Respiratory Emergencies in Dogs
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Respiratory distress in dogs is not only frightening for owners. Veterinarians faced with having to make quick decisions fear making the wrong choice or not making the right choice fast enough. There is a fairly short list of differential diagnoses for causes of dyspnea in dogs. Some of these causes can look very similar but treatments may vary widely. Some treatments for one condition may not help (or even harm) another cause of dyspnea. With an understanding of the common causes, how they look alike, and how they can be differentiated, the medical team can quickly rank possible causes and treat accordingly.

Initial observation and diagnostics
Try to determine the nature of the problem first with observation. A rapid shallow respiratory pattern suggests restrictive disease while a slow deep inspiratory pattern is seen with airway obstruction. With the restrictive pattern, auscultation can help differentiate pleural space disease (pneumothorax, hydrothorax) from parenchymal diseases (pneumonia, pulmonary edema). Signalment and history can help determine a cause of upper airway obstruction (playing with small toys, brachycephalic airway diseases, laryngeal paralysis). Once the patient has been sedated and calmed, treated for shock and hyperthermia, definitive diagnostics can be performed. Some of the important emergency diagnostics will be reviewed.

Imaging
Animals presenting with upper and lower respiratory signs should have a thoracic radiograph. Bronchial patterns develop as the peribronchiolar tissues become inflamed and the airways thicken. Interstitial patterns develop with thickening of the fibrous structures of the lung. Alveolar patterns characterized by “Air bronchograms” are caused by fluid accumulation in the alveoli. Thoracic and cervical radiographs can be used to diagnose collapsing trachea, tracheal or laryngeal foreign bodies, and tracheal or laryngeal masses. Taking inspiratory and expiratory views of the trachea or through the use of fluoroscopy it is possible to assess dynamic changes in airway diameter.

Thoracocentesis
When pleural fluid or air accumulation is suspected, a thoracocentesis can treat the impaired tidal volume while making the diagnosis. The character of the fluid or presence of air will be valuable in understanding the cause of the problem. Thoracocentesis should be approached as a quick “yes-no” question. With one person manipulating the needle and another holding the syringe and tubing. The syringe is aspirated as soon as the needle is through the chest wall and the team quickly determines if air or fluid is causing the problems. If no air or fluid is easily withdrawn, the needle is withdrawn to avoid iatrogenic lung injury. Ultrasound is a useful modality to determine the presence and location of fluid if available.

Airway cytology
Transtracheal and transoral airway wash are useful techniques for the diagnosis of diseases of the respiratory system and easily performed in most dogs in about 15 minutes. It should be performed following assessment of the thoracic radiographs and is indicated for all coughing dogs and cats with interstitial, bronchial, or alveolar lung patterns that are not suspected to be due to cardiogenic disease or coagulopathy. The goal of the wash is to collect fluids from the trachea, bronchi and lower airways for cytology, culture, and antibiotic susceptibility. Animals rarely develop subcutaneous emphysema and pneumomediastinum following airway wash and so should be observed in the hospital for several hours following completion of the procedure.

