Respiratory Medicine: The Ins and Outs

by

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Welcome to Hamburger University!
Respiratory Medicine: The Ins and Outs  
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OVERVIEW
Today’s goal is to provide an overview of common respiratory diseases in practices, and to include discussions on options for diagnostics, therapeutics and prognosis. We will cover conditions including upper airway obstruction, asthma, pulmonary vascular disease, and pleural space disease as well as review options for collecting samples, providing supplemental oxygen and for advances in diagnostic imaging.

AIRWAY OBSTRUCTION
Airway obstruction is a true emergency. Successful resolution requires a rapid combination of pattern recognition, excellent judgment and technical acuity. Airway obstruction may be divided into several categories, including laryngeal paralysis (lar par), brachycephalic airway syndrome (BCAS), allergic reactions, neoplasia and foreign objects both in and around the airway (eg. sticks/abscesses). In the first 3 categories in particular, the obstruction will be magnified by tissue edema created by high flow rates as the patient tries to breath around the obstruction. Additionally, hyperthermia is common as effects to cool by panting are thwarted, and increased muscle activity is associated with aggressive respiratory efforts.

PATTERN RECOGNITION: Airway obstruction caused by anatomic abnormalities is most common on hot and humid days, or following some sort of exertion. Most anatomic airway obstruction dogs have historical or physical examination evidence of pre-existing disease. Lar par dogs are usually older large breed dogs, with a preponderance of Labrador retrievers. Most dogs have been “noisy” breathers for awhile (months to years), with a relatively sudden onset of severe distress. In contrast, brachycephalics are immediately recognizable by their physical appearance. Overweight Bulldogs and Pugs seem over-represented in specific crisis, although any brachycephalic breed may be affected. Brachycephalics are also seemingly commonly affected when hospitalized for other conditions, such as corneal ulcers or knee disease. Knowledge of this is vital to the practitioner, because while some dogs (eg. Beagles) may bark and whine for hours seemingly without consequence, the anxious whiny Bulldog is a potential disaster waiting to happen. Dogs with allergic reactions typically are apparent on physical examination, and intra-airway foreign bodies typically rapidly appreciated. Upper airway obstruction can either be life-threatening or rapidly progress to such.

EXCELLENT JUDGMENT: As clinicians, we all strive for excellence in judgment. The decision making in upper airway obstruction involves answering the following questions:

1) Should I give sedation? Should I give steroids?
2) Do I need to intubate?
3) What is likely to happen if I do that?
4) Do I need to do a tracheostomy?
5) Where are we going long term?
Mild sedation is warranted for many dogs with upper airway obstruction, particularly those with chronic and dynamic conditions. As the respiratory inspiratory flow rates increase, mobile soft tissues are “sucked” into the lumen of the airway, resulting in increased obstruction to flow as well as edema of the tissues themselves. Laryngeal saccules will evert, and as well decrease the laryngeal diameter, which will increase the airway resistance. Acepromazine in low doses ± butorphanol is often very helpful to limit the dynamic component. Dogs that are hyperthermic should also be actively cooled with room temperature IV fluids and wet down with tepid water. A single dose of rapidly acting glucocorticoids is often warranted due to extreme tissue edema. Hyperthermia in dogs serves as a strong incentive to pant, and this will further increase airway edema. For dogs that have severe obstruction (evidenced by cyanosis, or extreme distress), sedation alone is not enough and the pet should be anesthetized and rapidly intubated. DO NOT WATCH A DOG WITH MARKED DISTRESS WAITING FOR THEM TO GET BETTER.

Intubation is often life-saving. Most clinicians and technician are proficient at intubating dogs and cats with normal anatomy. The challenges that accompany airway obstruction reflect the underlying disease. Pets with laryngeal collapse or paralysis are easy to intubate, as their obstruction is far more dynamic. If the pet is only moderately distressed, it is reasonable to take a few seconds to evaluate function (abduction/collapse) but if there is severe distress this may be postponed until a future date. Brachycephalics are often challenging to intubate under the best of circumstances. Useful aids include a long laryngoscope blade, and multiple tube sizes. Clinicians should consider learning to intubate blindly using digital palpation only, to use a bronchoscope which is primarily advanced into the trachea and then the tube is advanced past that, or intubating first with a long polypropylene catheter and then advancing the tube over that. If you are unable to intubate within ~ 20-30 seconds, an urgent tracheostomy should be performed, although if you are prepared this is relatively unlikely. As a side note, you should not plan to do an oral examination on a pet with a potential upper airway obstruction without having all the supplies needed handy. This is because sedation for examination may result in the collapse of the residual airway lumen.

Following intubation, the next step is to support the pet and to decide what to do next. The next step with reflect the disease process. It is important to decide between leaving the pet sedated and intubated versus performing a tracheostomy. The step chosen typically reflects the underlying disease. The following are my biases from the Northeast part of the US, clinicians in other parts of the world may have other choices.

**Laryngeal paralysis:** Most laryngeal paralysis dogs are older retrievers. In most, sedation and cooling are adequate. The owners should be advised regarding palliative surgical options, with the understanding of the risk to benefit ratio, and particularly of the real potential for aspiration pneumonia. If the dog is intubated, following sedation and cooling, extubation should be attempted. If not possible, and surgical palliation is not immediately available, a tracheostomy should be performed. My rationale for this is that these dogs are at an already increased risk of aspiration and leaving them intubated will greatly increase the risk of this. Additionally, these tend to be older and occasionally
decrepit dogs, and arthritis and other consequences of prolonged immobility are rapidly apparent. Consideration should be given to performing a tracheal wash for evaluation of airway cytology and bacterial culture.

**Brachycephalic airway syndrome:** While BAS is multi-factorial, most often elongated soft palate and subsequent eversion of the laryngeal saccules are responsible for the crisis situation. Intubation even for a day or longer until either the swelling can be reduced or palliative surgery may be performed is a reasonable option. Bulldog tracheostomies are not to be taken lightly. They have small trachea overall and thick necks, these features combined with a tracheostomy may prove fatal. Ultimately, it important to evaluate the larynx for concurrent laryngeal collapse. As a preemptive strike, it is wise to evaluate and surgical palliate the upper airway of brachycephalics prior to a crisis developing.

**Allergic reactions:** Most allergic reactions cause hives (urticaria) and little more. True anaphylactic reactions appear to be exceeding rare in dogs and cats. A number of dogs develop facial swelling and edema, with occasionally airway obstruction following the chewing on sticks or other objects. Treatment should involve glucocorticoids, antihistamines, and rest. If airway occlusion is present, a tracheostomy is advised.

**Foreign object:** Most foreign object airway obstructions are only partial, as complete obstructions would also certainly result in the patient’s death prior to arrival. Methods of removing obstruction include a modified Heimlich maneuver, hemostats or long towel clamps, or bronchoscopy. A temporary tracheostomy may be advised if the object is not rapidly removed. Dogs with severe quillings (porcupine) may be very challenging to treat, as, when the porcupine is attacked, quills often fill the larynx making intubation contra-indicated.

**Neoplasia:** suspected neoplasia airway obstruction is less common, but may develop over weeks to months. Thyroid and laryngeal neoplasia are particularly common. The clinician should recall that the thyroid is a very vascular structure, and hemorrhage may occur after aspiration of a neck mass, potentially resulting in airway obstruction. If a laryngeal mass is identified and biopsied, it is uncommon to not require a temp tracheostomy as post-procedural swelling often reduces the lumen of the airway even further.

