Management of Respiratory Emergencies in Small Animals

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KEYWORDS

- Upper airway
- Respiratory distress
- Cough
- Pulmonary edema
- Oxygen
- Ventilation
- Pleural effusion

KEY POINTS

- Respiratory distress is a common presenting complaint for animals brought to the emergency room, and it is important for clinicians to feel comfortable diagnosing and treating these animals.
- Prompt recognition of the localization of the source of respiratory distress, based on history, pattern recognition, and physical examination findings, will help to determine the underlying cause and is key to determining an appropriate therapeutic course.
- Careful handling, minimizing stress, and rapid and focused treatment are crucial in the management of all patients in respiratory distress.

Respiratory distress is a common presenting complaint for dogs and cats in the emergency room and may develop during hospitalization for noncardiopulmonary disease as well. Appropriate management and a favorable outcome require rapid recognition, assessment of the underlying cause, and timely interventions. This article focuses on current recommendations for emergent diagnostics and management of dogs and cats either presenting to the emergency room with respiratory distress or developing respiratory distress while hospitalized.

The initial approach to a patient with respiratory distress involves localization of the affected region(s) of the airway, lungs, or pleural space, and creation of an initial list of differential diagnoses based on patient history, signalment, and physical examination findings.

Localization is key in determining the best step in management. Respiratory dysfunction occurs because of difficulty in getting oxygen into the lungs (eg, upper airway obstruction, pleural effusion, or lower airway disease) or with difficulty in gas exchange (eg, abnormalities at the alveolar-capillary membrane caused by edema, neoplasia, or hemorrhage).

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UPPER AIRWAY DISEASE

Normal upper airway physiology reflects negative pressure ventilation; this involves a drop in pressure in the lumen of the upper airway during inspiration, which permits room air to move into the lungs down the pressure gradient. Increased resistance, resulting from narrowing of the upper airway lumen, requires increased inspiratory pressure for equivalent flow rate and clinically may be recognized as loud or stridorous breathing. Increased resistance may be associated with a fixed and/or dynamic obstruction. Specific sites commonly associated with upper airway obstruction include the larynx (eg, due to paralysis, collapse, or masses), the nasopharynx (eg, due to abnormalities in the soft palate or pharyngeal tissues), the cervical trachea, and the nasal passages (Fig. 1). Brachycephalic dogs and cats are at increased risk of upper airway obstruction because of their abnormal anatomy, and older large-breed dogs are at increased risk of laryngeal paralysis.

LOWER AIRWAY DISEASE

Lower airway diseases include bronchial disease, such as feline lower airway disease ("asthma") and canine chronic bronchitis. Lower airway disease may present as an emergency in cats with moderate to severe bronchoconstriction, and dogs can present for increased coughing and wheezing. Respiratory distress from lower airway disease is associated with expiratory airflow obstruction. During inspiration, the airways are open, but often collapse or narrow during expiration, resulting in increased expiratory effort and often an expiratory “push” on physical examination. Many dogs with lower airway disease/chronic bronchitis are overweight or obese (Fig. 2). Eosinophilic bronchopneumopathy (or bronchitis), commonly seen in northern-breed dogs,
may also result in lower airway disease. Eosinophilic bronchopneumopathy is associated with coughing, gagging, and a marked bronchial pattern. Tracheal wash cytology is strongly eosinophilic, and concurrent peripheral eosinophilia may or may not be present.

PULMONARY PARENCHYMAL DISEASE

Pulmonary parenchymal diseases include cardiogenic and noncardiogenic pulmonary edema, pneumonia, pulmonary contusions, hemorrhage, interstitial disease, and neoplasia. Parenchymal diseases are characterized by decreased pulmonary compliance, or the development of “stiff” lungs, that require higher inspiratory pressures to reach the same tidal volume. Inspiratory and expiratory efforts may both be increased. Specifically, there is often fluid in the alveolar space, resulting in collapse of the alveoli, or thickening of the alveolar-capillary membrane, which decreases the efficiency of gas exchange.