Laryngeal paralysis
Can be either congenital or acquired and is a common cause of emergency visits in large breed dogs. The paralysis may be either unilateral or bilateral. Acquired laryngeal paralysis is more common with many proposed etiologies. The recurrent laryngeal nerve innervates the arytenoid processes of the larynx. One of the longest nerves in the body, it is susceptible to a variety of degenerative processes. Damage to the nerve anywhere along its course by trauma, surgery, neoplasia, polyneuropathy or even hypothyroidism can lead to a loss of innervation of the intrinsic laryngeal muscles. Animals with laryngeal paralysis will present with varying degrees of exercise intolerance, stridor, voice change, inspiratory effort, cyanosis and hyperthermia. They will have a pronounced inspiratory stridor with a loud, deep (obstructive) breathing pattern. Often an obvious inspiratory wheeze will be heard loudest over the larynx. Diagnosis is by direct examination under a light plane of anesthesia. A low dose of propofol is administered to allow the mouth to be held open while visualizing the glottis. If the animal becomes apneic with the sedative a 1 mg dose of doxapram HCL IV can initiate a large breath. Normally the arytenoid cartilage adducts on inspiration. With laryngeal paralysis the arytenoids may actually be drawn together during inspiration causing inflammation and edema. Laryngeal paralysis may be unilateral or bilateral. Increased airway pressures can lead to everted laryngeal sacculles further compromising the laryngeal lumen.
Because panting is such an important method of controlling body temperature, subclinical laryngeal paralysis may only become evident on hot days or following strenuous exercise. The body temperature can quickly climb to dangerous levels, necessitating treatment for heat stroke. Dyspnea from upper airway obstruction can cause the animals to become anxious and more dyspneic. A vicious cycle begins as the more distressed they become, the harder they try to breathe. Handling these animals can be difficult and often the best treatment is sedation. Acepromazine is a predictable sedative. A dose of 0.02 – 0.04 mg/kg not to exceed 0.25 mg will break the cycle of distress. Acepromazine should be given cautiously in dehydrated or shock patients as it may cause a drop in blood pressure. Patients with prolonged hyperthermia should be hospitalized and observed for complications. The kidneys, GI tract, liver and nervous tissue can all be damaged by excessive heat. Disseminated intravascular coagulation is another common complication. Once the patient is stable and signs of heat stroke have resolved definitive treatment for laryngeal paralysis can proceed.

**Pulmonary edema**

Non-cardiogenic pulmonary edema occurs occasionally in dogs and cats secondary to electric cord bites, sepsis, following near drowning or choking, snake bites, uremia, smoke inhalation, upper airway obstruction, and the adult respiratory distress syndrome (ARDS). Dogs that chew on electric cords often present with acute onset of dyspnea and oral burns, which may or may not be associated with dysphagia or ptyalism. The syndrome occurs most commonly in the young. Pulmonary edema develops rapidly, generally within hours. Common physical examination abnormalities include oral burns, dyspnea, and pulmonary crackles. Thoracic radiographs show mixed interstitial and alveolar patterns that are most prominent in the dorsal portions of the caudal lung lobes. The pathogenesis of edema is thought to be increased pulmonary capillary hydrostatic pressure and increased alveolar-capillary permeability. Increased pulmonary capillary hydrostatic pressure is likely due to a centrally mediated burst of sympathetic activity, which causes constriction of resistance and capacitance vessels leading to a shift of blood from the splanchnic viscera into the circulation. This ultimately results in overcirculation of the pulmonary vasculature. Increased peripheral vascular resistance increases pulmonary capillary hydrostatic pressure and pulmonary venous pressures increase as the left ventricle pumps against increased outflow resistance. Treatment includes administration of low dose narcotics, diuretics, and oxygen (mask, nasal insulation or oxygen cage). Morphine and other pure narcotic agonists at lower doses can have a good clinical effect. At low doses it sedates dyspneic animals while drawing excess fluid from the lungs via splanchnic vasodilatation.

The clinical signs and physical examination abnormalities associated with near drowning, smoke inhalation, and snakebite are similar to those with electric cord bites with the exception of oral burns. Historical findings confirm near drowning and smoke inhalation. Puncture wounds and a swollen face or extremities may be found on animals with snakebite. Administration of bronchodilators may also aid in the treatment of some cases. Smoke inhalation causes dyspnea by inducing carbon monoxide poisoning and damage to respiratory tissues by heat and noxious gasses. Laryngeal spasm, loss of ciliary function, decreased surfactant activity, bronchospasm, increased alveolar-capillary permeability, impaired phagocytosis, and sloughing of airway mucosa frequently occur. Bronchial patterns occur first with interstitial and alveolar edema developing later if edema develops. Treatment is similar to electric cord bite and near drowning.

Pulmonary edema occasionally develops secondary to upper airway obstruction in dogs. Laryngeal and pharyngeal diseases are most common. Inspiratory and expiratory stridor, dyspnea, crackles, and cyanosis are common physical examination abnormalities. Mixed interstitial and alveolar lung infiltrates are detected in the perihilar and dorsocaudal lung fields. Treatment can include administration of oxygen, diuretics and glucocorticoids, as well as tracheostomy if needed. Edema is primarily related to decreased intrathoracic pressure resulting in decreased interstitial hydrostatic pressure and hypoxia resulting in increased alveolar capillary permeability.

