RESPIRATORY DISTRESS IN DOGS AND CATS

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Respiratory distress is a stressful emergency as the patient presents with minimal reserve and is an imminent risk of respiratory or cardiac arrest. In addition, completing thorough physical examination and point of care diagnostics such as radiographs can add stress, which can lead to acute decompression and arrest. Prompt treatment is warranted to identify where the lesion is present and the underlying cause, help alleviate the sensation of difficulty breathing for the patient and provide diagnostic and therapeutic options.

There are 8 “big causes” of respiratory distress based on the anatomic localization of the lesion. The lesion may often be an anatomical abnormality, airway collapse, pulmonary edema of cardiac and non-cardiac causes, infection, inflammatory, and trauma.

1. Upper airway
2. Lower airway
3. Parenchymal
4. Pleural space disease
5. Chest wall disease
6. Abdominal distension
7. Look a likes
8. Pulmonary hypertension/ pulmonary thromboembolism (PTE)

Upper airway

Upper airway disease is a common cause of respiratory distress in dogs and less often in cats. Upper airway includes the nares, nostril passages, the pharynx and larynx and extra thoracic trachea up to the thoracic inlet.

Clinical signs

Clinical signs commonly found in dogs and cats with upper airway disease and distress includes loud breathing noises. Sterdor refers to increased nasal airway sounds, and is similar to loud snoring in humans. Stridor refers to a raspy breathing and tends to be associated with lesions within the laryngeal area, most often the larynx itself. A goose, dry honking cough may be present with lesions present at the level of the extra thoracic trachea such as a collapsing trachea. Upper airway disease is often seen with an INSPIRATORY respiratory effort, as the animal is struggling to move air Inside of the airway, given the location of the lesion.

Common complications

Hyperthermia is a common complication from upper airway obstruction, as an inability to cool.

If present, the pet will need to be actively cooled to 39.5C, by wetting the hair and possibly placing a fan over the animal. Care will be taken to not cool below 39.5C, as rebound hypothermia is a common complication of active cooling.

Non cardiogenic edema is also seen in upper respiratory obstructions in dogs (and less likely in cats). See below for diagnosis and treatment.

Differentials

Below is a non-exhaustive list of differential diseases which can cause upper airway:

DOGS:

- Brachycephalic airway syndrome
- Laryngeal paralysis
- Mass
- Abscess
- Neoplasia
- Tracheal collapse

CATS

- Nasal pharyngeal polyp
- Rhinitis
- Inflammatory laryngeal/ granulomatous disease
- Neoplasia
- Abscess
- Mass
- Foreign body

Treatment of upper airway disease:

1. Oxygen supplementation is the first and essential step of stabilization. Supplemental oxygen may be provided via a variety of options, including flow-by, face mask, nasal oxygen, e-collar and cellophane wrap ("oxygen hood") initially. During the initial assessment and stabilization phase, flow by oxygen is preferably used, and after providing sedation, the pet is place in an oxygen chamber.

2. Anxiolysis is the next step to provide to animals with upper airway disease. Anxiolysis/sedation will help slow the respiratory effort and make breath more efficient in moving oxygen inside the alveolis. However, those animals have minimal reserves and are at imminent risks of arrest, therefore the sedation should have no to minimal effects on the respiratory and cardiovascular system.

    - Butorphanol, is a Kappa agonist and a mu antagonist, which provides excellent sedation, good anxiolysis and minimal analgesia. It is reversible with naloxone. It is the first line agent for animals in upper and lower airway respiratory distress. It is a cough suppressant, with no effect on the respiratory drive and minimal effects on the cardiovascular system.

- Methadone, hydromorphone are acceptable alternatives, although they provide less sedation/ anxiolysis and more analgesic effects.
- Acepromazine is a alpha antagonist, which provides excellent anxiolysis and sedation and no analgesia. Acepromazine provides a dose dependent arterial vasodilation, which at high dose can lead to hypotension. At low dose, the small arterial vasodilation is beneficial as it will increase stroke volume by decreasing the afterload.
- Other options includes benzodiazepines such as midazolam and diazepam. These drugs provide sedation, possible anxiolysis and no analgesia. The effect is idiosyncratic, and in some cases, animals will become hyperexcitable with the administration of those drugs. When administered in combination, this is less likely.

