Respiratory distress is a common presenting complaint in veterinary medicine that requires the veterinary team to work quickly and efficiently to evaluate and stabilize the patient. Initial assessment of the patient should begin with an observation of the animal’s breathing pattern as this may increase one’s suspicion for upper airway disease, pleural space disease or pulmonary parenchymal disease. Animals with upper airway obstructions may have significant inspiratory stridor while animals with pleural space disease may have a restrictive, asynchronous breathing pattern. Auscultation should focus on describing the patient’s lung sounds, auscultation of the laryngeal/ tracheal region and evaluating the patient for a murmur or arrhythmia. In unstable patients an abbreviated physical examination may need to be performed.

While the patient is being examined a detailed history should be obtained from the clients including any prior respiratory or cardiac disease, duration of clinical signs, change of clinical signs with activity or time of day, vaccination/preventative status, history of vomiting/regurgitation, prior medical history and current medications.

Supplemental oxygen should also be administered to all patients presenting for difficulty breathing. Oxygen therapy can be delivered in a number of different manners depending on the equipment at your disposal.

Short term supplementation:

Flow-by oxygen supplementation can be supplied by holding oxygen tubing which is hooked up to an anesthetic machine or a humidified oxygen source approximately 2 cm from the patient’s nostrils. These tubes can be held perpendicular or parallel to the patient. Inspired oxygen rates of 25 - 40% can be obtained with an oxygen flow rate of 2 - 3 L/min. Oxygen can also be administered by a face mask that is attached to oxygen tubing. Tight fitting masks can deliver up to 50 - 60% oxygen when flow rates of 8 - 12 L/min are utilized, while lower flow rates (2 - 5 L/min) should be utilized with loose fitting masks. It is important to note that not all patients will tolerate face mask therapy and that both flow-by oxygen and face masks are not effective long term methods to supply supplemental oxygen.

Prolonged oxygen supplementation
Prolonged oxygen therapy can be provided by oxygen hoods, oxygen cages, nasal oxygen lines, intubation or mechanical ventilation.

Oxygen hoods can be purchased from manufacturers or they can be created from an Elizabethan collar, Saran wrap and tape. The Saran wrap is placed over ¾ of the front of the E-collar and taped in place. The remaining ¼ of the E-collar is left open for ventilation. Oxygen tubing is then placed through the back of the collar and taped in place. Oxygen flow rates of 1 - 2 L/min can provide approximately 30 - 40% oxygen depending on the fit of the collar. Hyperthermia can occur with oxygen hoods and the patient’s temperature must be closely monitored.

Oxygen cages can be created from human pediatric incubators where humidified oxygen is infused into the incubators or commercially available units can be purchased where the oxygen concentration, humidity and temperature can be controlled. Oxygen concentrations up to 50 - 60% can be administered through these cages. The temperature of most oxygen cages should be around 70°F to prevent hyperthermia. Oxygen cages are excellent sources of supplemental oxygen in patients requiring prolonged oxygen therapy, but they are not practical for larger patients or for some patients requiring frequent treatments.

For those patients who are too large for an oxygen cage nasal lines may be placed. Human nasal prongs can be used in medium to large breed dogs, but these lines can become dislodged easily. In this author’s experience nasal oxygen catheters are tolerated much better than nasal prongs and are not as easily dislodged. To place nasal oxygen lines the patient’s nasal cavity should be numbed with topical 2% lidocaine or proparacaine. The catheter (5 - 10 French red rubber catheter depending on the size of the patient) should then be measured from the tip of the nose to the lateral canthus of the eye and that area marked with a permanent marker. The tip of the catheter is then lubricated with sterile lube and inserted into the ventral nasal meatus (ventromedial) to the predetermined length. The catheter is then secured to the nostril and lateral maxilla with suture. Humidified oxygen can then be administered through the catheter.

Unilateral or bilateral nasal oxygen tubes may be placed. Oxygen flow rates of 50 - 100 ml/kg/min (per catheter) can provide up to 30 - 70% oxygen. High flow rates may cause irritation to the nares and may not be tolerated by the patient.

