.
- Acute incidence of rhinitis, coughing, serous or mucopurulent nasal discharge are seen in acute events among young offspring.
- In case of erosion of the mucosa, there is a purulent, partially bloody nasal discharge.
- In severe cases, small or large blood flocculus may occur during sneezing. Sometimes there is even obvious bleeding.
- There was no constant association between atrophic changes and acute rhinitis.
- In some cases, rhinitis may not cause atrophy, or there is no deformity, rhinitis is not seen, but atrophy is very prominent.
- Facial deformity is a symptom of severe disease in rapidly growing young pigs.

Pathological Findings:

- Pathological findings are found in the nasal mucosa and in the bones.
- Changes in the nasal mucosa develop from the front to the back.
- It is initially characterized by serous, catarrhal and then purulent rhinitis.
- The nasal mucosa is covered with this exudate.
- Sometimes it is pale and dry.
- The antero-dorsal region of the ventral turbinate is most changed.
- The presence of a inflammation in the mucosa, which covers the bulla tympanica, gives the impression that the disease begins with the infection of the upper respiratory tract.
- Inflammation can spread to all sides of the mucosa and sinuses.

- Atrophy is seen macroscopically in turbinates, and sometimes in other bones in the nose and sinuses.
- The consistency softens.
- In some regions, hypertrophy is also shaped as a secondary response.
- Percent deformity occurs with deviation of the maxilla to one side or dorsale.
- Asymmetry in the nasal and paranasal sinuses and total or partial atrophy in the nasal concha are formed.
- The most appropriate method for identifying atrophic rhinitis in pigs l, 2. To make perpendicular cross-section between premolar teeth.
- Thus, the size of the ventral turbinates is seen as comfortable.

- In these sections, sometimes small, deformed bones are found and sometimes there are no turbinates.
- Since atrophy is usually asymmetrical, the young pigs are exposed to the distorted side as the nose grows.
- Median septum is twisted.
- If atrophy is symmetrical, the nose is raised upwards.
- Due to changes in the nose, the animal has difficulty breathing.

Microscopic Findings:

- In the microscopic examination, ossa turbinate and facial bones have progressive characteristics.
- At the beginning of the disease and in young pigs, osteoclasts are seen to be increased.
- Osteoblast proliferation is also insufficient.
- Therefore, osteogenesis is not complete.
- In advanced cases, fibrous tissue forms the fibrous framework of ossa turbinates.
- Sometimes a gelatinous matrix devoid of cells is formed.
- Structural changes such as hypertrophy can also be observed in blood vessels and arterioles.

Differential diagnosis

- Diagnosis is not difficult when there are deformations.
- In the absence of this finding, it may be confused with the inclusion of rhinitis and other rhinitis.

Coexisting Diseases with Rhinitis in Dogs

- In dogs, there is no specific infection that infects only the nasal cavity and sinuses.
- Acute rhinitis can usually be formed as part of general respiratory diseases caused by various viruses.
- These viruses canine distemper virus, canine adenovirus 1 and 2, canine parainfluenza, reovirus and canine herpesvirus.
- Viral lesions in the respiratory tract are usually transient, but the effects of these viruses on other tissues may be fatal, for example, as encephalitis in the distemper.
- As with other species, viral infections of the respiratory tract may also result in subclinical bacterial rhinitis and sinusitis.
- The most common agents in bacterial rhinitis in dogs are B.bronchiseptics, E.coli and P.multocida.

Coexisting Diseases with Rhinitis in Cats

Feline Respiratory Disease Complex

- The viral rhinotracheitis of cats, which are characterized by inflammation of the upper respiratory tract, is the most important part of this complex.
- Another important disease in this group is Feline calicivirus infection.
- Feline reovirus and cats-adapted Chlamydia psittaci (cat pneumonitis agent) infections are less important.
- Mycoplasma felis is associated with viral and chlamydial infections, and has an oppurtunist role in causing mucopurulent conjunctivitis.

Feline Viral Rhinotracheitis

- Feline is a viral disease characterized by inflammation of the upper airway generated by herpesvirus-l (FHV-1).
- Severe fibrinous rhinotracheitis and possibly acute viral or seconder bacterial pneumonia are usually seen in lethal cases.

- There are rarely ulcers in the tongue and only seen in severe cases.
- However, in calicivirus infections, vesicular and ulcerative lesions are frequently seen on the tongue, hard palate or nose.
- Eye lesions are mostly in the form of purulent conjunctivitis, but may progress to ulcerative keratitis.

Microscopic Findings:

- Along with respiratory and conjunctival lesions, multifocal necrosis, which is characteristic of active herpes virus infections, and epithelial cell death caused by intranuclear viral replication are seen.
- P.multocida, B.bronchiseptica, streptococcus spp. and Mycoplasma felis, which are involved in the formation of fusible lesions when mixed with secondary bacterial infections.

Microscopic Findings:

- The most common virus replication and necrosis occurs 2-7 days after infection and herpesvirus inclusions are seen in the nuclei of infected cells.
- These are large, acidophilic and inclusions (Cowdry A type) inclusions in their environment. They are good in acidophilic fixatives such as bouin.

- It is difficult to find these inclusions in dead animals, they can be selected very rarely 7 days after infection.
- Therefore, the inclusion body is not a reliable finding in the diagnosis.
- The cells with the inclusion bodies are also large and pale.
- Epithelial cell organization is disrupted and erosion and ulcers are formed.
- In these regions, acute inflammation reaction with fibrin and many neutrophil leukocytes occurs.
- Acute inflammation and focal necrosis are seen in tonsils and local lymph nodes.

Caliciviral Infection *(Feline Infectious Coryza, Feline Influenza)*

- In recent years, one of the two major respiratory diseases of cats has been calicivirus infection.
- The other is feline viral rhinotracheitis.
- Mostly clinical findings are covered by herpes virus infection, but sometimes two viral diseases are formed together.
- The most important feature of calicivirus is the affinity of the mouth and lung epithelium.
- The most important signs of the disease are oral ulceration and primary pneumonia.
- It causes lighter inflammatory changes in the upper respiratory tract and conjunctiva.

Clinical Findings:

- Fever, rhinitis, conjunctivitis, nasal and tear discharge, vesicular and ulceration in the mouth and sometimes pneumonia are seen.
- As a result of bacterial complications, purulent rhinitis is formed and tear discharge increases.
- These lesions are also shaped in rhinopneumonitis.
- However, in the case of calicivirus infection, ulceration of the mouth is very prominent, but unlike the other disease, keratitis or corneal ulcer is toast.
- Macroscopically, ulcers are found in the mouth, in the tongue, on the hard palate and at the tip of the nose.
- The severity and distribution of the lesions depend on the tropism and the severity of the infection.

- Ulcers are initially present in the transient vesicle stage.
- They are found in the dorsal and lateral sides of the tongue and in the hard palate.
- Serous or mucoid rhinitis and conjunctivitis are less common.
- Intersititiel pneumonia is shaped, and this finding separates the feline viral rhinotracheitis from the bronchointertitiel pneumonia.

Microscopically:

- Alveolar type I cells show interstitial pneumonia with necrosis.
- This is related to the tropism of the virus's type I alveolar epithelial cells.
- In the first few days necrosis in these cells is formed, it is accompanied by serofibrinous and neutrophilic exudation and sometimes with the shaping of the hyaline membrane.
- At the beginning of the second week, hyperplasia begins in type II alveolar epithelial cells (fetalization, epithelization).
- Hyaline membranes are formed in the alveolar lumen.
- 30 days after the infection, the fibrosis is shaped as the connective tissue cells gradually increase in the alveolar walls.
- As a result of bacterial infections, it turns to other forms of pneumonia.

Differential diagnosis

Feline rhinotracheitis can be confused with :

- Chlamydia sp.,
- Mycoplasma sp.
- Other bacterial agents in complicated conditions.

Feline Chlamydiosis

- It is a persistent respiratory system infection created by Chlamydia psittaci of cats.
- Infection results in mild conjunctivitis (similar to human trachoma) and rhinitis.
- However, light and transient bronchointerstitial pneumonia occurs in severe cases.
- This pneumonia was previously called feline pneumonie Feline reovirus and Mycoplasma species also cause mild upper respiratory tract infection.
- The clinical findings and lesions are covered by viral rhinotracheitis, calicivirus infection and chlamydiosis infections.
- Many respiratory system infections in cats are associated with the immunosuppressive effect of Feline leukemia virus.