**Cats:** Upper airway obstruction is less common in cats than in dogs. In young cats (particularly purebred cats), nasopharyngeal polyps may develop and result in airway obstruction. In older cats, neoplasia is relatively common. In cats in particular have a predisposition to laryngospasm, and placement of a tracheosomy tube should be considered prior to the recovery from anesthesia. It is not a good plan to “see how they do,” as asphyxiation is likely.

**TECHNICAL ACUITY**
As with all procedures, experience breeds comfort. Clinicians and technicians should evaluate multiple airways and prepare for emergencies. Digital and sylet guided intubation should be attempted in non-crisis settings, so that they are not impossible in the crisis setting. Tracheostomies should be practiced on cadavers if possible. Equipment
for the difficulty airway should be carefully maintained, and kept in a convenient location.

**Asthma/Chronic Bronchitis**

Asthma is a common, although poorly defined, condition in cats. Some authors attempt to classify cats with lower airway diseases into asthma or chronic bronchitis. Clinical signs include cough (occasionally productive), wheezing and respiratory distress. Some cats with asthma cough their entire lives but seem otherwise unaffected while some cats have episodes of severe respiratory distress separated by months to years. At this point, perhaps asthma can be best described as a lower airway disease with some cats having primarily components of asthma (reversible bronchoconstriction) and some cats having primarily components of chronic bronchitis.

The history of affected cats is generally either chronic coughing or an acute onset of respiratory distress. In some cats, seasonal variations are detected and city cats seem to have a higher incidence than cats from more rural environments. Anecdotally, cats from smoking households appear to be at increased risk.

Physical examination may be normal in cats with chronic cough or may reveal moderate to severe respiratory distress. Pronounced crackles or wheezes may be present on auscultation. The expiratory phase in particular may appear pronounced or lengthened. Some cats have a history of severe respiratory infection during kittenhood.

Radiographs will often demonstrate an increased bronchial pattern (‘donuts’), with hyperinflation. Occasionally the right middle lung lobe is collapsed. Radiographs are also very useful for excluding other causes of respiratory distress such as congestive heart failure. Radiographs may also appear normal.

Routine blood testing is usually normal although peripheral eosinophilia (> 1500 μL) may be present in asthmatic cats. A transoral tracheal wash may be performed to evaluate cytology of the airways. Asthmatic cats often have an increased eosinophil count although other inflammatory cells may be present in wash samples as well. Occasionally, a bacterial infection may be present and may complicate diagnosis and therapy. It is important to realize that at tracheal wash may be stressful in a cat that has respiratory compromise. In our practice, tracheal washes are performed much more frequently in cats with chronic cough than those with acute distress. Our protocol involves non-stressful placement of an intravenous catheter, pre-oxygenation for 5 minutes, then induction with propofol (2-10 mg/kg slow IV to effect), placement of sterile endotracheal tube and then washing with 2-3 ml aliquots of sterile saline. A sterile specimen cup may be placed at the end of the tracheal tube to collect any secretions that are expectorated. The sample should be submitted for cytology and aerobic culture. Some cats appear to have a secondary bacterial infection, particularly those with right middle lung collapse. Mycoplasma infection may also play a role in triggering an asthmatic response in some cats.
In most cases, it is appropriate to exclude other causes of airway inflammation or cough. *Aelurostrongylus abstrusus* (lungworm) infections may also develop in cats. Clinical signs may appear initially similar to feline asthma. However, affected cats are usually younger and live primarily outdoors as a snail/slug intermediate host is required. Larvae may be identified on a tracheal wash or via a Baermann fecal. Fenbendazole is used for treatment. Occasionally other internal parasites may migrate through the lungs and cause an allergic response (ascarids) in young cats. Heartworm infection is another possible trigger for allergic lung disease in cats. In recent years, heartworm infection has been described more frequently in cats in all parts of the United States, even indoor cats. Diagnosis may be challenging as affected cats have a small worm burden, but may be made through serology or occasionally echocardiography. Treatment of the asthmatic cat includes glucocorticoids (prednisone or long-acting reposital preparations such as Depo-Medrol®) and bronchodilators. (See table 1) Commonly used bronchodilators include theophylline and beta-2 agonist like terbutaline or inhaled albuterol. Newer proposed treatments include leukotriene antagonists, cyclosporine A, anti-interleukin 5 antibodies or cyproheptadine. Individual cats may vary in their response to various therapies. In an emergency setting, the severely asthmatic cat (status asthmaticus) should be treated with oxygen, minimal handling, injectable rapid-acting glucocorticoids, and possibly beta-2 agonists. If a response is not seen within 6-12 hours, it may be wise to re-consider the diagnosis. Long-term therapy tends to reflect both the owner and the clinician’s preferences. My usual choice is prednisone or Depo-Medrol® for those cats that have had moderate to severe signs. Some cats do very well with either theophylline or terbutaline. I have also treated some cats with Accolate® successfully, although these were cats that did not tolerate glucocorticoids well due to other conditions (eg diabetes/ congestive heart failure).

