

# Bronchial disease in the dog and cat

**Nicole Van Israël DVM CESOpht CertSAM CertVC Dipl ECVIM-CA (Cardiology) MSc MRCVS**  
ANIMAL CARDIOPULMONARY CONSULTANCY (ACAPULCO), EUROPEAN SPECIALIST IN VETERINARY  
CARDIOLOGY  
RUE WINAMPLANCHE 752, 4910 THEUX, BELGIUM.

This is the first of a series of two articles describing the more common lower respiratory diseases in dogs and cats. Although they have been separated into two articles (bronchial disease and pulmonary parenchymal disease), one needs to be aware that there is often overlap between the anatomical localisation of the different pathologies.

## **CANINE INFECTIOUS TRACHEOBRONCHITIS (KENNEL COUGH)**

### **Aetiology**

Kennel cough is an infectious disease where the pathogens can act on their own or in a group. The most commonly isolated agents are *Bordetella bronchiseptica*, Parainfluenza III, but also canine distemper, canine herpes virus, adenovirus II and reovirus. However more recently *Mycoplasma* spp. appear to be emerging (often associated with bronchopneumonia).

Transmission occurs mainly by inhalation or by direct contact with oronasal secretions.

### **Clinical signs**

Single agent infection (often viral) results in mild clinical signs but concurrent bacterial infection can cause complications that may be life-threatening. A paroxysmal acute cough, occurring after kennelling, hospitalisation or contact with an infected animal (park, dog playgrounds) is the hallmark of canine infectious tracheobronchitis. It is often associated with retching and expectoration. The cough can be associated with anorexia, lethargy and fever, but the latter is not always present and can be transient. Depending on the causative agents, serous to mucopurulent nasal and ocular discharge can be present (Fig. 1). Puppies are often more severely affected.



**Fig. 3:** Nasal mucopurulent discharge is a common associated feature in more severe kennel cough cases.

### **Diagnosis**

The history and the clinical signs suggest this diagnosis. Additional assessment is rarely necessary unless the animal is severely ill and the cough is persistent for more than three weeks.

Thoracic radiographs might be indicated if there is suspicion of pulmonary parenchymal involvement. In those cases with associated bronchopneumonia, bronchoscopy and broncho-alveolar lavage (BAL) might be indicated to isolate the causative agent and to guide appropriate antibiotic therapy (Fig. 2). A (trans)tracheal (Fig. 3) wash is an alternative when there is no access to BAL.



**Fig. 2:** Bronchoalveolar lavage fluid should be submitted for cytology and culture and sensitivity.



**Fig. 3:** A tracheal wash is a good alternative for those that have no access to fancy equipment such as (video) bronchoscopy.

### **Treatment**

In most cases the disease is self-eliminating and no treatment is warranted. Isolation from other animals is always recommended. Antibiotics are rarely required and should only be used where there is a clear indication (severe bronchopneumonia, pyrexia, pulmonary parenchymal involvement). Their use should be determined on the basis of culture and

sensitivity from the airway, and an antibiotic should be chosen that reaches appropriate therapeutic concentrations in the bronchial wall. The author's preference, in the absence of excessive mucus secretion is a trimethoprim/sulphonamide combination. Doxycycline is an alternative although this is a bacteriostatic drug. Quinolones, although effective, should not be used as first line treatment. Nebulisation with gentamycin might reduce the population of *Bordetella bronchiseptica* in the airway of infected dogs but its use is only recommended as an adjuvant treatment. Corticosteroids and anti-tussives are strongly contraindicated in the acute phase of the disease.

### Prophylaxis

Parenteral vaccination with different types of vaccines and strains is widely available but protection is not always 100%. Duration of protection depends on the individual vaccine. An intranasal vaccine including Bordetella and Parinfluenza strains, inducing a local immunity, is also available.

