Introduction, initial assessment and stabilization

Respiratory distress in small animal patients is a true emergency which requires rapid stabilization, prompt recognition and treatment of the underlying problem, determination of diagnostic and therapeutic options, and an assessment of prognosis. The first steps in the management of the dyspneic patient include recognizing that the respiratory system is compromised, performing a physical examination, providing supplemental oxygen, and obtaining a brief but focused history from the owner.

Physical examination

Animals that present in respiratory distress must be handled carefully to minimize stress and struggling and the initial physical examination may be limited.
to assessment of mucous membranes, capillary refill, and thoracic auscultation. Commonly the clinician may note increased respiratory rate and/or effort, shallow chest excursions, excessive respiratory noise, extension of the head and neck, nostril flare, mouth breathing, elbow abduction, and an inability to lie down or be comfortable. Patients on the verge of respiratory arrest may have limited movement of the chest wall and a paradoxical respiratory pattern due to respiratory muscle fatigue (1-4).

It is imperative to assess airway patency at presentation by observing that the patient is able to move air during breathing; the animal should not be stressed by attempting to open the mouth. If complete airway obstruction is diagnosed, rapid sequence sedation, intubation and possibly positive pressure ventilation are indicated. Cyanosis is not a reliable indicator of hypoxemia, as it does not develop until the partial pressure of oxygen in arterial blood (PaO₂) is <50mmHg, and cannot be detected in severely anemic or hypoperfused patients. Therefore the presence of pink mucous membranes should not be perceived as an indication of adequate oxygenation (1,4). A brief period of thoracic auscultation at this time should include auscultation of the heart to detect arrhythmias or murmurs, auscultation of the lungs with particular attention to areas of dullness or abnormal lung sounds such as crackles or wheezes, and auscultation of the cervical trachea to detect loud sounds indicating a possible airway obstruction.

Many dyspneic patients, especially cats, are intolerant of handling. Therefore supplemental oxygen, regardless of the cause of respiratory distress, is imperative for all patients with respiratory compromise. If possible, a peripheral intravenous catheter should also be placed at presentation to provide vascular access.

**Methods of oxygen supplementation**

Methods of oxygen support include flow-by, mask, hood delivery, nasal oxygen catheters, oxygen cage, and positive pressure ventilation (3-7).

Flow-by oxygen is the provision of oxygen through a tube held in front of the patient’s face. Inexpensive and uncomplicated, it does require some patient restraint and someone to hold the line to the patient’s mouth and nose. It is most useful for short-term provision of supplemental oxygen during the initial assessment of the animal and during brief procedures such as radiographs and catheter placement (4-7) but may be insufficient for patients that are panting or moving. Flow rates should be between 100-200 mL/kg/min, but high flow rates may not be well tolerated and are comparatively wasteful. The effectiveness of flow-by oxygen can be improved via an oxygen mask: a plastic cone that attaches to the oxygen line. With similar flow rates to those given above, this technique provides higher percentages of inspired oxygen (especially with a well-fitted mask and a recumbent patient). Mobile patients will require restraint to keep the mask in place, and many distressed animals resist placement of the mask over their face. An oxygen hood can be fashioned from an Elizabethan collar partially covered with plastic wrap. An oxygen line is inserted inside the collar, with the plastic cover vented to allow expired heat and gases to escape. Commercially available hoods, with an adjustable collar and perforated holes for expired gas release, also exist. Hoods are generally well tolerated, can achieve high percentages of inspired oxygen and allow for patient monitoring and procedures without interrupting oxygen delivery. However, there is no control over the amount of inspired oxygen and some patients, especially those that are panting, may overheat. Humidification should be used for long term oxygen hood therapy (3-6). Nasal catheters can provide high percentages of inspired oxygen via an indwelling catheter (see Table 1) or human nasal oxygen prongs (which are less invasive and useful in quiet or recumbent patients - Figure 1), using flow rates of 0.5-3 L/min. This technique is useful in patients that are restless or panting, or too large or intolerant of an oxygen cage, and is less wasteful than other methods. It allows patient monitoring and further procedures without interruption of oxygen therapy, but given the time needed for catheter placement this method is usually reserved for more long-term treatment (rather than initial stabilization) and humidification should be added in this situation (3,4,6,7). Oxygen cages are extremely useful as they provide accurate and (if indicated) high concentrations of humidified oxygen, allowing patient observation without restraint. However cages are expensive and can be wasteful as
The use of human nasal oxygen prongs in a dog with respiratory disease.

