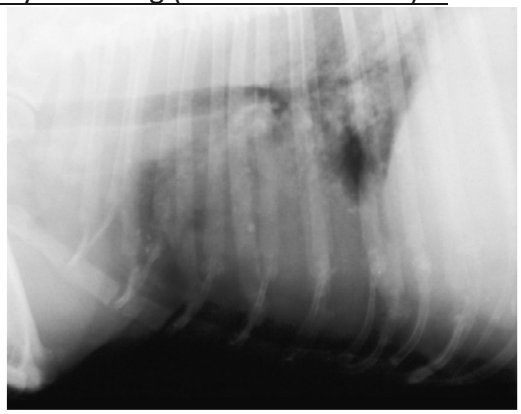
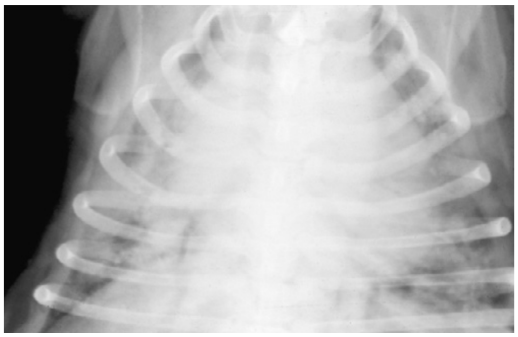
Idiopathic Pulmonary Fibrosis (IPF)

- Cryptogenic fibrosing alveolitis [CFA] in the United Kingdom and Europe
- Diagnosis of pathological exclusion, where there is no alternative explanation for the cause of lung fibrosis and the lung pathology has clearly identifiable pathological changes
- Pathological end-result of lung parenchymal inflammation
- There are specific conditions in which lung fibrosis is an inevitable pathological consequence of the disease
    - Associated with paraquat poisoning in dogs
    - Complication of Cushing's syndrome
- Though there is extensive understanding of the etiology of the diseases that secondarily cause lung fibrosis, little is known of the possible causes of IPF
    - Potential involvement of environmental pollutants?
- Breed-prevalent?
    - West Highland white terrier
- Underlying pathological mechanisms of fibrosis are complex and incompletely understood
    - End-stage lung fibrosis represents an aberrant remodeling process in response to injury
    - The reason for scar formation rather than return to normal structure and function is unknown
        - Fibrotic response due to up-regulation of gene expression for a range of cytokines?
- Both acute and chronic inflammatory mechanisms are implicated in the induction and maintenance of fibrosis
- The incidence of IPF in the dog and cat is unknown
- Gender bias towards males has also been reported for IPF in humans
    - Males twice as likely to be affected
    - In one study of West Highland white terriers, 17 were male and 12 were female, giving an approximate ratio of 60% to 40%
- Median age of onset is approximately 9 years in the West Highland white terrier
- Slow onset and progression of the disease
    - Coughing occurs late in the disease
    - +/- exercise intolerance, dyspnea, tachypnea, cyanosis, syncope
- Approximately 30% of cases are presented because of dyspnea
- PE (dogs): diffuse pulmonary crackles, with wheezes and rhonchi auscultated in many cases
    - +/- varying degrees of dyspnea, tachypnea, and cyanosis
- Limited information on disease progression
    - Slowly progressive
    - Deterioration is inevitable irrespective of treatment, with eventual respiratory failure
    - Expected survival time from the onset of clinical signs varies widely

        - Three to 41 months has been reported in the West Highland white terrier, with a median survival of 15.5 months
- Major differential in dogs with IPF is chronic bronchitis
    - In contrast to IPF, dogs with chronic bronchitis often have minimal radiographic changes and have bronchoscopic evidence of bronchitis
- Other ddx: respiratory infections, pulmonary infiltration with eosinophils, and infiltrative neoplasms
- Thoracic radiography is important in the diagnosis of IPF in the dog because collection of diagnostic biopsy material is unlikely
    - Radiographic changes can vary but tend to reflect the severity of the clinical presentation with varying degrees of a diffuse interstitial pattern and right- sided cardiomegaly
    - Sensitivity and specificity of radiography is very poor, and there is a very poor correlation with severity of disease, unless honey- combing (advanced disease) is present