I personally tend to start with butorphanol and if no effect is seen within 1-2 min will administer acepromazine shortly after, as the onset of effect tends to be longer- 5 to 10 min for full effect.

Sedation with alfaxolone may cause respiratory depression, and therefore I usually use that drug as a rapid induction agent, if cyanosis, hypoxemia and severe effort remains despite flow by administration of O2 and anxiolysis/ sedation.
Dexmedetomidine is an effective, reversible sedative and anxiolytic that **should also be used with caution in patients with cardiovascular disease or instability (i.e: with severe respiratory distress)**. However, low doses are effective at controlling anxiety (0.5–2 µg/kg IV dogs or 3–5 mg/kg IM dogs and cats). Its reversal agent, atipamezole, should be readily available for intramuscular administration (or IV in a life-threatening emergency) in an equal volume to that of the dexmedetomidine administered.

Table with recommended drugs and dosages for sedation/ anxiolysis in cases of respiratory distress

Drug	Dose	Route
Butorphanol	0.2-0.4 mg/kg	IV, IM, SC
Acepromazine	0.007-0.015mg/kg	IV, IM, SC
Midazolam	0.2-0.5 mg/kg	IV, IM, SC
Diazepam	0.2-0.5 mg/kg	IV only

Drugs for rapid induction intubation:
Alflaxolone: 1-4 mg/kg to effect IV
Propofol 2-4 mg/kg to effect IV

Both drugs will cause respiratory depression and the pet will need to be bagged once intubated to restore ventilation and oxygenation until the effects dissipates and the animal starts breathing on it's own again.

The clinician with a dog or cat with an upper airway obstruction should be ready to rapidly induce the animal for endotracheal intubation or worse case scenario an emergency

tracheostomy if intubation is not possible. (see anesthesia notes for all the tricks and tips to intubate a dog and cat). Placement of a very small ET tube is preferrable to having to perform a tracheostomy.

- Whether or not the animal requires rapid intubation, upper airway disease will often cause severe inflammation of the airway due to increased resistance within the airway flow. Therefore administration of a **low dose gluco-corticosteroid** is recommended. Dexamethasone SP 0.1-0.2 mg/kg IV, IM is commonly administered to help treat the edema associated with the inflammation. Non-steroidal medication are contraindicated, as these patients commonly have poor GI perfusion due to the hypoxemia and administration of an NSAID will increase the risk of GI ulceration, and possible GI upset.

Diagnostic tests:
PLUS is the first diagnostic imaging test that should be done in respiratory distress patients as it allows to identify presence of parenchymal disease (presence of B lines), pleural effusion and/or pneumothorax. Additionally cardiac POCUS also allows to identify enlarged left atrium to support cardiogenic cause of B lines (pulmonary edema secondary to left sided congestive heart failure) or pleural effusion in cats (left sided CHF). PLUS can be performed following initial triage examination, or with the patient in the oxygen cage following sedation and being placed in a calm environment.
In patients with upper airway disease, there is usually no significant findings on PLUS, or we may see presence of caudo dorsal B lines when we suspect non cardiogenic pulmonary edema (secondary to the upper airway obstruction).

3 view thoracic radiographs are an important diagnostic test in all patients with respiratory distress, but those should only be obtained once the animal is stable. Radiographs of the cervical trachea, larynx, and nasopharynx aid in the diagnosis of laryngeal and tracheal masses, extraluminal tracheal or laryngeal compressive disease, nasopharyngeal collapse or masses, cervical and thoracic inlet tracheal collapse, and foreign bodies. They are also helpful to evaluate presence of non-cardiogenic pulmonary edema secondary to upper airway obstruction within the lungs, cardiogenic pulmonary edema, pneumonia, intrathoracic tracheal or bronchial collapse or neoplasia. Fluoroscopy may be required to help assess dynamic changes within the airway diameter and collapse along the airway during all phases of respiration and coughing.
Occasionally, additional imaging such as a CT scan, endoscopy, biopsy, fin needle aspirates may be required to obtain the final diagnosis and treatment.