To place indwelling nasal oxygen catheters, one or both nostrils should first be infused with lidocaine. A suitable catheter should be selected, measured to the medial canthus of the eye, and marked at that depth prior to insertion. With an assistant restraining the dog’s head, the catheter should be advanced into the nasal passage gently but quickly, as the initial sensation of passing through the rostral part of the ventral meatus is the most uncomfortable part for the patient. Once inserted to the mark, the catheter should be secured using tissue glue or sutured with a tape butterfly or a Chinese finger-trap suture pattern. Any remaining length of catheter should be secured to the top of the patient’s head to avoid irritating the ears or whiskers. Most patients will also require an Elizabethan collar to prevent them from scratching or dislodging the catheter.

Oxygen concentration decreases rapidly when the door is open. If severe respiratory distress cannot be relieved using other techniques, intubation and positive pressure ventilation is the best way to control the airway, deliver oxygen or positive end-expiratory pressure, to relieve the patient’s anxiety and discomfort. Heavy sedation or light anesthesia, constant intensive monitoring, specialized equipment and training are required for this technique (4-7).

Regardless of the method of oxygen supplementation chosen, it is important to remember that prolonged periods (>24 hours) of high oxygen concentrations (>60%) should ideally be avoided to reduce the risk of oxygen toxicity due to free radical formation (4,6,7).

Localization of respiratory compromise, diagnostics, and therapeutics
A more complete physical examination can be performed to localize the origin of respiratory distress. Based on this examination, the causes of respiratory distress can be assigned to one of five categories: airway obstruction, pulmonary parenchymal disease, pleural space disease, thoracic wall abnormalities, and “look-alikes”. Identification of the probable site of the problem, combined with the signalment and history, allows the determination of a list of likely differential diagnoses, necessary diagnostics and immediate therapeutic options.

Airway obstruction
These patients may have inspiratory and/or expiratory stridor or stertor, head and neck extension, heat and exercise intolerance, prolonged inspiration, cyanosis, a honking or dry voice changes, history of heart or pulmonary disease, use of heartworm preventative, and the presence of concurrent systemic illness, such as vomiting, anorexia, and endocrine diseases. A current medication list should also be obtained. Once a physical examination has helped determine the anatomic origin of the respiratory distress and the animal has been stabilized, a more complete history can be obtained and a definitive diagnostic and therapeutic plan reviewed with the owners.

Table 1.
Nasal oxygen catheter placement

To place indwelling nasal oxygen catheters, one or both nostrils should first be infused with lidocaine. A suitable catheter should be selected, measured to the medial canthus of the eye, and marked at that depth prior to insertion. With an assistant restraining the dog’s head, the catheter should be advanced into the nasal passage gently but quickly, as the initial sensation of passing through the rostral part of the ventral meatus is the most uncomfortable part for the patient. Once inserted to the mark, the catheter should be secured using tissue glue or sutured with a tape butterfly or a Chinese finger-trap suture pattern. Any remaining length of catheter should be secured to the top of the patient’s head to avoid irritating the ears or whiskers. Most patients will also require an Elizabethan collar to prevent them from scratching or dislodging the catheter.
cough, respiratory distress, retching, and collapse. Dogs may be hyperthermic and cats may have intermittent open mouth breathing. A prolonged inspiratory phase of respiration (because the upper airway is sucked closed on inspiration) may be noted and wheezes may be heard, particularly on expiration. Coughing is commonly seen in cats with asthma, and lower airway obstructive disease is associated with increased expiratory effort. Referred upper airway noise may be differentiated from pulmonary parenchymal sounds as the sound intensity and pitch is louder on auscultation of the larynx and trachea (1,2,4,8,9).