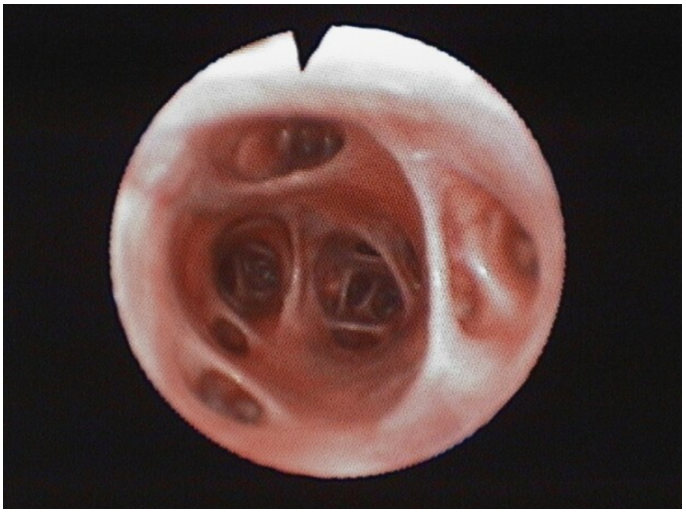
- Open lung biopsy is the main method for definitive diagnosis of lung fibrosis and the other interstitial lung diseases in humans
    - Though biopsy is necessary for confirmation of lung fibrosis, in a British Thoracic Society study, diagnosis of CFA in humans was still made on the basis of clinical findings in 60% of cases
- A strong tentative diagnosis can be made on the basis of the clinical presentation of chronic-onset coughing and dyspnea, diffuse pulmonary crackles, and radiographic changes
- Bronchoscopy and bronchoalveolar lavage (BAL) may be useful tests in canine IPF because they may allow exclusion of chronic bronchitis
    - In the majority of cases of canine IPF the BAL samples are normal or have low to moderate mixed populations of inflammatory cells
- On gross pathology, the lungs tend to be firm, heavy, and noncollapsable
- Right ventricular changes have also been noted (e.g., right ventricular hypertrophy and dilatation)
- Medical treatment in the dog relies on glucocorticoids (prednisolone) and bronchodilators
- Additional approaches to drug therapy in human patients include immunosuppressive and cytotoxic drugs (e.g., azathioprine and cyclophosphamide)
- Antifibrotic drugs (such as colchicine) of benefit?
- Survival times in humans are best in young patients, especially if they are female

    - Presence of right-sided cardiomegaly and right axis deviation (suggestive of cor pulmonale) are poor prognostic indicators

Bronchiectasis

- Pathological destruction of the elastic and muscular components of the bronchial wall leading to chronic abnormal dilation and distortion of the bronchi
    - Chronic airway infection and inflammation and resulting bronchiectatic changes
    - Damage to the epithelial cells lining the airways induces squamous metaplasia and ciliary loss, which leads to impairment of the mucociliary apparatus
    - Dysfunction of the mucociliary apparatus allows pooling of mucus, exudate, and microbes in the distal airways
- Reversible dilation of the bronchi has been described in acute pulmonary diseases in humans (e.g., pneumonia, tracheobronchitis, and atelectasis) and must be differentiated from true bronchiectasis
- May be congenital or may develop secondary to acquired disease, with the latter being much more common
- Humans: acquired causes
    - Diseases that cause bronchial obstruction (e.g., asthma, chronic bronchitis, panbronchiolitis, neoplasia, foreign body, hilar lymphadenopathy, recurrent aspiration pneumonia, and broncholiths)
    - Necrotizing or suppurative pneumonia
- Dogs: acquired bronchiectasis usually develops as a result of eosinophilic bronchitis, chronic bronchitis, bronchiolitis, or bronchopneumonia
- Cats: although allergic bronchitis (feline asthma) and chronic bronchitis are common clinical disorders, bronchiectasis is rarely found in association with these diseases
- Middle age or older, consistent with the higher incidence of acquired versus congenital bronchiectasis
- American cocker spaniel dogs and Siamese cats breed prevalence?
- Trend for male overrepresentation was noted in cats, no sex predisposition in dogs
- Hx/PE:
    - Chronic cough; purulent or mucopurulent sputum production; wheezing; dyspnea; recurrent fever; hemoptysis; and, in advanced stages, anorexia and weight loss
    - Dogs gag; tachypnea; and occasionally fever
    - Retrospective study in cats, only 5 of 12 had clinical signs referable to the respiratory system (i.e., cough, tachypnea, and dyspnea)
- Two key components in the diagnostic evaluation:
    - Dilated airways must be recognized and localized because this pathologic process by itself is responsible for ongoing bronchopulmonary inflammation
    - The underlying disease process that led to the development of bronchiectasis must be identified
- Bronchiectasis can be detected by survey thoracic radiography, bronchography, high resolution computed tomography (HRCT), bronchoscopy, and histology

