

## ACUTE RESPIRATORY DISTRESS IN THE DOG AND CAT

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The signs of respiratory distress include things such as an increased respiratory rate, increased respiratory effort, orthopnea, and possibly noise associated with respirations. When a dog or cat presents in respiratory distress, the #1 priority is to provide oxygen therapy. It is important not to create too much stress while providing the oxygen. For dog, oxygen cages and flow by oxygen therapy tend to be the easiest ways to provide oxygen since they are the least stressful (as long as the dog fits into the available oxygen cage). Be aware that if a dog is too large relative to the size of the oxygen cage, it may be too hot and/or humid in the cage for the dog to be comfortable. Due to the increased temperature and humidity associated with panting, it is rare that a non-temperature and humidity controlled oxygen cage (such as simply using a cage door on an existing cage) is appropriate for a dog. In contrast for cats oxygen cages and flow by oxygen therapy tend to be the easiest and least stressful ways to provide oxygen. Be aware that if a cat is too large relative to the size of the oxygen cage, it may be too hot and/or humid in the cage for the cat to be comfortable. Alternative options to provide oxygen to cats would be to place a cat carrier in a plastic bag filled with oxygen, to use an Elizabethan collar covered with plastic wrap and filled with oxygen, or to use nasal oxygen cannulas.

### Upper airway obstruction

After the animal has been given oxygen to stabilize it, the next order of business is to determine if it is an upper airway or lower airway disorder. Upper airway disorders are usually accompanied by loud breathing (stertor or stridor), inspiratory dyspnea, visible anxiety in the patient, and sometimes cyanosis. Upper airway disorders occur more commonly in dogs than cats. The most common types in dogs are brachycephalic airway syndrome, laryngeal paralysis, and collapsing trachea. Brachycephalic airway syndrome occurs in breeds such as pugs and bulldogs, laryngeal paralysis occurs in Labrador retrievers and other large breed dogs such as German shepherds and golden retrievers, and collapsing trachea occurs in small breed dogs such as Yorkshire terriers. When upper airway obstructions occur in cats, they are commonly related to masses and polyps in the oropharyngeal area. Masses occur secondary to infectious disease like viral infections, can be neoplastic, or can be nasopharyngeal polyps in kittens or younger cats. While uncommon, cats can also have laryngeal paralysis.

Acute treatment of an upper airway obstruction can be achieved by following the mnemonic NOSE: Noisy breathing means **O**xxygen, **S**edation, and **E**ntubation if needed. As mentioned before, *oxygen* should be administered in the least stressful way that still allows you to administer drugs and other treatments to the patient. Options include flow-by oxygen, an oxygen cage, intranasal oxygen, enclosing a carrier in a garbage bag, or even using an E-collar with the front covered by plastic wrap. *Sedation* can be achieved with drugs such as butorphanol (0.2-0.5 mg/kg) IV or IM and/or acepromazine 0.1-0.2 mg/kg IV or IM. Acepromazine can take 20-30 minutes to reach full effect so refrain from re-dosing it prematurely. Typically administering more than a total of 0.5 mg/kg acepromazine will not achieve further sedation. In some cases

dexmedetomidine can be used for sedation at a dose of 2-10 µg/kg IM or IV in dogs or 10-20 µg/kg IV or IM in cats. Dexmedetomidine can also be used as a constant rate infusion (1-3 µg/kg/hr) after bolus administration if necessary. Remember that animals with upper respiratory signs can easily overheat (leading to further distress and panting) or can become hypoglycemic if they have a prolonged bout of anxiety/panting/distress (see below for further details).

If you have waited 10-15 minutes with oxygen and sedation and there is no improvement in the animal's condition, the dog may need to be *entubated* (*yes I know it is spelled intubate!*). Usually administering propofol IV to effect for intubation is the easiest technique. Keep the animal relaxed and comfortable using propofol as needed for at least 30-60 minutes prior to allowing the animal to wake up. In many cases, the dog will continue to keep the tube in place despite being 'awake' since they realize how much easier it is to breathe. We typically encourage this for as long as the animal tolerates the tube; there is no rush to remove the tube. Administer flow by oxygen at the end of the tube or 100% oxygen from an anesthesia circuit if necessary (which it is often not the case). If the dog immediately becomes dyspneic once the tube is removed (even with concurrent acepromazine, butorphanol, or other sedatives) it may need to be re-intubated and kept heavily sedated/anesthetized with propofol and/or other drugs until definitive diagnostics and treatment have been completed.

