Proceeding of the LAVC
Latin American Veterinary Conference
Oct. 24-26, 2011 – Lima, Peru

Next LAVC Conference:

Apr. 24-26, 2012 – Lima, Peru

Reprinted in the IVIS website with the permission of the LAVC

http://www.ivis.org/
Respiratory Therapy

Helio Autran de Morais, DVM, PhD, ACVIM (Internal Medicine & Cardiology)

Tracheal Collapse

Tracheal collapse is a dynamic reduction in the luminal diameter of the large conducting airways with respiration. It affects mostly older dogs of small breeds, particularly Yorkshire terriers and Chihuahua. Dogs with tracheal collapse have a dry honking cough and also may have ronchus. Clinical signs are worsened by exercise, heat, humidity, obesity, and infections. Cyanosis and syncope may be seen in severely affected dogs. Inspiratory wheezes may be present in dogs with cervical collapse, whereas expiratory wheezes will occur in patients with intrathoracic collapse. Crackles will occur if there is concomitant small airway disease. Respiratory sinus arrhythmia secondary to increased vagal tone is usually present, whereas a loud second heart sound and splitting of the second heart sound may occur in patients with pulmonary hypertension.

Collapse of the trachea can be identified in radiographs in approximately 60% of the cases. Inspiratory films will reveal collapse in the cervical trachea, whereas expiratory films show the collapsed trachea inside the chest. Fluoroscopy is helpful to identify dynamic collapse of the trachea, increasing diagnostic sensitivity.

Bronchoscopy is used to grade the severity of the collapse and to identify presence of small airway disease. Samples for cytology can be obtained during bronchoscopy or doing a wash using oral intubation (not transtracheally). Cytology is unremarkable in uncomplicated collapse, but may show inflammation and bacteria if there is a secondary infection.

Therapy is directed at weight reduction, decrease environmental stress and treatment of complications, particularly infections. Prednisone can be used short-term (5-7 days) to decrease inflammation. Bronchodilators will not help unless there is also small airway disease. Antibiotics are used if there is a positive culture, signs of infection in cytology or a sudden worsening of the clinical signs. Only antibiotics that penetrate the hematobronchial barrier should be used. Cough suppressants (e.g., butorphanol: 0.5 – 1.0 mg/kg PO q4-8h; or hydrocodone: 0.22 mg/kg PO q4-8h) help decrease the irritation-cough-irritation cycle. In acute crisis, butorphanol (0.05 mg/kg SC) will help by sedating as well as suppressing cough. Acepromazine (0.025 mg/kg SC q8-12h) may enhance sedative effects. Surgery or placement of an intratracheal stent should be considered in patients that do not respond to medical therapy.

Infectious Tracheobronchitis (Kennel Cough)

Infectious tracheobronchitis is a contagious respiratory disease of dogs caused primarily by Bordetella bronchiseptica associated or not with other bacteria and viral agents. It is more common in young, debilitated animals housed in crowded conditions. Dogs with uncomplicated infections have a dry hacking cough in an otherwise normal dog. Presence of pneumonia, fever or anorexia is associated with complicated infections. Chest radiographs are usually unremarkable in the uncomplicated disease and are useful to rule-out other conditions causing loud cough. Radiographic evidences of pneumonia can be found in dogs with complicated disease. Transtracheal wash should be performed in dogs with complicated disease to obtain material for cytology, culture (including Mycoplasma cultures) and sensitivity.
Uncomplicated disease is usually resolved within 14 days without therapy. Patients should, however, be isolated to decrease spreading of the disease. Patients with systemic signs, evidence of pneumonia or that have been sick for longer than 14 days should receive antibiotics. Antibiotics should be selected based on culture and sensitivity and the ability to achieve therapeutic concentration in the bronchial tree. Good empirical choices are tetracyclines and quinolones. Support therapy with nebulization and airway humidification, rest, proper nutrition and hydration should also be introduced.

**Left main stem bronchial compression**

In dogs, the left main stem bronchus is dorsal to the left atrium, making it prone to be compressed during left-sided heart disease associated with increase in left atrial size. This is more common in small breed dogs with mitral endocardiosis, but may also occur in some dogs with dilated cardiomyopathy. Therapy is directed at decreasing left atrial size. In patients without heart failure, ACE inhibitors are used initially. If there is inadequate response, hydralazine (0.5 – 3.0 mg/kg q12h PO) can be added to the protocol.

