

# Coughing Dogs March 2017

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## INTRODUCTION: OVERVIEW OF THE ISSUE:

A cough is an explosive release of air from the lungs through the mouth. It is generally a protective reflex to expel material from the airways, but inflammation or compression of the airways can also stimulate cough. Acute onset cough is usually infectious and often self limiting but when it comes to chronic cough, definitive diagnosis is needed for successful management to be achieved. Some clients with coughing dogs would like to simply receive medication to stop the cough, however, treatment is most likely to provide long-term relief if an accurate diagnosis of the underlying cause is made so the veterinarian understands what they are dealing with (especially if there is an exacerbation of the cough). Cough is associated with many lower respiratory tract diseases like collapsing airway, chronic bronchitis, broncho-malacia and in people, gastro-oesophageal reflux and post-nasal drip are also common causes of cough.

### INTRODUCTION:

#### THE DIFFERENCE BETWEEN THE COUGH REFLEX AND EXPIRATORY REFLEX (CR VS ER)

Cough depends on the site and type of stimulation. Stimulation of the larynx causes an immediate expiratory effort not preceded by deep inspiration, whereas stimulation of the lower airways produces an obvious inspiratory phase. The upper airways are extremely sensitive to mechanical stimulations, while lower airways are more chemo-sensitive. NB – In the lung periphery, at the bronchiolar and alveolar level, there are probably no cough receptors!

**The cough reflex (CR)** is to remove or expel harmful substances, such as foreign bodies, mucus or debris, from the airways and preserve the normal health of the respiratory tract. The typical features of coughing can be summarised in three phases,

- Initial deep breath
- Rapid and powerful expiratory act against the closed glottis
- Opening of the glottis, closing of the nasopharynx and forceful expiration through the mouth, accompanied by a typical vocalisation caused by the vibration of the vocal cords

**Expiratory reflex (ER).** A defensive mechanism to prevent the entry of material into the airways. This is induced by mechanical or chemical stimulation of the vocal cords or upper trachea and consists of forced expiratory effort against a closed glottis but it is not preceded by an inspiration. ER sounds like a 'huff' sound, often described by the owners as "huffing" or 'clearing the throat'.

The distinction between CR and ER is very important. Cough will draw air into the lungs to augment the force of the subsequent expulsive phase, promoting clearance of mucus and foreign material from trachea and bronchi. The ER will prevent the entry of material into the airways. The two reflexes have different sensory pathways since one starts with an inspiratory act and the other with an expiration. Furthermore, the medicines to inhibit the two reflexes are different since codeine, at the antitussive dose, has minimal effect on the ER.

### INTRODUCTION: KEY CAUSES

A common complaint in small and medium-size dogs is coughing and in middle-age to older dogs a heart murmur is often heard on physical exam. If a middle-age to older dog presents with a murmur and cough, these main differentials for the cough can be considered including:

- Primary respiratory disease (Chronic Tracheo-bronchitis, collapsing trachea).

- Left atrial enlargement vibrating against a malacic left mainstem bronchus
- URT [Upper Respiratory Tract] disease
- Rarely congestive heart failure (pulmonary oedema) or
- Pulmonic hypertension.

All these may occur individually or simultaneously in a patient.

## HISTORY AND PHYSICAL EXAM:

A full history and physical exam are always needed. Determine vaccination history and whether the dog could have been exposed to infectious diseases. Infectious respiratory diseases are more common in young animals while bronchitis, tracheal collapse and acquired heart disease (degenerative mitral valve disease) are more common in middle-age to older dogs.

Because the list of differential diagnoses for cough is long, prioritization through a careful history is essential. Questions to ask include:

- How would you describe the cough (including quality and loudness)?
- How long has the cough been present?
- How frequent does the cough occur, when does it occur, and what is its pattern?
- Who smokes in the house or around the dog?
- Has there been any change in bark quality or intensity?
- Has there been any vomiting or regurgitation?
- Has there been a change in attitude or appetite?
- Do you have a video of the episode?

Upon presentation, many patients with **tracheal collapse** are overweight and may have concurrent heart disease and hepatomegaly, thus physical exam should not focus solely on the respiratory system. In many cases oxygen therapy and tranquilization may be needed before physical exam can occur due to the stressed and anxious nature of these dogs. Palpation of the cervical trachea may elicit paroxysmal coughing and cyanosis. In some cases the lateral borders of the cervical trachea are palpable since the trachea has lost its cylindrical configuration. Auscultation may reveal a heart murmur as well as a "snapping" sound heard at the end of exhalation. **Exposure to smoke** or other irritants can exacerbate any inflammatory airway disease and while obtaining such a history does not lead to a specific diagnosis, significant relief of clinical signs can often be achieved by eliminating the irritant from the dog's environment. A history of **voice change** is suggestive of laryngeal disease, usually laryngeal paralysis or severe reflux onto laryngeal folds. A history of **vomiting or regurgitation** increases suspicion for aspiration pneumonia. Diseases such as infectious tracheobronchitis and canine chronic bronchitis are usually **not** associated with a decrease in attitude or appetite