**Pneumonia**

Bacterial pneumonia in dogs is rarely a primary disease. Occasionally, *Bordetella bronchiseptica* or *Mycoplasma* spp. can induce pneumonia due to their adverse affects on mucociliary function. Most cases of bacterial bronchopneumonia are secondary to immunosuppressive diseases or previous inflammatory insults including viral infection, aspiration, and irritant inhalation. Owners should be carefully questioned concerning potential exposure to other animals and clinical signs associated with immunosuppressive diseases or aspiration.

Most animals with bacterial pneumonia will be clinically ill. Common complaints include depression, anorexia, dyspnea, productive, moist cough with a terminal retch, and exercise intolerance. Some animals with pneumonia will present only with cough. Physical examination findings commonly include fever, crackles and wheezes, and muffled lung sounds in cases with consolidated or abscessed lung lobes. Many dogs will have increased tracheal sounds, a tracheal cough, and pharyngeal inflammation due to transport of inflammatory cells up the mucociliary apparatus to the mouth. Thoracic radiographs usually reveal a mixed alveolar, bronchial, and interstitial pattern. Aspiration pneumonia generally has radiographic lesions that are most pronounced in the right middle lung lobe. Animals with opacity of the right middle lung lobe should be evaluated for esophageal and gastrointestinal disease or respiratory stridor. Esophageal diseases leading to regurgitation and aspiration may be evident on evaluation of thoracic radiographs. Laryngeal paralysis, which is characterized by inspiratory stridor, can predispose dogs to aspiration.
One of the most important treatments of bacterial pneumonia is hydration. The mucociliary apparatus function best in a well-hydrated animal and is essential for the clearance of infection. Affected animals should receive parenteral fluid therapy until able to maintain hydration orally. Airway hydration can be accentuated by nebulization or by placing the animal in a closed bathroom while running hot water through the shower. Common bacterial isolates include *Bordetella bronchiseptica*, *Pasteurella multocida*, *Klebsiella* spp., *Streptococcus* spp., and *Escherichia coli*.

**Canine bronchitis**

Causes a cough occurring on most days usually in the absence of other active disease. With long standing inflammation histologic changes include fibrosis, epithelial hyperplasia, glandular hypertrophy, and inflammatory infiltrates. Canine chronic bronchitis is likely a consequence of a chronic inflammatory process initiated by infection, allergy, or inhaled irritants or toxins. Uncontrolled, inflammation leads to mucosal damage. Excessive mucus secretion, and airway obstruction impair normal clearance mechanisms. Tracheobronchial weakness can further contribute to the ongoing cycle of cough and inflammation. The inflamed airways are also prone to dynamic collapse. These patients typically present with an expiratory wheeze and increased expiratory effort. They can be distinguished from the cardiac patient with pulmonary edema who will have primarily an inspiratory dyspnea with pulmonary crackles. Treatment is aimed at reversing inflammation while opening the airways with a bronchodilator.

**Pleural space disease**

Diseases of the pleural space cause a decreased tidal volume and a restrictive breathing pattern. Characterized by the rapid, shallow respirations and dull lung sounds, fluid and air in the pleural space can be diagnosed and treated by rapid thoracocentesis following the procedure above. Once removed, fluid can be examined to determine the likely cause. A negative thoracocentesis may be due to fibrous adhesions and small pockets of fluid. Diaphragmatic hernias may also cause significant pleural restriction but yield a negative tap. These patients should be given supplemental oxygen while ultrasound is performed or radiographs taken. Large volumes of air or fluid, continuous production of air, or supplicative inflammation are indications for tube thoracostomy. Pneumothorax and some inflammatory conditions may require continuous suction. Disposable suction devices are available to hook onto surgical suction units. These “3-bottle” devices allow easy regulation of suction (20 cm H₂O desirable), a water trap to prevent air from being drawn into the chest should suction become interrupted, and a collection chamber to quantitate fluid production.