Aerosol therapies are very popular among some clients and in certain regions. Aerosol therapy is divided into acute and chronic therapy. Albuterol (Salmbutol) is used for acute bronchodilation, while inhaled steroids (flovent) are used for the maintainence phase. The interested reader is directed to www.fritzthebrave.com for more information on inhaled medication. In summary, in order to understand aerosols, it is important to review the both the technical aspect of aerosol delivery and also the normal physiological response to particulate inhalation (or the clearance of inhaled particles based upon their deposition). The deposition of aerosol particles within the respiratory tract is dependent upon their size as well as the tidal volume, inspiratory flow rates and ability to breath-hold. Optimal particle size for delivery to the trachea is 2-10 um and 0.5-5 um in the peripheral airways. Particle size is dependent upon the type of nebulizer or metered dose inhaler (MDI). Classically, in dogs and cats, aerosols are delivered via a nebulizer. The nebulizer is connected to a source of compressed air or oxygen. The drug is placed within a chamber and the nebulizer unit which is connected to a baffle which generates the particles. The patient is typically placed within a chamber and receives the nebulization treatment for a specific length of time. It is important to distinguish medical grade nebulizer from humidifiers which may merely generate water vapor. As in human medicine, aerosol therapy is considered potentially desirable as a method to limit systemic absorption and to direct therapy to the source of the problem.
Diseases that are considered particularly amenable to aerosol therapy include feline asthma (as well as canine chronic bronchitis or kennel cough complex in puppies.) Drugs that are considered beneficial when administered via the aerosol route include bland salineantibiotics (particularly aminoglycosides), glucocorticoids, and bronchodilators (eg. Beta-2 agonists such as albuterol). Doses are somewhat arbitrary, as human dosing is based upon cooperation with instructions to inhale deeply and to momentarily breath-hold. Aerosol therapy has been proposed as a method of limiting complications of systemic glucocorticoids, with treatments with inhaled glucocorticoids or use of the inhaled beta-2 agonists for immediate relief of bronchoconstriction. Rationale recommendations for treatment of asthmatic cats include first controlling the crisis with oral or injectable glucocorticoids, then to transition to inhaled glucocorticoids. It is prudent to warn clients that inhaled glucocorticoids are expensive (~ $100-200) particularly when contrasted with prednisone. Some cats do well with intermittent treatments with inhaled beta-2 agonists during crisis. It is NOT appropriate to treat the cat with inhaled beta-2 on a regular basis as this has been shown to increase the likelihood of complications in people. For cats with a single bout, aerosol therapy may be more expensive and troublesome than it is worth; however, it cats that appear moderately affected, it is worth discussing. Most cats do tolerate inhaled therapy, particularly in a home environment, but some cats are very challenging to treat.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>MOA/Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisone</td>
<td>5 mg PO q 12 hours, then taper</td>
<td>Anti-inflammatory</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Effective, inexpensive, rare system side effects, usually first-line of therapy</td>
</tr>
<tr>
<td>Methylprednisolone acetate (Depo-Medrol ®)</td>
<td>10-20mg/ cat IM or SC q 2-4 weeks</td>
<td>Anti-inflammatory</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Effective, good for &quot;difficult to pill&quot; cats</td>
</tr>
<tr>
<td>Terbutaline (Brethine®)</td>
<td>0.625mg/cat PO q 8 to 12 hours (1/4 tablet) or 0.01 mg/kg IV or SC</td>
<td>Beta-2 agonist</td>
</tr>
<tr>
<td>2.5 mg tablets</td>
<td></td>
<td>Variable efficacy long-term; Excellent parentally in crisis</td>
</tr>
<tr>
<td>Theophylline (Theo-Dur®)</td>
<td>20 ‰ 25 mg/kg PO q 24 hours AT NIGHT</td>
<td>Methylxanthine</td>
</tr>
<tr>
<td>100 mg tablets</td>
<td></td>
<td>Variable efficacy, may cause restlessness or GI upset</td>
</tr>
<tr>
<td>Zafirlukast (Accolate®)</td>
<td>5 mg/cat PO q 12 to 24 hours</td>
<td>Leukotriene-receptor antagonist</td>
</tr>
<tr>
<td>20 mg tablets</td>
<td></td>
<td>Promising, may be additive with Beta-2 agonists</td>
</tr>
<tr>
<td>Cyproheptadine (Periactin®)</td>
<td>1-2 mg/cat PO q 12 to 24 hours</td>
<td>Serotonin antagonist</td>
</tr>
<tr>
<td>4 mg tablets</td>
<td></td>
<td>Variable efficacy, not first choice</td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>10 mg/kg PO q 12 hours</td>
<td>Immunosuppressive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe, chronic life-threatening asthma Rarely used clinically, Monitoring</td>
</tr>
</tbody>
</table>
The following table lists common drugs used by aerosol therapy. Doses are largely empirical and should be adjusted as clinically warranted.

<table>
<thead>
<tr>
<th>Drug</th>
<th>MOA</th>
<th>Dose</th>
<th>Indications</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol</td>
<td>Beta-2 agonists</td>
<td>1 puff/5 kg prn</td>
<td>Bronchoconstriction</td>
<td>Tachycardia, Restlessness</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Asthma</td>
<td></td>
</tr>
<tr>
<td>Flovent 110 mcg</td>
<td>Glucocorticoid</td>
<td>1 - 2 puffs/cat twice a day</td>
<td>Asthma, Allergic disease, Chronic bronchitis</td>
<td>Infection (Candidia), Expense</td>
</tr>
</tbody>
</table>

The prognosis is usually good, although some cats have recurrent bouts and require frequent medications.
Pulmonary vascular disease
Pulmonary vascular disease or pulmonary vascular obstructive disease (PVOD) is a catch-all term for conditions that affect the pulmonary circulation. These conditions are of particular importance as they may result in severe respiratory dysfunction. As a review, the pulmonary and systemic circulation are in series, with the cardiac output of the left and right heart (in the absence of cardiac shunt) being equal. However the pressure in the pulmonary vasculature is low in relation to the systemic circulation, with the mean pulmonary arterial pressure averaging less than 15 mmHg and a systolic pressure of < 25 mmHg at rest and less the 30 mmHg during exercise. Elevated pulmonary pressures result in syncope, weakness and fatigue, and may ultimately result in right heart failure. Knowledge of normal intra-cardiac pressure is essential to recognize the effects of disease.

NORMAL CARDIAC AND VASCULAR PRESSURES

Strain on the right heart is assessed by measurement of the pulmonary vascular resistance (PVR), which is defined as the [mean pulmonary arterial pressure - mean left atrial pressure (or pulmonary capillary wedge pressure)]/cardiac output. Increasing PVR is associated with increased work for the right ventricle. If R heart pressures exceed left
heart pressure in the presence of an intra-cardiac shunt, there will be right to left shunting.

In simplicity, two things can go wrong with the pulmonary vasculature that will affect the pressures. The first is a clot or clots may lodge in the pulmonary vasculature, while the second is that there is vasoconstriction or hypertrophy of the pulmonary arteries.

**Pulmonary thromboembolism (PTE)** is a more clinical recognized cause of pulmonary dysfunction in critically ill dogs and to a much lesser extent, the critically ill cats. As an overview, in order for a thrombus to form, there should be disruption in one or more aspect of the Virchow’s triad which includes vascular damage, blood stasis and hypercoagulability. A thrombus is defined as a blood clot that forms in situ, while an embolus is a clot that lodges somewhere after first forming in another site. It is often unclear as to the initial source of the clot; thus, the term thromboembolism is used. In human medicine it is widely appreciated that thrombi form in the deep veins, include the iliacs and occasionally the leg veins. It is, by far, less clear where they come from in dogs and cats. It seems possible that they are forming in veins, however; despite the advances in diagnostic imaging, it is rare that one is visualized.

A number of risk factors have been associated with PTE in dogs, but as a general rule, critical illness and trauma are common sources.

Diagnosis of PTE is typically based upon an acute onset of respiratory distress in a patient at risk. Other differential diagnoses include aspiration pneumonia, acute respiratory distress syndrome (ARDS), and volume overload. Thoracic radiographs may document oligemia. Angiography is considered the goal standard, but is uncommonly performed. CT and echocardiography are particularly useful for identifying these conditions, with echo able to document right ventricular failure from PTE (eg. Massive PTE) and CT being useful to visualize the thrombi in pulmonary arteries.
Evolving questions in veterinary medicine include questioning the need for and the type of prophylaxis. There have been no well-designed studies to try to answer these questions. However, it is my opinion that in all critically ill dogs, prevention should be at least considered. Methods to prevent thrombi include heparin (Unfractionated versus low-molecular weight) and aspirin or clopidogrel (Plavix).

Pulmonary hypertension is a global term for anything that results in increases in the pulmonary arterial pressures. PTE may result in pulmonary hypertension, but for this discussion we will consider the disease separately. Specific diseases that may result in include primary pulmonary hypertension, which is pulmonary hypertension without an underlying cause identified. It is thought to be most common in small breed elderly female dogs. Clinical signs include shortness of breath, exercise intolerance, syncope and ascites. Cor Pulmonale is defined as the developed of right sided heart failure due to pulmonary hypertension due to lung disease. Common lung diseases that are associated with cor pulmonale include chronic bronchitis/"COPD" and pulmonary fibrosis. Eisenmeger syndrome (or physiology) is a specific form of pulmonary vascular disease associated with an intracardiac shunt, often a VSD or large ASD. In this case, the initial shunting is left to right, but over time, the volume overload to the pulmonary vasculature hypertrophies, and as the pressure increases above that in the LV, right to left shunting ensures. This is a relatively common complication of large left to right shunting defects. Dogs and cats will tend to present at 1-2 year or older with signs more often attributable to neurological sequelae of either hyperviscosity due to polycythemia or stroke. Two relatively rare conditions that may exist in animals are portopulmonary hypertension and hepatopulmonary syndrome; these are considered the major pulmonary vascular
complication of hepatic disease. Other sources of respiratory distress, including large volume ascites or pleural effusion, pneumonia, congestive heart failure or chronic obstructive pulmonary disease (COPD) are also common in people with hepatic disease.