### 'TRACHEOBRONCHIAL SYNDROME'

This syndrome describes a group of dogs that continue to cough after a kennel cough infection despite the absence of infectious agents. The exact mechanism of the cough remains unclear but antitussive and low-dose corticosteroid therapy is often successful in abolishing the cough. However some dogs may progress to develop chronic bronchitis.

## CHRONIC BRONCHITIS

### Definition

Chronic bronchitis is an incurable disease characterised by chronic inflammation of the bronchi, associated with hypersecretion of mucus. Its chronicity is defined by the persistence of a chronic cough for a period of at least two months in the last year. Chronic bronchitis will cause chronic airflow obstruction and is therefore also referred to as COPD (chronic obstructive pulmonary disease).

### Prevalence

It is more commonly seen in middle-aged and older small breed dogs. Terriers might be predisposed. Chronic bronchitis can occur in older cats but is relatively rare. It should be differentiated from feline asthma.

### Aetiology

The exact aetiology is unknown, but kennel cough, environmental pollution (cigarette smoke and other irritants), allergenic challenge, mucociliary defence deficiency might contribute to this multifactorial disease.

### Clinical signs

A persistent, slightly productive cough associated with production of sputum is observed (although the sputum might be swallowed and remains unnoticed by the owner). Exercise intolerance is a common feature in severely affected animals. Cyanosis can

occur but is rare (Fig. 4). Obesity is commonly a complicating factor (Fig. 5).



**Fig. 4:** Wide stand (elbows abducted) and mild cyanosis in a 16-year-old Terrier with chronic obstructive pulmonary disease secondary to severe chronic bronchitis.



**Fig. 5:** Obesity often compromises the dog with chronic bronchitis and COPD and weight reduction should be an important part of the treatment strategy.

### Diagnosis

The diagnosis of chronic bronchitis requires the presence of the following:

1. chronic cough
2. evidence of excessive bronchial mucus production
3. absence of other chronic cardiorespiratory disease.

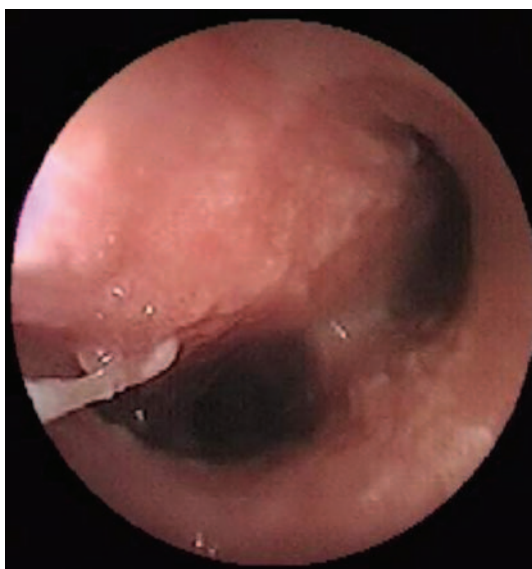
An expiratory dyspnoea will be present. Auscultation might reveal the presence of a marked sinus arrhythmia due to increased vagal tone, and wheezes and end-inspiratory crackles might be heard over the lung fields.

Thoracic radiography is very helpful in supporting the diagnosis. A marked bronchial pattern (dough nuts and tramlines) can often be recognised (Fig. 6). In the more chronic cases 'cor pulmonale' and air entrapment (lung overinflation and flattening of the diaphragm) might be observed. The presence of localised alveolar densities suggests the presence of concurrent infection.

Bronchoscopy is the gold standard for the diagnosis of chronic bronchitis. The mucosa appears thickened, irregular and oedematous, and in the more severe cases polypoid formations and saccular and irregular dilation of the small bronchi



**Fig. 6:** Detail of the radiographic appearance (doughnut and tram lines) of the bronchial wall in a dog with chronic bronchitis.