The final diagnosis as what is causing the upper airway obstruction is often obtained with a sedated upper airway examination. However, heavy sedation/ anesthesia, if not required by the patient's response to initial stabilization attempts carries significant risk upon anesthesia recovery if definitive intervention is not pursued concurrently. The clinician

should be able to recognize normal upper airway anatomy and function when performing an upper airway exam.

Lower airway disease:

Lower airway disease includes diseases which affect the intrathoracic trachea, the bronchi, bronchioles, basically all the intrathoracic airway tree. Most lower airway disease are chronic conditions, but acute exacerbation can result in acute presentation to a veterinary hospital.

Clinical signs
Clinical signs associated with lower airway disease includes coughing (often thought to be coughing up hairballs), lethargy, and vomiting have also been reported. On physical examination, tachypnea, expiratory respiratory effort, presence of wheezes on auscultation and an abdominal push are classic for lower airway disease.

Differential diseases for lower airway disease:

Canine	Feline
• Bronchitis • Bronchomalacia • Intra thoracic tracheal collapse • Bronchopneumonia • Lung worms • Neoplasia	• Feline asthma • Neoplasia • Abscess • Mass • Feline parasitic infection

Diagnostics tests:
Point of care ultrasound will often show no significant abnormalities. In some rare cases, of feline parasitic bronchial disease, parenchymal disease may be present and B lines may be seen.

When stable, thoracic radiographs can be diagnostic for most lower airway diseases processes. However, in many cases, especially in feline patients, empirical treatment is initiated prior to obtaining radiographs, as it will help with stabilization, and prevent decompensation while performing the diagnostic imaging.

Treatment:
Initial therapy is similar to that of upper airway disease with oxygen supplementation and sedation/ anxiolysis.

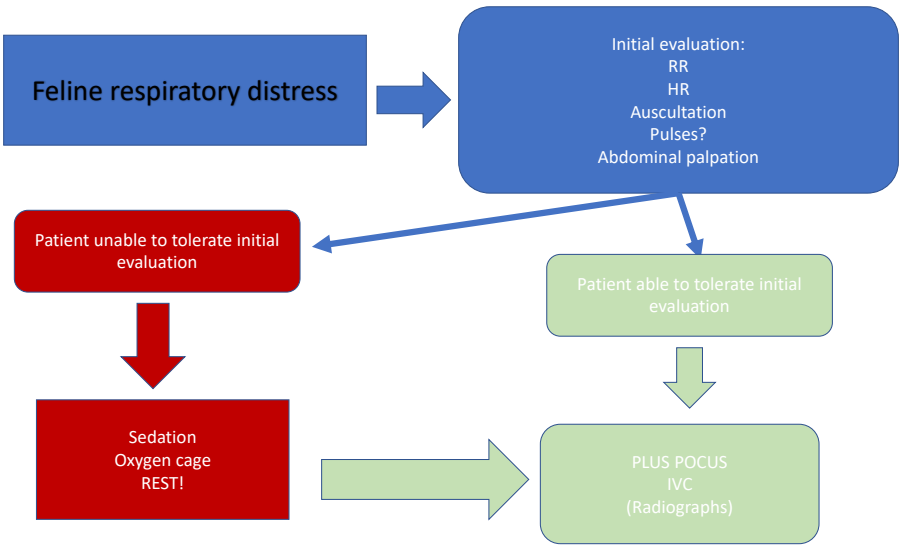
For feline asthma, treatment is twofold:

