THE PET IS BREATHING HARD, THE FIRST HOUR

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Key pointers

- 1. Exclude pleura space disease by focused ultrasound, radiographs or thoracocentesis
- 2. Exclude upper airway obstruction by breed profiling, auscultation and history
- 3. Cold cats have heart failure; old cats rarely get polyps or new onset asthma
- 4. Dobies lie about heart failure
- 5. Oxygen is a good idea, but you need an action step
- 6. One dose of anything (other than euthanasia solution) never hurt anyone.

Respiratory distress represents a true emergency and is represents a major cause of animals presenting to the emergency service. Prompt treatment is warranted to attempt to identify the underlying cause, to limit the sensation of difficulty breathing and to provide diagnostic and therapeutic information for clients of affected animals. Some forms of respiratory distress are easily treatable, while others are much more limited and may only be able to be palliated. For the clinician in practice, success in patient management revolves around developing a knowledge base of potential causes of respiratory distress as well as "pattern recognition" of common emergent problems affecting dogs and cats. The goals of this talk is to illustrate various methods for classifying respiratory distress, to highlight common emergency conditions resulting in respiratory distress and to provide guidelines for emergent management.

While an understanding of potential causes of respiratory distress are mandatory, the first steps in the clinical evaluation of a patient presenting with respiratory distress are to provide a supplemental source of oxygen and to obtain a brief history from the client. All emergency facilities should have a form of supplemental oxygen available. Supplemental oxygen may be provided via a variety of options, including flow-by, face mask, nasal oxygen, e-collar and cellophane wrap ("oxygen hood"), oxygen cage and intubation with intermittent positive pressure ventilation (IPPV). Intubation and IPPV is the best option for providing high levels of supplemental oxygen, removing respiratory fatigue, and eliminating patient fear and anxiety although due to demands; staffing, financial and emotion should not be undertaken lightly unless it is felt the problem can be promptly ameliorated, such as an airway foreign body or dog with laryngeal paralysis and heatstroke from inadequate cooling. High flow oxygen therapy is gaining popularity as an additional method of providing supplemental oxygen.

History, as always, is often very helpful. In some cases, the precipitating cause of the respiratory distress is straight-forward, such as with traumatic injuries, while in other cases, the onset may be more insidious. Animals with pre-existing medical conditions, such as cardiac disease, neoplasia or megaesophagus may also be predisposed to the development of respiratory distress. Owners should be questioned as to past medical conditions, history of routine veterinary care, including heartworm prophylaxis, and finally, the progression of the signs of respiratory distress should be described. Specifically, distress may be acute in onset, or more progressive. In cats in particular, the development of respiratory distress may be preceded by anorexia, lethargy or abnormal behavior.

Respiratory distress may be further characterized by the location of the lesion or the underlying pathophysiological condition. Often, localization of the lesion can help to guide the clinician to the most likely cause. Specifically, respiratory distress may be localized to upper airway, lower airway, parenchymal or pleural space disease. Common pathophysiological causes for respiratory distress include anatomical abnormalities, airway collapse, pulmonary edema of cardiac and non-cardiac causes, infection, inflammatory and trauma. For the emergency clinician, the most appropriate first step is to localize the lesion and then to review specific differentials based upon signalment, history and other physical examination findings.

Upper airway diseases may be appreciated by loud stridorous breathing, with an increased inspiratory time. Many dogs are hyperthermic on initial presentation due to decreased ability to cool. The upper airways represent the primary source of resistance to airflow. Upper airway obstructions can be either dynamic or fixed. Dynamic obstructions are characterized by the paradoxical movement of tissues into the lumen of airway during inspiratory inspiration. Common dynamic obstructions include laryngeal paralysis and tracheal collapse, while fixed obstruction include extraluminal obstructions such as neoplasia or cellulitis and intraluminal obstructions such as laryngeal tumors or nasopharyngeal polyps. Both dynamic and fixed severe upper airway obstructions will also result in the development of airway mucosal edema and possibly everted laryngeal saccules due to irritation from the increased air flow rates through a narrow lumen.