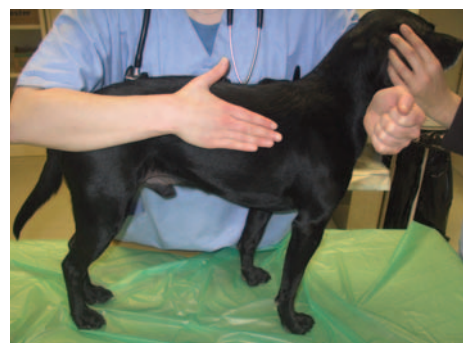
**Fig. 7:** Endoscopic image of the airways of a dog with chronic bronchitis.

(bronchiectasia) can be observed (Fig. 7). Excessive thick tenacious mucus is often present and occludes the smaller airways. Bronchial collapse during expiration might be observed in some patients. Bronchoalveolar lavage with cytology and culture must be considered in all cases to assess for secondary bacterial infections and other cardiorespiratory disease. Tissue biopsy is not required for confirmation. Non-invasive pulmonary function tests such as whole body plethysmography might give additional information in determining the presence and severity of obstructive disease (COPD) and it can give information with regard to treatment efficacy. The cost of the equipment and its availability limits its use in daily practice.

Full haematological assessment is always indicated to assess for pneumonia (neutrophilia with left shift) and chronic hypoxia (relative polycythaemia and reticulosis). Serum biochemistry might be useful to screen for aggravating underlying disease e.g. hyperadrenocorticism, but only if appropriate clinical signs are evident.

### Treatment

Eliminate the causative agent if one is identified. For opportunistic secondary bacterial infections antibiotics are required. Initially use a lipophilic antibiotic that crosses the blood-bronchus barrier (trimetoprim/sulfa, quinolones, clindamycin at bactericidal dosage alone or in combination in severe infections). In the absence of a secondary bacterial infection prescribe corticosteroids at an anti-inflammatory dose reducing to the minimum effective dose e.g. alternate day therapy. Bronchodilators (e.g. theophylline) might be helpful in some cases. Mucolytics and nebulisation assist airway clearance, and respiratory physiotherapy (coupage) (Fig. 8) is an emerging technique with probably underestimated value. Expectorants have no proven efficacy and anti-tussives should only be used on a short-term basis to avoid excessive accumulation of mucus in the airways. Weight control is imperative and animals should preferably be on the thin side. The collar should be replaced by a harness.



**Fig. 8:** Coupage (respiratory physiotherapy) can be of marked therapeutic value in animals with excessive mucus secretion and bronchopneumonia.

## BRONCHIECTASIA

### Definition

Bronchiectasia is a pathological irreversible dilation of the bronchi.

### Aetiology

It most often develops secondary to bronchopneumonia or chronic bronchitis. Chronic inflammation, the resistance to airflow due to bronchial obstruction by mucus and an excessive respiratory effort leads to destruction and remodelling of the airways.

### Clinical signs

The clinical signs are those from the underlying disease.

## Diagnosis

Radiography often shows a pathognomic saccular or tubular dilation of minor or major bronchi (Fig. 9). At the end of the dilated bronchus atelectasis or consolidation of the lung lobe can often be noticed. Bronchoscopy is useful in confirming the airway destruction (and associated mucus and pus accumulation) and might be helpful in identifying the primary disease (Fig. 10).

## Treatment

Manage the underlying disease to stop or slow the progression of bronchiectasis but be aware that existing