**Chronic bronchitis in dogs**

Chronic bronchitis is a chronic inflammation of unknown origin of the bronchial tree that may involve lobar bronchi and the smaller airways. Regardless of cause, chronic bronchial inflammation results in increased tracheobronchial secretions, cough and architectural changes in the bronchial tree. Airway epithelium suffers hypertrophy, metaplasia, and ulceration. Goblet cells and submucosal glands undergo hypertrophy and increase production of mucus, whereas bronchial mucosa and submucosa may become edematous and infiltrated with inflammatory cells.

Typically, chronic bronchitis affects adult dogs (8 years or older) of small breeds. There is no sex predilection. The hallmark of chronic bronchitis is chronic cough (at least two months in duration). The cough often terminates with a gag suggesting that it is productive, although it may be dry in some dogs. Cough may worse with exercise. Tachypnea, shortness of breath, and cyanosis may also be exacerbated or precipitated by exercise. In advanced cases, dogs may display a marked expiratory effort with a prolonged expiratory phase of breathing. Auscultatory findings are variable and non-specific. Wheezing due to mucus plugging and expiratory airway collapse and crackles are present in a large number of dogs with chronic bronchitis. The abnormal sounds are best heard after maneuvers that force the patient to take deep inspirations (e.g. occlusion of nostrils for a few seconds).

*Thoracic radiographs* are usually abnormal, but they may be normal in early cases. Bronchial thickening are the most common radiographic abnormalities. Pulmonary hyperinflation, bronchiectasis, tracheobronchial collapse, right middle lung lobe atelectasis, bronchopneumonia, and right heart enlargement due to cor pulmonale also can be found in radiographs of dogs with chronic bronchitis. *Bronchopulmonary cytology* may reveal increased mucus, hyperplastic epithelial cells, and a variable mixture of inflammatory cells. Neutrophils are usually the predominant inflammatory cell with smaller numbers of lymphocytes and eosinophils. Culture may be sterile or yield growth of a number of different bacteria. Airways of dogs with chronic bronchitis may look erythematous with a roughened or granular appearance during bronchoscopic
examination. Accumulation of a thick mucus, mucus plugs, and tracheobronchial collapse also can be observed.

Treatment of chronic bronchitis is guided by the clinical signs, cytologic evaluation and culture, the extent of radiographic changes, and response to therapy. Therapy may alleviate clinical signs. Control rather than cure is achieved, because many structural changes in the airways are irreversible. Environmental stress (e.g. tobacco smoke) and factors that may exacerbate clinical signs (e.g. excitement) should be identified and minimized. Weight reduction is advised to obese patients because diaphragmatic function is impaired, small airways close earlier than normal and ventilation may be impeded in these patients. Inhalation of humidified air via a vaporizer or nebulizer may help liquefy secretions. Animals should be lightly exercised or be coupaged and encouraged to cough after these procedures. Infections should be treated as guided by the tracheobronchial cultures. Bronchodilators may help in relieving reversible airway obstruction. They also decrease mucosal edema and have anti-inflammatory effects by preventing mediator release from inflammatory cells. In addition, theophylline (10 – 20 mg/kg q12h) increases strength of respiratory muscles and decreases the work associated with breathing. Beta2-adrenoceptor agonists (e.g. terbutaline; small dogs: 0.625 – 1.25 mg q12h; medium-sized dogs: 1.25 – 2.5 mg q12h; large dogs: 2.5 – 5 mg q12h) are effective bronchodilators because they act as functional antagonists or airway constriction regardless of the cause. They also stimulate secretion of airway mucus resulting in a less viscous secretion and enhanced ciliary activity. Cough suppressants may be used in selected cases. Dogs with nonbacterial bronchitis that have a cough that is distressful to the owner or that is followed by exhaustion or collapse are potential candidates for antitussive therapy. One should bear in mind however, that suppression of the cough reflex will be detrimental for the clearance of airway secretion in chronic bronchitis. Antinflammatory therapy with steroids is most effective in dogs with nonbacterial bronchitis, particularly those with eosinophilic bronchitis. Steroids can be used systemically or by inhalation.

**Chronic Bronchitis/Asthma in Cats**

Chronic bronchitis and asthma are frequently encountered clinical conditions in cats. Several subclassification of feline bronchial disease have been attempted. Differences, many times subtle, in presentation and laboratory findings have allowed investigators to suggest specific etiologies for these cases. It is important to remember, however, that regardless of the cause, cats with bronchial disease have similar clinical signs, and that the treatments used are also similar. Cats that have periods of normalcy between crises likely have asthma.