Specific Rhinitis ve Sinusitis
Mycotic Rhinitis

- Because of local destruction of the nasal mucosa or impaired immunity, defenses or other systemic effects make the nasal cavity available for opportunistic fungal or yeast infections.
- The rhinitis cases with funghi are formed in all animal species and are characterized by the formation of granulomatous nodules in the nasal mucosa.
- There are various types of funghi that infect pets.

Aspergillosis

- A.Fumigatus frequently infects dogs, rarely other animal species.
- The lesions are in the form of chronic, necrotic granulomatous foci. There is an exudate which contains large amounts of necrotic fungal hyphae.
- The live surface hyphae can be seen as a thin blue-green cover.
- The lesion progresses slowly, leading to destabilization of the vertebrae and sometimes the nasal septum.
- Occasionally, the nasal, maxillary and palate bones are broken.
- As in other fungal infections, local or general immunity disruption is thought to play a role in predisposition.
- Similar lesions also occur with penicillium species.

Cryptococcosis

- Cryptococcus neoformans frequently causes granulomatous rhinitis in cats, in sporadic events, in horse, dog and other animal species.
- In cats, lesions are gelatinous rather than granulomatous.
- This is related to the presence of large amounts of polysaccharide capsular material around the agent.
- Macrophage, epithelioid cell and lymphocyte reaction are usually few.
- The deterioration of the defense system and the masking of the capsular polysaccharide in the absence of inflammatory reaction play a role.

- The lesions are either polypoid nodular or larger.
- Generally, they are slowly destroying.
- Cats usually face swelling.
- Lesions appear on the skin and mouth mucosa when they spread to the adjacent bones in the nasal cavity.
- In the local spread, lesions in the eye and brain, and in the wider spread, lymph nodes and lungs or various visceral organs are shaped.

Rhinitis Parasitaria

Oestrus ovis-

- The infestation of tissues with the larvae is called myasis.
- Oestrus ovis is found in the nasal passages and sinuses of sheep, and often presents with upper respiratory tract inflammation and obstruction.
- Goats and humans rarely infect.
- The larva settles on the nose, replaces two shirts and usually falls into the ground.
- Some of them adhere to cones and sinuses, grow rapidly, but are not able to go into spaces.

- The clinical finding is sneezing, nasal discharge, possibly wheezing.
- Catarrhal-mucopurulent rhinitis, erosions in the mucosa, hypertrophy of the sinus mucosa and rarely meningitis are pathologically related to the physical irritation.
- Meningitis is taken by the penetration of larvae from the ethmoid region into the head or by the penetration of the bacteria by the olfactory pathway.

Linguatula serrata-

- This arthropod is parasitic in the form of tongue and 2 males of females are l0-l2 cm long.
- They are especially found in carnivores, but sometimes they are found in grass eaters and humans.
- The adult parasite settles in the nasal cavity, which can be seen in the middle ear in the sinuses.
- It has an irritant effect and causes catarrhal inflammation.

Schistosoma nasale

- In Asia, goats, horses and cattle are seen.
- Rhinitis, mucopurulent nasal discharge, sneezing and difficulty breathing (wheezing).
- The lesion is similar to granulomatous rhinitis (nasal granuloma).

Limnatis species

- Various types are attached to the upper respiratory mucosa.
- The host is taken by contact if the cause is water.
- Limnatis species infects herbivores, carnivores and humans in Europe and Africa.
- The main effect is anemia and loss of condition.
- Besides, breathing difficulties, sometimes oral breathing and asphyxia are formed in relation to the edema. In such cases, there is bloody discharge in the mouth or nostrils.

Habronema-

- They make a granuloma in the nasal cavity.
- They are similar to those formed in other parts of the skin (summer wound).

Allergic Rhinitis

- Little work has been done on allergic rhinitis in domestic animals.
- It is seen as sporadic cases in dogs, cats and horses and is similar to Hay-fever (nasolacrimal urticaria), which is shaped against pollen in humans.
- Hay fever in human and domestic animals is usually considered a type I hypersensitivity reaction, and mast cell degranulation results in acute rhinitis and conjunctivitis.
- In addition, seasonal rhinitis is frequently seen in cattle and occasionally in sheep.
- The disease occurs in the summer season, when the flower is blooming.
- As a herd problem, it can also occur as an individual disease, as seen in many animals.

Clinically :

- Eye-nasal discharge, sneezing, itching of the nose, nodding and possibly epistaxis are seen.
- Nasal discharge, pruritus, lacrimation and sneezing are the main findings.
- The nasal mucosa is pale, swollen due to edema fluid.
- Erosion may occur at the tip of the nostrils.
- The exudate is initially serous, then has mucopurulent properties or is in the flocculent form of mucus and cell debris.
- Eosinophils are also essential components of exudate.

Microscopically:

- Hyperplastic or ulcerated and eosinophils infiltrate the intact nasal epithelium.
- The glandular epithelium is hypertrophic, with a large amount of mucus secretion.
- If the drainage channels are blocked by mucosal lesions, mucus accumulates in the channels and consequently removes this exudate.

Microscopically:

- In more severe cases, the diphtheric membrane increases in the mucosa and fibrinoid necrosis is formed in many small mucosal vessels.
- Nasal granuloma is considered to be more chronic allergic rhinitis and is considered to be the result of recurrent inhalation of an antigen that has not yet been identified.
- Lesions are seen in the posterior part of the nasal vestibula, in the anterior region of the nasal septum and ventral meatus.

Pharyngitis

- It is seen as part of the inflammation of the upper respiratory and digestive system or both.
- Various nonspecific factors can cause lesions in this region and related clinical findings.
- In all types, foreign body traumas may occur in the caudolateral pharynx region.
- These may be caused by the wounds associated with the bite as a result of misuse of the catheters (especially during manipulations such as improper feeding of the sheep), causing clogging of the collar used in dogs and cats.
- Local edema and inflammation occur in small traumas.
- Cellulitis, which ends with death, is shaped more seriously. Sometimes foreign bodies are found.
- It causes dysphagia, difficulty in breathing and foreign body ingestion according to size and location.
- Dogs in the pharynx with chicken bone, garbage and needle injuries can be shaped as a result of injuries.

Laryngitis

- Because of the anatomical localization of the larynx, inflammatory changes are seen.
- It usually occurs with infections of the upper respiratory tract.
- Inflammatory changes are catarrhal, purulent or pseudomembrane. It is acute or chronic.
- In necrobacillosis in the calves and pigs, especially with oral lesions, diphtheroid-necrotic membranes are formed.
- Also in piglets, diphtheroid laryngophthiasis may also be caused by streptococci. Abscesses of C.pyogenes infections in calves and piglets are formed.
- Such disorders occur in sheep and young cattle in the pasture, especially in the case of enzootic or sporadic events, especially when the mucosa is injured by the penetration of the agent (injury to the hard dry grass and mucosa).
- Such infections can then lead to scarring and deformation in the larynx.

- Fibrinous, necrotic-diphtheroid laryngitis in the larynx is caused by bacterial effects.
- Cytogens in cattle, cattle plague can also cause serous-catarrhal and then fibrinous pseudomembraneous laryngitis may occur.
- Pus and cattle actinomycosis is associated with fungus or disseminated lesions.
- At the horses, non-primary, generalized from other regions can be observed related to the symptoms (nodule, ulcer, scar).
- In chronic-open lung tuberculosis in cattle, fungal structures in the larynx and the opening of these ulcers are the result of their opening.

Tracheitis

- Tracheitis occurs as a result of upper respiratory tract or other respiratory system or systemic diseases.
- For example, dogs are associated with bronchitis in youth disease.
- IBR in cattle, CGB, rhinotracheitis of cats, rhinopneumonitiste in horses are shaped. In horses, opening of abscesses in strangles forms pus, tracheitis, abscess and phlegmon. In dogs, in addition to bronchopneumonia it occurs catarrhal pneumonia, and microscopic examination of the mucosa epithelium inclusion bodies are seen.
- Hemorrhagic necrotic or pseudomembrane tracheitis, croupous pneumonia and sero-fibrinous pleuritis can be seen in acute events in sheep.