PLEURAL SPACE DISEASE

Pleural effusion or pneumothorax may result in respiratory distress because of compression of the lungs and relative limitation of lung expansion. The pleural space may develop pathology due to air, pure or modified transudates, exudates, or solid tissue (eg, neoplasia, organs) filling the pleural cavity and resulting in decreasing ability of the lungs to expand and to ventilate. Pleural effusion is not a final diagnosis, and may develop from several different conditions. Common underlying etiologies include trauma, which may result in pneumothorax or diaphragmatic hernia, right-sided or biventricular heart failure, neoplasia, or infection (pyothorax). Anticoagulant
Rodenticide intoxication may result in a hemorrhagic pleural effusion, and it may be accompanied by mediastinal and pericardial hemorrhage as well. Traditionally, pleural space disease has been reported to result in a restrictive pattern of breathing, characterized by fast and shallow breaths. Paradoxic chest wall movement has also been associated with pleural space disease; however, in a recent study of respiratory parameters of animals in respiratory distress, it was found that a fast and shallow breathing pattern was not associated with pleural space disease in dogs and cats, whereas an asynchronous or inverse breathing pattern was associated with pleural space disease.2

PROFILING AND PATTERN RECOGNITION

Profiling and pattern recognition is a commonly used, but underappreciated, technique when evaluating the patient with respiratory distress. Profiling, when applied to animals, simply reflects the recognition that some ages and breeds of pets are more likely to be affected with a specific disease than another. For example, a geriatric Labrador retriever may be much more likely affected with laryngeal paralysis than an English bulldog puppy, which is more commonly affected with pneumonia. Profiling and pattern recognition may help direct the clinician to a more likely diagnosis, but clearly a complete assessment of each individual pet is warranted. Table 1 provides a list of breeds of dogs and cats with common clinical respiratory problems.

<table>
<thead>
<tr>
<th>Breed of Dog</th>
<th>Common Respiratory Problem</th>
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<tbody>
<tr>
<td>Yorkshire terrier</td>
<td>Tracheal collapse</td>
</tr>
<tr>
<td>Toy poodle</td>
<td>Tracheal collapse; chronic bronchitis; chronic valvular heart disease</td>
</tr>
<tr>
<td>Norwich terrier</td>
<td>Brachycephalic airway syndrome; laryngeal collapse</td>
</tr>
<tr>
<td>Pug</td>
<td>Brachycephalic airway syndrome</td>
</tr>
<tr>
<td>Cocker spaniel</td>
<td>Bronchiectasis; chronic bronchitis</td>
</tr>
<tr>
<td>West Highland white terrier</td>
<td>Interstitial lung disease (pulmonary fibrosis), chronic bronchitis</td>
</tr>
<tr>
<td>Labrador retriever</td>
<td>Laryngeal paralysis</td>
</tr>
<tr>
<td>Bulldog</td>
<td>Brachycephalic airway syndrome</td>
</tr>
<tr>
<td>Golden retriever</td>
<td>Spontaneous pneumothorax</td>
</tr>
<tr>
<td>Northern breeds (husky/malamutes)</td>
<td>Spontaneous pneumothorax; eosinophilic bronchopneumopathy</td>
</tr>
<tr>
<td>Doberman pinscher</td>
<td>Cardiogenic pulmonary edema secondary to dilated cardiomyopathy</td>
</tr>
<tr>
<td>Young puppies</td>
<td>Noncardiogenic pulmonary edema secondary to electric cord injury, and so forth; long-acting anticoagulant rodenticide; <em>Bordetella bronchiseptica</em> pneumonia</td>
</tr>
<tr>
<td>Hunting breeds</td>
<td>Pyothorax; blastomycosis in endemic areas</td>
</tr>
<tr>
<td>Young cats</td>
<td>Nasopharyngeal polyps; upper respiratory infection</td>
</tr>
<tr>
<td>Pointed cats (Siamese/Himalayan)</td>
<td>Allergic airway disease (“asthma”)</td>
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</tbody>
</table>
PERTINENT HISTORY