Bronchial diseases are more common in middle aged (2-8 year old) female cats, but cats of all ages can be affected. The Siamese breed may be overrepresented. The most common clinical sign is cough. The coughing episodes may vary from mild to severe and be paroxysmal in nature. Cats also may have dyspnea, wheezing, occasional sneezing, and vomiting. Severely affected cats may show open mouth breathing, a respiratory effort disproportionate to the animal’s level of exercise, and cyanosis. Some owners report seasonal exacerbation of the clinical signs. The peak seasonal incidence, however, is variable. Duration of clinical signs may vary from less than 24 hours to several years. Some cats may have cough and dyspnea for months or years with asymptomatic periods between crises. On physical examination, most affected cats have abnormal lung sounds including crackles and wheezes. Some cats may also manifest expiratory dyspnea and tachypnea. Mild hyperthermia is present in about 25% of the cases.
Chest radiographs are essential in evaluating cats with bronchial disease and usually show a bronchial pattern. A mild to moderate interstitial pattern may be seen concurrently. Lung lobe collapse, overinflation of the lungs, patchy alveolar infiltrates, and emphysematous changes may also be observed. Parasitic infection of the lungs (e.g. Aelurostrongylus abstrusus, Capillaria aerophilia, and Paragonimus kellicotti), and heartworm disease should be ruled-out as potential causes for the clinical signs in affected cats. Bronchial cytology typically shows increased mucus and inflammatory cells, usually eosinophils or neutrophils. Positive bacteria cultures are encountered in approximately 25% of cats. Most bacteria found in cats with chronic bronchial disease can also be isolated from the respiratory tree of normal cats. The main exception is Mycoplasma sp. found more commonly in cats with chronic airway disease.

The goals of therapy are to control secretions, improve alveolar ventilation, and normalize excessive reflexes. Mild affected cats may be treated conservatively with weight reduction, avoidance of trigger events and external stimuli for secretion (e.g. dusts, tobacco smoke). Fenbendazole (50 mg/kg/5 days) should be used routinely to treat lungworms. Albuterol can be used as an inhaler (1-2 puffs as needed). Cats with mild signs that occur daily can be managed with inhalers: albuterol and fluticasone (110 µg q12h) or oral bronchodilators (Theo-dur tablets or Slo-bid capsules: 25 mg/cat q24h at night; or terbutaline: 0.625 mg/cat q12h) and short-acting steroids. Cats with moderate signs that occur daily and are “never” normal require at least a short course of prednisone for 10 – 15 days in addition to the inhalers.

Cats presenting an acute exacerbation of a long-standing bronchial disease should be managed in an emergency basis. After a physical examination with as little restrain as possible to rule out other possible reasons for the clinical signs, the cat should be stabilized with supplemental oxygen and bronchodilators. All other diagnostic steps should be delayed until the patient is more stable. Bronchodilators (terbutaline and immediate-release aminophylline) can usually be given orally because they reach peak plasma concentration in approximately 1 hour. Severely affected cats may require terbutaline subcutaneously at approximately 0.01 mg/kg. If no improvement is noted, the dose should be repeated once. Alternatively, albuterol can be used as an inhaler every 30 minutes for up to 4 hours. Intravenous corticosteroids are often recommended. In acute situations, however, steroids are more likely to be effective due to a mild bronchodilatory effect. Significant suppression of inflammation by the steroids takes time to occur.

Pulmonary Edema

Pulmonary edema is the accumulation of fluids in the interstitium and alveoli of the lung. There are two main basic mechanisms for edema development: increased hydrostatic pressure in the lung capillaries (“high-pressure edema”) and increase vascular permeability (“low-pressure edema). This classification helps understand the basic pathophysiological differences between the two types of pulmonary edema, but has limitations. Disruption of some or all layers of the alveolar-capillary unit occurs during elevated capillary hydrostatic pressures, a phenomenon termed “pulmonary capillary stress failure”. Pulmonary capillary stress failure represents a process that blurs the distinction between high-pressure and low-pressure pulmonary edema, as the disruption of the alveolar-capillary membrane by high hydrostatic pressures may render it more permeable to fluid and proteins. The resulting edema fluid has a higher concentration of protein than would be
expected in conventional high-pressure pulmonary edema. These observations may explain some features seen in high-altitude pulmonary edema and neurogenic pulmonary edema.