Portopulmonary hypertension is characterized by portal hypertension in association with pulmonary hypertension (mean pulmonary arterial pressure > 25 mmHg at rest or > 30 mmHg during exercise). An estimated 2-10% of people with cirrhosis will have PPHTN. The mechanism for the development of pulmonary hypertension seems to be independent of portal hypertension although portal hypertension not liver disease per se is required for the development of PPHTN. Hypotheses include high cardiac output resulting in shear stress on the pulmonary vasculature, which may result in the proliferation of endothelial and smooth muscle cells and subsequent pulmonary arterial hypertension. High cardiac output is associated with portal hypertension and liver disease due in part to splanchnic volume overload and bowel-wall congestion which subsequently causes the release of endotoxin and cytokines.

Most individuals with PPHTN have only mild to moderate changes, but in some individuals, disease may progress to severe pulmonary hypertension. Genetic components are thought to perhaps explain why despite similar hemodynamic changes, only a small percentage of patients develop PPHTN. Interestingly PPHTN does not improve following liver transplantation and in fact severe PPHTH is a contraindication to transplantation. Other treatments are directed toward standard management of hepatic dysfunction and pulmonary hypertension, including intravenous infusion of epoprostenol or orally active pharmaceuticals such as bosentan or sildenafil.

Hepatopulmonary syndrome is the other clinical pulmonary vascular anomaly associated with liver disease. In human medicine, HPS is defined by the triad of 1) hepatic dysfunction 2) arterial hypoxemia and 3) evidence of intrapulmonary shunt. Intrapulmonary shunt, as seen in the dog of this report, is most commonly identified by saline bubble contrast documenting spontaneous contrast in the left side of the heart 2-4 seconds after appearance in the right heart. This delay in contrast is reflective of the intrapulmonary shunting of the blood, as opposed to the immediate appearance of bubbles supported intra-cardiac shunting. Arterial hypoxemia typically identified by arterial blood gas analysis include demonstration of an increased A-a gradient although in pediatric patients, pulse oximetry may be employed as a secondary option. Hypoxemia is often more pronounced in a standing position and improves when a patient is recumbent. The incidence of PPHTN and HPS in veterinary medicine remains to be determined.

**Evaluating pulmonary vascular dysfunction**

A variety of tests can provide usually information regarding the pulmonary vasculature. However, the most practical test to use is the echocardiogram since the most important question to answer is: *Is the right heart functionally well?* In animals with significant pulmonary vascular disease of any sort, subjective and objective echocardiograph data is useful. Subjective information includes recognition that as the normal LV pressure is much higher than the normal RV pressure, there is septal flattening; this will suggest that there is increased RV pressures, and limitation of volume delivery to the left heart. The
right heart may look hypertrophied. It is important to exclude pulmonary stenosis as a cause of right heart changes prior to incriminating the pulmonary vasculature, but this is typically easily done. If available, and if there is a tricuspid regurgitant jet, Doppler may be use to estimate systolic pulmonary artery pressures using the Bernoulli equation, with the estimated pressures $= 4 \times \text{velocity}^2$

Other testing that might be warranted is the cardiac catheterization, this is considered the gold standard for evaluation of pulmonary pressures, but its use requires advanced training and specialized equipment. Chest radiographs are advisable in all cases of suspected cardiac or pulmonary dysfunction; however the findings may be non-specific. Other advanced imaging, including CT thoracic scanning may be advised for lung disease, and may be coupled with angiography. In animals where disease is less defined, post-mortem biopsy may be helpful in establishing etiology and helping future patients.

**Treatment of pulmonary vascular disease**

As with all diseases, treatment of the underlying condition is most likely to result in clear benefit. However, provided that cure is not a possibility, options include anticoagulants, and cardiac/pulmonary medications.

Anticoagulation in the short term is often most easily accomplished with intravenous heparin as a CRI; however, LMWH (eg. fragmin or lovenox) may be equally efficacious. Long-term common anticoagulants include anti-platelet drugs such as aspirin (0.2-5 mg/kg per day-) or clopidogrel (less clear dosing) are popular. Unfractionated heparin is not practical for long term use at home, although the LMWH are good (but pricy). Coumadin is a popular anticoagulant in people, but relies on frequent monitoring and may be quite challenging in dogs.

- **Key Point** - strongly consider anticoagulant in all dogs with pulmonary vascular disease, not just those with historical PTE. This is important because blood flow (stasis component of the Virchow’s triad) is affected by pulmonary hypertension and even small PTE may be synergistic with pulmonary hypertension.
- Cardiopulmonary medications that might be useful include supplemental oxygen (which is a potent vasodilator), sildenafil and pimobendan.
Pleural effusion
Identification of pleural space disease in a patient represents a major diagnostic and therapeutic target for the clinician. Pleural space disease may consist of an effusion, pneumothorax, or a space-occupying mass (eg. Contents of a diaphragmatic hernia). Pleural effusion develops when there is an altered balance between the normal production and drainage of pleural effusion. In the normal individual, a few milliliters of pleural effusion coat the visceral and parietal pleura, and serve to lubricate the serosal surface and permit normal respiration. Pneumothorax describes the presence of air within the pleural cavity, and may be traumatic, spontaneous or iatrogenic in origin.

Pleural effusion may be suspected based upon physical examination, and confirmed via thoracic radiographs or ultrasonography. Computed tomography is also a useful method of identifying pleural effusion, but rarely the first diagnostic step. Thoracocentesis is useful for both as a diagnostic and therapeutic procedure. In some cases, thoracocentesis may be performed prior to documentation of pleural effusion by diagnostic imaging, particularly in cases of severe distress. In many cases, if ultrasonography is not readily available, a single DV radiograph, even improperly positioned, can highlight the presence or absence of effusion. Properly positioned thoracic radiographs may be performed following removal of effusion.

Tips for successful thoracocentesis
1) Determine if you anticipate a diagnostic or therapeutic centesis. In almost all cases, as much effusion as possible should be removed, which is often in contrast to abdominocentesis.
2) In a young dog with pleural effusion, evaluate the clotting time PRIOR to tapping the chest. Anticoagulant rodenticide toxicity is a common cause of pleural effusion (usually with a widen mediastinum as well) and thoracocentesis should be either entirely avoided in this situation or postponed until after correction of the coagulopathy.
3) Based upon imaging results, anticipate the volume of effusion that is present. An average sized cat may have 200-300 ml of effusion (30-50 ml/kg) while a retriever-sized dog may easily have 2-3 liters of effusion. This step is important, as removing 50 ml from a Labrador may help your diagnostic efforts, it would not be expected to therapeutic. Additionally, if you remove a greater volume that anticipated, that may suggest either on-going leak (in cases of pneumothorax) or inadvertent drainage of the abdomen (in cases of bi- or tri-cavitary effusion).
4) Prepare supplies and determine need for sedation, for dogs with large volume effusions, sedation with either butorphanol and diazepam or propofol [recall this is a potent cardiovascular depressant] is often beneficial for patient comfort and for decreasing the time required. For example, removing 3 liters of effusion is about 5-10X faster with a 16 ga catheter than with a 22 ga needle. For most cats, a butterfly catheter is adequate, but in very large cats a longer needle may required.
5) Recall that for animals with chronic effusions, there is an increased risk of iatrogenic pneumothorax so taps with a catheter fed into the chest may be less traumatic than needles.
6) Dogs and cats have fenestrated mediastinums, so the specific side that the effusion is removed from is less important than often given credit for.
7) Save adequate volumes of the effusion for analysis and record the total volume removed.
8) If desired, perform post-tap radiographs or other imaging.
9) Monitor patient’s recovery, in most cases marked improvement in respiratory rate and effort is evident immediately. Thus if respiratory distress persists or worsens, it is concerning that either iatrogenic pneumothorax has developed, or that there is underlying parenchymal or airway disease. Following removal of a large volume of effusion, a dog may cough for the next 12-24 hours as previously atelectatic lung re-inflates.
10) Do not delay performing centesis for pets that are short of breath; this is an urgent/emergent procedure.