Humidified oxygen should be provided to any patient requiring prolonged oxygen therapy to help prevent drying and irritation of the nasal mucosa. Patients that have received more than a few hours of non-humidified air can develop dehydration of the nasal mucosa, respiratory epithelial degeneration, impaired mucociliary clearance and can be at increased risks for nasal and respiratory infections.

Once supplemental oxygen has been administered to the patient further evaluation of their underlying disease can proceed. Respiratory disease can be divided into 3 broad categories: upper respiratory disease, lower respiratory disease (pulmonary parenchymal disease) and pleural space disease.

**Upper respiratory disease**
The upper respiratory system includes the nasal passages, nasopharynx, oropharynx, larynx and cervical tracheal. Upper airway disorders can occur secondary to foreign bodies, neoplasia, infection, edema or degenerative neuromuscular disease (ie. laryngeal paralysis). Clinical signs associated with upper airway disease may vary based on the site and severity of disease. One of the hallmark signs of upper airway disease is stridor, a high pitched noise usually associated with inspiration, however the presence of stridor does not exclude concurrent lower airway disease. Laryngeal and pharyngeal disease can also result in dysphonia (change in bark/meow).

Upper airway disease develops secondary to narrowing of the airways. This narrowing may be static or dynamic. Static disease is present at all times and typically results in clinical signs of both inspiratory and expiratory dyspnea. Dynamic lesions may produce dyspnea during inspiration or expiration pending on the site of obstruction. Intrathoracic obstruction typically results in increased effort during expiration while extrathoracic obstruction results in increased effort during inspiration.

Animals with upper respiratory obstruction can present in varying degrees of distress. Immediate stabilization should focus on administering supplemental oxygen and sedation. Acepromazine (0.005 - 0.02mg/kg IV or 0.01 - 0.05 mg/kg IM) and Butorphanol (0.1 - 0.5 mg/kg IV or IM) can be used independently or in combination for sedation. For patients in severe distress where sedation and supplemental oxygen are not improving their distress, intubation and/or tracheostomy following a propofol titration (0.05 - 2 mg/kg IV titration) may be necessary for stabilization. Most animals with upper airway obstruction will have a marked improvement and normalization of their breathing patterns and mucous membrane colors with intubation. Antiinflammatory glucocorticoid therapy (dexamethasone sodium phosphate 0.05 - 0.15 mg/kg IV or IM) , if not contraindicated, may be administered to help decrease upper airway inflammation. These patients also commonly present hyperthermic. Active cooling with room temperature intravenous fluids, fans and room temperature baths should be instituted in patients with a temperature > 104.5 and active cooling stopped. Ice packs should not be placed on the patient as this causes local vasoconstriction. Placing alcohol on the pads of the feet is also not effective as the surface area is too small to be effective. Once the patient is stable the underlying cause of their upper airway disease can be addressed.

Pulmonary parenchymal disease

Pulmonary parenchymal disease may occur secondary to numerous diseases including pneumonia, hemorrhage, edema (cardiogenic or noncardiogenic), infiltrative disease, inflammatory disease or pulmonary thromboemboli. Auscultation of patients with pulmonary parenchymal disease will typically reveal increased bronchovesicular or harsh lung sounds, pulmonary crackles or wheezes. The distribution of these abnormal lung sounds can assist doctors in determining the underlying cause of disease. For instance, auscultation of perihilar crackles is highly suspicious for congestive heart failure while crackles over the right middle lung lobe is more suspicious for pneumonia. Any patient in respiratory distress should receive supplemental oxygen as discussed above. If congestive heart failure is suspected furosemide (2 -
4 mg/kg IV, IM, SQ) should be administered. Once the pet has been stabilized thoracic radiographs can be obtained to further evaluate pulmonary disease.

Below we will discuss a few causes of pulmonary parenchymal disease. Please note that this lecture does not discuss all causes of pulmonary parenchymal disease.