Trying to characterise the intensity/timing/productive nature of the cough:

A **chronic loud, harsh cough** can be associated with airway inflammation, such as chronic bronchitis, or large airway collapse like tracheal collapse (the classic 'goose honk' sound) and often ends in a gag. These dogs are often in good body condition and have a good appetite and activity level. A **soft cough** is often linked to pneumonia or pulmonary oedema. **Timing of the cough** -if pulling on the collar exacerbates it, then it may be tracheal in origin. Coughs related to cardiac disease are heard more at night or after sleeping. Dogs with heart disease or pneumonia have an acute, often soft cough and may have difficulty breathing. Remember, the timing of coughs may be biased by when the owner is actually with the dog.

NB - The presence of a murmur does not mean heart disease is the cause of the cough.

## Diagnostic tests:

Although the typical tests used in the work up for the coughing dog are radiographs, CT, fluoroscopy, bronchoscopy and tracheal or bronchial washes, other tests are needed in the workup of the coughing dog:

Faecal float, blood smear, FBC, urinalysis and full chemistry panel if the dog is systemically unwell. Blood-gas analysis, pulse oximetry as well as fine needle aspirates of the lungs can be indicated too. In dogs with murmurs, echocardiography, ECG and blood pressure may be needed. Measurement of **NT-proBNP** has been shown to be helpful in the differentiation of airway disease vs. that of CHF in dogs that do Not have heart disease. It showed a high sensitivity and average specificity to differentiate between cardiac vs non cardiac respiratory causes of cough or dyspnea BUT false-positive results exist and currently no rapid test exists. [Most clinicians would be unwilling to wait 24 hours before treating patients with severe clinical signs consistent with CHF]

**Imaging** - for diagnosis of tracheal collapse, bronchitis and an enlarged heart, radiography of the neck and thorax is helpful for diagnosing tracheal collapse. Both inspiratory and expiratory lateral films as well as dorsoventral (DV) views should be taken so as to demonstrate the degree of tracheal collapse in all areas of the trachea (and mainstem bronchi). Evidence of cardiomegaly is often identified and pulmonary disease may also be seen.

**Normal Thoracic Radiographs** - It is common for dogs with chronic cough to have normal thoracic radiographs. The most common cause of this is tracheal collapse and chronic bronchitis, with or without tracheo-bronchomalacia. A bronchial pattern with increased interstitial markings is typically seen on thoracic radiographs of dogs with chronic bronchitis, but changes are often mild and difficult to distinguish from clinically insignificant changes associated with aging. In one study, thoracic radiographs had a sensitivity of 50-65% for the diagnosis of chronic bronchitis. Detection of airway collapse requires insp. and exp. radiographs of the thorax and of the neck during inspiration but as collapse is dynamic, it is difficult to ever rule out the diagnosis without fluoroscopy, and/or bronchoscopy. Additional considerations for dogs with acute cough but normal thoracic radiographs include canine infectious tracheobronchitis, influenza, acute aspiration, and acute foreign body inhalation and pulmonic hypertension. Gastroesophageal reflux is one of the most common causes of normal chest radiographs and a ER [Huff] in people.

*Literature reviews note that many veterinarians, who see a normal chest radiograph will ID the heart as enlarged when it is just fat accumulation around the heart!*

**Tracheoscopy and bronchoscopy** are considered the gold standard tests for evaluating tracheal and bronchial collapse and diagnosis of laryngeal disease requires direct visualization with endoscopy. Before insertion of the bronchoscope, it is important to evaluate laryngeal function. It has been reported that as many as 1/3 of tracheal collapse patients may also have laryngeal paralysis, although in our experience the percentage is much lower. With the dog sedated just enough to visualise the laryngeal folds [we use Propofol], administer IV Dopram and monitor laryngeal movement. After evaluation of laryngeal function, tracheoscopy/bronchoscopy is performed. Tracheal collapse is graded as follows: Grade I - a 25% reduction in lumen diameter; Grade II - a 50% reduction in lumen diameter; Grade III - a 75% reduction in lumen diameter; and Grade IV - no lumen present and cartilages are completely flattened or inverted. If no collapse is seen, a broncho-alveolar lavage should be performed and fluid sent for cytology and culture.