Disease processes associated with pleural effusion; Guidelines for recognition and management

Pleural effusions typically develop because of increased hydrostatic pressure, decreased oncotic pressure, lymphatic leakage or increased vascular permeability. In some cases, a combination of two or more causes may co-exist. Often, evaluation of the patient’s signalment, history and physical examination can directed the diagnostic efforts. Additionally, evaluation of the effusion itself, both grossly and microscopically may provide important clues as to the source of the effusion. The most common effusions are discussed below.

MODIFIED TRANSUDATES:
Cardiac disease: Right-sided cardiac disease and pericardial effusion are two common causes of pleural effusion in small animals

*Cardiac disease,* specifically cardiomyopathy is a particularly common cause of effusion in cats. Other physical examination findings that are supportive of cardiomyopathy include a murmur and/or gallop and a low rectal temperature. Cats with pleural effusion associated with cardiomyopathy tend to be older, and the clinician should recall that a cat that has previously had effusion, a recurrence of shortness of breath is often (always?) associated with recurrent effusion. In dogs, pleural effusion is seen in end-stage valvular disease and cardiomyopathy. Occasionally, isolated right-sided defects may also result in effusion. Echocardiography is the diagnostic test of choice to evaluate cardiac structure and function. Analysis of BNP is emerging as a patient-side test for evaluation of atrial stretch. Fluid analysis is a modified transudate and/or occasionally chylous. Treatment of cardiac disease induced pleural effusion involves treatment for the underlying disease, draining as much effusion as possible, and increasing the furosemide dose and/or adding another diuretic such as spironolactone or spironolactone-hydrochlorothiazide.

Pericardial effusion is another common cause of pleural effusion. Physical examination findings consistent with pericardial effusion include muffled heart sounds, jugular venous distention, and pulsus paradoxus. Echocardiography is diagnostic. The pleural effusion is a modified transudate. Pericardiocentesis will result in rapid re-absorption of the pleural effusion (by removing the right-sided heart failure component). Treatment of pericardial
effusion reflects the underlying cause of the pericardial disease and may include pericardiectomy.

**Malignant pleural effusion**: Malignant pleural effusion (MPE) is another cause of a modified transudate. Neoplasia may be divided into lymphoma and non-lymphoma in regards to treatment options. *Lymphoma* commonly results in MPE and an associated anterior (cranial) mediastinal mass. In cats, the cranial aspect of the chest is usually uncompressible and in dogs, hypercalcemia is common (most are T-cell lymphoma). Treatment of MPE associated with lymphoma involves standard chemotherapeutic protocols, and is usually associated with a good short term outcome.

*Non-lymphoma neoplasia* may also result in pleural effusion. Thymoma is common in older dogs and cats. Metastatic neoplasias (carcinomas) are also possible, such as with primary lung tumors, mammary tumor or other carcinomas. Treatment of a suspected metastatic effusion usually shifts to palliative care, and may include periodic centesis, and/or infusion of local chemotherapy, such as carboplatin. In moderately effusive states, a reasonable quality of life main be maintained for many months. Recently, the concept of a pleural-port has been introduced and to date met with fair to good reviews.

**EXUDATES**

**Chylothorax**: Chylous effusion may also have properties of a modified transudate. Chylous effusion is rich in triglycerides and tends to look milky or pinkish (*strawberry milkshake*). Chylous effusion may be either primary [idiopathic] or secondary to a variety of conditions such as congestive heart failure, heartworm infestation, or an anterior mediastinal mass. Appropriate work-up involves evaluation for an underlying cause, and specific therapy if possible. If not possible, then therapy may be surgical (thoracic duct ligation, ± pericardiectomy and omentalization). Surgical therapy has a success rate of 50-70%, so it is essential that a "cure" is not promised. Long-standing effusion may result in restrictive pleuritis, with on-going respiratory difficulties.

**Pyothorax**: Pyothorax represents a collection of infected material within the thorax. Most cases are thought to be due to penetrating wounds (including bite wounds) or foreign bodies. In dogs, pyothorax is considered a surgical disease, while in cats, medical management is often adequate. Infection is common anaerobic, and broad-spectrum antibiotics are warranted. For animals treated medically, pleural lavage is occasionally employed, using 10-30 ml/kg of warm heparinized (10 iu/ml) saline infused and withdrawn every 4-8 hours.

**Hemothorax**: Hemothorax is most commonly associated with trauma or anticoagulant rodenticide intoxication; however in these cases thoracocentesis may not be performed. In cases where hemothorax is detected, the most common cause by far is neoplasia (eg. rib tumor), although other causes such as lung lobe torsion or pancreatitis may also be responsible.
PNEUMOTHORAX
Pneumothorax may be classified as traumatic, spontaneous or iatrogenic.

**Traumatic** pneumothorax is the most common and usually easily recognized due to other signs of trauma. Some animals have concurrent pulmonary contusions, or other thoracic trauma. Animals presenting emergently with respiratory distress following trauma should under urgent thoracocentesis. Auscultation may document decreased lung sounds, but this may be masked by correspondingly louder sounds from pulmonary contusion. 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.

**Spontaneous** pneumothorax are defined as pneumothorax that develops without evidence of trauma. Historically, dogs will present with signs of either shortness of breath or difficulty settling down. Northern breeds are predisposed. Causes include bulla and blebs, as well as neoplasia or foreign bodies (quills). Cats with lower airway disease may also develop spontaneous pneumothorax, as may patients with heartworm disease. Primary spontaneous pneumothorax associated with bulla or bleb is considered a surgical disease, even if the air leak spontaneously stops, it is still likely to recur without surgical resection.

**Iatrogenic** pneumothorax is a pneumothorax created by the veterinary professional. These are most commonly associated with thoracocentesis in a patient with abnormal pleura. The normal pleura will heal rapidly if inadvertently punctured, however; if the diseased pleura is punctured, the normal rapid sealing will not occur. This is a big risk in particular with chylous effusions, or any exudative process.

**OTHER PLEURAL SPACE CONDITIONS**
Other conditions may affect the pleural space; these include most commonly diaphragmatic hernia, thoracic bite wounds and a variety of neoplasia. Diaphragmatic hernias (DH) are usually traumatic in origin, although congenital disease has been described. DH should be surgically repaired as soon as the patient is stable. Older literature initially suggested that a delay of 24 hours was associated with a better outcome, but later studies have suggested that early repair is preferable. Chronic DH is associated with a high peri and post-operative complication rate, including the development of re-expansion pulmonary edema (Non-cardiogenic/high protein) and/or pneumothorax.