Emergently, upper airway obstruction should be suspected in a dog with loud, noisy breathing. Therapy for a suspected dynamic obstruction should include sedation and supplemental oxygen. Sedation is beneficial in the dynamic obstruction in reducing the anxiety associated with inspiration because with increased inspiratory efforts there is a resulting paradoxical decline in airway diameter. Low doses of acepromazine (0.03-0.05 mg.kg intravenously) alone or in combination with butorphanol (0.1 mg/kg intravenously) are often effective. Hyperthermia should be treated by active cooling with room temperature (not cold!) intravenous fluids, and by placing the dog in a cool area. Due to airway swelling and edema, a single dose of short acting anti-inflammatory glucocorticoid is advisable. If the dog has not improved within 15-30 minutes or if distress is worsening, more aggressive therapy is warranted. The dog should be heavily sedated or anesthetized and intubated. The emergency clinician should be competent to evaluate airway function and anatomy and to perform a tracheostomy if needed. Additionally, as many upper airway conditions require management and/or surgical intervention, the emergency clinician should be fluent in discussions with clients concerning long-term outcomes. The most common causes of upper airway obstruction may vary depending on location but in our practice include laryngeal paralysis, tracheal collapse, brachycephalic airway syndrome and severe cellulitis. While a complete discussion on the management of these conditions is beyond the scope of this chapter; however, despite their similarities, some differences due to the underlying disease do exist concerning optimal management of affected patients.

Laryngeal paralysis primarily affected older large breed dogs, particularly retrievers. Usually, the clinical signs of noisy breathing have been present for some length of time prior to a crisis. Crises often occur during the first hot and humid days of the spring or summer. Dogs will commonly respond well to sedation. Dogs that do not rapidly improve should be sedated and have laryngeal function evaluated and be intubated. If palliative surgery is not readily available, dogs may have a tracheostomy performed or may be keep briefly sedated/intubated until normothermia and eupnea ensue. In our practice, we will commonly maintain dogs on a propofol CRI in 0.9% saline (20 ml of propofol in 1 liter of NaCl) titrated to effect for 30-60 minutes. If after this time period a dog can not be extubated, due to the higher risk of aspiration pneumonia, it is better to perform a tracheostomy than keep a patient intubated.

Conversely, in dogs with severe brachycephalic airway syndrome or tracheal collapse, it may be impossible to remove a tracheostomy tube after placement. This means that it avoidance of a tracheostomy is preferable in these dogs as compared with dogs with laryngeal paralysis. If a tracheostomy is unavoidable; plans should be made for surgical correction of the obstruction as soon as feasible. Brachycephalic dogs may also develop laryngeal collapse, which is not amenable to laryngoplasty, and may ultimately necessitate a permanent tracheostomy.

In cats, upper airway obstructions are less common, but may be caused by nasopharyngeal polyps or infiltrative laryngeal diseases (neoplasia or granulomatous). Occasionally cats with severe pleural effusion will have the appearance of severe inspiratory distress. If sedation for an oral examination for a cat with a suspected upper airway obstruction is planned, then supplies should be collected ahead of time for an emergency tracheostomy. The laryngeal lumen of affected cats can be only a millimeter or two in diameter and may require an urgent tracheostomy. If a biopsy of a laryngeal mass is performed in a cat, a tracheostomy is almost always required due to subsequent airway swelling. Cats may also have a permanent tracheostomy placed, although it less well tolerated than it is in dogs.

Respiratory distress may also result from lower airway disease, parenchymal lung disease or pleural space disease. Thoracic radiographs are essential to help clarify the degree of pulmonary or pleural space involvement. However, it is important to recall that radiography can be stressful, particularly in cats that are experiencing respiratory distress. Lower airway diseases include chronic bronchitis and feline asthma. Chronic bronchitis rarely presents emergently, although flare-ups in some patients do occur. Chronic bronchitis is defined as the presence of a cough on most days for the preceding two months, without evidence of other underlying cause. Canine chronic bronchitis commonly affects small breed dogs. On auscultation, a mitral murmur is commonly heard. Conversely, feline lower airway disease may present as an emergency. In cats, airway disease appears to represent a continuum with some cats having primarily inflammatory airway disease with cough and excessive mucus production, while other cats are the more prototypical "asthmatics" with reversible bronchoconstriction. Cats with severe bronchoconstriction will often present emergently. It is important to distinguish the airway disease from congestive heart failure. Cats with airway disease typically are normothermic and have had a history of cough. Both heart failure and airway disease may be accompanied by crackles.

Parenchymal lung disease is often responsible for respiratory distress. Common causes of parenchymal lung disease include pulmonary edema (cardiogenic and non-cardiogenic), pulmonary contusion, pneumonia and neoplasia. Heart failure in cats is usually appreciated by hypothermia combined with an increased respiratory rate and effort. Jugular venous distension may be present. A gallop or a murmur may be auscultated. Due to the hypothermia, cats with congestive heart failure (CHF) will commonly have slow heart rates (130-140 bpm). Heart disease in dogs is usually either chronic valvular disease or dilated cardiomyopathy. Animals with a history of trauma or possible trauma and that are presenting with respiratory distress can be assumed to have some component of pulmonary contusion (and/or pneumothorax). Therapy for respiratory distress associated with

pulmonary infiltrates include supplemental oxygen and therapeutic agents directed towards the presumptive underlying cause. The distribution of the pulmonary infiltrates may be very useful to help determine the underlying problem. In dogs, cardiogenic pulmonary infiltrates will most often surround the perihilar region, while in cats the distribution of pulmonary edema may vary. Bacterial pneumonias will typically have a cranioventral distribution. Neoplasia will usually result in a nodular pattern, although metastatic disease may appear variable.