Obtaining an appropriate, thorough history is imperative in the patient with respiratory distress. Identifying the duration and progression of clinical signs (e.g., coughing, vomiting, anorexia, febrile), any prior therapies, current medications (e.g., prednisone), previous medical problems, history of recent anesthesia or sedation, potential exposures to toxicants, any environmental changes, and other underlying indicators of metabolic disease (e.g., polyuria/polydipsia) is important. Some animals with respiratory distress may have chronic pulmonary disease with acute exacerbations.

Cats with lower respiratory disease may have a history of “trying to cough up hairballs,” although no hairball ever materializes. Smaller dogs with congestive heart failure (CHF) typically have a long-standing heart murmur, and often a recent history of weight (muscle) loss. Dogs or cats that have sustained trauma should be monitored closely for pneumothorax or progressive pulmonary contusions.

For dogs that develop respiratory distress during hospitalization, common causes include aspiration pneumonia, fluid overload, pulmonary thromboembolism, and, less commonly, acute lung injury/acute respiratory distress syndrome. Cats that develop respiratory distress while hospitalized are most often diagnosed with fluid overload.

INITIAL ASSESSMENT AND ACTION STEPS

After initial patient evaluation, the history, signalment, and patient profile should lead the clinician to the most likely anatomic localization of respiratory distress, and help the clinician create a list of differential diagnoses. Although stressful handling may not be tolerated in animals in respiratory distress, particularly cats, it is also important to recognize that simple observation is rarely therapeutic. Major action steps for a patient with respiratory distress include the following:

1. Supplemental oxygen therapy
2. Sedation/Anxiolytic therapy
3. Control of hyperthermia
4. Thoracocentesis (if indicated)/thoracostomy tube placement
5. Pharmacologic therapy specific for the underlying disease
6. Assessment of oxygenation (pulse oximetry/arterial blood gas analysis)
7. Tracheostomy (if upper airway obstruction cannot be relieved)
8. Intubation and positive pressure ventilation (PPV) for imminent respiratory failure
9. Thoracic radiographs, ultrasound, or echocardiography. Note that diagnostic imaging of any form is never therapeutic; however, detection of the specific underlying pathology may permit the clinician to proceed with targeted therapy, which increases the potential of therapeutic benefit. Imaging may be brief, and include an ultrasound scan for effusion (thoracic focused assessment with sonography [TFAST]), a single radiograph view, or an assessment of the left atrial size via rapid echocardiography. More extensive imaging should be pursued only when the patient has been stabilized.
10. Laboratory evaluation (e.g., venous or arterial blood gas, packed cell volume, total solids, coagulation testing)

Supplemental Oxygen Therapy

Oxygen therapy should be initiated promptly in all patients presenting in respiratory distress; however, keep in mind that patients with pleural space disease or upper airway obstruction will benefit more from therapeutic intervention (e.g., thoracocentesis, sedation for relief of airway obstruction), and should always have an initial brief
assessment done before placement in an oxygen cage to exclude a more definitively treatable cause of distress. Initially, oxygen can be administered via flow-by oxygen, an oxygen cage, an oxygen mask, or via an oxygen hood. Longer-term oxygen therapy can be administered via oxygen cage, nasal oxygen cannulas, transtracheal oxygen, or intubation and administration of PPV. The approximate FiO₂ achieved with different methods of oxygen administration is shown in **Table 2**.³⁴ Keep in mind that benefit of nasal oxygen cannulas is reduced in panting dogs, and an alternate form of oxygen supplementation should be used.

