Proceedings of the 34th World Small Animal Veterinary Congress
WSAVA 2009

São Paulo, Brazil - 2009

Next WSAVA Congress:

Reprinted in IVIS with the permission of the Congress Organizers
The respiratory system undergoes various anatomical, physiological and immunological changes with age. Muscular strength of the respiratory apparatus consistently declines and the chest wall stiffens leading to increased work of breathing. The lung parenchyma loses its supporting structure causing dilation of air spaces, and the lung elastic recoil decreases further compromising ventilation. The alveolar dead space increases with age, affecting arterial oxygen without impairing the carbon dioxide elimination. The respiratory centers become less sensitive decreasing the cough reflex, sensation of dyspnea and diminishing ventilatory response to hypoxia and hypercapnia. Decreased sensitivity to hypoxia or hypercapnia results in a diminished ventilatory response in cases of heart failure, infection or airway obstruction. The most commonly encountered respiratory diseases in old dogs are tracheal collapse, chronic bronchitis, pneumonia and pulmonary fibrosis.

**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 toy breeds, particularly Yorkshire terriers and Chihuahuas. 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. 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. 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.

**Chronic bronchitis**

Chronic bronchitis is a chronic inflammation of unknown origin of the bronchial tree that may involve lobar bronchi and the smaller airways. Regardless of the 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) of small breeds. There is no sex predilection. The hallmark of chronic bronchitis is chronic cough that often terminates with a gag. Tachypnea, shortness of breath, and cyanosis may be present, and be exacerbated or precipitated by exercise. Auscultatory findings are variable and non-specific. Expiratory wheezes and crackles are present in a large number of dogs.

*Thoracic radiographs* are usually abnormal, but they may be normal in early cases. Bronchial thickening are the most common radiographic change. Bronchiecstasis, 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 erythematos with a roughened or granular appearance during bronchoscopic examination. Accumulation of a thick mucus, mucus plugs, and tracheobronchial collapse also can be observed. Treatment is guided by the clinical signs, cytologic evaluation and culture, the extent of radiographic changes, and response to therapy. Therapy alleviate clinical signs, but control rather than cure is achieved. 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. Dogs should be lightly exercised or be coupaged and encouraged to cough after these procedures. Infections should be treated as guided by the cultures. Bronchodilators (e.g., theophylline: 10-20 mg/kg q12h) may help in relieving reversible airway obstruction. They also decrease mucosal edema and have anti-inflammatory effects by preventing mediator release from inflammatory cells. 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) also are effective bronchodilators. They stimulate secretion of airway mucus resulting in a less viscous secretion and enhanced ciliary activity. Cough suppressants may be used in cautiously used in dogs with nonbacterial bronchitis that have a cough that is distressful to the owner or is followed by exhaustion or collapse. 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.

Bacterial Pneumonia
Bacterial pneumonia is the inflammation developed in response to the presence of virulent bacteria in the pulmonary parenchyma. It is usually secondary to aspiration or systemic infection (hematogenous pneumonia). Clinical signs can varie from mild signs related to infection until severe depression and evidence of systemic inflammatory response syndrome. Some dogs may have purulent nasal discharge, dyspnea and fever. Crackles may be heard particularly in the cranio-ventral lung fields. Cough, if present, is usually soft. Bordetella bronchiseptica and Streptococcus zooepidemicus are primary pathogens leading to pneumonia in dogs. In most cases, however, the bacteria are opportunistic invaders. Gram-negative aerobes: Escherichia coli, Pasteurella multocida, Klebsiella pneumoniae, and Pseudomonas aeruginosa are most commonly isolated from dogs with pneumonia. Staphylococcus spp, Streptococcus spp and Mycoplasma spp. can also be isolated occasionally, whereas anaerobes are usually seen in patients with pulmonary abscess.

In patients with aspiration pneumonia, radiographic examination usually reveal an alveolar pattern in the cranio-ventral lung fields or in the region of the right middle lung lobe. Cytology obtained by transtracheal wash may show a neutrophilic inflammation with degenerate neutrophils. Bacteria can be found in less than 50% of samples. Anaerobic and aerobic cultures are thus mandatory to identify the organisms and to determine their antibiotic susceptibility. Therapy of the stable patient (still eats, temperature < 40°C, no left shift) consists of antibiotics at home for 10 days, nutritional support and rest. Reasonable empiric antibiotic choices include amoxicillin + clavulanic acid, cephalaxin or trimethropin + sulfonamides. Antibiotic choice should be reevaluated based on culture and sensitivity or if there is no improvement in 72 hours. A stable patient that gets worse should be hospitalized and rehydrated. A new antibiotic should be selected based on culture and sensitivity. The unstable patient should be hospitalized, kept hydrated and receive nutritional support and IV antibiotic therapy. Dogs with complicated pneumonia usually have an aerobic gram negative, especially E. coli. The first choice of antibiotics is cefazolin: 15-25 mg/kg q6-8h or ampicillin: 20-40 mg/kg q6-8h + enrofloxacin: 2,5 mg/kg q12h.
**Pulmonary Fibrosis**

Pulmonary fibrosis is a poorly characterized disease of dogs. The etiology is not known although multifocal alveolitis similar to the one associated with pulmonary fibrosis in human beings has been suggested. This is reinforced by the fact that in animal models of pulmonary fibrosis, alveolitis usually precedes development of fibrosis. Dogs with pulmonary fibrosis are usually old, and small terrier breeds may be predisposed. There is usually a long and progressive history of shortness of breath. Cough is absent unless the patient also has chronic bronchitis or secondary bacterial pneumonia. Bilateral, diffuse, very-loud crackles are the classic physical findings. Exercise may cause tachypnea, open-mouth breathing and cyanosis. Radiographs may reveal mild cardiomegaly due to cor pulmonale or decreased lung expansion. A mild diffuse interstitial pattern is usually evident with rounding of the peripheral lung edges and flattening of the diaphragm. Arterial blood gases usually reveal hypoxemia and hypocapnia. Bronchoalveolar lavage may help in the diagnosis of pulmonary fibrosis. In patients with no evidence of bronchitis, alveolar fluid (obtained by bronchoalveolar lavage) containing more than 20% of neutrophils suggest the presence of alveolitis and potentially fibrosis. There is no effective treatment for pulmonary fibrosis in dogs. Obesity should be corrected and exposure to airway irritants should be minimized. Sildenafil (1mg/kg q8h) causes arterial vasodilation in the pulmonary vasculature improving blood flow. Patients with pulmonary fibrosis with moderate to severe pulmonary hypertension may greatly benefit from sildenafil. Those patients may show a great improvement in exercise tolerance increases and a decrease in associated clinical signs.