## **Let's Discuss *the most Common Causes of Cough in dogs:***

**1. Tracheal collapse** is a progressive disease caused by cartilage flattening and results in tracheal and/or bronchial collapse. The cause is only partially known and is likely multifactorial but ends in cartilage rings losing their rigidity and typically collapse in dorsoventral fashion resulting in decreased tracheal lumen opening and interferes with

air flow to the lungs. During inspiration the cervical trachea collapses and during exhalation the intra-thoracic trachea and mainstem bronchi collapse.

*Clinical signs* - coughing (often described as a goose-honk sounding cough), abnormal respiratory noises, gagging, "spitting up" phlegm, exercise intolerance, inactivity, cyanosis, syncope, and death. Because there is often coughing involved in the disease process, this causes chronic inflammation which results in more coughing, increased inflammation, more coughing and then decreased function of the mucociliary transport mechanism. Once the "cough cycle" is in place, it can often be difficult to "break".

*Medical management is geared toward the goal of breaking the cough cycle.*

Tracheal collapse occurs most often in toy and miniature breeds of dogs. Breeds that are typically affected with tracheal collapse include Yorkshire terriers, Chihuahuas, Toy Poodles, and Pomeranians [middle-aged and older dogs]. Signs generally worsen with age, and tracheal collapse is considered a progressive disorder. Heat, exercise, tracheal infection, compression of the trachea, excitement, noxious stimuli, eating and drinking may result in, or exacerbate, coughing.

### **TRACHEAL COLLAPSE VS. TRACHEOBRONCHOMALACIA [TBM]**

Tracheal collapse in dogs has been thought of as a congenital cartilage defect of small breeds that results in a honking cough, and that sometimes progresses to signs of upper airway obstruction. Several studies have demonstrated ultrastructural differences in the tracheal cartilage of toy breed dogs with collapsed tracheas, compared to those with normal tracheas, such as areas within the rings that are hypocellular or contain fibrocartilage or fibrous tissues, and decreased chondroitin sulfate and calcium. In many of these dogs, signs do not develop until later in life. One plausible explanation for the delay of signs is that they are initiated by an "acute on chronic" event. In this scenario, an exacerbating problem develops in an affected dog which results in increased respiratory efforts, airway inflammation, and/or cough. Exacerbating problems could include infectious tracheobronchitis, heart enlargement or failure or parasitic disease, with contributions from obesity, exposure to tobacco smoke, reflux or poor oral health. Changes in intrathoracic and airway pressures during increased respiratory efforts or cough contribute to narrowing of the trachea and stretching of the dorsal ligament. With severe collapse. Inflammation also contributes to an ongoing cycle of cough and of collapse. Damage to the tracheal epithelium and changes in mucus composition and secretion impair airway clearance. Previously tolerable irritants and organisms may perpetuate inflammation and cough.

We now know that many dogs with tracheal collapse also have bronchomalacia, with narrowing of principal and/or lobar bronchi and that not all dogs with tracheal collapse are toy breeds and that some dogs have bronchial collapse without tracheal collapse. In human medicine, the broader term tracheobronchomalacia is used. Further complicating the diagnosis and management of dogs with TBM is that this syndrome not only shares similar presenting signs as those of chronic airway inflammation (e.g., idiopathic chronic bronchitis, eosinophilic bronchopneumopathy, bacterial bronchitis, parasitic disease), TBM can be seen concurrently with these diseases. In some dogs, it is likely that the chronic airway inflammation actually causes the TBM through inflammation and cough, perhaps with contributing factors related to the genetic make-up of an individual dog related to cartilage structure or balance of pro- and anti-inflammatory mediators. It is possible that the TBM contributes to airway inflammation, as described above with respect to congenital disease.

## **2. LARYNGEAL PARALYSIS [ER OR HUFF]**

Laryngeal paresis/paralysis is a disease that typically occurs in older, working breed dogs. Common signs include some combination of cough, noisy breathing, exercise and heat intolerance and reduced bark. The primary clinical problem is abnormal, asymmetric or loss of movement of the laryngeal folds and associated arytenoid cartilages. Specialist surgeons generally prefer to lateralize the most affected laryngeal fold and associated arytenoids but don't fully open the airway to prevent FB pneumonia.