**Sedation/Anxiolytic Therapy**

Sedation is most often used to ameliorate the dynamic component of upper respiratory obstruction associated with laryngeal paralysis, tracheal collapse, or brachycephalic airway syndrome. Low-dose acepromazine and/or butorphanol can be used. Recent anecdotal evidence has suggested that either doxepin or trazodone may be useful for laryngeal paralysis; however, there are no clinical trials supporting this. Diazepam or midazolam may also be used in combination with butorphanol; however, benzodiazepines alone are not effective at sedating dogs, and should therefore not be used as a single agent. Excessive respiratory effort leads to increased energy expenditure and may contribute to hyperthermia and/or respiratory failure, resulting in a vicious cycle and an exacerbation of clinical signs.

It is challenging to decide whether to use sedation in animals with nondynamic respiratory distress, such as heart failure, and must be weighed carefully; if sedation is administered, the patient should be closely monitored for respiratory depression or exhaustion. Patients with imminent respiratory failure (e.g., exhibiting air hunger, orthopnea, respiratory exhaustion) that fail to respond to therapy should ideally be sedated, intubated, and administered PPV; this will provide a protected airway, patient comfort, and allows the clinician the ability to perform diagnostics in a more controlled manner and without patient discomfort or stress. Additionally, the use of PPV may provide a humane option during the decision making on a patient’s ongoing care.

**Control of Hyperthermia**

Upper respiratory disease may result in severe hyperthermia, which will further contribute to respiratory distress. **Fig. 3** shows a pug with severe upper airway obstruction, who had a rectal temperature higher than 110°F (>43.3°C). Prompt active cooling, with room temperature intravenous (IV) fluids, fans, alcohol to the footpads,

<table>
<thead>
<tr>
<th>Oxygen Administration Technique</th>
<th>Mean FiO₂ Achieved (%)</th>
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<tbody>
<tr>
<td>Oxygen cage</td>
<td>21–60</td>
</tr>
<tr>
<td>Flow-by oxygen</td>
<td>24–45</td>
</tr>
<tr>
<td>Face mask, loose fitting</td>
<td>35–55</td>
</tr>
<tr>
<td>Oxygen hood</td>
<td>30–50</td>
</tr>
<tr>
<td>Unilateral nasal catheter</td>
<td>30–50</td>
</tr>
<tr>
<td>Bilateral nasal catheter</td>
<td>30–70</td>
</tr>
<tr>
<td>Intratracheal catheter</td>
<td>40–60</td>
</tr>
<tr>
<td>Positive pressure ventilation</td>
<td>21–100</td>
</tr>
</tbody>
</table>
cool towels to the body, and so forth, is imperative to prevent secondary disseminated intravascular coagulation (DIC) and multiple organ failure. Cooling measures should be discontinued at a temperature of 103.5°F (39.7°C). Sedation and/or intubation to relieve the upper airway obstruction may also be warranted, and may help control hyperthermia.

**Thoracocentesis**

Removal of pneumothorax or pleural effusion is therapeutic in alleviating clinical signs of dyspnea. In animals with moderate to severe distress, thoracocentesis **before** imaging is warranted, as one cannot evaluate cardiac size, presence of metastasis, or mass lesions easily when pleural effusion is present. Dogs with pleural effusion and anemia should be evaluated for anticoagulant rodenticide **before** thoracocentesis. The recent emergence of limited-use ultrasound scanning in the emergency room (TFAST) will aid the clinician in appropriately treating the dyspneic patient (see the article “Emergency Management and Treatment of the Poisoned Small Animal Patient” elsewhere in this issue, for more information).