- Tracheotomy is characterized by chronic diffuse tracheitis.
- A severe reaction is formed at the edge of the wound, the mucosa is swollen, and in the final stage, intense scar tissue is formed.
- Focal chronic polypoid tracheitis occurs occasionally in dogs and cats.
- Thickening of the throat may be too high to cause stenosis or difficulty breathing.
- The cause is unknown, and various pathogenic factors, such as nasal polyps, are thought to play a role.
- Thyroid epithelium in squamous metaplasia A vitamin deficiency and severe iodine toxicosis is found.

Canine Infectious Tracheobronchitis (*Kennel Cough*)

- It is quite contagious infection characterized by sudden coughing at dogs.
- The expression used for the disease is not specific, such as colds of people or shipping fever of cattle.
- The infection is usually shaped by mixing dogs of different origins.
- Therefore, it is frequently seen in animal care homes, commercial sales areas and veterinary clinics.

- The disease is clinically hard, persistent and usually sees cough as seizures.
- Cough bouts include healthy animals.
- In some cases coexistence with cough is not observed, while in some cases rhinitis, pharyngitis, tonsillitis or conjunctivitis may occur. Occasionally, secondary pneumonia is formed.
- The etiology of infectious tracheobronchitis of dogs is complex.

- Many pathogens and environmental factors are responsible here. The most important factor in the disease is Bordetella bronchiseptica. Canine adenovirus 2 (CAV-2) and canine parainfluenza type 2 virus (CPV) are frequently involved in canine distemper virus.
- The severity of the disease is greater in the inclusion of more than one factor or in the challenges of environmental conditions including stress.
- Although less important in the disease, infectious canine hepatitis virus, reovirus type I, canine herpes virus and mucoplasma cyanos also play a role.
- These factors are involved as single or mixed. The effect of Reoviruses with Mucoplasma, P.multocida and other Gram-negative bacteria is not clear

- Depending on the agent, gross findings may occasionally be absent, or a fever chart is formed that ranges from catarrh to mucopurulent tracheobronchitis.
- Serous mucopurulent rhinitis and cranioventral bronchopneumonia can also be seen in cases where the disease progresses.
- Tonsillar, retropharyngeal, and tracheobronchial lymph nodes are always bulging and red.
- Microscopically, varying degrees are tracheobronchitis and bronchiolitis.
- The findings range from focal superficial necrotic tracheobronchitis and bronchiolitis to more severe mucopurulent inflammation.
- In general, epithelial degeneration and necrosis and necrotic areas of normal pseudo multiple-layer structure is seen in the disorganization. There is no significant reaction to the lamina propria.

- B.bronchiseptica infections are caused by intense neutrophil infiltration and mucopurulent rhinitis, tracheobronchitis and bronchiolitis.
- The lesions then progress to the proximal and distal part of the airway and possibly lead to bronchitis and bronchopneumonia.
- Bacteria adhere to cilia, occurred by the ciliostasis. If there are enough bacteria on the cilia, Gram (-) bacteria are easily stained and light can be seen under microscope.
- In contrast, focal necrosis of the tracheobronchial epithelium is formed if the lesions belong to pure viral infections.

Special Inflammations

- Tuberculosis develops as in the upper respiratory tract (knot and ulcer). Because they are rich in lymph follicles, they are more common in the spathyre retromucosum.
- Glanders lesions are formed as secondary depending on the nose and lung ruam.
- Actinobacillosis-mycosis is shaped by direct inhalation in cattle, or spread from other organs in the secondary. It causes lesions in the form of cauliflower.

Parasitary Diseases of Larynx ve Trachea

- Parasitic infestations of this region cause obstructive results.
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- Syngamus trachea- Trekeya is also found in the bronchi. It is seen in red because it absorbs blood. It causes catarrhal-bloody inflammation in the mucosa.
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- Capillaria aerophila - A 2-3 cm long, cylindrical parasite found in the trachea and bronchi of dogs, foxes and occasionally cats. The eggs enter the airways, mucus in the pharynx comes. It is then ingested and excreted by the ghetto. The larva reaches the infective phase in the egg and remains there until it is removed by the appropriate cone-catcher. Fertilization occurs in intestines. The larvae are delivered to the lung within a week and are grown into trachea within 6 weeks.
- The effect of C.aerophila depends on the number. Mild infestations are overlooked and cause slight catarrhal inflammation. In severe infestations, severe irritation and obstruction of the airways are observed. Then, chronic cough and discrete breathing difficulties are shaped. Subclinical bacterial bronchopneumonia can also be formed.

- Filaroides osleri- In the shape of thread, 5- 15 mm. length is a parasite. It is found in dogs and other carnivores. The typical lesion is the shaping of submucosal nodules in the bifurcation area of the trachea. The parasite has been found in many regions, but is very rare. They are not similar to other metastronionic agents, they do not need an arachonacist. The first stage larvae are direct infectious. Thin-walled eggs containing the first phase larvae are coughed up, swallowed, and hatched from the hatchery as infective larvae in the stool. The offspring are infected with the saliva of the mothers or the larvae of the stool. The filaroides osleri of the wild carnivores will cause special damage. Because these offspring get infection easily during vomit feeding. Larvae come from the stomach to the lungs with blood.
- Lesions 1 cm. or larger, nodules that are visible to the naked eye. The large ones are oval and they are parallel to the long axis of the trachea. Lesions do not typically produce acute bronchitis or tracheitis. They usually cause a cough or breathing difficulty in the form of seizures. Nodules are gray or whiteish and parasites are easily visible under the mucosa.

- The histological section shows parasites. Small nodules have non-adult parasites, adults have adults with strong ring curves.
- The parasitic trachea and wide bronchi are found in tissue gaps between the cartilage rings around, in adventitia and lymphatic channels.
- Live parasite causes minimal reaction and is formed by lymphocytes and plasma cell infiltrations in lamina propriada with a thin capsule.
- The superficial nodules are covered with epithelium, where only the female parasite has small holes to remove the tail and lay its eggs.
- Makes a foreign body reaction consisting of dead parasitic neutrophils and giant cells.
- Non-adult parasites can be found in lung lymphatics and rarely in alveoli without significant tissue reaction.

Besnoitia bennetti - It is a coccidia and causes papillom-like lesions in the larynx. Africa, Central and South America and England were reported. The life cycle of the parasite is still unknown. Macroscopically 2 cm. until the nodules are seen. Histological examination shows that these nodules are seen as thick-wall parasitic cysts and have acanthotic extensions that have made fingerprints on them. Sometimes the epithelium can become ulcerated. If the cyst tears, it causes a foreign body reaction.

Spirocerca lupi forms the nodules occasionally in the bronchus and bronchus. It is considered as a perverse localization and is very rare.

BRONCHUS AND BRONCHIOLE

Foreign Bodies in Bronchus

- In the bronchi, aspirated blood may be encountered during cutting.
- There is water in suffocation.
- During the drug ingestion, foreign bodies are seen due to misuse in careless swallows or intracranial diseases.
- Swallowed substances may cause gangrene in the bronchi. Or, the inflammation site formed due to the foreign body is encapsulated. It may cause bronchiectasis by expanding into the pouch.
- The abscesses in the surrounding area open to the bronchi and cause abscess contents in the bronchi. In such cases, bronchitis develops later.

Form Changes in Bronchus

- These changes occur in the form of narrowing and obstruction of the bronchi or expansion of the bronchi.

I. Stenosis and obstruction in the bronchial lumen (obstruction, obturation) -

- Stenosis is usually characterized by the swelling of the mucosa during bronchitis and the accumulation of excess exudate in the lumen. In some cases, spastic contraction of the bronchi occurs. Continuous pressures related to granulomatous inflammations, peribronchitis and neoplastic cases around the bronchi cause compression stenosis.
- Obstruction of the bronchus can also be caused by the obstruction of the lung parasites by lumen as it is formed by aspiration of foreign bodies.