High-pressure edema is usually secondary to left-sided congestive heart failure and many times called “cardiogenic pulmonary edema”, whereas low-pressure pulmonary edema are termed “noncardiogenic”. Fluid in noncardiogenic pulmonary has a higher concentration of proteins and the edema occurs with normal capillary wedge pressure. The increased vascular permeability can occur with a wide variety of pulmonary and systemic disorders including vasculitis, acute respiratory distress syndrome, electric shock, neurogenic edema and uremic pneumonitis. Patients with pulmonary edema are usually presented with expiratory or mixed dyspnea with normal to increased lung sounds and presence of abnormal sounds (e.g.; crackles). Radiographs are helpful in the diagnosis and differentiation between cardiogenic and noncardiogenic edema based on the distribution of the edema.

The initial goals of therapy in cardiogenic pulmonary edema include increasing arterial $P_2$, reducing oxygen demand, establishing a diuresis, and unloading the ventricles while supporting blood pressure, tissue perfusion and renal function. Supplemental oxygen therapy and sedation are used as needed to reduce distress or air hunger. Pulmonary edema sufficient to cause respiratory failure and respiratory muscle fatigue is an indication for artificial ventilation. Diuresis is established with furosemide at an initial IV bolus of 2–5 mg/kg that can be followed by serial IV or IM boluses of 1-4 mg/kg every 6 to 8 hours or more frequently when necessitated by insufficient clinical response. The use of constant rate infusion (CRI) of furosemide also may be used to treat dogs and cats with life-threatening pulmonary edema. In healthy dogs and in human patients with CHF, furosemide CRI increases urine output and minimizes electrolyte disturbances when compared to repeated bolus injections. After the initial IV bolus of furosemide, the furosemide dosage required for the next 24 hours is estimated, and then infused by syringe pump. Supplemental boluses also can be given if required during the CRI. Nitroglycerin ointment can be used to decrease preload, whereas nitroprusside can be used to decrease afterload in dogs with florid pulmonary edema. Two percent nitroglycerin ointment (¼ to 1 inch of the 2% ointment, topically q12h) acts primarily as a systemic venodilator, and this treatment is well-tolerated by both dogs and cats. Although some question exists about the efficacy of topically-administered nitroglycerin, the anticipated venodilation should work in concert with furosemide to lower venous and capillary hydrostatic pressures. The need for arteriolar dilators in the hospital setting depends on the cause and severity of CHF. Although vasodilator therapy has the potential to induce systemic hypotension, such treatment generally is safe in dogs when baseline ABP exceeds 95 mm Hg. For dogs with severe CHF, sodium nitroprusside (1–5 μg/kg/min IV by constant rate infusion), enalapril (0.5 mg/kg PO q12h), and hydralazine (1–2 mg/kg PO q12h) are effective vasodilators in the hospital setting. Each drug can increase stroke volume and reduce pulmonary edema. Afterload reduction is particularly useful in the treatment of severe mitral regurgitation arising from canine endocardiosis or when left ventricular dysfunction is evident, as in dogs with dilated cardiomyopathy.

Cardiac output, arterial blood pressure, and tissue perfusion are supported when necessary by providing inotropic support. In dogs or cats with severe systemic hypotension (ABP <80 mm Hg), inotropic support with dobutamine (2.5–10 μg/kg/min) or dopamine (2–10 μg/kg/min) is indicated. Catecholamines most often are administered to dogs in CHF caused by dilated cardiomyopathy. Occasionally, this approach is used in patients with severe mitral regurgitation or pulmonary embolism. Cats with any form of cardiomyopathy may develop cardiogenic shock characterized by bradycardia, hypothermia, and hypotension. Treatment with dobutamine can be life-saving in
affected cats. Infusions should be titrated to a systolic ABP of 90 to 120 mm Hg and can be combined with slow external warming in an oxygen incubator. When treatment with catecholamines is impractical, oral administration of the calcium sensitizer pimobendan should be considered once this drug is available for general use.

There is no specific pharmacological treatment for noncardiogenic pulmonary edema. Diuretics are often ineffective and, despite widespread use, there is no evidence that corticosteroids are helpful. Support therapies include: controlling the causative factor, ventilatory support and maintaining the patient well hydrated.