Common causes of airway obstruction in dogs include: brachycephalic airway syndrome, laryngeal paralysis, inflammation or edema of the pharynx or larynx, airway infections and/or abscission, foreign body, coagulopathy induced hemorrhage, neoplasia, tracheal and mainstem bronchial collapse, and bronchitis (1,4,8,9). In cats, the most common causes of airway obstruction are feline asthma, nasopharyngeal polyps, pharyngeal and laryngeal neoplasia, inflammatory and granulomatous laryngeal disease, and viral nasal infections (2,4,8,9).

Since airway obstructions may impede oxygenation, ventilation, or both, useful diagnostics include pulse oximetry (Figure 2) and arterial or venous blood gas analysis. Hypoventilation is defined as arterial carbon dioxide partial pressure (PaCO₂) >43 mmHg in dogs and >36 mmHg in cats, resulting in primary respiratory acidosis. PaCO₂ ≥60 mmHg is consistent with significant hypoventilation and warrants definitive therapy to relieve the airway obstruction. When arterial blood gas sampling is not possible, venous carbon dioxide partial pressure (PvCO₂) can be used. Hypoxemia is defined as arterial partial pressure of oxygen (PaO₂) <80 mmHg and a value of PaO₂ ≤60 mmHg (which corresponds to <90% pulse oximetry) is consistent with severe hypoxemia and requires therapeutic intervention.
In these patients initial stabilization should be aimed at provision of supplemental oxygen, establishing vascular access, and applying cooling measures. Sedative and/or anxiolytic therapies can relieve respiratory distress and decrease respiratory drive, thus decreasing the degree of airway collapse. Intravenous fluid therapy, wetting of the fur and the use of a fan will aid cooling. Anti-inflammatory doses of corticosteroid may also be considered; this can be life-saving in patients with severe airway edema or inflammation but should be used selectively as it may impede a definitive diagnosis of lymphoma (1,8). Cats with feline asthma may benefit from parenteral bronchodilators such as terbutaline if they have no evidence of heart disease. Inhaled drugs (e.g. albuterol, fluticasone) may be substituted if complicating systemic disease is present. In the majority of patients, sedation, cooling and head/neck positioning to optimize airway patency are sufficient for stabilization.

For those with complete airway obstruction, or when cooling and sedative efforts are ineffective, induction of anesthesia and intubation is required. If endotracheal intubation cannot be achieved, an emergency tracheostomy must be performed.

The cause of an upper airway obstruction can usually be diagnosed by a sedated upper airway/laryngeal examination, cervical and thoracic radiographs, fluoroscopy, rhinoscopy/laryngoscopy/tracheoscopy/bronchoscopy, and/or computed tomography (CT). If a hematoma caused by a coagulopathy (e.g. secondary to rodenticide) is suspected, prothrombin time (PT) and partial thromboplastin time (PTT) should be performed. Transtracheal lavage, endotracheal lavage, or bronchoalveolar lavage should be considered in patients suspected to have lower airway (bronchial) or concurrent pulmonary parenchymal disease.

**Pulmonary parenchymal disease**

Dogs with pulmonary parenchymal disease are often depressed; signs may include: panting or breathing open-mouthed, with nostril flare, coughing, head and neck extension, and anxiety. Cats only breathe through an open mouth when they have severe respiratory compromise and (in contrast to dogs) rarely cough. Physical examination findings may include weakness, tachypnea, tachycardia, fever, mucopurulent nasal discharge, harsh lung sounds, and/or crackles. A heart murmur and/or arrhythmias are usually heard in dogs that have respiratory signs due to congestive heart failure. The same is not always true of cats with congestive heart failure, when cardiac auscultation may be normal. Pulse quality may be diminished whilst cyanosis may be seen in severely hypoxemic patients although hypoperfusion may produce pallor of the mucous membranes (1,2,4).