Bronchiectasia

- Local and permanent dilatation in one or more bronchus is defined as bronchiectasis.
- Bronchiectasis is frequently congenital malformation, which is rarely seen later.
- Subsequent causes of bronchiectasis are chronic inflammations (tuberculosis, mucous membranes, abscesses), end of aspiration secondary or another abnormality such as immotile cilia syndrome.
- Sometimes it occurs as a result of extraction (pull) of the tissue that develops from the surrounding tissues.

- Bronchiectasis has 2 important anatomical aspects. These are saccular and cylindrical bronchiectasis.
- Saccular bronchiectasis is rare and defines the limited expansion of the bronchus or bronchiolar wall outwardly in the form of a pocket.
- The ectatic wall is usually thinner than normal.
- They are readily seen in lungs fixed under tracheal pressure.
- This type of bronchiectasis occurs as a result of focal necrotic bronchitis and bronchiolitis and occasionally occurs in sheep and cattle.

- Sometimes the bronchiolitis-emphysema complex of horses (Soluğanlık) can be found in small airways.
- Cylindrical bronchiectasis is the extension of the bronchus or its entire length. In cattle, it is always shaped as sequelae of chronic suppurative bronchitis.
- Therefore, bronchiectasis of this type follows (cylindrical) bronchopneumonia and occurs in the airways in the cranioventral regions where bronchopneumonia occurs.
- There are several stages in the formation of bronchiectasis. One of them is the collection of exudate in the lumen and the lysosomal enzymes of the neutrophils and the deterioration and weakening of the bronchial wall with the effect of oxygen radicals.

- The other is diffuse atelectasis in the alveolar parenchyma where the affected airways function.
- The loss of alveolar tissue volume causes traction in the wall of the inflamed airway during inspiration and the airway expands over time.
- Because the bronchial wall is weak, it expands to provide parenchymal volume during breathing.
- In the lower airway obstruction and atelectasis, the air flow in the bronchial lumen is slow, so that the lumen expands even in the coughing effort.

- The mucosilic cleansing is also less effective because the cells of the flesh are destroyed.
- This leads to the accumulation of more mucus and inflammatory exudate in the bronchi, followed by the formation of bronchiectasis.
- Absence of lobular septum in the cattle and the absence of collateral ventilation reduce the resorption effect of bronchopneumonia, thus more common atelectasis with airway blockade.
- Therefore, bronchiectasis in cattle and bronchopneumonia follow.
- In bronchiectatic lungs, irregular dilated bronchioles are seen in the cranioventral regions.
- They are caudal-viscous, filled with yellow green pus. The parenchyma is atelectasis and sometimes fibrotic.
- Atelectasis is common or complete in the anterior lobes.
- Atelectasis, emphysema and bronchopneumonia are confused with caudal lobes.

- Since the lobular margins are separate in the cattle lungs, the expansion of the central bronchiole and the surrounding alveolar collapse makes small lobes (hillock) in each lobule and therefore resembles the surface ananasha.
- This surface appearance is mixed with fibrous pleural adhesions.
- When the lobe slices are cut, the induction of the parenchyma and the thin-walled enlarged bronchi filled with exudate are very well seen because the bronchi are transverse.
- In severe cases, dilated bronchi loba give a honeycomb or cystic appearance.

Microscopically:

- Depending on the severity and chronicity of the lesion, the bronchial wall shows a different degree of reconstruction with granulation tissue.
- Mucus, dead tissue, large amounts of inflammatory cells, often also have some blood.
- The mucosa may be

destroyed by ulceration

up to the muscle

layer or exhibits

atrophic, metaplastic

and hyperplastic changes.

- A large amount of leukocytes are infiltrated into the bronchial wall and the lamina propria shows granulation tissue structure characterized by progressive fibrosis.
- Necrotic tissue descends deep into the mucosa, cartilage and submucosal glands can be destroyed.
- Destructive and suppurative lesions are formed on all floors of the bronchial wall and adjacent alveoli and are equal to the lung abscess.
- Bronchiectasias are more common in cattle.
- It may occur as a result of tuberculosis in the larynx. infections.
- The parasites are the main causes of bronchiectasis at pigs, sheep and goat
- Occasionally, all types of localized bronchiectasis follow obstructs by foreign bodies, granulomas or tumors.

- Changes in atrophy and hypertrophy may also occur in the bronchiectatic wall. In this case, atrophic and hypertrophic bronchiectasis are mentioned.
- In atrophic bronchiectasis, exudate-expanding bronchial lumen, as well as the appearance of mucosal paper is thinner.
- Bronchial connective tissue and bronchial glands have also been destroyed.
- A rarely seen hypertrophic bronchial wall shows dilatations in the form of a stiff and chronic inflammatory sac.
- The exudate, which is reusable and often purulent, darkens and calcifies. Thus, the constructions called bronchial stones (bronchiolite) occur.

The conclusion of bronchiectasis -

- The course of bronchiectasis is chronic and not preferred.
- Complications such as bronchopleural fistula, septic thrombosis and hemorrhage or metastatic abscess formation and septic embolism and secondary amyloidosis occur beyond bronchopneumonia.

Bronchitis

- Bronch and bronchioles are located between the upper and lower airways, and are associated with upper respiratory tract and lung inflammation.
- It is usually shaped by the descending spread of the factors taken from the upper airway.
- Ascendence infection is particularly important in verminous and granulomatous pneumonia.
- In the same way, lung abscesses in the pseudotuberculosis also open to the bronchi and persistent caseous bronchitis.

- Among the factors, infectious ones occupy the main place.
- In addition, irritant gases, allergens, foreign bodies can develop.
- Cold, environmental factors and deficiency of the defense of the organism play a preparatory role.
- Bronchitis is defined as acute or chronic according to the course of the inflammation.
- Acute bronchitis is usually caused by severe upper respiratory tract lesions or pneumonia.

Acute Bronchitis

- According to their morphological structure, they are divided into catarrhal, purulent, mucopurulent, fibrinous, ulcerative or fibrinonecrosis (diphteric). Sometimes they become granulomatous.
- The lesions on the obstructed bronchial pathways vary depending on the cause. For example, in hypersensitivity reactions, eosinophils, and in some viral infections, inclusion bodies are seen.
- In the vast majority of cases, the lesions are non-specific and define the severity and duration of the effect rather than the cause.

- These epithelial cells lose their silanes, are degenerate and necrosis, are separated from the basement membrane, and then are desquamated into the lumen (exfoliation).
- The goblet cells between these cells are also swollen and contain mucus.
- Other gland cells are similar.
- Edema, hyperemia, and a small number of neutrophil leukocyte infiltrations are encountered.
- This is followed by the collection of leukocytes through the lumen in the epithelium.
- If the inflammation is transient, it is immediately repaired by proliferation of the remaining basal and intermediate cells.
- If the defect is large, the solid cells in the periphery also play a role in proliferative regeneration.

- Catarrhal bronchitis is the simplest form of inflammation.
- Macroscopically, there is mucous exudate in the bronchial lumen and swelling of the mucosa due to hyperemia and edema.
- Mucous exudate is formed by the increase of secretion Since the relative numbers of secreting epithelial cells and the density of the glands vary from one species to another, the amount and structure of the epidemic will also differ in animal species.
- Cilial epithelial cells are the most sensitive cells to various factors, so the first disorder in microscopic examination is seen in these cells.

- The course of bronchitis after initial catarrhal phase depends on the severity and course of the irritant substance and effect.
- If bacterial causes are present or if bacteria have been mixed sequentially, they become suppurative, fibrinous, necrotic, pseudomembrane or ulcerative inflammation in purulent or more severe cases.
- Purulent bronchitis usually occurs as a result of bacterial infections.
- The bronchial lumen is yellowish in color and has a thick exudate.

- Microscopic examination shows a large amount of neutrophil leukocytes in the mucosa of the propria.
- The veins are more hyperemic and the destruction of the epithelium of the mucosa with edema is greater.
- Purulent bronchitis tends to localize in cattle.
- Cylinderic or saccular bronchiectasias may cause.

- Ulcerative bronchitis is characterized by serious viral and bacterial infections, where the epithelial layer has a large area of destonation and lamina propria has been exposed.
- It is usually shaped as a result of chronic purulent bronchitis.
- Fibrinonecrotic bronchitis is characterized by exudate, where a thick and yellow membrane is formed and the membrane is firmly attached to the mucosa at many points.
- The reaction of this severity affects the larynx, trachea and cranioventral parts of the lung.
- In the clinic, the masses showing the bronchial pattern with cough are expelled.
- It's usually lethal.
- Although it is caused by bacterial agents, CGB, IBR and cattle plague and mucotic bronchitis are the primary causes.