Always check to ensure that an upper airway obstruction animal is not hyperthermic. Panting is the best way to remove heat and an obstruction will limit the ability to remove heat. Obesity, stress, and the oxygen cage itself can lead to hyperthermia which makes the animal continue to pant and be tachypneic. In addition, dogs can develop hypoglycemia during stressful respiratory distress bouts if they use up their stored glycogen responding to the stress. Seizures can result from hypoglycemia or lead to low blood glucose. Make it part of your treatment/stabilization plan to evaluate upper respiratory animals for hypoglycemia as soon as they can safely tolerate a blood draw. (We commonly draw blood while quickly placing a catheter to facilitate further drug administration.)

### **Lower airway disease in dogs**

#### **“Loud auscultation”**

Lower airway disease in dogs is commonly marked by an increased respiratory rate ± a cough. The animals can be open or closed mouth breathing and may or may not show a markedly increased respiratory effort. Unlike upper respiratory cases where any thoracic auscultation conveys referred upper airway noise making it nearly impossible to evaluate the lungs, auscultation of the thorax is important in lower respiratory dogs. A “loud” auscultation with crackles or wheezes can occur most commonly with pulmonary edema or pneumonia. Pulmonary edema can be due to heart failure (ie. cardiogenic pulmonary edema) or non-cardiogenic pulmonary edema. Dogs of any age can develop pulmonary edema that is cardiogenic in origin. Dogs can go into heart failure from congenital causes at a young age or develop heart failure secondary to chronic mitral valve disease or progressive diseases such as dilated cardiomyopathy at an older age. They may or may not have an arrhythmia and certain breeds such as Boxers, Dobermans, and small breed dogs suffer most commonly from heart

failure. In contrast, younger dogs are more likely to develop non-cardiogenic pulmonary edema than older dogs since it occurs after seizure activity, drowning, choking and electrocution; all these things tend to affect younger dogs more so than older dogs. In contrast, dogs with pneumonia can be of any age and may have a fever and/or an elevated white blood cell count on a complete blood count. Since bacterial and fungal pneumonia are common in dogs, knowing travel history and any relevant history of mingling with other dogs in a boarding-type situation can be important. Also, doing a complete physical exam looking for things like draining skin lesions can be important in pneumonia cases as is obtaining a full history including vomiting/regurgitation information.

Pulmonary edema occurs when fluid fills the interstitial space between the alveolus and capillary, limiting diffusion of oxygen from the alveolus to the blood. In non-cardiogenic pulmonary edema, the fluid buildup occurs secondary to increased permeability of the capillary wall leading to fluid extravasation into the lung tissue. Cardiogenic pulmonary edema occurs because increased fluid volume in the capillaries leads to increased capillary hydrostatic pressure which pushes fluid out of the capillary into the interstitial tissue and possibly the alveolus.

Differentiation of cardiogenic vs non-cardiogenic pulmonary edema occurs largely from radiographs (in addition to signalment and history). Cardiogenic pulmonary edema has an interstitial to alveolar pattern that in dogs is worst in the perihilar region and caudodorsal lung fields. In contrast, non-cardiogenic pulmonary edema tends to be a bilaterally symmetric alveolar pattern that is strongest in the caudal lung fields.

*Cardiogenic pulmonary edema* is treated by FON: furosemide, oxygen and narcotics for sedation (if needed). Furosemide is given at a dose of 2 mg/kg IM or IV repeated every 20-30 minutes up to 6-8 mg/kg total. Intramuscular furosemide works at approximately the same speed as IV furosemide and can be much less stressful to administer in dyspneic animals. Oxygen can be given by any of the techniques mentioned previously but it is important to provide oxygen with as little stress as possible. Morphine (0.2-0.5 mg/kg IM or IV) or butorphanol (0.2 mg/kg IV or IM) is also commonly given to reduce anxiety and discomfort in heart failure patients.

*Non-cardiogenic pulmonary edema* is treated by oxygen and time (typically 24-48 hrs+) to allow the increased capillary permeability to resolve and the interstitial fluid to be reabsorbed. In many situations, non-cardiogenic pulmonary edema cases receive furosemide during initial stabilization or perhaps later; in some cases the furosemide seems to improve the dog's condition. However, this is not due to induction of water loss in the kidney but to the bronchodilatory effects of furosemide. Therefore, many clinicians will use other bronchodilators such as albuterol, terbutaline or theophylline and feel that there is some improvement in dogs with non-cardiogenic pulmonary edema. However, it is unclear if the improvement is due to the drugs or the passage of time.