    - Different radiographic patterns can be seen in patients with bronchiectasis
        - Major forms include cylindrical, saccular, cystic, and varicose bronchiectasis
    - Survey thoracic radiography may not be a sensitive test for bronchiectatic changes because imaging of the bronchial walls is dependent on inflammation and fibrosis of the airways, conditions typical of advanced disease
- Gross examination of bronchiectatic airways reveals prominent dilation and luminal filling with purulent secretions
- Histological examination of the lungs reveals dilation of the affected airways and various degrees of airway wall remodeling with granulation tissue and fibrosis
- Microscopically, the lumen of the airways is usually filled with mucus, proteinaceous material, and inflammatory cells
- Bronchiectasis is irreversible
    - Goals of therapy are to control clinical signs and slow the progression of disease
    - As most cases of bronchiectasis in dogs and cats are acquired secondary to an underlying disease, addressing the primary pathological process is vital to attempt to halt the progression of destruction of the bronchial walls
    - Airway humidification may help loosen secretions and avoid inspissation and subsequent airway obstruction
    - Anti-inflammatory
        - Beneficial role of inhaled steroids has been shown in humans with bronchiectasis
- Bronchiectasis can lead to bronchopneumonia, pulmonary hemorrhage, bronchiolitis obliterans and emphysema, chronic respiratory insufficiency, and cor pulmonale
- Focal bronchiectasis treated with surgical lobectomy is associated with a good prognosis



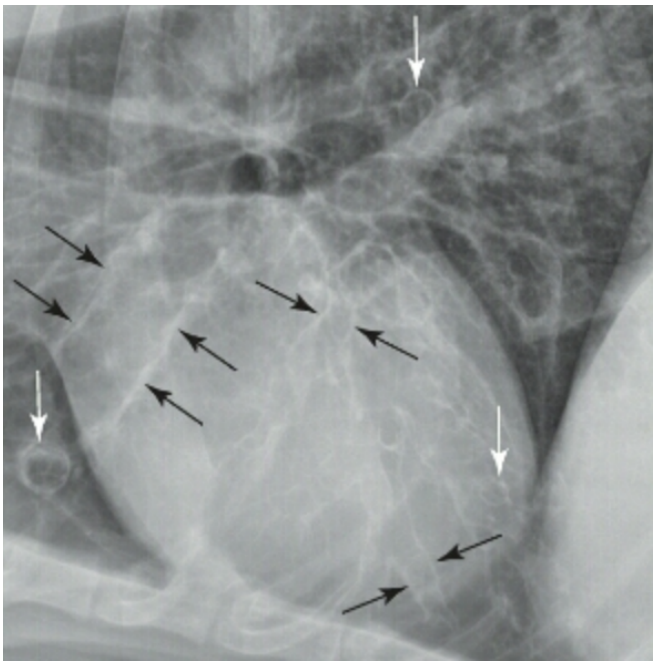
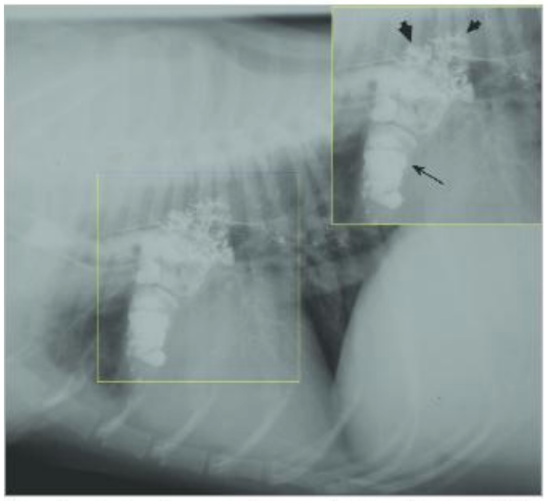
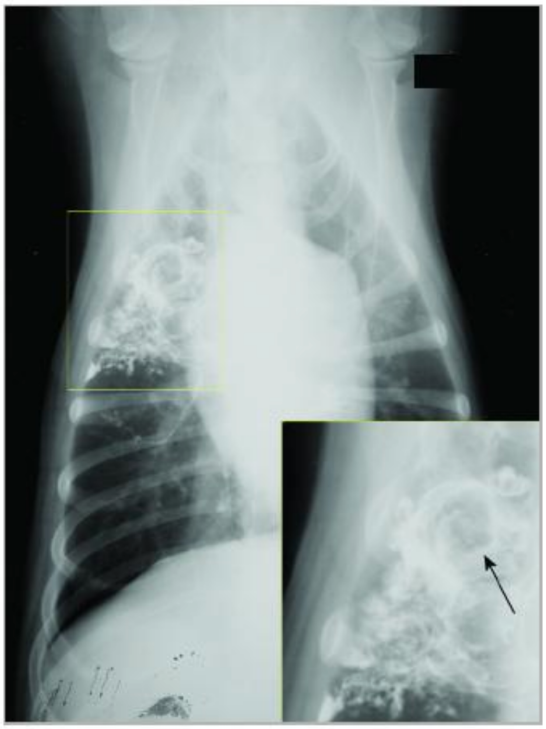