- Severe necrotic bronchitis is characterized by necrosis in the mucosa.
- Severe parasitic invasions occur in aspirated foreign bodies and bronchiectasis. .
- It develops through various microorganisms.
- The bronchial mucosa in the form of this inflammation is greenish and has a stinking appearance.
- Acute bronchitis, which is predicted to be allergic, is clinically called asthma or allergic bronchitis.
- Acute bronchitis can usually heal with neutralization or disappearance of the agent.
- Basically, degeneration of the injured bronchial epithelium, separation from the basement membrane and exfoliation are seen.
- Under normal circumstances, this loss is soon followed by exudative inflammation and repair. Repair is by regeneration of the bronchial epithelium.

- Usually, the inflammation in the large bronchus heals without leaving any traces.
- In severe cases, the bronchial wall may sometimes be damaged.
- Rarely, mild fibrosis in bronchial lamina propria and granular tissue-like polyps may be formed.
- Minor bronchi, especially bronchiolar inflammation, results in bronchopneumonia.
- Inflammation of large bronchi is less limited and less important than minor bronchi and especially bronchioles.
- Large bronchi are found in interstitial tissue other than the lung lobes.
- Epithelial pseudo-multilayer and well supported with secretory and ciliated cells.
- Peribronchial connective tissue is abundant and mature.
- The lumen is too wide to be occluded by the exudate and this exudate can be easily removed with the cough reflex.
- For this reason, even in severe events, the inflammation is spread only to the surrounding parenchyma.
- In contrast, small bronchus and bronchi are present in the parenchyma, whereas in bronchioles, there is a wider spread. Because they have less peribronchial connective tissue.

- Inflammation of the large bronchi is less important for the lungs, especially the inflammations of the bronchioles usually result in parenchymal disorders.
- Bronchopneumonia, atelectasis or emphysema are the most important results.
- Often bronchitis or tracheobronchitis rarely causes death.
- They often cause problems clinically. The best example of this type of inflammation is Kennel cough (Infectious tracheobronchitis) of dogs

Chronic bronchitis

- These bronchitis are characterized by more severe and ongoing epithelial injuries.
- It is usually based on bacterial, parasitic or allergic causes.
- The importance of these causes varies according to animal species.
- Chronic catarrhal or mucopurulent bronchitis is important in dogs and bronchial irritation and mucous hypersecretion cause chronic persistent cough.
- The disease is usually seen in small breeds, especially in obese animals.
- In postmenopausal cases of chronic catarrhal bronchitis, mucous or mucopurulent exudate is present in the tracheal bronchial tree.
- In the tracheal-bronchial junction, exudate is seen in the airways, ranging from sticky, white or green to brown.
- Sometimes exudate makes foamy accumulation in airways.
- The bronchial mucosa is thick, often hyperemic and edematous.
- Occasionally, lumene polypoid extensions are formed.

Microscopically:

- Thickening and folding of the mucosa produces infiltration of the mucosal glands and severe lymphocytes in the lamina propria, plasma cells and occasionally macrophages and neutrophils.
- In superficial epithelium, Goblet cells have hyperplasia and focal ulceration and squamous metaplasia.
- Intraluminal mucus is mixed with plenty of neutrophils.
- Fibrosis and hyperemia and the amount of edema in the bronchiole wall depend on the severity of age and lesion and whether there is a new exacerbation in a short time.
- Small bronchopneumonia foci are present in up to 25% of cases.

- In severe chronic bronchitis, hypertrophy is found in the smooth and medium-sized pulmonary arteries.
- Shaped pulmonary hypertension causes cor pulmonale, which can sometimes be clinically defined.
- The most common complication is bronchiectasis with alveolar atelectasis.

- Such chronic bronchitis (chronic catarrhal) is seen in the most beautiful cigarette smokers and these people need constant coughing to remove excess mucus secretion.
- In some cases, excess mucus cannot be completely removed from the airways and leads to chronic obstructive bronchitis.
- The pathogenesis of chronic bronchitis in dogs is unknown. As with infectious tracheobronchitis, there is no improvement in this syndrome.
- Regardless of the cause of healing in acute outbreaks, the normal defence mechanism is disrupted and the inflammation is shaped at the end of the war between bacteria and leukocytes.
- The most important infectious agent in dogs is B.bronchiseptica.

Bronchiolitis

- The junction points of the bronchiolar and alveolar canals are the most vulnerable to aerogenesis. Bronchiolitis are associated with absolute bronchitis and pneumonia.

Acute bronchiolitis

- The disorder that occurs in the bronchioles varies depending on the nature, severity and continuity of the effect.
- However, it is very difficult to obtain information about etiology according to histological structure.
- Only some specific viral inclusion bodies and other exogenous agents may help in the diagnosis.
- Sometimes, as in the viral pneumonia of the calves, inflammatory components can give information about the disease.

- The epithelial covering of the bronchiolar region is highly susceptible to injury, including certain respiratory viruses (necrotic herpes viruses, some adeno and myxovirusa), oxidant gases (NO2, SO2, O3) and toxic substances (3-methylindole).
- In this sensitivity of the bronchioler epithelium, probably (1) high oxidative and free radicals, (2) the presence of Clara cells rich in mix-function oxidase (by shaping local toxic metabolites) in this region, and (3) lung alveolar macrophages and leukocytes in this region of the lung. tandans are considered to play a role.
- The first response of the bronchioles with irreversible injuries of these effects is the necrotion of the bronchiolar epithelium and pouring into the lumen. Thus the basement membrane remains naked.
- If the effect disappears or does not persist, the epithelium completely regenerates itself within a few days and then differentiates into the normal bronchiolar epithelium.
- It is accepted that the regenerative potential in this region is in Clara cells and that they make proliferating ciliate and secretory cells.
- However, it is not known whether the minor components of bronchiolar epithelial cells play an important role in cases where this is a general rule or in cases where there is not a normal cilia or secretory granules.

- The other important response of the bronchiolar epithelium is hyperplasia and is especially shaped by some adenoviral infections.
- In these cases, the role of ciliated cells or secretory cells in hyperplasia is indistinguishable.
- Under normal conditions, the phagocytic cells in the region eliminate the exudate and degenerative-necrotic epithelial cells in the bronchiolar lumen and thus prepare the basement membrane for the localization of new cells.
- However, it should also be considered that this exudate disrupts the local antibacterial defense and increases the predisposition to pneumonia as a result of the loss of lung function with atelectasis in the distal alveoli.
- In severe injuries, usually fibrin-rich exudate is collected and this material cannot be easily removed from the bronchiolar basement membrane. Fibrocytes and fibroblasts are adhered.

- This focus is then organized to form microscopic polyps that extend into the bronchiolar lumen.
- This organization of exudate is completed within 7-l0 days.
- Their external faces are ultimately covered by silylated epithelium. This lesion is usually defined as brochiolitis fibrosa obliterans or organized bronchiolitis.
- In all mild injuries, neutrophil infiltration is present in the bronchial wall, usually in the surrounding loose connective tissue, and in the surrounding blood and lymph vessels.
- Neutrophils predominate at the beginning of the fire.
- Then mononuclear cells are increased.
- Eosinophils are essential components of allergic conditions and are infiltrated by IgE antibodies, such as asthma, parasitic bronchitis, in the sensitization of tissue mast cells.
- Cell-mediated immunity plays a role in the accumulation of lymphoid cells in peribronchios, peribronchial and perivascular regions.
- This lymphoid hyperplasia is particularly prominent in mucoplasm infections (peribronchioler cuffing). However, they are shaped wherever there is persistent antigenic stimulus.

- Cellular immunity is as follows: The antigenic material from the microorganisms or the modified host cell membrane sensitizes lymphocytes, particularly T cells, after they have been processed by macrophages.
- They are collected locally and react with other antigens and form soluble lymphokines.
- These lymphokines induce inflammation, proliferation of lymphocytes, accumulation of macrophages, and thus destruction of microorganisms.
- The local excretion of the agent also increases with the severity of the local inflammatory lesion.