1) Reduce the inflammation within the respiratory tract and the production of mucus. This is done by administration of fast acting dexamethasone sodium phosphate, at 0.1 -0.2 mg/kg IV, IM, or SC. If IV administration is feasible without overly stressing the patient, then it can be given through that route, but most often the drug is given IM, and the patient is then placed in an oxygen chamber until the drug takes effects.
2) Bronchodilation. Feline asthma causes airway constriction, which further decreases the ability to expire the breath (which is already challenging given the presence of mucus in the airway). Bronchodilators can be administered in one of 2 ways:

- Injectable: terbutaline is an injectable selective beta 2 agonist which causes bronchodilation. Dose is 0.01 mg/kg and can be given IM or IV. Similarly, if IV administration is possible without overtly stressing the cat, this can should be the route of choice, but we will prefer IM administration and decreased stressed, over IV route.

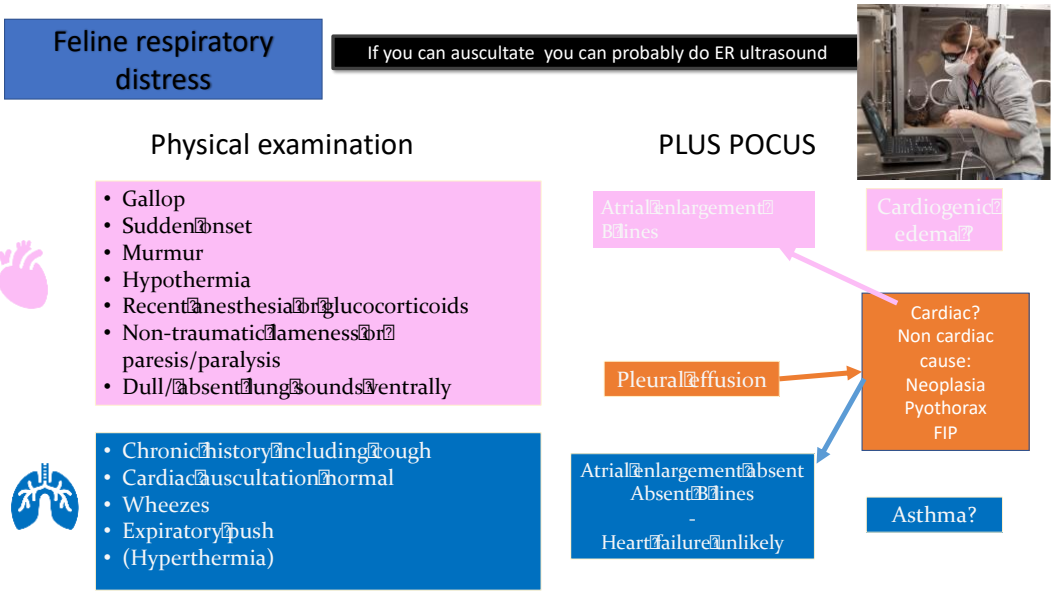
- Local administration: Salbutamol puffers. Salbutamol is also a short-acting beta-2 agonists (SABAs), such as terbutaline, and has a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol needs to be administered via a spacer, which is a tube connected to a small fitting mask which is placed over the cat's nose and mouth. The puffer should be vigorously shaken prior insertion into the spacer and delivery of the puff. Once the puff delivered, the clinician or technician should keep the aero chamber system sealed on the cat's mouth and nose for 10 seconds or 6 good big breaths. The procedure should be repeated twice to deliver the appropriate dose. It is very important to shake the puffer prior delivering each puff to mix the drug with the delivery air. Failure to do so, would decrease the dose delivered. Similarly, administration of 2 puffs "back-to-back" without a 10-20 sec pause would also result in a lower dose administered as the spring contained inside the puffer needs to reload and it takes about 20 seconds to do so.
  This video shows to correctly use an inhaler (intended for human patients suffering from asthma, but the concepts shown are the same):
  https://www.asthma.org.uk/advice/inhaler-videos/pmdi/

In some cats with respiratory distress, the patient is so affected, that they cannot sustain even a basic triage examination. In those cases, minimal handling is indicated, and the clinician should treat the most likely causes of respiratory distress:



Provide sedation: butorphanol 0.2- 0.4 mg/kg IM

The cat should be placed in an oxygen chamber and left to calm himself.
Once more relaxed and after improvement of the respiratory rate and effort, auscultation and POCUS can be performed within the oxygen chamber to limit stress and gain valuable diagnostic information.