Figure 7. Bronchogram.



Lateral projection demonstrating saccular bronchiectasis within the right cranial lobar bronchus (*arrow* in *inset*) and normal left cranial lobar bronchus dorsally (*arrowheads*).



Ventrodorsal projection showing the severely dilated bronchus (*arrow* in *inset*).

Chronic Bronchitis in Dogs

- Chronic bronchitis is essentially an incurable disease of insidious onset usually seen in middle age or older dogs of the small breeds
- It is characterized clinically by a chronic, persistent cough and characterized pathologically by chronic inflammation of the airways, as well as mucus hypersecretion
- The cough is usually productive with gagging, but because dogs do not expectorate, the production of excess mucus may be difficult to recognize
- Chronic bronchitis in dogs is defined as a condition of chronic or recurrent excessive mucus production in the bronchial tree for at least 2 consecutive months in the preceding year, and manifested clinically by chronic coughing
    - Diagnosis of chronic bronchitis requires fulfillment of three major criteria:
        - Chronic cough
        - Evidence of excessive mucus or of mucus hypersecretion
        - Exclusion of other chronic cardiorespiratory diseases
            - (e.g. congestive heart failure, chronic bacterial pneumonia, pulmonary neoplasia, parasitism, airway collapse and fungal pneumonia)
- The most common functional sequela of chronic bronchitis is chronic airflow obstruction, which is generally referred to as chronic obstructive pulmonary disease

(COPD)

- It is generally accepted that the development of chronic bronchitis is the result of a vicious cycle of airway damage and patient response
    - Persistent infection or chronic inhalation of airborne irritants can result in sustained injury to the bronchial epithelium
    - Stimulating metaplastic transformation of the ciliary epithelium, hyperplasia and hypertrophy of mucus-secreting glands and cells, and hyperemia and cellular infiltration of the bronchial mucosa
- The viscid mucus contains a large number of neutrophils and macrophages admixed with varying amounts of cellular debris and edema fluid
    - Smaller bronchi are often occluded by thick mucus plugs
- The bronchial mucosa is usually hyperemic, thickened, and edematous
    - Polypoid proliferations often project from the mucosa into the bronchial lumen
- Patchy pneumonia is a complicating factor in about one quarter of the dogs
- Emphysema is a much less important lesion in the dog than in humans, and is primarily confined to the edges of the lung lobes
- Once bronchiectatic airway changes occur, they are irreversible
    - As all these changes impede normal defense mechanisms, bacterial colonization of the airways commonly results
    - Collapse of the major airways impedes expiratory airflow and efficient clearance of mucus from the bronchial tree, exacerbating the clinical condition of patients with chronic bronchitis
- Airway diameter is reduced in chronic bronchitis by a combination of the following mechanisms:
    - Edema and cellular infiltration of airway walls
    - Copious quantities of tenacious intraluminal mucus
    - Localized endobronchial narrowing associated with fibrosis of the lamina propria and polypoid proliferations of the mucosa
    - Spasticity of bronchial smooth muscles causing reactive airway narrowing (may not be as significant in dogs as in humans)
    - Collapse of larger bronchi associated with weakening of the bronchial walls subsequent to chronic inflammatory activity
    - Plugging of smaller airways by tenacious mucus
    - Obliteration of bronchioles as a result of inflammatory activity
    - Emphysema develops following flooding of the alveoli with mucus
- The persistent airway inflammation associated with chronic bronchitis is responsible for