Chronic bronchiolitis

- On the other hand, if the bronchiolar injury is mild but persistent, the main finding in the lesion area is epithelial cell hyperplasia and metaplasia.
- Squamous metaplasia is characterized by the continuous destruction of irritant gases (SO2, NO2, O3) and other chemicals.

- The goblet cell metaplasia describes the transformation of secreting bronchiolar cells into mucus-making goblet cells and is usually seen in humans.
- However, animals with chronic bronchitis are also formed in older animals without active bronchitis and bronchiolitis.
- It is not known whether discrete or continuous smoking, air pollution and infections in humans cause this metaplasia.
- It is known that secreted bronchiolar cells (Clara) transform into goblet cells.
- In this case, the lung function decreases and increases the predisposition to bronchopneumonia.
- In this case, the physiochemical characteristic of bronchiolar secretion changes and the serous fluid secreted by Clara cells, which is normally secreted by the Clara cells, becomes a sticky material with mucous secretion of the goblet cells.
- As a result of increased viscoelasticity of the mucus, bronchiolar secretion cannot be removed with cessation and thus leads to obstruction of distal airways.
- Under these conditions, this group of disorders is usually defined as chronic obstructive pulmonary disease.

- The cough is mandatory to remove mucus from obstructive bronchioles.
- Another result of bronchioler obstruction is emphysema and atelectasis.
- The most typical example of chronic obstructive pulmonary disease is the parenchymal disease (chronic bronchiolitis-emphysema complex) of the horses.

Soluganlık (Chronic bronchiolitis- emphysema complex of horse, heaves, Equine Asthma Syndrome)

- This disease is characterized by horses, generalized bronchiolitis and emphysema. **Emphysema** means tissue puffed up by air. ... **Chronic bronchiolitis-emphysema complex** in the **horse** is also known as "heaves" or "broken wind" or the more scholarly "**chronic** obstructive pulmonary disease." As with many disease syndromes with multiple names, the causes and **pathogenesis** are poorly understood.
- The disease is also recently described as chronic obstructive disease, with local names such as heaves or brocken wind.
- The most common clinical finding is chronic bronchiolitis.
- Emphysema, which describes the expansion and destoluction of airways, is less common.
- Sometimes in the lungs that have been cut, the alveoli may be swollen by air retention.
- Very rarely, emphysema can be seen without significant bronchiolitis.
- Emphysema, though generally associated with generalized bronchiolitis, is usually located in the cranial lobes.

Macroscopically:

- In the lungs, usually no findings can be detected. However, in severe cases, emphysema can be seen.

Microscopically:

- The main feature of chronic bronchiolitis is epithelial hyperplasia, goblet cell metaplasia, peribroncherious fibrosis and lymphocyte and plasma cell infiltration.
- The lumen of the bronchiole was narrowed with exudate and peribronchiolar fibrosis.
- The mucus in the exudate is the main component and sometimes becomes so much that it passes through the adjacent alveolar canal and alveoli.
- The basic cell component of bronchiolitis is eosinophil leucocytes.
- These are sometimes very severe in intraluminal exudate, intraepithelial and peribronchiolar regions.
- The number of mast cells around the bronchioles also increases.
- There are neutrophils and eosinophils, but sometimes the mucopurulent bronchiolitis may be formed.

- The importance of allergy, infection and toxicity in the formation of bronchiolitis has not been established.
- The occurrence of the disease against clinically moldy herbs, sediments or fixed powders and aerosols has been perceived as an allergic response to inhaled allergens (eg Microspora faeni, A. fumigatus and grass dust).
- In experimental cases, pneumotoxins in the blood, especially 3-methylindole, were found to destroy the epithelium of bronchiol in horses.
- Considering the presence of goblet cell metaplasia and mucus hypersecretion, histamine, prostaglandins and leukotrienes secreted during Type I allergic response (anaphylaxis) are thought to have stimulating effect on mucus secretion.
- This explains the increase in goblet cell, mucus hypersecretion, eosinophil and mast cell increase. It should be remembered that it is not possible to precisely differentiate allergy bronchitis, chronic allergic bronchitis and bronchiolitis in domestic animals.

LUNGS

Postmortal Changes

Postmortal hypostase

- It is the collection of blood by the effect of gravity in the veins on the side where the animal lies.
- Postmortal hypostasis begins as agony. It is more pronounced in the amount of CO2 in the blood, and in animals that die from septicemic diseases.
- The blood fluid then exits the vessel and is mixed with pulmonary edema.
- The part of the lung on the side of the hypostasis is dark red and this is mixed with hyperemia. Therefore, it is necessary to examine the absolute other lung.
- In back position animals, hypostasis is symmetrically shaped in both lungs.
- When blood coagulates within the veins, it mixes with thrombosis that occurs before death

Atelectasia pulmonum

- The term atelectasis describes the partial or complete collapse of the lungs or the inability to expand the alveoli by air. Briefly, alveoli is airless.
- In the fetal stage, air is not present in the lungs and is called fetal atelectasis.
- These lungs may be partially swollen with a viscous fluid called bronchial fluid or lung fluid that is present in the bronchoalveolar cavities and is locally secreted.
- This fluid normally moves along the tracheal-bronchial pathway, travels to the oropharynx, eventually reaching the amniotic fluid.
- At birth, the lungs are rapidly reabsorbed by the pulmonary lymphatic vessels, and instead of the inspected air, the alveoli undergoes normal expansion.
- This is called fetal atelectasis because lung tissue does not expand with air at all, if respiratory movements have never been performed in early birth. This atelectasis is diffuse.

Atelectasis, which is shaped after birth, is examined in 2 parts as congenital and acquired.

- In the first few breaths of newborns (neonatal), lung tissue cannot be swollen by air. Congenital atelectasis (Atelectasia congenita, neonatal atelectasis)

- At the end of the lungs containing air, the airless air is called acquired atelectasis (Atelectasia acquisita, alveolar collapse, postnatal acquired atelectasis).

- Congenital atelectasis is usually formed in the neonatal period in the obstruction of the airways, usually in the aspiration of amniotic fluid during delivery (meconium aspiration syndrome) or anoxic destruction of the respiratory center in the brain stem, malformations of the central nervous system, injury to the brain during delivery, abnormalities in the lungs and related structures. .

- These atelectasis are mostly localized. Because animals breathe, but as much as they need, the parenchymal tissue is swollen with air.
- These lung parts are very small, such as in the fetus, the consistency is hard (meat consistency), does not show crepitation and sink in water.
- They are dark blue in color due to dilatation of the alveolar capillaries.
- Large alveolar cavities are seen in the microscope with a large and vascular interalveolar septum.
- In some alveoli, those under hypoxia stimulation can be found in flat cells aspirated from the uterus, or in amniotic material in the swallowed granular structure.
- These types of congenital atelectasis are defined as neonatal hyaline membrane disease (neonatal or acute respiratory distress syndrome).

- Diabetic is usually seen in premature infants born from alcoholic mothers.
- A similar situation is observed especially in foals, and rarely in lambs, dogs and piglets.
- When they try to inhale Thai and piglets they make a barking sound and therefore the disease is called barkers.
- Hypoxic brain damage is seen in most of the living foals.
- These animals are meaningless and do not show any signs of normal fear. With this finding, the disease is called wanderers. The disease is mostly diagnosed in necropsy.

- In these animals, it is seen that quite large lung areas resemble fetal lungs.
- These lung parts are heavy, fleshy, airless and bluish.
- The body is edema.
- Microscopically, the interalveolar septum is very close to each other and the bronchioles are enlarged.
- Many terminals and respiratory bronchi are thick, homogeneous eosinophilic hyaline membranes.
- Since the membranes were formed in a very short time, the animal had not yet breathed.

Acquired atelectasis

a. Obstructive or rezorbtive atelectasis :

- Exudate bronchi and bronchiol lumen, aspirated foreign material, parasites or tumors are shaped by blockage.
- The most common form of atelectasis.
- The severity of obstruction atelectasis depends on whether the obstruction is complete and whether there is a possibility of collateral ventilation.