### **3. CHRONIC BRONCHITIS [CBD]:**

Chronic bronchial disease (CBD) is a general term used to describe a complex, progressive respiratory syndrome characterized by excessive mucous secretion within airways and thickening (hyperplasia of smooth muscle and epithelium) in the bronchial tree and frequent coughing. Chronic bronchitis is, in most cases, a non-infectious inflammatory condition affecting the lining (mucosa) of the large airways – the trachea and bronchi. Chronic coughing is the hallmark clinical sign in dogs with bronchial disease. However, CBD can induce severe, acute-onset paroxysmal coughing episodes and the patient is presented in respiratory distress. Collapse/syncope are occasionally reported by clients in acute episodes. In our experience, acute respiratory distress associated with CBD is likely to be accompanied by acquired airway (not necessarily tracheal) collapse. Neither age nor gender seems to be predisposing factors to the development of CBD in dogs. While the disease is most common in dogs over 5 years of age, younger dogs can be affected. Among dogs, clinical signs associated with CBD appear to be most prevalent in small and toy breeds, particularly toy poodles, Pekingese, Yorkshire terriers, Chihuahuas, and Pomeranians. At least one author has suggested a hereditary predisposition to CBD in dogs. It is perhaps more appropriate to consider these breeds at risk of developing severe clinical signs of bronchial disease, since CBD clearly occurs in mixed breed and large breed dogs as well as smaller breeds. Obesity and advanced dental/periodontal disease are common, independent findings among small and toy dog breeds with CBD and are regarded as additional complicating (contributing??) factors in the clinical patient.

Detection of abnormal respiratory sounds during thoracic auscultation is not a consistently reliable indicator of CBD. Wheezing on expiration, if present, is considered a hallmark sign of chronic bronchial disease. The ability to elicit coughing by simple manipulation of the cervical trachea is an inconsistent finding in dogs with CBD and an uncommon finding in affected cats. Crackles, when present, can be attributed to the presence of fluid, usually viscous respiratory secretions, in constricted airways.

Dogs with chronic small airway disease are predisposed to bronchial and intrathoracic tracheal collapse. Therefore, during coughing episodes, it is oftentimes possible to auscult airway collapse. Toward the end of expiration, particularly during cough, airway collapse is evident during thoracic auscultation as a loud, discrete thump, referred to as an end-expiratory click or "snap." The sound is generated as the main bronchi and intrathoracic trachea collapse abruptly. Tracheal collapse can culminate in respiratory distress and syncope in dogs during paroxysmal coughing episodes. It is possible for affected dogs to die subsequent to airway obstruction and respiratory arrest during an acute episode.

Chronic bronchitis and tracheal collapse often occur in small and toy breeds that are similar breeds to those which also develop heart murmurs as a result of chronic valve disease. Patients with bronchitis frequently respond to corticosteroid administration while bronchodilators and cough suppressants may be useful as well. Secondary bacterial infections generally are not considered to cause bronchitis; however, patients with severe dental tartar have bacterial shedding and antibiotic therapy with dental cleaning may be of therapeutic benefit. In most cases, the specific cause of chronic bronchitis in dogs is not identified. Most chronic bronchitis is neither infectious, nor contagious. The literature on chronic bronchial disease in humans attributes the underlying cause to 3 factors: age, inhaled particulate material (especially smoke from tobacco), and bacteria. Clients willing to treat a pet with chronic bronchial disease must accept the fact that treatment is aimed at control, not cure.

Dogs with chronic bronchitis generally have a persistent hacking cough. Some people describe it as sounding like a goose honking. However, any trachea-bronchial inflammation/irritation can produce a similar sounding cough. Often, the coughing occurs during the night or when the dog first starts to move around upon waking. It also commonly occurs with excitement or exercise.

In the early, non-obstructive stages of CBD, a generalized interstitial lung pattern is usually present, although bronchial changes predominate. Thickening of bronchial walls, indicated by the "doughnut" appearance of end-on bronchi, and "tram lines," the longitudinal shadows associated with thickened bronchi, can be seen. Bronchial calcification alone, commonly seen as a normal age-related change in old dogs, should not be interpreted as bronchitis.

As CBD progresses, there is a tendency for the small airways, bronchi and, eventually, the intrathoracic trachea to collapse during exhalation, particularly during the expiratory phase of cough. The prevalence and severity of tracheal collapse appears to be most severe in adult, miniature, and toy dog breeds. Although chondrodysplasia and trachealis muscle dysfunction have been implicated in the pathogenesis of tracheal collapse, the functional diameter of the small airways in dogs with chronic bronchitis is also an important cause of bronchial and tracheal collapse, particularly in older dogs. Acquired airway collapse is a significant and complicating factor in dogs (especially small breeds) with CBD. Acquired changes in intra-thoracic airway aerodynamics lead to lower intra-thoracic airway pressure during exhalation (cough) and can lead to rapid, intermittent, but total, collapse of the airway, especially at the level of the carina (tracheal bifurcation). These can be heard during auscultation as the expiratory phase of cough (end-expiratory 'snap') is abruptly interrupted (video of acquired airway collapse will be shown during the presentation).

#### *Bronchoalveolar Lavage and Culture:*

Cytology of specimens collected during BAL may contain only mucous and normal respiratory epithelium in spite of the severity of the patient's clinical signs. Neutrophils, eosinophils, macrophages, lymphocytes, goblet cells, and even bacteria may be seen. However, in our hands, the diagnostic value of cytologic examination of tracheobronchial washings collected during tracheal aspiration or bronchoalveolar lavage is limited.