Parenchymal disease:

Parenchymal disease is most often an acute presentation, with clinical signs of coughing, increased respiratory rate and effort. Auscultation may reveal presence of increased/ loud bronchovesicular sounds, crackles and possibly a heart murmur.
A fever may be present in case of inability to move air, or due to the presence of an infectious or inflammatory disease process.

Diagnostic tests:

Following careful physical examination, POCUS may yield some valuable information, with detection of B lines which represents a fluid/air interface. Depending on the history, physical examination findings and the localization of the B lines, the differential for the cause of the B lines varies. Assessment of the left atrial to aorta ratio is also recommended in cases of respiratory distress, especially when a heart murmur and/or B lines are detected on POCUS. If the left atria is enlarged (i.e. we can fit 4 aortas within the left atria, left sided congested heart failure with pulmonary edema is the most likely cause of respiratory distress). For example, in a dog with a history of vomiting, presence of a fever, crackles and B lines found in the right ventral medial thorax is most suggestive of aspiration pneumonia. In a cat with acute onset of respiratory distress, a gallop, mid thorax B lines and an enlarged left atrium, left sided congestive heart failure is suspected.

Radiographs, ideally three views should be performed **only** once the patient is stabilized. Radiographs will allow identification of pneumonia, congestive heart failure, lower airway disease, presence of chest wall disease etc...

Differentials for parenchymal disease:

Canine	Feline

• Pneumonia
  • Aspiration
  • Community acquired pneumonia
• Hemorrhage
  • Trauma = contusions
  • Coagulopathy
• Neoplasia
  • Mass/ nodules
  • lymphoma
• Pulmonary edema
  • Cardiogenic pulmonary edema
  • Non cardiogenic edema
• Eosinophilic bronchopneumopathy
	• Pneumonia
	• Hemorrhage
	  • Trauma= contusions
	  • Coagulopathy
	• Neoplasia
	• Pulmonary edema
	  • Cardiogenic pulmonary edema
	  • Non cardiogenic pulmonary edema
	• Parasitic pneumonia
	•

Treatment:
Parenchymal disease is the most likely to benefit **from supplemental oxygen** and this should be the first line of treatment. Sedation should be provided in cats. In dogs, sedation is not always administered if supplemental oxygen provides enough relief from the sensation of dyspnea.

Specific treatment should be instigated when a presumptive diagnosis is obtained. Treatment of cardiogenic pulmonary edema is covered in the acute cardiac emergencies, and pneumonia is covered in the internal medicine course.

Non cardiogenic edema is secondary to disruption of the endothelial wall and movement of water within the alveoli. This has been seen with electrocution, near drowning (immersion in salt or non-salty water), upper airway obstruction, and seizures. Presentation is acute and the underlying cause is not always obvious, as the injury may not have been witnessed. Diagnosis is performed with POCUS and thoracic radiographs. On POCUS, B lines are seen in the caudal dorsal area, but in severe cases can been seen in all lung field. The left atrium to aorta ratio is normal. On thoracic radiographs, an interstitial to alveolar pattern is seen in the caudo dorsal lung fields. There is an absence of enlargement of the pulmonary veins which rules out cardiogenic pulmonary edema. Treatment is mainly supportive with supplemental oxygen. Administration of furosemide is controversial in its efficiency in clearing the edema, given that their no increase in intravascular volume or interstitial pressure. One dose (1-2 mg/kg IV) can be administered, but prolonged administration may lead to renal injury without evidence of benefit. Most animals will improve within a few days of oxygen supplementation unless they develop an acute lung injury/ acute respiratory distress syndrome.

Pleural space disease.