*Pneumonia* can occur in animals of any age but infectious pneumonia is most common in younger dogs and aspiration pneumonia is more common in older animals. Differentiation of the causes of pneumonia depend largely on the location of the lung pattern in concert with signalment and history. Classically, infectious pneumonia (bacterial, viral, or fungal) most

commonly affects the cranial lung lobes and creates an alveolar to interstitial pattern on radiographs. Fungal pneumonia creates a diffuse nodular pattern on radiographs that may appear similar to metastatic neoplasia. In contrast, aspiration pneumonia is marked by an alveolar to interstitial pattern that occurs most commonly in the right middle lung lobe and may occur concurrently with megaesophagus or another comorbidity such as neuromuscular weakness, previous seizure activity, or recumbency.

When treating pneumonia, the presumed or confirmed cause dictates treatment. Bacterial pneumonias are treated with antibiotics with a Gram positive aerobic spectrum such as penicillins or 2<sup>nd</sup> or 3<sup>rd</sup> generation cephalosporins. If there is reason to believe that Gram negative aerobes are involved or you wish to have more complete antibiotic coverage, fluoroquinolones or aminoglycosides are used. It is rare to have an anaerobic bacterial pneumonia. Fungal pneumonia is treated with fluconazole or itraconazole. Fungal pneumonia cases are much more likely than bacterial pneumonia to require mechanical ventilation due to respiratory failure. Respiratory failure can occur 48-72 hours after initiating treatment due to death of the fungal organisms and concurrent inflammation. Viral pneumonia lacks a specific anti-viral treatment and cases are managed supportively.

### **“Quiet auscultation”**

When dogs with lower airway disease have a quiet auscultation, the most common reasons are pneumothorax or pleural effusion. Certain breeds such as huskies and malamutes are prone to spontaneous pneumothorax and animals with a history of trauma commonly have pneumothorax. Pleural effusion can occur in any breed or age of dog and be caused by toxins (ex. anticoagulant rodenticide- hemothorax), heart failure, chylothorax, or pyothorax. The presence of a heart murmur or fever on physical exam in addition to history can help to suggest the reason for the pleural effusion.