Common causes of pulmonary parenchymal disease include parasitic or bacterial pneumonia, pulmonary edema (cardiogenic or non-cardiogenic), pulmonary contusions, smoke inhalation, pneumonitis (chemical and uremic), aspiration pneumonia, fungal or viral infection, pulmonary thromboembolism, neoplasia, pulmonary fibrosis, and acute respiratory distress syndrome (ARDS). Cardiogenic edema occurs frequently in cats, whereas aspiration pneumonia is uncommon in this species (1-4,10).

Thoracic radiographs are particularly important in this situation. For example, classic radiographic changes associated with pneumonia include a cranio-ventral alveolar pattern ([Figure 3](#)), whereas cardiogenic pulmonary edema in dogs is generally associated with a heavy perihilar interstitial to alveolar pattern ([Figure 4](#)). In contrast, non-cardiogenic pulmonary edema is associated with a caudo-dorsal interstitial to alveolar pattern ([Figure 5](#)).

Pulmonary parenchymal disease causes hypoxemia primarily by mismatch of ventilation and perfusion, but also due to diffusion impairment and intrapulmonary shunt. Pulse oximetry and blood gas analysis are important in determining the severity of disease, and hematology and biochemistry may assist diagnosis. Lower respiratory tract sampling via transtracheal, endotracheal, or bronchoalveolar lavage can be important diagnostics to determine the origin of pulmonary parenchymal disease, especially in patients with atypical radiographic changes or when multiple disease processes are suspected, and cytology, bacterial culture and
sensitivity, and fungal culture are usually indicated. Emergency therapy for patients with pulmonary parenchymal disease depends on the clinician’s educated guess about the most likely etiology.

**Pleural space disease**

Clinical signs in these patients may include a short, shallow restrictive breathing pattern, increased respiratory rate, nostril flaring, orthopnea, an abdominal component to respiration, and a reluctance to lie down. Cats may have open mouth breathing. The degree of respiratory distress depends on the rate of development of the pleural space-occupying lesion, especially pleural effusion. Paradoxical respiration may be noted in patients with a diaphragmatic hernia. Thoracic auscultation may reveal decreased or dull lung sounds ventrally (pleural effusion) or dorsally (pneumothorax), an auscultable fluid line, muffled cardiac sounds, or borborygmi if stomach or intestines are in the chest. Thoracic auscultation changes may be unilateral or bilateral and are not always equal. Cats with a mediastinal mass have decreased chest wall compressibility (1,2,4,11,12).

Common causes of pleural space disease include pneumothorax and pleural effusions. Pleural effusions may be pure or modified transudates due to congestive heart failure, vasculitis, and lung lobe torsion; pyothorax, chylothorax, hemotorax, feline infectious peritonitis, and neoplastic effusions are also common. Diaphragmatic hernia and pleural space masses also occur (1,2,4,11,12).

If pleural disease is suspected, it is imperative to evacuate the pleural space as quickly as possible (Table 2). Oxygen supplementation and vascular access is established if it can be done without stress. Thoracocentesis is both therapeutic and diagnostic, and any fluid obtained submitted for cytology and culture. Thoracocentesis is not indicated for patients with pleural space masses, diaphragmatic hernias, or hemotorax secondary to coagulopathy that is not causing significant respiratory compromise. Other diagnostics (e.g. radiography or ultrasound) should be delayed until after the animal has been stabilized by thoracocentesis, allowing the pulmonary parenchyma to be visualized effectively.
**Table 2.**

**Thoracocentesis**

Sedation is often not necessary unless the patient is very distressed or agitated; most animals can be restrained in sternal recumbence for the procedure. The patient’s chest wall should be prepared in the region where the lung sounds are most muffled. This is generally in the upper third of the caudodorsal thorax between the 8th and 9th ribs for pneumothorax, and in the ventral two thirds of the thorax between the 6th and 8th intercostal spaces for pleural effusion. Alternatively, an ultrasound probe can be used to find pockets of fluid amenable to aspiration. When aspirating air, a 22 or 25 gauge straight needle or butterfly needle is preferred; larger gauge needles (18 or 20 g) are often needed for pleural fluid aspiration in dogs. The needle is attached to tubing, a three-way stopcock, and syringe. The cranial border of the rib is palpated, and the needle is advanced through the skin, bevel up, and slowly into the thoracic cavity on the cranial border of the rib. The needle should be slowly advanced while an assistant aspirates the system; the needle is then held still as soon as air or fluid is visible in the tubing. Air and fluid should be aspirated until negative pressure is achieved, at which point the needle may be advanced further into the thorax, redirected, or repositioned if additional air or fluid is suspected.