#### **4. Gastro-Oesophageal reflux:**

Gastro-oesophageal reflux is one of the most common causes of cough in people. The two problems often exacerbate each other, with the efforts of cough causing reflux and the presence of reflux leading to cough. URT stenosis causes severe inspiratory pressures also thought to predispose to reflux. The relationship between reflux and cough in dogs is not well established. Recent studies of brachycephalic dogs showed a strong association with concurrent gastrointestinal signs such as ptyalism, regurgitation, and vomiting. If thorough evaluation has failed to identify respiratory or cardiac disease, treatment with a proton pump inhibitor [PPI] and metoclopramide may be attempted. In people, a beneficial effect may not be seen for weeks, making it difficult to attribute improvement in signs directly to therapy.

#### **5. ASSOCIATION BETWEEN COUGHING AND CHF?**

It has been reported in the veterinary (but not human) literature that CR and ER are the result of pulmonary oedema. This appears to be incorrect and can easily be explained by the fact that these reflex's are not present in the deeper respiratory tract. Patients with pulmonary oedema will present with tachypnoea/dyspnoea. They may occasionally cough if there is a massive accumulation of fluid in the alveolar space severe enough to reach higher airways and stimulate coughing receptors. We should therefore approach the problem from a different angle. Most cardiac patients are geriatric animals (primarily toy to small breeds) who suffer from concomitant airway disease. When these patients are presented to us with a loud heart murmur and coughing, we often don't observe a deep inspiration preceding the cough. It is, in fact, an ER originating from the stimulation of receptors in the upper airways (larynx and trachea). In most cases, these dogs are bright, alert, not dyspnoeic and present with an obvious sinus arrhythmia, which are all signs not consistent with congestive heart failure. All other causes of cough should also be considered, including infectious tracheobronchitis, tracheal collapse, bronchomalacia, gastro-oesophageal reflux, gagging reflex, etc.

For many years, *cardiomegaly*, and, in particular, *left atrial enlargement*, have been indicated as an important cause of coughing in dogs with mitral valve disease. In approximately 300 dogs with cardiac disease there was a very weak association between cardiac size (measured on radiography and echocardiography) and the presence of cough. However, fluoroscopic studies conducted in some older dogs with cardiomegaly have shown dynamic bronchial collapse synchronous with their heart beats and/or diaphragmatic movement. Therefore, the most realistic explanation of this phenomenon is the concomitant presence of bronchomalacia and cardiac enlargement. Young puppies with severe cardiomegaly due to congenital defects (e.g., patent ductus arteriosus) rarely cough, since their airways are healthy and do not collapse under the pressure of the enlarged heart. In summary, dogs with cardiomegaly may cough, but the primary cause of this cough is likely to be bronchomalacia and it should be properly treated as airway disease, rather than heart failure.

#### *So when is it Heart Failure?*

The diagnosis of CHF requires a group of clinical findings. In small-breed dogs, where MMVD is the most common diagnosis, this requires identification of a loud murmur, lack of a sinus arrhythmia, evidence of sleeping tachypnoea (if mild) or tachypnoea on physical examination if more severe, radiographic or echocardiographic evidence of marked LA enlargement (unless acute chord rupture is suspected or identified) and radiographic evidence of pulmonary oedema. If necessary, a properly conducted furosemide trial can help differentiate CHF from other diseases. In our clinic, we use the sleeping respiratory rate as our test for CHF. If a fast asleep dog breaths at a rate of under 30 breaths per minute it is very unlikely to be in CHF!

### **THERAPEUTIC PLAN FOR THE COUGHING DOG:**

*Let's try a diuretic trial and if it improves its cardiac. Furosemide has well recognised anti-tussive properties, acting primarily at peripheral level and is a bronchodilator so many of the causes discussed before will respond, if only for a while!*

#### **THE FIRST PRIORITIES IN COUGHING DOGS:**

Make a definitive diagnosis and then the points below apply to most airways diseases:

- Avoidance of irritants; Cigarette smoke, dust, sprays from deodorants, perfumes, house cleaning products, are all potential irritants. Carpets should be vacuumed frequently. Cleaned cotton sheets should be used to cover the dog's bed.
- Avoidance of excessive, strenuous exercise and collars; Gentle and long walks are more indicated than fast short runs. Light exercise can assist in dislodging bronchial mucus and helps open small airways by promoting increased lung volumes associated with a standing position. A harness should be worn instead of a collar when the dog is walked on a lead.
- Weight control - Fat tends to accumulate in the chest and reduce the lung volume. This can cause compression of the airways and stimulate cough. Weight reduction will improve respiration, exercise capacity and cardiovascular function with dramatic results.
- Relief of airway obstruction; Nebulisation helps bring up mucus from the lungs, bronchi and trachea because it thins the mucus and lubricates the irritated respiratory tract. There are several nebulisers / humidifiers available but the owner can also take the dog into the bathroom, close the door and turn on all of the hot taps to create a steam bath, allowing the dog to breathe in the warm, moist vapours. During, or immediately after, the steam bath, the owner can perform 'coupage'. This loosens some of the deeper secretions and helps them move into the airways, causing coughing that will remove mucus from the chest. Nebulisation should be carried out for 10–15 minutes, at least twice daily.
- Inhalation steroids (if have inflammatory disease); If the dog does not improve following the above non-medical interventions, inhalation steroids can be

considered. These drugs will act directly on the airways and reduce the local inflammation responsible for the excessive mucus production. Since inhaled steroids are not absorbed in the body, there are minimal side effects observed.

These drugs are given to the dog using a dedicated spacer, however, oral steroids are often needed and work more effectively than inhaled meds.

The American Lung Association has a useful website with non-proprietary recommendations for improving indoor air quality ([www.lung.org](http://www.lung.org)).

### **Therapy for Specific Causes of Chronic cough:**

#### **Tracheal collapse:**

Always requires medical management, while severe cases may require "surgical" intervention.

*Medications, as well as environmental changes:* The primary focus of medical management is to break the cough/inflammation cycle: antitussives, steroidal anti-inflammatories, antimicrobials, bronchodilators, anxiolytics, and sedatives. Patients that present in respiratory distress also greatly benefit from oxygen therapy, sedation, steroid administration if indicated, minimal handling, and a cool and restful environment. Often dogs will become refractory to one antitussive and may need to be switched to a different antitussive, or have an additional antitussive added to a regimen. In order to break the cough cycle, it may be necessary to use a short term course of steroids (preferably not longer than 7 to 10 days). Bronchodilation is useful, particularly when mainstem bronchial collapse is present. Antimicrobial therapy may be indicated, and drug selection should preferably be based on culture. Other agents that are occasionally indicated are mucolytics and/or nebulization for patients with excessive mucous production or infection.

*Changes to the environment include:* use of a harness rather than collar; avoidance of the use of a leash at all; exercise restriction; avoiding the outdoors during the heat of the day; maintaining a cool and dry environment; maintaining a stress free environment; avoidance of smoke, pollens, dust, or other allergens; good dental hygiene; weight reduction; treatment of any other respiratory conditions that may be contributing to coughing; and management of cardiac problems. Since exercise, heat, and humidity can all result in increased respiratory effort and coughing, patients should have their exercise restricted and should never be allowed outside during the heat of the day. If animals are able to go on short walks, these walks should be during cool parts of the day. Likewise, extremes of cold should also be avoided as coughing can result. Weight reduction is critical in the management of tracheal collapse and so is good dental hygiene. I've also had patients respond better to medical management after having a dental cleaning. Most of these patients have terrible teeth and dental disease, and improvement of the environment of the mouth results in fewer respiratory infections. Extreme care must be taken with patients who are being anaesthetized for dentals (or scoping procedures) as they can "crash" during recovery from anaesthesia. Additionally the presence of an endotracheal tube results in increased coughing since tracheal mucosal inflammation has occurred.

The "*surgical*" interventions are usually only recommended in patients with Grades III/IV collapse that have become refractory to appropriate medical management, patients having life-threatening episodes of cyanosis and dyspnoea, or those patients that are at risk for euthanasia if coughing isn't improved. The options for "repair" of tracheal collapse include placement of extraluminal tracheal rings or intraluminal stent placement. Since both these procedures can have catastrophic complications, they are considered to be salvage procedures, and not 1st tier options in the management of the disease. The placement of extraluminal tracheal rings, is the preferred technique for the patient with cervical and thoracic inlet collapse and it is possible to stretch the trachea sufficiently to place rings in the thoracic inlet portion of the trachea and caudally to the level of the first or second rib.

Placement of an intraluminal stent under fluoroscopy is a second option for dealing with tracheal collapse in any portion of the trachea except for the carina and mainstem



bronchi. Because complications can be severe following this procedure, it is also considered a salvage procedure. Placing stents in younger or middle age patients should be carefully thought through as the stent might not "outlive" the patient. Other complications that can occur, include granuloma formation, stent migration, laryngospasm if the stent is too close to the larynx, trauma to the tracheal mucosa resulting in hemorrhage or increased mucous production, implant shortening or deformation, and worsening of cough. Much improvement, however, can be seen following stent placement in patients that have severe cyanosis and dyspnea.