Pleural space disease is a common emergency presentation in dogs and cats. Pleural space disease presents with increased respiratory rate and effort. The breath pattern is abnormal with short and shallow breath, as the normal chest wall compliance is impaired by the presence of either fluid or air between the two pleurae. Abdominal effort is common. Thoracic auscultation yields an uneven breath sound. With presence of air, lung sounds are dull/ diminished in the dorsal hemithorax and increased in the ventral hemithorax. In cases of pleural effusion, the opposite is found, with dull/ diminished lung sounds in the ventral hemithorax and increased lung sounds in the dorsal hemithorax. In cases of diaphragmatic hernia, dull/ diminished bronchovesicular sounds may found in the caudal thorax, and borborygmi may be auscultated if intestines are herniated.

Diagnostics tests
Diagnosis is usually found on POCUS, with presence of pleural effusion seen in the ventral thorax. Pneumothorax is also diagnosed via POCUS with absence of a lung sliding, presence of an abnormal curtain sign (see POCUS lecture notes for complete findings).

Thoracic radiographs may be warranted in cases of diaphragmatic hernia to confirm the diagnosis.

Treatment:
Animals with pleural space disease (pleural effusion and pneumothoraxes) benefit most from a thoracocentesis. Thoracocentesis has the benefit of being both diagnostic and therapeutic procedure.
In cases of pleural effusion, measurement of total solids and in house cytology (ideally on a spun down sample) should be performed bedside. The rest of the sample should be sent out for a clinical pathologist's analysis and cytology.
Specific treatment for pleural effusion will not be covered here.

Differentials for pleural space disease

Diaphragmatic hernia	***Pneumothorax***	***Pleural effusion***		
		Low protein transudate	*High protein transudate*	*Exudate*
Trauma Congenital	Traumatic Spontaneous	Neoplasia, Cardiac disease Lung lobe torsion Hypoalbuminemia	Neoplasia, Cardiac disease Lung lobe torsion, Chylous	Pyothorax Fungi Neoplasia Pneumonia FIP

Pneumothoraxes
Pneumothoraxes can either be spontaneous or traumatic.

- Spontaneous pneumothoraxes: are secondary to rupture of a bleb (small bubble found at the surface of the lung) or bulla (bubble found within the parenchyma) and without any history of trauma. Spontaneous pneumothoraxes should be suspected in all cases of acute respiratory distress with suspicion of pleural space disease on exam and POCUS.

- Traumatic pneumothoraxes are the most common presentation of pneumothoraxes in small animals. Any type of trauma can lead to development of a pneumothorax (vehicular, high rise, bite wound, firearm/weapon injuries, etc..

- Treatment:

Treatment of pneumothoraxes relies on thoracocentesis to relieve the pressure within the pleural space and allow expansion of the lungs for improved ventilation and oxygenation. In cases in which air fills quickly (if more than 3 thoracocentesis are required within a 24-hour period) then placement of a thoracostomy tube is recommended. In cases in which negative pressure cannot be obtained during a thoracocentesis, we are concerned for the presence of a possible tension pneumothorax.
Tensions pneumothorax occurs when the lesion within the lung parenchyma creates a one-way valve, where the leaked air builds up in the pleural space and is trapped within the pleural space. This creates an increase in pressure which causes compression of the structures within the thoracic cavity: compression of the lung parenchyma and inability to ventilate and oxygenation, and compression of the vasculature, specifically the veins which are more collapsible than the arteries, thus decreasing venous return and there of cardiac output. Tension pneumothorax can occur in both traumatic and spontaneous pneumothoraxes. As you see, tension pneumothorax if left untreated will cause fast cardiorespiratory arrest.

Treatment of tension pneumothorax consists in placement of a thoracotomy tube and placement of a continuous suction device, called pleurovac. This allows constant aspiration of the air leaked within the pleural space and aims to allow creation of a fibrous scar tissue that would seal the leak. A CT scan is strongly recommended in all cases of pneumothoraxes, specifically in cases of tension pneumothorax to identify the source of the leak and see whether a seal is possible, or whether surgery with lung lobectomy is recommended.