BOX 52-1
Possible Causes of Chronic Bronchitis in Dogs

Atmospheric Pollution

Passive Smoking

Chronic exposure to smoke in poorly ventilated confined spaces

Respiratory Tract Infections

Chronic fungal infection
Chronic bacterial infection
- *Bordetella bronchiseptica*
- *Mycoplasma spp.*

Viral infection
- Canine distemper virus
- Adenovirus (types 1 and 2)
- Herpesvirus

Parasites
- *Filaroides milksi; Filaroides herthi*
- *Crenosoma vulpis*
- *Capillaria aerophila*
- *Dirofilaria immitis*

Genetic or Acquired Defects

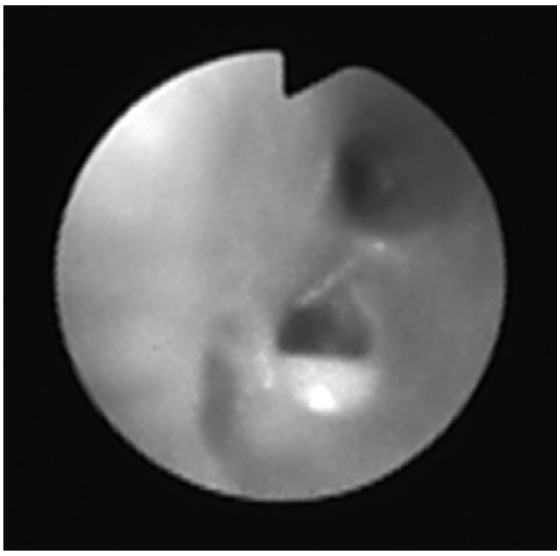
α_1-antitrypsin deficiency
Mucociliary defects
Immunodeficiency

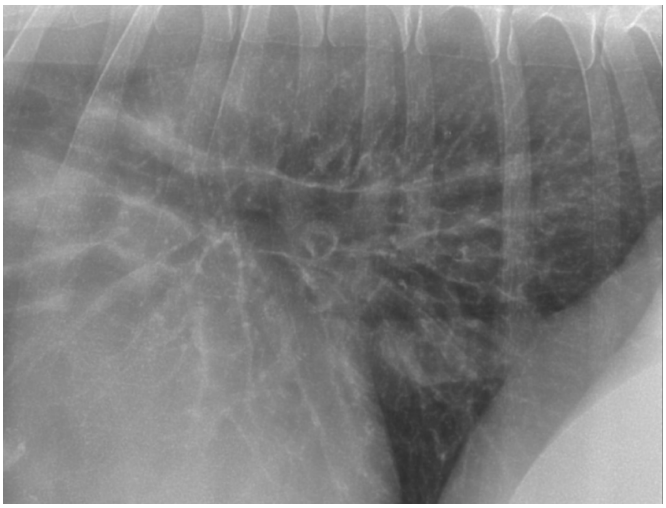
Hypersensitivity (Allergic) Lung Disease

the development of refractory airflow obstruction
    - Disease of small airways, therefore, must be diffuse and extensive before airway resistance is enhanced sufficiently to bring about clinical signs
    - Small airway disease in the dog must be remarkably extensive before clinical signs of shortness of breath associated with COPD are observed (Pouiselle's law)
- Enhanced airway resistance and a decline in maximum expiratory airflow rate are characteristic findings
    - Primarily manifests clinically as expiratory dyspnea
- Causes of chronic bronchitis are poorly understood in the dog, and usually remain unknown in individual patients
    - Major difficulty in determining the cause of chronic bronchitis is because the disease is detectable only in its advanced stages
    - Largely because chronic bronchitis has an insidious onset and lengthy pathogenesis, and the diagnosis is largely based on a descriptive clinical definition
- Clinical signs usually seen in patients with chronic bronchitis include:
    - Persistent, intractable, productive cough with gagging and production of sputum, which is typically swallowed and thus difficult to document
    - Cough may be unproductive, resonant, harsh, hacking during the day and productive during the evening or early morning hours
    - Paroxysmal cough precipitated by exercise or excitement
    - Obesity
    - Cyanosis, collapse, exhaustion, and exercise intolerance
    - Pronounced sinus arrhythmia
    - Expiratory dyspnea
    - Varying periods of remission followed by exacerbation of coughing (exacerbations may be in association with changes in weather, particularly cold weather)
    - Systemic signs of illness may be seen during severe exacerbations or episodes of bronchopneumonia
- Paninspiratory crackles and expiratory wheezes are the most commonly heard adventitious (abnormal) breath sounds.
    - Though many dogs with chronic bronchitis have normal auscultation findings
- Most important differential diagnoses that must be ruled out are cardiac diseases (typically chronic mitral regurgitation), chronic bacterial pneumonia, pulmonary neoplasia, foreign body bronchitis, hypersensitivity airway disease, dirofilariasis, pulmonary parasites, fungal pneumonia, dysphagia, and megaesophagus
- Since the diagnosis of canine chronic bronchitis is largely based on the history of chronic cough, diagnostic tests are performed to rule out other causes of chronic cough
    - Thoracic radiographs from dogs with nonobstructive chronic bronchitis usually show bronchial wall thickening or generalized increased airway-oriented interstitial density or both
        - Peribronchial and interstitial densities in thoracic radiographs of older