### **Chronic Bronchitis:**

Corticosteroids are used to reduce inflammatory damage to the mucosa and reduce excessive production of secretions in dogs with chronic bronchitis, but may or may not be useful in dogs with airway collapse. Prednisone or prednisolone can be used at relatively high doses initially (0.5–1.0 mg/kg BID for 5–7 days) and then tapered to once daily while maintaining control of cough. As the dose is lowered, a longer course of therapy may be needed and the goal is to attain alternate day therapy. Some dogs may be tapered completely off medications; however, exacerbation of disease is treated with an increase in prednisone to the dose that effectively controlled clinical signs. Animals that cannot be controlled on glucocorticoids or those that suffer excessively from side effects associated with steroid use can be treated with inhaled steroids. Oral steroids are generally continued during the first several weeks of inhaled therapy because of a delay in the onset of efficacy for inhaled medications. Oral steroids will also help reduce mucus in the airways and improve penetration of the topical drug.

Administration of drugs via the inhalational route is valuable because it delivers a potent amount of drug at the site of disease. The respiratory tract has a large surface area for topical delivery, and drugs that can be administered locally avoid potentially harmful systemic side effects. Because animals will not inhale on command, a facemask and spacing chamber are needed for delivery of medication from a metered dose inhaler (MDI). The most commonly recommended steroid for use in controlling signs of chronic bronchitis is fluticasone propionate. Initial therapy with BID dosing appears to be used most commonly. Some dogs that fail to respond to anti-inflammatory therapy may benefit from the addition of a bronchodilator to improve expiratory airflow or to support a reduction in the dose of steroid required. Bronchodilators seem to have a role in managing bronchial collapse also, although they do not physically open the large, collapsed airways. Bronchodilators commonly used include the methylxanthines (extended-release theophylline at 10 mg/kg BID) or beta agonists such as terbutaline (0.625–5 mg/dog BID). Theophylline has variable metabolism across individuals, and side effects include vomiting, diarrhoea and agitation. Often, these can be avoided by starting initially at a dose of 5 mg/kg PO BID for a few days, then increasing the dose to 10 mg/kg PO BID if the dog tolerates it. Extended-release properties of theophylline are maintained when the drug is split in half, but the pill cannot be quartered or ground up and remain effective.

When inflammation has been controlled but cough persists, a narcotic cough suppressant may be required. This occurs most often in dogs with concurrent airway collapse.

For dogs with excessive mucus production, saline nebulization can be helpful.

Nebulization can be achieved using an ultrasonic nebulizer, vibrating mesh nebulizer, or compressed air nebulizer. To administer drugs, a facemask is essential; however, for purposes of airway hydration, an aquarium, sealed cage, or animal carrier covered in plastic can be used as a holding chamber to trap the mist.

Finally, management of chronic respiratory disease generally requires weight loss

The role of **long-term antimicrobial therapy** may be underappreciated in managing patients with CBD. Although bacterial **infection** (pneumonia) is seldom recognized as a co-factor in dogs with CBD, many pulmonologists do consider "low-grade" bacterial colonization within the small airways to be a key factor in CBD. Although relatively uncommon, opportunistic infections (pneumonia) involving normal respiratory flora can become life-threatening in dogs with significantly compromised respiratory defense

mechanisms, particularly tracheobronchial collapse and diminished mucociliary transport. The role of *B. bronchiseptica* as a complicating factor in the pathogenesis of CBD must not be underestimated. When *in vitro* culture and sensitivity results are not immediately available, the clinician is justified in prescribing antimicrobial. In the author's experience, antimicrobial therapy plays a critical role in the long-term management of CBD in dogs. Several antimicrobial agents are available for use. Those most commonly prescribed are listed below:

- Doxycycline (5 mg/kg, orally, q12 h).
- Azithromycin (5-10 mg/kg, orally, once daily)
- Enrofloxacin (5 – 10 mg/kg, orally, once daily) caution when using with methyxanthine (aminophylline or theophylline) bronchodilators. ...see below.

### **Congestive Heart Failure Due to MVD:**

We start ALL meds, only once SRR exceeds 30 breaths per minute. Currently we follow the acronym - **FAV**ourite Drug**S**; **F**urosemide **ACE** Inhibitors **V**etmedin, **S**pirinolactone

**Furosemide** administered orally sid-tid, with tid less likely to result in nocturia, up to a maximum total daily dose of 8-12 mg/kg. Be aware of pre-renal effects, and potential hypokalaemia (tends to be countered by ACE inhibitors/aldosterone) and aim to use the lowest effective dose. Use SRR to first determine if heart medications are actually needed and then use SRR to taper to lowest effective dose.

**ACE inhibitors:** any "pril" will do as they all have the same action counteracting the effects of the RAAS (induce vasodilation and reduce pre-load). Usually very safe, we use twice daily from the onset, but some animals can go hypotensive initially so we monitor BP.