- **Aspiration pneumonia** is a common cause of respiratory distress in dogs. This is primarily diagnosed based on thoracic radiographs where an interstitial to alveolar pattern is noted in the right middle and cranioventral lung lobes although any lung lobe may be affected. Pneumonia is treated with oxygen support, nebulization/coupage, fluid therapy, antiemetics (if indicated) and broad spectrum antibiotic therapy.

- **Cardiogenic pulmonary edema** in dogs typically has a perihilar distribution while a diffuse patchy infiltrate can be seen in cats. Auscultation will typically reveal cardiac abnormalities (murmur and/or arrhythmia) but not all cats will have audible cardiac abnormalities. Pulmonary venous congestion and cardiomegaly may also be noted on radiographs. Echocardiogram is necessary to determine the underlying cause of cardiac disease but is not necessary for emergency treatment and stabilization.

- **Non-cardiogenic pulmonary edema** can occur secondary to several diseases including seizure activity, upper airway obstruction or electrocution. Radiographically these patients typically have an interstitial to alveolar pattern in their caudodorsal lung fields and clinical signs can occur within minutes of the underlying insult. Treatment for noncardiogenic pulmonary edema is largely supportive and the use of diuretics is controversial.

- **Animals** can also develop pulmonary parenchymal disease secondary to hemorrhage. Spontaneous hemorrhage can occur secondary to anticoagulant rodenticide ingestion, thrombocytopenia or other coagulopathies (ie. DIC, liver failure). Treatment involves correcting the underlying disease and supplemental oxygen therapy. Pulmonary hemorrhage can also occur secondary to trauma resulting in pulmonary contusions. Contusions can develop immediately following trauma but can also be delayed up to 12 hours. Treatment is once again supportive. Pulmonary contusions typically resolve within 24-48 hours.

- **Allergic airway disease** encompasses a number of different feline and canine diseases including but not limited to feline asthma, parasitic airway disease, allergic bronchitis and pulmonary infiltrates with eosinophils. These diseases can results in airway inflammation, bronchoconstriction, increased bronchial secretions, and pulmonary air trapping. Dogs will typically present with a history of coughing while cats may present for retching or coughing. Pulmonary wheezes with increased expiratory effort may be noted during examination. A diffuse bronchial pattern with hyperinflation of the lungs and flattening of the diaphragm may be seen on radiographs, especially in cats with “feline asthma.” Stabilization of these patients includes supplemental oxygen therapy, bronchodilators (such as albuterol, terbutaline and aminophylline) and glucocorticoids.

Pleural space disease
Animals with pleural space disease typically present with a restrictive breathing (rapid, shallow breaths) or asynchronous breathing pattern where the chest and abdomen do not rise and fall at the same time. Pleural space disease occurs secondary to fluid, air or other soft tissues compressing the lungs limiting their inflation. Auscultation of these patients may reveal decreased or absent lung sounds. When this occurs ventrally it is suspicious for pleural effusion while absent lung sounds dorsally is concerning for a pneumothorax. Pleural effusion can occur from any number of causes including congestive heart failure, neoplasia, infection, coagulopathy, lung lobe torsion, vasculitis, hypoalbuminemia, chylous effusion or diaphragmatic hernia. The development of a pneumothorax may occur secondary to trauma, a ruptured bullae/bleb, secondary to chronic lower airway disease or iatrogenic.

Patients with significant pleural space disease can rapidly decline and go into respiratory arrest. These patients should be handled as little as possible and once again sedation may be necessary. When available a thoracic ultrasound should be performed to evaluate these patients for the presence of pleural effusion. Radiographs should not be taken until the patient is stabilized as the stress could cause respiratory arrest. Thoracocentesis and/or chest tube placement may also need to be performed to stabilize these patients.

**Summary**

Respiratory distress is a common presenting complaint in veterinary medicine. Thoracic auscultation in combination with a detailed history may allow the clinician to deduce the likely cause of clinical signs so that empirical therapy can be initiated while the patient is being stabilized.