Animals with suspected cardiogenic pulmonary edema should be treated with initially with diuretics (furosemide 1-4 mg/kg iv or im, q 1-6 hours), cage rest and supplemental oxygen. If a rapid improvement is not observed, additional therapy with vasodilators (nitroprusside titrated to effect) is warranted. In practice, despite published guidelines, measurement of blood pressure during infusion of nitroprusside is usually not performed in order to limit patient stress, loss of supplemental oxygen (by opening cage door) and technical difficulties in getting accurate numbers. Specifically, it may be challenging or impossible to place an arterial line for direct blood pressure determinations in an animal with congestive heart failure, oscillometric techniques are commonly inaccurate with small dogs or cats or during ectopy and Doppler techniques are time-consuming and require a patient with respiratory distress to be restrained. Dobutamine, as a continuous rate infusion (CRI) is very useful in dogs with dilated cardiomyopathies. Intravenous fluids should not be administered to a patient with heart failure, although they should be permitted *ad lib* access to water. Hemodynamically significant arrhythmias should be treated. Patients should be transitioned to long-term medications after stabilization. While an echocardiogram is not considered an emergent procedure, it is useful for if an emergency clinician has access to an ultrasound machine for that individual to gain basic knowledge of echocardiography, including assessment of left atrial size, contractility and presence or absence of pericardial effusion.

Non-cardiogenic pulmonary edema may occur for a variety of reasons. In the emergency room; upper airway obstruction, seizures and electric cord injury are common triggers for the development of non-cardiogenic edema. Non-cardiogenic pulmonary edema is typically high-protein and results due to permeability shifts in the capillaries rather than hydrostatic forces as with cardiogenic edema. There is not specific therapy that has been proven beneficial for hastening recovery from non-cardiogenic edema. Treatment recommendations include cage rest and supplemental oxygen. More specific therapy with either diuretics or colloids has been advocated by various clinicians although no consensus statement exists. The vast majority of dogs with non-cardiogenic pulmonary edema will rapidly improve within 24-48 hours.

Pulmonary contusions are common after traumatic injury, particularly in dogs. Animals severe affected with pulmonary contusion will be short of breath rapidly after the injury, although radiographically infiltrates will often worsen over the first 12-24 hours. Dogs with contusions commonly have small to moderate volume pneumothoraces as well. Contusions will generally heal rapidly. One study in dogs was unable to support the use of either prophylactic antibiotics or steroids. Diuretics are also not indicated for animals with pulmonary contusion.

Dogs with pneumonia may present to the emergency room with respiratory distress. Bacterial pneumonia is very rare in cats. Pneumonia can be sub-divided in to community-acquired such as severe bronchopneumonia (infectious kennel cough complex) or aspiration pneumonia in a dog with laryngeal paralysis or megaesophagus or hospital-acquired for a dog that develops pneumonia while hospitalized for treatment of another condition. Therapy for pneumonia includes broad-spectrum antibiotics, physiotherapy and intravenous fluids. Ideally, a bacterial culture is performed prior to the institution of antibiotics.

Animals will infrequently present on emergency with dyspnea due to metastatic disease, although cough and lethargy are common. Spontaneous pneumothorax may occasionally develop in a patient with pulmonary neoplasia. Treatment of suspected neoplastic disease is directed at supportive care. Common metastatic tumors include hemangiosarcoma and mammary gland adenocarcinoma. Occasionally, further imaging is indicated to attempt to localize a primary tumor; however, generally this is futile. Pulmonary lymphoma may respond well to therapy. It is also important to exclude a recent travel history in dogs with a nodular pulmonary pattern as the systemic mycoses can mimic metastatic disease. Other less common causes of pulmonary infiltrates include eosinophilic pneumonitis and pulmonary fibrosis.

Pleural space disease will commonly result in marked respiratory distress. Common causes are pleural effusion, pneumothorax and diaphragmatic hernia. Pleural space disease may often be suspected clinically based upon a restrictive (short and shallow) breathing pattern. Thoracic radiographs are very useful in documenting the extent of the pleural space disease. Diaphragmatic hernia should be corrected as soon as the patient is considered stable enough for surgery. In traumatic injuries, concurrent pulmonary contusion may markedly worsen gas exchange, thus anesthesia and surgery may be postponed until clinical improvement. However, if significant herniation exists, including the presence of the stomach intra-thoracically, surgical repair become urgent. Anesthesia may still be safely performed with pulmonary contusions, although in addition to the positive pressure ventilation required due to the loss of diaphragmatic integrity, a small amount of positive end-expiratory pressure (PEEP) may be beneficial to help recruit collapsed alveoli. In chronic hernias, re-expansion pulmonary edema may result in severe respiratory failure. Thus correction of chronic hernias should be undertaken with care and gradual re-inflation of the lung.