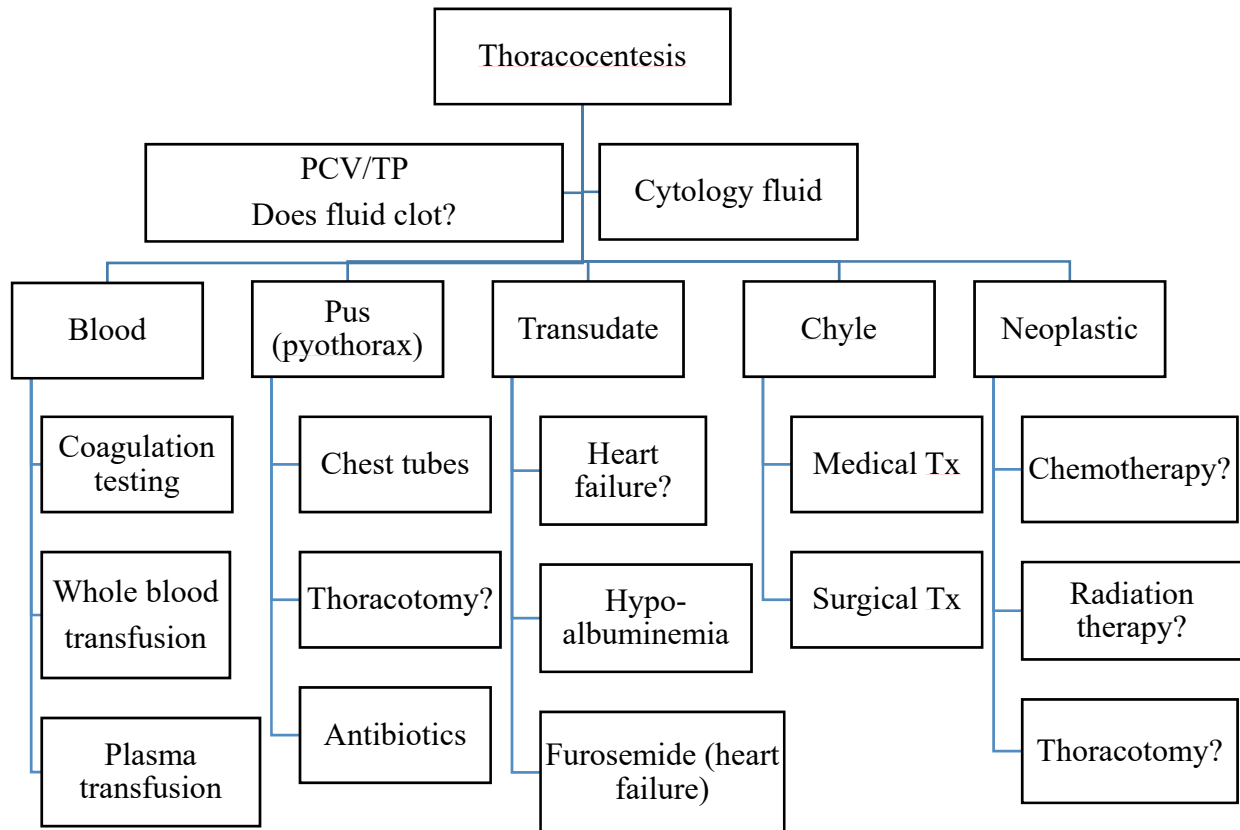
*Pneumothorax* tends to occur in animals less than 7 years of age since younger dogs are most likely to suffer from trauma and spontaneous pneumothorax tends to occur in young or middle-aged dogs. Spontaneous pneumothorax is a disease of larger breed dogs with Siberian huskies over-represented. Typically the source of the air are blebs or bullae that grow from the internal or external elastic layer of the pleura and subsequently rupture. However, occasionally chronic lung disease in older dogs can lead to spontaneous pneumothorax. Remember that coughing or even retching/vomiting are common clinical signs of pneumothorax in dogs. Dogs with traumatic pneumothorax very commonly have blunt force injury to the chest wall that leads to damage to the lungs and a pneumothorax without damaging the thoracic wall aside from possibly closed rib fractures. In contrast, in some cases bite wounds or other penetrating injuries of the chest wall lead to a pneumothorax. And rarely rib fractures/flail chest can secondarily damage the lungs leading to a pneumothorax.

Pneumothorax is first and foremost treated by thoracocentesis. The majority of pneumothorax cases need a single chest evacuation and do not have significant amounts of air refill the thoracic

cavity. However, in rare cases air continues to form in the chest from damage to the lungs and a chest tube +/- continuous suction drainage is required.

*Pleural effusion* can occur in all ages and breeds of dog. The cause of pleural effusion is dependent on the signalment of the dog and can be grouped into three large categories. Group A (dogs < 2 years) commonly suffer from pyothorax, transudate due to heart failure, or hemothorax (trauma or toxin). Group B (dogs 2-7 years old) have diseases such as pyothorax, chylothorax, transudate from heart failure, hemothorax, or neoplasia. Group C (dogs >7 yrs old) have heart failure-related transudate and neoplasia most commonly.

In order to determine the cause of the pleural effusion, thoracocentesis for at least a diagnostic sample needs to be performed. If the fluid appears RED, a PCV/TP should be immediately performed on the fluid along with placing a sample in a red top tube to see if it clots. Free blood in the chest will not clot in a red top tube and will have a PCV/TP roughly equivalent to or higher than the periphery. If the fluid is not red, it should be evaluated cytologically first. The following flowchart indicates an approach to pleural effusion in dogs. Depending on the fluidtype diagnosed, medical treatment and/or surgical treatment may be indicated (see below).



## Lower airway disease in cats

### **“Loud auscultation”**

Lower airway disease in cats can be divided categorically based on the animal’s signalment. It can also be thought of in terms of which diseases have a “loud” vs “quiet” auscultation. Cats with “loud” thoracic auscultation (ie. crackles and wheezes) typically have either *cardiogenic* or *non-cardiogenic pulmonary edema* or *feline asthma*. Cats can also get *pneumonia* which is bacterial or viral in origin.

In general, pulmonary edema occurs when fluid fills the interstitial space between the alveolus and capillary, thus limiting diffusion of oxygen from the alveolus to the blood. With non-cardiogenic pulmonary edema, the fluid buildup occurs secondary to increased permeability of the capillary wall leading to fluid extravasation into the lung tissue. Cardiogenic pulmonary edema occurs because increased fluid volume in the capillaries leads to increased capillary hydrostatic pressure which pushes fluid out of the capillary into the interstitial tissue and possibly the alveolus.