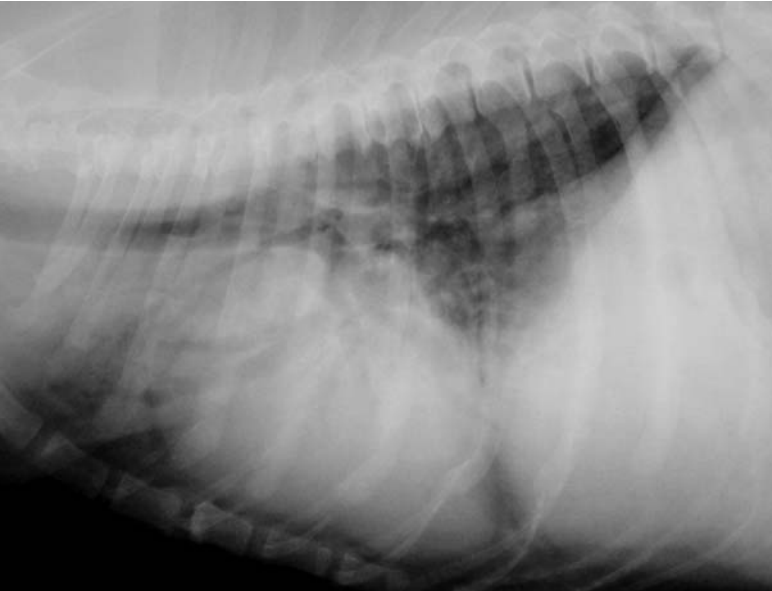


Fig. 9: Radiographic image of a dog with severe bronchiectasia and cor pulmonale.



Fig. 10: Endoscopic image of the airways of a dog with bronchiectasia. Note the dilated bronchus.

lesions are often irreversible. If the bronchiectasia is severe focal pulmonary lobectomy may be considered. Palliative treatment is as for chronic bronchitis.

## FELINE ASTHMA

### Definition

Asthma is defined as a reversible bronchoconstriction that causes wheezes and dyspnoea.

Inflammation of the lower airways often complicates the disease presentation.

### Prevalence

Feline asthma is a very common disease. In the United States Siamese appear to be predisposed but in the UK all breeds are equally affected.

### Pathophysiology

Two different groups have been identified:

1. The first group shows signs of bronchoconstriction (cough and wheeze) but the pathology is associated with very little eosinophilic inflammation. This group of cats certainly has an airway smooth muscle hypersensitivity to allergens.
2. In the second group the bronchoconstriction is associated with severe eosinophilic airway infiltration. Pathophysiologically a Type I and III hypersensitivity has been advanced as the inflammatory pathway, with predominantly mast cells liberating inflammatory mediators like histamine and serotonin.

### Clinical signs

In the acute phase a fulminant, life-threatening, expiratory dyspnoea associated with cyanosis is the most common clinical presentation. In the more chronic cases a chronic non-productive cough associated with intermittent wheezes can be noticed.

### Diagnosis

The history and physical examination are important factors in achieving a diagnosis. Thoracic radiography is rarely pathognomic and the absence of lesions does not exclude the presence of asthma. However the presence of bronchial markings, lung field hyperinflation (flattening of the diaphragm) and collapse of the right middle lung lobe are confirmative indicators. Bronchoscopy often shows hyper-reactive airways with occasionally spasm of the smooth muscles. Bronchoalveolar lavage may show the presence of granulocytes, with often an increased percentage of eosinophils. Culture of the bronchoalveolar lavage may reveal bacteria or *Mycoplasma* spp (ask your lab for culture and transport modalities). A peripheral eosinophilia is present in 10–20% of cases. *Aleurostrongylus* should be excluded as a causative agent of the eosinophilic inflammation (Baermann sedimentation technique for search of larvae in faecal samples collected on three consecutive days).

### Treatment

1. **Acute phase:** Use an oxygen chamber (FiO<sub>2</sub> 40%), rapid-acting corticosteroids (dexamethasone 1 mg/kg IV, IM, SC or methylprednisolone succinate 10 mg/kg IV, IM). Intravenous beta-agonists (terbutaline, albuterol, salbutamol) should be considered but the associated tachycardia can compromise animals with concurrent cardiac

disease. Adrenaline (20 microg/kg IM, SC) is reserved for cases of severe refractory life threatening bronchoconstriction. Cats in an acute crisis should be handled with extreme care because they easily tilt the balance from life to death. Additional diagnostic tests should be postponed until the cat is more stable.