Pneumothorax may be classified as either traumatic or spontaneous. Traumatic is the most common. For animals with a known history of injury, needle thoracocentesis may be performed as guided clinically. Due to the high density of tissue thromboplastin, the previously healthy injured lung will heal rapidly, thus chest tubes are not commonly required in the trauma patient. A good guideline is that three or more thoracocenteses ("Three strike rule") within 24 hours is sufficient justification for placement of the chest tube in the traumatic pneumothorax. It is exceedingly rare to have a patient with trauma require a thoracotomy for resection of the traumatized lung. Conversely, most cases of spontaneous pneumothorax require surgical resection of the affected lobe. Spontaneous pneumothorax is defined as pneumothorax occurring without trauma. Common causes include bulla/blebs and neoplasia (primary or metastatic). Additionally, cats with lower airway disease may occasionally develop spontaneous pneumothoraces. (White) For affected dogs, rapid surgical exploration and resection has been associated with decreased morbidity and expense. (Puerto)

Home oxygen therapy

Home oxygen, either prescribed via a medical supply house or via a veterinary specific company (eg Pawprint oxygen) can be very useful for pets with chronic conditions where transport with oxygen is needed or for short term supplementation at home. Quality of life for pets is clearly important, and oxygen therapy, if used wisely, can improve quality and quantity of life, and with veterinary input save costs if oxygen therapy alone is keeping a pet hospitalized.

Spectrum of Care options

If an animal has severe difficulty breathing, almost all pets should be admitted or euthanized. For mild or moderate disease, it is appropriate to consider outpatient care, particularly if suspected target is treatable. Pro-NT BNP testing can direct therapy towards heart or not heart. \$4 medications can help save costs, and ideal rechecks may be curtailed

References

- 1. Waddell LS, Brady CA, Drobatz KJ. Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986-1999) J Am Vet Med Assoc. 2002 Sep 15;221(6):819-24.
- 2. Puerto DA, Brockman DJ, Lindquist C, Drobatz K. Surgical and nonsurgical management of and selected risk factors for spontaneous pneumothorax in dogs: 64 cases (1986-1999) J Am Vet Med Assoc. 2002 Jun 1;220(11):1670-4.
- **3.** Drobatz KJ, Walker LM, Hendricks JC. Smoke exposure in cats: 22 cases (1986-1997). J Am Vet Med Assoc. 1999 Nov 1;215(9):1312-6.
- 4. Drobatz KJ, Walker LM, Hendricks JC. Smoke exposure in dogs: 27 cases (1988-1997). J Am Vet Med Assoc. 1999 Nov 1;215(9):1306-11.
- 5. Drobatz KJ, Saunders HM, Pugh CR, Hendricks JC. Noncardiogenic pulmonary edema in dogs and cats: 26 cases (1987-1993)J Am Vet Med Assoc. 1995 Jun 1;206(11):1732-6.
- 6. White HL, Rozanski EA, Tidwell AS, Chan DL, Rush JE. Spontaneous pneumothorax in two cats with small airway disease. J Am Vet Med Assoc. 2003 Jun 1;222(11):1573-5, 1547.
- 7. Clercx C, Peeters D, Snaps F, Hansen P, McEntee K, Detilleux J, Henroteaux M, Day MJ. Eosinophilic bronchopneumopathy in dogs. J Vet Intern Med. 2000 May-Jun;14(3):282-91.
- Corcoran BM, Cobb M, Martin MW, Dukes-McEwan J, French A, Fuentes VL, Boswood A, Rhind S. Chronic pulmonary disease in West Highland white terriers. Vet Rec. 1999 May 29;144(22):611-6.
- **9.** Powell L, Rozanski EA, Tidwell A, Rush JE. A retrospective analysis of pulmonary contusion secondary to motor vehicle accidents in 143 dogs: 1994-1997. J Vet Emerg Crit Care 9:127-136, 1999.

- **10.** Stampley AR, Waldron DR. Reexpansion pulmonary edema after surgery to repair a diaphragmatic hernia in a cat. J Am Vet Med Assoc. 1993 Dec 15;203(12):1699-701.
- **11.** Schmiedt CW, Tobias KM, Stevenson MA. Traumatic diaphragmatic hernia in cats: 34 cases (1991-2001). J Am Vet Med Assoc. 2003 May 1;222(9):1237-40.