**Thoracic bite wounds** commonly penetrate the chest, even if the puncture site seems small. Thus, all thoracic wounds should be cleaned and completely explored, and the surgeon prepared to enter the chest as needed for further repair. Broad-spectrum antibiotics are warranted. Chest wall and rib neoplasia may also present with signs of pleural effusion. Advanced imaging is often required to highlight the lesion and to
determine if a surgical option exists. CT scanning is evolving as an invaluable tool in surgical and oncological planning.

Figure 1 CT images of an 18-month old Labrador, who presented for evaluation of shortness of breath and a palpable mass. The biopsy of the mass confirmed osteosarcoma, and the CT highlighted the mass and also evidence of metastasis (seen at the periphery of the lung in the right hand image at the top left lung), which altered the treatment plan.

**Sampling for cytology and culture**
Definitive diagnosis of pulmonary disease remains elusive at times. Cytological or histopathological samples are useful to help better classify the underlying cause as well as determine both prognosis and treatment course. Thus, it is prudent for the criticalist to have a strong grasp of the various techniques and options available for sampling. Additionally, as many patients with cardiopulmonary disease are relatively unstable, it is wise to recognize the potential risk and benefits of the testing.

The goal of the presentation is to describe the techniques as well as the potential benefits and risks of available methods to assess lung pathology. Available techniques include oral examination with biopsy, bronchoscopy, bronchoalveolar lavage, transtracheal aspirate, transoral tracheal aspirate, fine needle lung aspirate (with or without ultrasound guidance), and thoracocentesis with evaluation of cytological characteristics of the pleural effusion.

As an overview, pulmonary disease is classically localized to upper airway, tracheal/bronchial (lower airway), pulmonary parenchyma and pleural space. Differential diagnoses will vary based upon patient signalment and history. Some patients may require extensive evaluations, while others do not require further evaluation short of a complete history and physical examination. For example, a young dog with pulmonary infiltrates in a pattern consistent with pulmonary contusion after having been hit by a car requires no further diagnostic tests. Conversely, an older dog with a recurrent bout of cough and pulmonary infiltrate may require extensive testing.
Larynx
Common laryngeal diseases include laryngitis (from excessive barking or tracheobronchitis) or laryngeal masses. Laryngeal masses are more common in older patients, particularly cats. Clinical signs of laryngeal masses include slowly progressive loud or stridorous breathing. Some animals have an apparent response associated with prior therapy associated with antibiotics or glucocorticoids. Neoplasia, specifically squamous cell carcinoma is the most common. A granulomatous proliferative (albeit non-neoplastic) condition has also been described in cats.

Biopsy samples are required to definitively identify neoplasia as well as to provide major prognostic information. Two points are of specific importance when considering a laryngeal biopsy. The first point is that all clinically significant obstruction laryngeal disease is not a quick fix. This is important from the perspective of the client as in contrast with some other disease processes, the presence of laryngeal mass carries a very guarded prognosis. The second major point is that due to the often gradual progression of infiltrative disease, there is often a very tiny remaining airway lumen at the time of oral examination. Any loss of active airway dilating activity may result in complete airway occlusion. Thus, the clinician should be prepared for an emergency tracheostomy with readily available support staff and supplies. Additionally, even in the airway is likely considered adequate to recover the pet from sedation; airway swelling subsequent to a biopsy may result in occlusion. Discussion of a temporary tracheostomy tube should be cleared with the pet's family prior to undertaking an oral examination.

A laryngeal biopsy may be obtained with endoscopy biopsy instruments, long-handled scissors (Metzenbaums) or a biopsy cup forcep. My preference is to use a large cup forcep with the goal to also debulk a major portion of the mass.

Lower airway disease
Common lower airway diseases include feline lower airway disease (f̄sthma), eosinophilic pneumonitis and chronic bronchitis. Cytological examination with bacterial culture and sensitivity testing is warranted in most cases. Options for collection of cytological samples include transoral tracheal wash, transtracheal wash (aspirate), and bronchoscopy with bronchoalveolar lavage. Bronchoscopy is considered the gold standard for evaluation of the lower airways and collection of cytological samples. However, due to financial constraints or concerns regarding anesthesia, other options are often pursued.

A transoral approach for collection of samples is warranted in all cats and small dogs. Many clinicians prefer to perform a transoral approach in all sized pets. Sample collection should not be pursued if subsequent sedation is considered risky (eg. Inadequate supplies/training/support staff or marked respiratory distress in the patient). Necessary supplies include supplemental oxygen, propofol, a laryngoscope, sterile endotracheal tube, sterile specimen cup, and sterile long red rubber urinary catheter (5 or 8 Fr) and sterile 3 aliquots of saline (3-5 ml for small pet, 5-10 for medium dog, and 10-15 for large dog). Saline is used to avoid cell lysis. The patient should be pre-oxygenated for 2-5 minutes and then anesthesia induced with propofol IV to effect. The sterile endotracheal
tube should be placed. Do NOT use any lubricant on the tube, although it is acceptable to use a smaller tube to facilitate intubation. The cuff may be gently inflated if desired and supplemental flow-by oxygen may be administered. Allow the pet to wake up a bit, so that a cough reflex is restored. I will often take initial advantage of the sedation in order to perform an oral examination. Monitoring with a pulse ox and EKG is wise. Pass the catheter through the endotracheal tube and then infuse the saline. Simultaneously, aspirate back on the syringe and move the head position to allow fluid to drain (expectorate) into the sterile cup. The collected fluid should be placed into an EDTA tube as well as a red top or culturette. Some clinicians like to use a self-contained suction system to actively aspirate samples. However, my anxiety in a teaching hospital is that it creates the impression that elaborate supplies are needed to perform a tracheal wash and thus may frighten students from performing this procedure in the future.

Fluid retrieved should be evaluated cytologically following a concentration technique (eg. Cytospin) and also cultured for aerobic bacteria. There is some evidence that Mycoplasma spp play a role in respiratory diseases of cats and dogs. Thus as indicated clinically, culture or PCR for these organisms would be warranted.

Transtracheal aspirate (TTA) was particularly popular in the days before the ready availability of propofol. TTA are somewhat technically challenging, particularly in big or uncooperative dogs. Supplies required to performed a TTA include sterile gloves, prep solution, local anesthesia, a through the needle catheter (eg. Intracath) and aliquots of saline. A TTA is performed by clipping and prepping the site over the trachea or cricothyroid membrane (larynx). Either is fine, although in larger dogs the lower you go in the airways the better samples you get. The site of intended puncture is identified and infiltrated with local anesthesia. The catheter is inserted into the airway and advanced distally. Coughing should ensue. If the catheter is adequately placed, air should be easily aspirated back though the catheter. The saline aliquots are similarly infused though the catheter and retrieved. Subcutaneous emphysema is a potential complication.

Bronchoscopy with collection of samples is the best way to evaluate the lower airways. Like any technique, practice and experience will improve your assessment and diagnostic yield (ie- do lots of bronchoscopy!). In contrast with GI endoscopy, at times the airways may be confusing. McKiernan and Amis developed the classic “road map” of the canine airways. This should be posted at a readily accessible area near the bronchoscopy suite.