dogs have been shown to correlate with significant histologic abnormalities
        - Dogs with obstructive chronic bronchitis (e.g., chronic bronchitis and chronic obstructive pulmonary disease) have radiographic evidence of pulmonary hyperinflation in addition to bronchial wall thickening and a generalized increase in airway oriented interstitial density
    - Bronchoalveolar lavage or tracheal wash to collect material for cytology and microbiology should be considered in all dogs suspected of having chronic bronchitis
        - Excess mucus with either normal or hyperplastic bronchial epithelial cells; and increased numbers of macrophages, goblet cells, neutrophils, and lymphocytes are typically present
        - Bronchopulmonary cytology supports the presence of infection based on the findings of intracellular bacteria or the toxic appearance of neutrophils
            - Most common isolates are *Bordetella bronchiseptica, Streptococcus* spp., *Pasteurella* spp., *Escherichia coli, Pseudomonas* spp., and *Klebsiella* spp.
- Bronchoscopy may reveal erythema and a roughened granular appearance of the mucosa
    - The mucosa of-ten appears thickened, irregular, and edematous
    - Occasionally polypoid or nodular proliferations are seen projecting into the bronchial lumen
    - Excessive thick tenacious mucus may be found in strands or small plaquelike accumulations within the airways
    - Small airways may be occluded by mucus plugs
    - Collapse of the dorsal tracheal membrane into the lumen of the airway is commonly observed in dogs with chronic bronchitis and indicates the presence of concurrent tracheal collapse
- Chronic bronchitis is largely a clinical diagnosis, tissue biopsy is not required for confirmation
    - Fibrosis; edema; and cellular infiltration of the lamina propria by lymphocytes, plasma cells, macrophages, and neutrophils are seen histopathologically
- Extremely severe cases may have medial hypertrophy of the small pulmonary arteries and muscularization of the pulmonary arterioles, associated with right ventricular hypertrophy as a result of chronic hypoxic pulmonary hypertension
- The structural alterations in airway anatomy associated with chronic bronchitis are not readily reversible, if at all
    - Bronchiectasis, tracheobronchial collapse, and emphysema are permanent, irreversible changes that complicate the management of these patients
- Management of patients with chronic bronchitis is divided into five major categories:
    - Avoidance of exacerbating factors and control of body weight
    - Relief of airway obstruction and inflammation

    - Control of cough
    - Control of infection
    - Oxygen therapy
- Housing in a clean, cool environment and using a harness rather than a collar is also recommended
- Three types of therapy are typically employed:
    - Antiinflammatory medications, bronchodilator medications, and treatments that promote removal of accumulated airway secretions
- The use of antibiotics should only be based on demonstrated evidence of bronchial infection.
- Chronic bronchitis is a common, progressive, and chronic airway disorder that can often be managed but is essentially incurable
    - Most dogs are only affected by a recurrent cough
- The major complications associated with chronic bronchitis are the development of COPD, bronchopneumonia, bronchiectasis, and, in severely affected dogs, cor pulmonale





Feline Bronchial Disease/Asthma

- Feline bronchial disease (feline asthma or bronchitis) is one of the most common respiratory diseases in cats
    - Recognized clinically by various combinations of cough, wheeze, exercise intolerance, and respiratory distress and is characterized pathologically by inflammation of the lower airways without an obvious identifiable cause
- Pathophysiology of feline bronchial disease has not been completely elucidated
- Clinical signs range from intermittent cough to severe respiratory distress
    - Attributable to airway obstruction caused by bronchial inflammation, with

subsequent smooth muscle constriction, epithelial edema, and mucous gland hypertrophy and hyperactivity