- Obstructive atelectasis with bronchitis results in local anoxia in the alveolar wall.
- Alveolar vascular permeability increases and the liquid part of the blood passes through the alveolar cavity and forms lung edema.
- In this way, the consistency of atelectasis and edema-shaped lung areas increases, the volume is either normal or increased.
- Liquid leaks from its section.
- The alveolar cavities in the microscope contain gas-mixed edema fluid.
- Alveolar cells are swollen.
- Emphysema is observed around this region. These areas may be complicated by bronchopneumonia.

b. Compression atelectasis :

- It is the result of physical pressure or spinal deformity caused by excess fluid, gas or large masses on the lungs. For example, pneumothorax, hydrothorax, hemothorax, chylothorax, embolism, intrathoracic tumors and meteorismus (pressure of the diaphragm into the chest cavity) can occur with pressure.
- In the atelectasis due to compression, the lung is pale due to ischemia.
- It is hard and has a thick consistency.
- The section is gray-red in color and the pleura is thick and folded.
- If there was fibrosis and adhesion in the pleura, all the lungs were collapsed.
- The alveolar walls of the microscope are close to each other.
- Bronchial and bronchial walls approach each other.
- There is nothing inside the alveoli and bronchi.
- If there is emphysema and edema in the surrounding area, the corresponding changes are observed.

c. Hypostatic atelectasis:

- It is shaped especially in large-scale animals that have to lie for a long time.
- It can also be shaped as a disorder due to prolonged anesthesia.
- In such animals, the air-blood balance is impaired, breathing is difficult, drainage is lacking, and surfactant activity is reduced.
- Here, the lung tissue retains its long-lasting recovery.
- It then follows a slight inflammation and then fibrosis.
- In addition, atelectasia with sharp edges, ribbon or lobular can be found in the cranioventral regions of the lungs during sheep.
- Most of them are shaped by bronchi and bronchioles. In some cases, the or this blockade was not found.

d. Massive atelectasis (massive lung collaps)

- Usually the end of the pneumothorax is shaped.
- However, long-term use of the respiratory device in the intensive care unit, 80% -1000 oxygen is formed in the lungs of cats and dogs.
- Oxygen was completely resorbed into the tissues, and postmortal examination revealed no gas in the lungs.
- These lungs are shrunk as a whole, dark red in color, and the blood leaks out of the cross-section.
- Generally, fetal atelectasis diffuse, obstructive type lobular, and others show a structure.
- In all types of atelectasis, the lungs are displaced to secondary infections.

- **As a result,** short-term atelectasis is improved.
- If it takes longer, these regions undergo induration and collapse.
- It results in edema, carnification or pneumonia.
- Cor pulmonale can be affected by the circulation.
- In atelectasis, death is shaped if adequate functional tissue is affected.
- If the animal lives, the change is reversible.

Emphysema pulmonum

Emphysema in its widest sense refers to tissue expansion by air or other gas. In the lung, there are 2 major forms:

1. **Alveolar (vesicular) emphysema**, which is rare in animals, air is present in alveoli

2. **Interstitial emphysema,** air is interlobular, subpleural and other important interstitial regions of the lung.

1. **Alveolar (vesicular) emphysema**, which is rare in animals, refers to abnormal and permanent enlargement of alveoli

resulting from destruction of alveolar septa, and an absence of obvious fibrosis. Emphysema thus differs from **overinflation**

of alveoli caused by air trapping, which is a reversible lesion that is commonly encountered.

2. **Interstitial emphysema** is the presence of air within interlobular, subpleural, and other major interstitial zones of the lung. *"Emphysema," unless otherwise qualified, should only be used for alveolar emphysema.*

Macroscopically :

Emphysematous areas of lung are grossly voluminous, pale,and puffy. The enlarged airspaces are often visible as small vesicles, and in severe cases, coalescence of airspaces can produce large air-filled bullae. Emphysematous bullae occasionally rupture to cause fatal pneumothorax.

Microscopically :

Enlargement and coalescence of airspaces are apparent histologically, and are most reliably assessed on lungs that have been infused with fixative to a volume approximating the in vivo state.

In true emphysema, fragments of alveolar wall may be apparent histologically, in contrast to the more frequent lesion of alveolar overinflation.

However, emphysema and overinflation are often difficult to distinguish. Emphysema may affect the terminal bronchioles and adjacent alveoli (centrilobular or centriacinar emphysema), or more uniformly involve the entire lobule (panlobular or panacinar).

- Current knowledge of the **pathogenesis of emphysema** is based on investigations of the human disease and of animal models. Emphysema is an important condition in humans, where it frequently coexists with chronic obstructive pulmonary disease attributable to cigarette smoking and is the result of an imbalance between proteases and antiproteases.
- *Neutrophil-derived serine proteases, particularly elastase, and matrix metalloproteinases from a variety of sources are the likely culprits,* and their concentrations are enhanced by the neutrophil and macrophage activation induced by chronic bronchitis.

Although antiproteases—α1-antitrypsin, secretory leukoprotease inhibitor, and tissue inhibitors of matrix metalloproteinases —

protect normal lung from proteolytic damage, their function may be reduced by genetic deficiency or by oxidative stress

from cigarette smoke and the resulting inflammation.

The emphysematous lung is dysfunctional because the loss of alveolar septa reduces the alveolar surface area; although the affected portion of lung may be larger than normal, gas exchange is reduced.

Loss of elastic recoil caused by degradation of elastin further compromises lung function.

Congenital lobar overinflation (congenital lobar emphysema) is rare in dogs, and results from collapse or obstruction of a lobar bronchus, such as by hypoplasia of the cartilage, leading to air trapping in the affected lobe.

The right middle or cranial lung lobes are greatly enlarged by alveolar overinflation and/or bullous emphysema, sometimes with bronchiectasis.

Pneumomediastinum, subcutaneous edema, or

pneumothorax may occur. Affected dogs typically have slowly progressive dyspnea and coughing, and surgical excision is usually curative.

Emphysema is acute or chronic according to duration.

Although acute emphysema is reversible, chronic emphysema is irreversible (desquamative emphysema) type which is associated with structural defects in the lungs.

The replicated air is either localized in one or several regions of the lung and is considered to be a partial emphysema, or occurs throughout the lung, which is called diffuse or universal emphysema. In view of these criteria, emphysema in the lung:

I. Alveolar Emphysema

a. Acute partial alveolar emphysema

aa. Acute diffuse alveolar emphysema

b. Chronic partial alveolar emphysema

bb. Chronic diffuse alveolar emphysema

II. Interstitial emphysema.

II.Intersititial emphysema

In interstitial emphysema, there are different sizes of air bubbles in the interstitial tissue between the lobules.

In the accumulation of air these air bubbles find a few centimeters and they are called bullae (bullous emphysema).

The term bullous emphysema does not document a further disease phenomenon, but rather a large collection of air.

Sometimes all three types of emphysema (alveolar, interstitial and bullous) found together.

- Acute and chronic obstructive emphysema
- Lung strongylosis
- Chronic interstitial pneumonia
- Severe cough
- Injury (rib fractures)
- Foreign bodies (bovine) occur in the formation of interstitial emphysema

At the end of :

- The end of emphysema depends on the prevalence. Generally, hypoxia or anoxic death occurs.
- Bullous, interstitial emphysema results in pneumothorax.
- Cor pulmonale is shaped by the effect of blood circulation.
- Emphysema is an extremely important primary disease in humans.
- In humans' emphysema, there is a permanent abnormal expansion of air gaps with terminal air to the terminal bronchioles, as well as destabilization of the alveolar wall (alveolar emphysema).
- This needs to be distinguished from the expansion of simple air cavities, which are congenitally formed (Down's syndrome) or later formed by old age (aged lung, sometimes called false senile emphysema) and no destabilization.
- The pathogenesis of emphysema in humans is still controversial.

Fog fever, acute bovine pulmonary emphysema and edema (Çayır / Mera Amfizemi)

- The disease is known as Meadow or Pasture Emphysema in our country, Fog Fever in England (it has nothing to do with atmosphere conditions).
- However, in the definition of the disease, acute bladder emphysema and emphysema, and in some regions, especially in the United States are also used terms such as toxic atypical interstitial pneumonia (AIP).
- The disease is shaped depending on the pasture.