**Pimobendan** administered orally as a vasodilator and positive inotrope (inodilator) at 0.3–0.6 mg/kg total daily dose divide bid. Rapidly effective and well tolerated, significantly improving quality of life in some patients. If no benefit seen, can discontinue.

**Spirolactone** is a weak potassium sparing diuretic for when aldosterone levels start to rise weeks to months after starting ACEi (aldosterone escape mechanism) or when furosemide dose is close to the upper end of the range. Prevents aldosterone induced muscle damage

### **Notes on - TRIAL BY LASIX IN CHF:**

Furosemide is often used to determine whether clinical signs are attributable to CHF. Owners will remark that the coughing subsides or improves dramatically with furosemide therapy. However, furosemide is also a potent bronchodilator and has activity at the larynx as an antitussive. Thus, elimination of a cough with lasix does NOT prove the CHF hypothesis. Instead, a proper furosemide trial consists of demonstrating a reduction in the sleeping or resting respiratory rate.

Remember – Monitor SRR at all times because congestive heart failure will be progressive in MVD cases requiring increased dosages and additional drugs. Co-amilozide (Moduret/Moduretic; hydrochlorothiazide & amiloride) is a diuretic combination that can be added in the latter stages.

Survival from onset of CHF is typically less than 12 months.

### **Medications used to manage cough:**

<b>Drug</b>	<b>Dose</b>	<b>Indications</b>	<b>Adverse effects</b>
Prednisone	1 mg/kg PO oid till no cough; then every 2 <sup>nd</sup> day for 1 week; then maintain a dose that controls the cough or pulse as needed.	Anti-inflammatory	Increased appetite, thirst, urination
Theophylline ER	10 mg/kg PO q12h	Bronchodilator	Tachycardia, anxiety

Hydrocodone	0.25 mg/kg PO q6–12h	Cough suppressant	Sedation
Furosemide	1–4 mg/kg PO, SC, IM, IV q6–12h	Diuretic	Dehydration, electrolyte abnormalities
ACEi [any 'pril]	0.5 mg/kg bd	If on a diuretic	Hypotension
Spirinolactone	1-2mg/kg bd	If on triple therapy	GI and possible anorexia
Codeine	0.5 to 2mg/kg 1-4x/day	To control cough	Constipation, sedation
Salbutamol [Venteese]	0.05mg/kg 1-4 x/day	Bronchidilator	Tachycardia, anxiety

## **PROGNOSIS:**

### **Collapsing Trachea:**

Prognosis variable. Typically, the younger the patient at the time of diagnosis, and the more severe the collapse, often tend to have the poorest prognosis for a normal lifespan. Many patients can be medically managed, and even some patients with grades III and IV can be medically managed. However, tracheal collapse is generally considered to be a progressive disease, and many animals may become refractory to medical management. The prognosis for the patient that has undergone extraluminal prosthetic ring placement is good assuming recurrent laryngeal nerve function is intact postoperatively. Many of these patients may be able to discontinue their medications after inflammation associated with surgery has subsided, although many dogs will still require medications, especially when intrathoracic trachea and/or mainstem bronchi are collapsed. Some patients ultimately end up needing a stent placed intrathoracically at some point in the future. The prognosis for the patient with tracheal stent placement is quite variable. In severely affected patients, you can "look like a hero" following stent placement. On the other hand, in the patient in which coughing is the primary clinical sign, you may be questioned as to whether you "actually did anything". They almost always still do some coughing post stenting.

### **CHRONIC BRONCHITIS:**

Owners should be aware that the prognosis for abolishing cough in dogs with bronchitis or airway collapse is guarded and many dogs require lifelong medication. A reasonable goal is to reduce clinical signs by at least 50%. Worsening of disease can lead to bronchiectasis or cor pulmonale. Visualization of bronchitic nodules or airway collapse during bronchoscopy likely indicates the irreversibility of the process.

### **SUMMARY**

Even when a heart murmur is present, if a dog presents for a chronic cough, has not had breathing difficulty and has a good appetite and activity level, then respiratory disease or mainstem bronchial compression should be high on the differential list. Thoracic radiographs will help differentiate respiratory disease, mainstem bronchial compression and heart failure. If a dog presents with a cough, respiratory difficulty, lethargy, anorexia and has a heart murmur present on physical examination, then heart failure should be high on the differential list and thoracic radiographs can confirm the diagnosis. Don't be tricked into assuming the presence of a heart murmur indicates the cough is related to heart failure. Get a detailed history and physical examination, obtain thoracic

radiographs and any other diagnostic tests indicated including a sleeping respiratory rate [SRR] to help determine the source of the cough.

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