2. **Chronic cases:**

- Hygienic measures: forbidden access to the bedroom (human dandruff is a common allergen), avoid dusty and/or perfumed cat litter, avoid air fresheners, use a special vacuum cleaner with filter
- Treat secondary infections with special attention to possible *Mycoplasma* infections
- Low-dose corticosteroid therapy often controls the clinical signs (prednisolone 1 mg/kg daily initially followed by alternate day therapy of 0.5-0.2 mg/kg). Once the animal is stabilised the author commonly uses fluticasone inhalations via an aerosol chamber/paediatric spacer. Fluticasone, a corticosteroid, is not systemically absorbed and the systemic adverse effects of long-term corticosteroid use are avoided. This technique is amazingly well tolerated by most cats (Fig. 11). Beta-2-agonists (salbutamol) can be used intermittently in the same aerosol chamber but should not replace corticosteroid therapy.



**Fig. 11:** Fluticasone administration via a paediatric spacer in a cat with asthma.

- Cyclosporine (10 µg/kg PO q 12h for the olive-oil based cyclosporine; aim for whole body trough levels between 250-500 nanog/ml) has been advocated in refractory cases. Cyproheptadine (1-4 mg/cat PO q 12h), a serotonin inhibitor, can be used to decrease the amount of steroids needed. Leukotriene-inhibitors have (in the author's experience) very little effect but are worth trying in the milder cases in view of the decrease in the amount of steroids that need to be administered.

© Illustrations Nicole Van Israël

REFERENCE AND FURTHER READING

- M. W. S. MARTIN and B. M. CORCORAN (1997). Cardiorespiratory diseases of the dog and cat. Blackwell Science.  
L. G. KING (2004) Textbook of respiratory disease in dogs and cats. Elsevier USA.

**CPD**

**CONTINUING PROFESSIONAL DEVELOPMENT SPONSORED BY PFIZER ANIMAL HEALTH**

*These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. Answers appear on page 99. In the editorial panel's view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.*

**1. Which of the following statements regarding canine infectious tracheobronchitis is incorrect:**

- It is also called kennel cough.
- The most commonly isolated agents are *Bordetella bronchiseptica* and Parainfluenza III.
- Transmission occurs mainly by inhalation or by direct contact with oronasal secretions.
- Corticosteroids and anti-tussives are strongly contra-indicated in the acute phase of the disease.
- Antibiotic therapy is a must in all cases.

**2. Which of the following statements regarding chronic bronchitis is incorrect:**

- Its chronicity is defined by the persistence of a chronic cough for a period of at least two months in the last year.
- Cigarette smoke might contribute to this multifactorial disease.
- It is more commonly seen in middle-aged and older small breed dogs.
- It is a disease that is only seen in dogs, never in cats.
- Bronchoscopy is the gold standard for the diagnosis of chronic bronchitis.

**3. Which of the following statements regarding bronchiectasia is incorrect:**

- Bronchiectasia is a pathological reversible dilation of the bronchi.
- It most often develops secondary to bronchopneumonia or chronic bronchitis.
- If the bronchiectasia is focal, pulmonary lobectomy should be considered.
- Radiography often shows a pathognomic saccular or tubular dilation of minor or major bronchi.
- The goal of treatment is to stop or slow the progression.

**4. Which of the following statements regarding feline asthma is incorrect:**

- Aleurostrongylus* infestation can mimic the clinical picture of chronic asthma.
- Thoracic radiography is pathognomonic and normal radiographs exclude the presence asthma.
- Feline asthma is a very common disease.
- In the more chronic cases a chronic non-productive cough associated with intermittent wheezes can be noticed.
- A peripheral eosinophilia is only present in a limited number of cases.

**stronghold**  
Spot-on for Fleas and Toxocara