- Decreased airflow in the small airways is caused by excessive mucus secretion, airway edema, cellular infiltrates, and smooth muscle hypertrophy and constriction
- Severe lower airway obstruction in cats with asthma can lead to lung hyperinflation because they are unable to exhale completely past the narrowed airways, resulting in air trapping
    - Can induce such dramatic intraluminal pressure for significant periods that permanent airway dilation (bronchiectasis) and loss of pulmonary elastic support structures (emphysema) may result
    - For unknown reasons, this process seems to affect the right middle lung lobe in cats with bronchial disease more often than other lobes
- Coughing may be initiated by a variety of factors in cats, including airway compression; the presence of foreign material, noxious gases, tissue, mucus, or fluid in the tracheobronchial tree; airway inflammation; or airway smooth muscle contraction
- Asthma is characterized by localized accumulation of inflammatory cells in the airway, particularly eosinophils and activated lymphocytes
    - Eosinophils appear to be primary effector cells in the development of asthmatic airway pathophysiology in cats as well as in humans
- Conditions that might be identified as stimulants of clinical signs in cats with bronchial disease include allergens, air pollution, and aerosolized irritants
- Respiratory infections increase airway hyperresponsiveness, possibly by causing or enhancing bronchial inflammation via stimulation of local cytokine secretion
- Symptoms are often chronic or slowly progressive; however, cats with severe exacerbations may present acutely with open mouth breathing, dyspnea, and cyanosis due to bronchoconstriction
- Cough is fairly specific for tracheobronchial disease in the cat because cats with pulmonary edema due to heart disease do not typically cough
- Many asthmatic cats can appear normal at rest, and thoracic auscultation may be unremarkable
    - Because bronchial disease is an obstructive disease of the small, lower airways, most affected cats exhibit a prolonged expiratory phase of respiration, and audible wheezes or crackles may be heard with or without the aid of a stethoscope, usually during expiration
- 20% of cats with bronchial disease have a peripheral eosinophilia
- Some cats have hyperglobulinemia, suggestive of chronic immunological stimulation
- Routine thoracic radiographs can be within normal limits in some cats with bronchial disease
    - Asthma should not be ruled out based solely upon normal rads
- The classic lung pattern in a cat with bronchial disease includes evidence of bronchial wall thickening (doughnuts or railroad tracks) because of airway inflammation
    - Air trapping may also be evident in the peripheral lung fields

- BAL/bronchoscopy generally provides evidence of airway inflammation, with increased numbers of eosinophils and/or neutrophils
    - Eosinophilic airway washes are not pathognomonic for asthma or bronchial disease
    - Samples should be submitted for culture of aerobic bacterial and mycoplasmal organisms and for antibacterial susceptibility testing though significance of a positive culture is unknown at this point
- Eosinophilic and/or neutrophilic bronchial inflammation with smooth muscle hyperplasia are common histopathological findings in cats with bronchial disease
- Hyperplasia and hypertrophy of goblet cells and submucosal glands are also common features, as is subsequent mucus accumulation with inflammatory cellular debris in the bronchial lumen
- Lobular and bullous emphysema, which may occur as a possible consequence of chronic obstructive airway disease, has been described in a small number of cats with bronchial disease
- No consistently reported strategy for the treatment of bronchial disease in cats
    - Four components of asthma treatment in humans:
        - Use of objective measurements of lung function to assess asthma severity and to monitor the course of therapy
        - Establishment of environmental control measures to avoid or eliminate factors that precipitate asthma symptoms or exacerbations
        - Utilization of comprehensive pharmacologic therapy for long-term management of disease that is designed to reverse and prevent airway inflammation and to manage asthma exacerbations
        - Employment of patient education that fosters a partnership among the patient, his or her family, and clinicians
- Emergency management
    - Initially, bronchodilator therapy (e.g., terbutaline 0.01 mg/kg IV, IM, or SC) should be used to combat acute bronchoconstriction
    - Inhaled bronchodilator medication (e.g., albuterol) may be used if the equipment is available and if the patient tolerates this method of administration
    - A positive response is expected within 30 to 45 minutes, and is indicated by a decrease in respiratory frequency and effort
        - If the cat does not respond in that time, a repeated dose of bronchodilator medication is warranted and a rapidly acting corticosteroid (e.g., dexamethasone 0.25 to 2 mg/kg IV or IM) should be administered
- Aminophylline exhibits weaker bronchodilatory activity than terbutaline and is not recommended as the first choice in emergency situations
- (Beta-blockers (e.g., propranolol and atenolol) should not be administered to cats in which bronchial disease is a possible cause for respiratory distress
    - Cats rely heavily on sympathetic tone for bronchodilation, and inhibition of beta-agonist activity may have dire consequences in these patients)