**Pulmonary parenchyma/interstitium**

The pulmonary parenchymal changes may include either alveolar or interstitial changes. Alveolar changes are most commonly either edema or pneumonia. Cardiogenic pulmonary edema is typically easy to recognize as cardiomegaly, murmur and left atrial enlargement are common. Sampling of pulmonary edema per se rarely is required. However, if an animal is intubated for support of respiratory failure and edema is present, it is appropriate to collect a sample for evaluation of cytology and protein content. Cardiogenic pulmonary edema (due to increased hydrostatic pressure) will have a very low total protein in relation to serum protein (Usually < 0.4) while non-cardiogenic pulmonary edema (due to vascular permeability) will have a protein content that
approximates serum protein. Samples from animals with suspected pneumonia should be collected via either a tracheal wash or bronchoscopy.

For animals with primarily interstitial lung disease, it is prudent to recognize that a tracheal wash will be fairly low yield (common "mild inflammation" no growth on culture). The best options for sampling the lung interstitium are either a fine needle aspirate or preferentially a lung biopsy (either via open lung biopsy or via thoracoscopic examination). A fine needle lung aspirate may be performed either blindly or with ultrasound (or CT guidance). FNA is commonly very helpful. In animals with diffuse disease, pneumothorax is a real possibility following aspiration, so pets should be carefully observed for at least several hours following the procedure.
Lung masses
Lung masses are an interesting quandary—many carcinomas exfoliate well so you might expect a good diagnostic yield. However, there are times that aspirate will document inflammation, and not neoplasia. Surgical resection of pulmonary masses is advised; so it is often unclear if pre-operative sampling will clearly change outcome. For example, aspirate of this mass documented inflammation with no neoplastic cells, but histopathology confirmed adenocarcinoma.

Pleural effusion
Pleural effusion is frequently sampled for both diagnostic and therapeutic ends. Cytological examination remains an essential aspect of evaluation of the pet with pleural effusion. Markers in pleural effusion are commonly evaluated in people but have rarely been looked at in animals. It is wise to recall that with malignant pleural effusions, neoplastic cells may be relatively hard to identify and large quantities of fluid may need to be evaluated to confirm the diagnosis.
EFFECTIVE OXYGEN SUPPORT – OVERVIEW

Supplemental oxygen is beneficial in a variety of situations, including hypoxemia, head trauma and critical illness. There are multiple methods of providing supplemental oxygen, with various advantages and disadvantages. It is prudent for the clinician to be prepared to provide supplemental oxygen via several routes and to be able to recognize the pros and cons of the different methods.

Options for providing supplemental oxygen include face mask or flow by, oxygen hood, nasal oxygen, transtracheal or nasolaryngeal oxygen, oxygen cage and mechanical ventilation. Patient factors to consider include: species, nasofacial confirmation (e.g. brachycephalic), degree of compromise, breathing pattern, complexity of patient care treatment and other possible causes of respiratory distress. Oxygen is most often available in tanks, although in larger facilities there may be a central source of oxygen. Oxygen tanks come in a number of sizes, but most commonly the smaller E tank which contains about 660 liters of oxygen and the larger tanks (H) which contain 6900 liters. Busy practices should ensure that they have enough oxygen in stock to get through a busy weekend. Practices with a central supply should be sure to have in-house several back-up canisters in case of oxygen system malfunction. An oxygen tank, which requires a large amount of oxygen to fill and maintain a cage, may go through an H tank every 24 hours. For oxygen administered by other routes, most often, oxygen flow rates are set at about 50-100ml/min, thus for a 30 kg (66 pound) Labrador, this translates to 3000-4000 Liters/day. When oxygen is going to be administered for
more than a few minutes, humidification is warranted to prevent the drying effects of forced air.

The percentage inspired oxygen contact obtained is dependant upon both flow rate and the respiratory rate and strategy of the patient. For example, oxygen sources typically are set at 50-100ml/min, while inspiratory flow rate are often in the order several hundred ml/SECOND. Thus, if you provide the above 30 kg dog with 2.5 liters/minute of supplemental oxygen, you are providing him with ~40 ml/minute of 100% oxygen. If his inspiratory flow rate is an average of 300 ml/second, you are providing 40 ml (100% oxygen) and 260 ml (21% oxygen) for a sum total of any FiO2 of 32%. [40 ml of 100% oxygen, plus 55 ml (0.21 X 260 ml) = 95ml of 100% oxygen within 300 ml/sec =32% oxygen}

Conversely, if the patient is panting, with a flow rate of 800 ml/sec, you will have 40 ml 100% oxygen plus 760 ml of 0.21% oxygen (160 ml), so 200 ml of oxygen in 800 ml of air flow/sec for a FiO2 of 25%. Panting dogs in particular may have a very hard time benefiting from nasal oxygen, as it very hard to get supplemental oxygen flow rates high enough to be beneficial.

**Face mask:** In very compromised pets or those recovering from anesthesia, it is simple and effective to provide oxygen by a face mask. Dogs and cats will typically not tolerate a face mask if they are not very sedate or ill. It is important to counsel support staff and new clinicians not to fight with the patient to hold the face mask, as this dramatically increases energy expenditure on the part of the patient.

*Supplies: Face mask, tubing, oxygen $*

**Flow by oxygen:** Occasionally, in order to avoid the struggles associated with face mask placement, individuals will hold oxygen tubing near a patient’s face with the hopes of increasing the FiO2. The likelihood of significant improvement is slight, and if oxygen is truly needed, a different route is preferred.

*Supplies: Tubing, oxygen $*

**Nasal oxygen:** A red rubber or other soft plastic tubing may be placed with the nasal passages to permit the delivery of supplemental humidified oxygen. Animals in extreme distress will often not tolerate the restraint required to suture (staple) in nasal lines. Nasal oxygen is relatively contraindicated in brachycephalic pets, pets with excessive panting, and may be more challenging in cats or very small dogs. The oxygen flow rate will reflect the diameter (French) of the tubing, thus with very small pets, with only a 3.5 or 5 Fr catheter being tolerated, the relative flow rate may be quite low.

**Procedure:** Select nasal catheter- Choose the largest size that will be tolerated by the patient- cats 3.5-5 Fr, Small Dogs 5-8 Fr. Medium dog 10-12 Fr, Large dogs 10-14 fr. Instill local anesthesia into nostril, use either lidocaine or proparacaine. Pre-measure the catheter to the medial canthus of the eye. Put a piece of white tape at the desired location. Pre-load the needle driver with suture (or use a stapler). Insert the tube, and promptly secure in place. Consider placement of an e-collar to prevent dislodgement of the tube. Place bilaterally if desired. Connect to humidified oxygen source.
Supplies: Nasal catheter, tubing, suture, scissors, oxygen, local anesthetic $

**Oxygen hood:** An oxygen hood may be either commercially purchased (eg. Jorgenson Laboratories, others) or made with cellophane wrap and an e-collar. The collar is secured around the patient’s neck and oxygen is introduced into the hood. The hood is vented to prevent carbon dioxide retention.  
*Supplies: Hood, oxygen and tubing $-$$

**Transtracheal oxygen:** Oxygen may be delivered trans-tracheally via a tracheostomy tube or with a catheter placed directly into the trachea. This technique does not work well in very small pets, as it is very challenging to safely pass a catheter into the lumen of the trachea without damaging the tissues.  
*Procedure:* The neck is sterilely prepped. If the patient is not obtunded, a local block may be performed. A through the needle catheter (eg. Intracath®) is passed into the trachea to the thoracic inlet and supplemental oxygen is provided at a lower flow rate (10-50 ml/kg). The catheter should be bandaged in place. Successful placement in the trachea should be confirmed (free aspiration of air, similar to a transtracheal wash).  
*Supplies: Through the needle catheter, oxygen, tubing, bandage materials, prep supplies $-$$.