Normally blood aspirated during thoracocentesis should clot quickly, unless a coagulopathy or a hemorrhagic effusion (e.g. due to neoplasia or lung lobe torsion) is present; if this occurs further testing to confirm these suspicions should be performed. If unexpected air is aspirated, the collection system should be checked for leaks. If no leaks are present, thoracoentesis should be temporarily aborted as long as the animal is still stable, because an iatrogenic pneumothorax may have been created (3,4,12,13). If negative pressure is not achieved during thoracocentesis, if the patient re-accumulates a significant amount of air within a short period requiring multiple thoracocentesis events, or for the medical management of certain pleural effusions such as pyothorax, thoracostomy tubes (unilateral or bilateral) may be needed and are best placed using general anesthesia (1,3,4,14).

**Thoracic wall abnormalities**

Abnormalities of thoracic wall function may occur because of dysfunction at several levels. In some cases, respiratory distress may be secondary to thoracic wall injuries or trauma. Those patients often demonstrate pain on manipulation or palpation of the thorax, and may have a flail chest with paradoxical motion of a segment of chest wall. Lacerations, bruising or concurrent pulmonary contusions may occur. Alternatively, abnormal thoracic wall and diaphragmatic function may result from defective neuromuscular control of breathing, which may be caused by disease in the brain, spinal cord, peripheral nerves or neuro-muscular junctions. Such patients typically lack diaphragmatic and abdominal muscle movement, and have clinical evidence of hypoventilation with elevated PaCO₂ levels on blood gas analysis (1,3,15).

Common causes of thoracic wall dysfunction include traumatic injuries, anesthetics and central respiratory depressants, severe hypokalemia, myasthenia gravis, botulism, tick paralysis, polyradiculoneuritis, congenital abnormalities, some snake envenomation, thoracic wall neoplasia, and spinal cord or phrenic nerve disease (1,3,15).

Diagnostics for these patients include blood gas analysis, pulse oximetry, capnography, and thoracic radiographs. Neurologic examination findings consistent with cervical spinal cord or brainstem dysfunction may support a neurologic origin. Measurement of acetylcholine receptor antibody titers, edrophonium response testing, and electromyelography (EMG) can aid in the diagnosis of myasthenia gravis. EMG and the confirmation of botulism toxin in serum, feces, or vomited material can support a diagnosis of botulism. There are no specific diagnostic tests for tick paralysis, polyradiculoneuritis, and snake venoms, though geographic location, signalment, and history may support a diagnosis (15).
**“Look-alikes”**

Whilst rare, some non-respiratory conditions can mimic respiratory disease. Examples include hyperthermia, compensation for metabolic acidosis, anemia, pain, stress, anxiety, hypovolemia, abdominal distension (cranial organomegaly and abdominal effusion), hyperadrenocorticism or corticosteroid therapy, and certain opioid medications (16). History, physical examination, thoracic radiographs, blood gas analysis and serum chemistry can be helpful to distinguish these conditions from real respiratory disease.

**Conclusion**

Respiratory distress is a common and serious emergency encountered by veterinarians. An understanding of the need for careful patient handling and the provision of supplemental oxygen is vital. Veterinarians should recognize the signalment, history, and physical examination findings seen with common respiratory diseases. An understanding of respiratory disease pathophysiology and localization as well as the diagnostic and therapeutic techniques essential for management of each category of dysfunction is crucial for successful treatment.

**REFERENCES**