- Chronic management
    - Decrease allergen/irritant exposure
    - The most consistent, reliable, and effective treatment for feline asthma or bronchitis is high-dose (initially), long- term, oral corticosteroids
    - Bronchodilators seem to be most useful in human and feline patients during acute exacerbations caused by bronchoconstriction
        - Beta-2 adrenergic agonists (e.g., terbutaline 0.625 mg PO BID)
            - Cats that have airway obstruction due to remodeling of the airways are less likely to show a positive response
            - Potential side effects of terbutaline administration include tachycardia, agitation and hypotension due to slight beta-1 agonist activity
        - Methylxanthine derivatives (e.g., theophylline and aminophylline) have been used extensively, and may be useful in some cats with bronchopulmonary disease
            - This class of drug appears to cause bronchodilation via a combination of mechanisms
                - May inhibit a phosphodiesterase isoenzyme, increasing cAMP concentrations and causing bronchodilation
                - May inhibit adenosine, a mediator of bronchoconstriction
                - May interfere with intracellular calcium mobilization
            - Inhibition of mast cell degranulation and increased strength of respiratory muscles
            - A suggested initial dosage of generic sustained-release theophylline is 10 mg/kg PO once daily in the evening
    - Cyproheptadine
        - *In vitro* studies have shown that serotonin, a mediator released from mast cells, contributes to airway smooth muscle contraction; and that cyproheptadine, a serotonin antagonist, significantly attenuates this response
    - Cyclosporine
        - Activated T cells play a large role in the pathophysiology of asthma, and it can be theorized that cyclosporine (a potent inhibitor of T cell activation) may be effective in asthma therapy.
    - Leukotrienes antagonists
        - Leukotrienes are inflammatory mediators that may contribute to the pathophysiology of certain forms of asthma in humans and in some animal models by causing airway smooth muscle contraction, increased microvascular permeability, stimulation of mucus secretion, decreased mucociliary clearance, and by acting as eosinophil chemoattractant agents
    - Antibiotics
        - Respiratory bacterial infections are rarely associated with clinical

bronchial disease in cats, and bacteria may be cultured from tracheobronchial washes in healthy cats
        - Antibiotics are rarely indicated or effective for the treatment of asthma in cats
- Effective therapy should eliminate or significantly minimize the clinical signs
    - The diagnosis of bronchial disease should be questioned if a significant response is not appreciated within 1 to 2 weeks of initiating proper treatment
- The majority of cats with bronchial disease respond to appropriate therapy, yet it should be assumed that lifelong treatment may be required

Canine Eosinophilic Bronchopneumopathy

- The authors use the term eosinophilic bronchopneumopathy rather than pulmonary infiltration with eosinophils or pulmonary eosinophilia, because EBP takes into account the fact that bronchial infiltration and parenchymal involvement are almost always present in these cases
- A cause is rarely identified, and most cases of EBP are considered idiopathic
- The syndrome of asthma has not been recognized in dogs, although the authors have observed apparent bronchial hyperactivity in some advanced cases of canine EB
- The cause of canine EBP remains unclear, although hypersensitivity to aeroallergens is suspected
- At initial presentation, cough is the most common clinical sign, occurring in 95% to 100% of dogs
    - The cough is usually harsh and sonorous, persistent, and frequently followed by gagging and retching
    - Early in the course of disease, gagging and retching might be confused with a disorder of the digestive tract
- Other clinical signs frequently reported include respiratory difficulty and exercise intolerance
- Nasal discharge is present in up to 50% of cases
    - Can be serous, mucoid, or mucopurulent and can be associated with a concomitant eosinophilic rhinitis in some cases
- General systemic health is not always affected unless concomitant disease is present
- Pruritus, with or without skin lesions, is another clinical complaint that is occasionally reported
- PE: thoracic auscultation can be normal but increased lung sounds, wheezes, or crackles are often found
- Diagnosis relies on radiographic and bronchoscopic findings, blood eosinophilia, tissue eosinophilic infiltration demonstrated by cytology of BALF or histopathologic examination of bronchial biopsies, and exclusion of known causes of eosinophilic infiltration of the lower airways
- Treatment relies on steroids