**Nasotracheal oxygen:** Oxygen may also be delivered directly in to the trachea by placement of a nasotracheal oxygen catheter. This technique is particularly helpful to bypass the pharynx and/or larynx.  
*Procedure:* This technique may be performed two ways. The first way is to simply pass nasal oxygen tubing farther in through the pharynx and attempt to advance further during inspiration. This may take a few attempts, as the tendency of the tube is to go down the esophagus. The second, which is my preference, is to place under sedation to evaluate the upper airway obstruction. In this scenario, the obstruction is first classified (eg. dynamic, fixed) and then if not immediately treatable, the nasal catheter is fed into the back of the nasopharynx and then grabbed with hemostats and directed into the trachea. A bit of dexterity may be required.  
*Supplies: Red rubber catheter, suture, tubing, oxygen, ± laryngoscope and sedation. $-$$

**Oxygen cage:** Supplemental oxygen may also be delivered by placing the pet within an oxygen-enriched chamber or cage. This technique is particularly helpful for animals that are too stressed to undergo any other form of supplemental oxygen (eg. Cats, small dogs) as it is very well tolerated. The disadvantages of an oxygen cage include that it is a relatively inefficient use of oxygen and, when the cage is opened, the oxygen concentration will re-equilibrate quickly with the room air.  
*Supplies- oxygen cage, source of oxygen $$$$-$$$$$

**Mechanical ventilator:** Positive pressure ventilation (PPV) with a mechanical ventilator provides the most control over the percentage oxygen, and the breathing pattern of the patient. In animals that are expected to be hypoventilating for more than a few minutes (eg. Some intoxications, neurological disease) or those with respiratory failure (PaO2 < 50 mmHg on supplemental oxygen), PPV may be life-saving. The decision to provide
PPV is beyond the scope of this brief discussion, but should be considered warranted in some cases. Practitioners associated with busy ER or Critical care services typically find PPV invaluable in caring for critically ill or injured pets. 

*Supplies* - *mechanical ventilator, adequate technical and professional staff to monitor pet.*

**Diagnostic imaging**

Thoracic disease represents a large burden in medicine; imaging is often vital to establish the cause of the disease, as well as to help judge respond to treatment and the extent of the disease. There is a multitude of imaging modalities; the objective of this hour’s talk is to review the merits and indications/caveats for each.

**Keys**

**Availability:**

- # universal
- ## fairly easy
- ### harder to come by
- #### difficult at times

**Cost:**

- $ <200
- $$ 200-500
- $$$ 500-1000
- $$$$ >1000

(Including anesthesia if needed; TCSVM costs US Dollars May 2009)
Thoracic radiographs #, $
Survey radiographs are readily available in all veterinary practices. They represent the first step in the evaluation of >95% of cats with signs of respiratory disease. Anesthesia is not required, and sedation is rarely needed. The main exceptions are cats with previous (and likely recurrent) pleural space disease, and those with evidence of upper airway obstruction. Radiographs are not therapeutic, and in cases of severe distress they should be postponed until the cat stabilizes. Radiographs document only 5 shades of grey; these include air, fat, fluid, bone and metal.

Radiograph interpretation is discussed in detail in another session but should certainly be considered the major imaging modality for patients with respiratory disease.

Figure 1 - Lateral thoracic films from a cat with respiratory distress. There is pleural effusion and suspicion of a mediastinal mass.
Ultrasonography ##, $$

Thoracic ultrasonography is ideal to better evaluate soft-tissue masses within the thoracic or on the thoracic wall. Mass evaluation may be coupled with fine needle aspiration (if major vessels may be avoided). TUS is well-tolerated, and can typically be performed with the cat manually restrained in lateral or DV recumbency. Residual effusion may help improve the imaging yield. Disadvantages of ultrasound include the relative inability to view within lung (due to air) and the inability to view the entire cavity in real-time.

TUS is indicated when a mass is suspected in the mediastinum or at the periphery of the lung. TUS is not indicated if the mass is centrally located, or in the presence of pneumothorax.

Figure 2 - Thoracic ultrasound image from the same cat as above; clearly delineating the suspected mass.
Echocardiography #, $$
Echocardiography is warranted when there is suspicion of cardiac disease. Cats, due to their propensity to have hypertrophic cardiomyopathy may have significant heart disease without radiographic cardiomegaly. Additionally, in contrast to dogs, a soft murmur may be associated with moderate to severe disease. Screening for cats with cardiac disease may be at some point replaced with biomarker testing, but for the time being, Echo remains the test of choice for suspected cardiac disease, with particular attention being paid to the existent of left atrial enlargement.

Figure 3 - A short-axis view demonstrating a marked left atrial enlargement.
Computed tomography $$$, $$$
CT is growing rapidly in its role of investigation of pulmonary disorders. As CT scanner progress, it becomes easier and easier to rapidly scan a cat’s thorax, which can help evaluate the entire lung parenchyma as well as thorax. Scans require anesthesia and PEEP in order to prevent lung collapse. For animals with diffuse pulmonary disease, scans are often very diagnostic. It may be prudent to couple anesthetized imaging with cytological sample collection. CT is warranted for any diffuse pulmonary disease, or in situations were metastatic disease is concern.

Figure 4 - A CT image of a cat with severe pulmonary fibrosis.

Figure 5 - A CT image of a cat with asthma and marked obesity.
Magnetic resonance imaging $$$$, $$$$
MRI is another emerging alternative imaging modality in veterinary medicine. MRI is generally poor for the lung, but great for other intra-thoracic structures. There is phenomenal contrast and ability to determine the extent of masses or other structures. MRI is currently the most expensive modality, but should be considered if outstanding soft tissue resolution is essential (eg. Pre-surgical planning).

Figure 6 - MRI image of a large mediastinal mass in a dog
Nuclear medicine $$, ####
Nuclear scan is a method of using radiopharmaceuticals to highlight the function of a specific organ. Scans are typically non-invasive, light sedation may be required. Scans for thoracic disease include those looking for evidence of ectopic thyroid tissues, or cardiac function. Nuclear scans may also be performed for ventilation-perfusion scan (eg. Pulmonary thromboembolism) although cats are less commonly affected with the condition than dogs.

Figure 7 - Thyroid scan of a hyperthyroid cat; note the absence of ectopic tissue in the chest.

Bronchoscopy $$, ##
Bronchoscopy is considered the test of choice for evaluating the airways of a patient. Direct visualization will permit better evaluate of the lesion as well as to collect samples for cytological evaluation and culture. Brief anesthesia is required.

Figure 8 - A tracheal stricture.
It is wise to have a standard approach to the pet with respiratory distress; be complete as to not miss anything, but recognize patterns.
Top Emerging Trends/Hot Topics in Respiratory Medicine (2009)

1) Nasopharyngeal Turbinate
2) Early correction of Brachycephalic airway disease
3) Introduction of Pimobendan
4) Recognition of pulmonary vascular disease
5) Sildenafil
6) Inhaled steroids for asthma/chronic bronchitis
7) Emphasis on prednisone for airway disease
8) Appropriate use of antibiotics
9) Tracheal stents