- Usually at the beginning of autumn (August-September), in the period when the short dry grass after the harvest turns to the juicy green grass, it is seen in the adult beef cattle grazing in these pastures (ın fog : pasture grazing, foggage = new growing herb after harvest).

- Although not fully proven, there is probably a role for L-tryptophan in the pathogenesis of the disease.

- L-tryptophan is metabolized to 3-methylindole during fermentation and reaches the lungs as it absorbs into the bloodstream.

- Here, the mixed-function enzymes found in the cells of the non-freite bronchiol epithelium (Clara cells) convert 3-methylindole into highly pneumotoxic metabolites.

- These cause a typical reaction in the lungs and shape of the lesions.
- The first lesion leads to severe necrosis in type I pneumocytes with bronchios cells.
- Then a change in the alveolar wall and lumen is formed.
- These animals are characterized by expiratory dyspnae, oral breathing, lungs, and even emphysema extending along the ridge.
- In the disease, respiratory distress occurs within 2 weeks following the change of pasture.
- In acute cases, the morbidity is around 10% -10% and in the herd, usually a few animals are severely affected, while others are unaffected. Letalite is recorded as 25-50%.
- In severe cases, death occurs 2-4 days after the onset of symptoms.
- In mild cases, the animal heals.

- Macroscopically, acute lethal cases contain large amounts of foamy edema fluid in large airways (trachea and bronchi).
- In the mucosa under the edema fluid, petechial and ecchymotic hemorrhages are seen with congestion.
- Severe interstitial emphysema in the lung and pulmonary edema with bullae usually occur.
- Subcutaneous emphysema is also seen at this stage.
- The lungs are enlarged, the parenchyma is red, and red-brown due to congestion and edema.
- The lobes are homogeneously affected and the section faces are moist.
- Lesions show sublobular or lobular localization.
- In general, the lesions in the dorso caudal region of the lung are diffuse spreading.

- Microscopic examination shows the edema and eosinophilic hyaline membranes in alveoli in acute lethal cases.
- The alveolar wall is also edematous, swollen and there are many eosinophils in this area.
- Type II pneumocytes have a single hyperplasia.
- In these subacute cases, type II alveolar epithelial cell hyperplasia (epithelization, fetal edema or adenomatosis) is very severe and persistent.
- Eosinophils and mononuclear cells are seen in peribronchial regions in the healing cases.
- In addition to these findings, fibrosis residues are also seen.

Pulmonary edema

Pulmonary edema is a frequent complication of many diseases and is therefore one of the most commonly encountered pulmonary abnormalities.

If severe, pulmonary edema has a catastrophic effect on lung function by reducing pulmonary compliance, blocking ventilation of the alveoli, obstructing gas exchange across the alveolar septa, and reducing the surface area of the air-liquid interface in the alveoli.

Edema of the lung is, in many respects, similar to edema

of other tissues, and is governed by the permeability of the vascular wall and by Starling forces—the balance of hydrostatic and osmotic pressures between the intravascular and interstitial compartments.

Distinctive aspects of edema in the lung include the importance of type I pneumocytes as barriers to fluid movement, the role of type II pneumocytes and club cells in active transport of water from the alveolus, the effect of surface tension on fluid movement, and fluid exchanges between the alveolar and bronchiolar airspaces and the pulmonary interstitium. It is useful to consider separately the means by which the alveoli are kept dry, and the ways in which excessive fluid is removed from the alveoli.

- Pulmonary edema is characterized by the localization of edema fluid from the interstitial to the alveolar.
- The main reason for this formation is the increase of permeability in capillary and type I alveolar epithelial cells. The other is the increased hydrostatic pressure.
- For this reason, pulmonary edema is considered to be either due to the increased permeability characteristic of the air-blood barrier or to the increased hydrostatic pressure (microvascular) or both factors.
- Thus, pulmonary edema is classified as hemodynamic and permeability edema.

- **Hemodynamic pulmonary edema** is characterized by an increase in fluid transudation due to a decrease in the hydrostatic pressure within the vessel or osmotic pressure in the blood.
- In hemodynamic pulmonary edema, pressure increases are usually due to heart.

Liver disease, hypoalbunemia, nephrotic syndrome, enteropathy, neoplasia at lymphatic system etc

- Permeability edema is characterized by excessive opening of the endothelial pores or the deterioration of the air-blood barrier (up to type I epithelium or endothelial cells).
- Increased permeability leads to rapid edema formation. This type of edema is shaped within the inflammatory response and is particularly due to local secretion of inflammatory mediators, for example leukotrienes, prostaglandins, platelet activating factor, cytokines and vasoactive amines.
- In some cases, permeability edema is shaped by direct destruction of the endothelium and alveolar epithelial cells. Type I pneumocytes are rapidly destroyed by pneumotrophic viruses (influenza, BRSV), toxic substances (NO_2, SO_2, H_2S, 3-methylindole) and in particular free radicals, resulting in the formation of alveolar edema.

- Inhaled corrosive gases (including 80% -1000 oxygen), bacterial toxins (especially in the enterotoxemia of sheep and pigs and septic shock),
- alpha-napthylthiourea, phosgene gas and lung embolization from other toxins (disseminated intravascular coagulation),
- type-I hypersensitivity in horses and cattle (anaphylaxis),
- pancreatitis,
- many allergic (milk allergy) and
- inflammatory reactions of the lung with the opposite drug reactions are also important causes of acute lung (permeability) edema.

- Secondary edema (neurogenic edema), which is shaped by acute brain trauma (damage to the vasomotor center) depends on both hemodynamic and capillary permeability disorder.

- On **gross examination**, edematous lungs are wet, heavy, and do not completely collapse when the thorax is opened; fluid oozes from the cut surface. The ratio of lung weight to body weight (or to heart weight in ruminants) is a useful measure of pulmonary edema. Edema is prominent in the pleura and the pulmonary interstitium, and may form shallow pools in the hilus of the lung or the mediastinum. In cattle and swine, the interlobular septa are obviously distended by clear fluid . Foam often fills the trachea and bronchi and flows from the nostrils, although this is a common and nonspecific finding in horses and sheep .
- The thoracic cavity may contain excess fluid. It is important to understand that the above features occur in severe cases, but pulmonary edema may be functionally significant yet grossly unremarkable.

Microscopically, edema fluid is often colorless and manifests simply as expansion and separation of constituents of the interstitial extracellular matrix, especially

surrounding blood vessels and the bronchovascular bundle.

With increased protein content, edema appears acidophilic,

especially within alveoli, where it is homogeneous or finely

granular except for occasional discrete holes that represent

trapped air bubbles.

Chronic edema is accompanied by a diffuse increase in the

number of alveolar macrophages, and in left -sided heart

failure, these may contain phagocytosed erythrocytes or

hemosiderin, the so-called “heart failure” cells.

Cor pulmonale (*pulmonary heart disease*)

- It refers to right heart ventricular hypertrophy and / or dilatation caused by lung hypertension in the lung parenchyma and vascular system.
- Here, hypertrophy and dilatation in the heart is shaped as a result of pulmonary hypertension.
- However, cor pulmonale which is shaped by secondary hypertension caused by cardiac disorders (such as right-left shunt, mitral stenosis) is also taken into this group.

- Cor pulmonale is divided into acute and chronic. Acute cor pulmonale is usually formed after massive pulmonary emboli.
- Here, rapid dilatation in the lung and right ventricle is shaped.
- Chronic cor pulmonale is more common than the acute one and is characterized by lung diseases that cause chronic pulmonary hypertension.
- In this case there is enough time to develop hypertrophy in the right heart.

- Hypertrophy of the right heart in chronic events is called Compensatoric or Concentric hypertrophic cor pulmonale.
- This hypertrophic heart is dilated over time. This is called Excentric cor pulmonale.
- In the latter case, right heart failure also occurs and disorders in the general circulation (such as edema, passive hyperemia in the liver) are also formed.
- In cattle, in less brisket disease (height disease) seen in horses and sheep, cor pulmonale is formed. The disease occurs in animals grazing over 2500 meters.

- Oxygen deficiency was caused by subcutaneous edema and chronic cor pulmonale.
- Ultimately, because small circulation encounters resistance in the lungs, the right heart tries to pump more blood and the blood pressure in the small circulation rises.
- Pulmonary hypertension and consequently hypertrophy of the right heart occur.