Cardiogenic pulmonary edema tends to be a disease of middle aged to older cats (ie. 5 yrs+) and should have an auscultable murmur present with the lung sounds. This murmur may not have been ausculted prior to the time the cat presented in heart failure. Cats will sometimes have an arrhythmia or gallop rhythm with heart disease as well. The most common type of heart disease in cats is hypertrophic cardiomyopathy although cats can rarely have congenital malformations or valvular disease. Certain breeds such as the Maine Coon cat are over-represented in cases of hypertrophic cardiomyopathy and will go into heart failure at a very young age. Heart failure in cats is treated by FON—*furosemide* (2 mg/kg IM or IV repeated every 20-30 minutes to a maximum of 6-8 mg/kg) and *oxygen* (with *narcotics* as needed for sedation).

In contrast, non-cardiogenic pulmonary edema occurs after prolonged seizure activity, drowning, choking and electrocution. It is therefore more commonly found in younger cats but can be seen in any age. Non-cardiogenic pulmonary edema is treated with oxygen and time +/- bronchodilators. In general, cats do not get non-cardiogenic pulmonary edema as commonly as dogs but it is still possible.

As with dogs, differentiation of cardiogenic vs non-cardiogenic pulmonary edema occurs largely from radiographs (in addition to signalment and history). Cardiogenic pulmonary edema in cats is an alveolar to interstitial pattern that can occur in nearly any lobe of the lungs. It is common to also see at least a small amount of pleural effusion in feline heart failure patients. In contrast, non-cardiogenic pulmonary edema in cats tends to be a bilaterally symmetric alveolar pattern that is strongest in the caudal lung fields.

Feline asthma tends to be a disease of younger cats (typically <5 yrs at their first presentation). They are most commonly indoor-outdoor cats and their signs are acute in onset. The owners may or may not recall previous shorter bouts of similar signs in the past. Asthmatic cats will often have periods of open mouth breathing as part of their presentation and might have a coughing history. Treatment for asthma emergently involves oxygen and administration of a

bronchodilator such as terbutaline (0.01 mg/kg SQ) or 1 puff of albuterol from a metered dose inhaler.

*Pneumonia* can occur in cats of any age but infectious pneumonia is most common in younger cats (esp. from shelter backgrounds). Differentiation of the causes of pneumonia depend largely on the location of the lung pattern in concert with signalment and history. Classically, infectious pneumonia (bacterial or viral) most commonly affects the cranial and ventral lung lobes and is an alveolar to interstitial pattern. With cats, many time animals with pneumonia have upper respiratory congestion and nasal discharge in their history.

When treating pneumonia, the presumed or confirmed cause dictates treatment. Bacterial pneumonias are treated with antibiotics with a Gram positive aerobic spectrum such as penicillins or 2<sup>nd</sup> or 3<sup>rd</sup> generation cephalosporins. If there is reason to believe that Gram negative aerobes are involved or you wish to have more complete antibiotic coverage, fluoroquinolones or aminoglycosides are used. Mycoplasma pneumonia is treated by fluoroquinolones. The treatment for pure viral pneumonia is supportive care.

### ***“Quiet auscultation”***

When a lower airway cat has a “quiet” auscultation, pleural effusion and pneumothorax are the most likely differentials. To determine if it is fluid or air in the thorax, thoracocentesis should be performed. Radiographs can be taken first in minimally affected cats, but if a cat is very dyspneic, TAP the chest before taking radiographs!

Pleural effusion is grouped into three main categories based on the cat’s signalment. Group A cats (typically < 5 yrs old) suffer from pyothorax, chylothorax, and possibly transudates from heart failure (especially if they are a breed predisposed to heart disease). Group B cats (5-10 years old) most commonly have diseases such as chylothorax, transudate from heart failure, or neoplasia. Older cats in group C (> 10 years old) most typically suffer from heart failure and neoplasia. Just as with dogs, the key to determining the type of effusion rests in performing cytology of the fluid. (And as with dogs, if the fluid appears to be red like blood, performing a PCV/TP and seeing if the sample clots in a red top tube is important to determine if there is free blood in the chest.) Treatment for each of the fluid types depends on the etiology of the fluid and includes surgical diseases such as chylothorax or refractory pyothorax or medically managed diseases like heart failure or neoplasia (see diagram above under canine lower airway disease).