Thoracocentesis is performed between the seventh and ninth intercostal spaces, ideally avoiding the caudal edge of the rib due to the location of the vessels and nerves. In smaller animals, a butterfly catheter is typically adequate, whereas in larger animals, an 18-gauge to 20-gauge, 1.0-inch to 1.5-inch needle or catheter may be required. The mediastinum in cats and dogs is less robust than in people, and may be fenestrated or incomplete; thus, recording volumes from the right or the left side may reflect only which side was tapped first, rather than the underlying pathology.
Rarely, complications from thoracocentesis may occur, including hemorrhage, cardiac puncture, or iatrogenic pneumothorax. The latter may occur more frequently in animals with long-standing effusions, such as chylothorax or with hyperinflated lung (eg, asthma). Iatrogenic pneumothorax develops due to either laceration of the pleura overlying the lungs, or the creation of excessive negative pressure in the pleural space, which may cause “ripping” of the pleura in trapped lungs rather than reexpansion of the lungs to fill the pleural space.\(^5\)

Thoracostomy tube placement may be necessary for management of pleural space disease. In an emergency setting, this is most commonly needed to treat pneumothorax, as large-volume pneumothorax can reform much more quickly than pleural effusions. In patients requiring more than 3 to 4 therapeutic, large-volume thoracocentesis procedures within a 24-hour period, placement of either unilateral or bilateral thoracostomy tube placement should be strongly considered.

In the vast majority of cases, placement of a thoracostomy tube should be done with the patient under general anesthesia and intubated for control of the airway and PPV. In rare cases in which the patient is unconscious or moribund, thoracostomy tubes may be placed without general anesthesia. See Box 1 for a brief guideline on thoracostomy tube placement.

Red rubber catheters or trocar catheters can be used for thoracostomy tubes. More recently, a thoracostomy tube has been developed that is placed using the modified Seldinger technique, which uses a smaller tube and may be placed more

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**Box 1**

**Guidelines for placement of a thoracostomy tube**

1. Determine clinical need.
2. Assemble needed technical help and supplies. Supplies needed include
   a. Sterile drape and supplies for aseptic preparation of the skin
   b. Sterile instruments and suture material
   c. Desired chest tube (eg, Argyle, red rubber)
3. Anesthetize and monitor the patient. Rarely, local analgesics are sufficient.
4. Place the patient in sternal or lateral recumbency. If placing bilateral tubes, sternal is advised; otherwise clinician preference.
5. Clip and prepare the site.
6. Drape the site.
7. Have an assistant pull the skin forward.
8. Make a small incision (<1 cm) into the skin and underlying musculature at approximately the 10th intercostal space.
9. Alert the individual monitoring anesthesia that entry into chest is imminent.
10. Place the chest tube through the incision into the chest.
11. Have the assistant release the skin, this creates a tunnel for the tube.
12. Aspirate the tube to ensure patency.
13. Securely suture the tube to the patient.
14. Place an adherent dressing if desired over the insertion site.
15. Confirm placement radiographically.
quickly, under sedation, and appears more comfortable for patients (Mila International, Lexington, KY).

Pharmacologic Therapy Specific for the Underlying Disease

The use of specific pharmacologic therapy is often considered essential in the patient with respiratory distress. Appropriate, titrated use of specific drugs is warranted, depending on the underlying disease process and pathophysiology of disease. Important pharmacologic drugs include antibiotics, diuretics, vasodilators, glucocorticoids, bronchodilators, and inhalation therapy.

Broad-spectrum antibiotics are warranted for the treatment of bacterial pneumonia or pyothorax. Appropriate antibiotics should be based on results of culture and sensitivity data, including ampicillin-fluoroquinolones or cefazolin-gentamicin–metronidazole. In some cases, a Gram stain may be useful to guide preliminary therapy. For young, rapidly growing neonates, the use of certain antibiotics may be relatively contraindicated (eg, tetracyclines, fluoroquinolones), and potential adverse effects should be discussed with the pet owner.

For animals with suspected hospital-acquired infections, antibiotics should be chosen based on known hospital resistance patterns. Hospital-acquired infections (HAI) should be suspected in dogs with a new fever or cough developing 48 hours or more after hospital admission, and they should be treated with antibiotics that have known efficacy against HAI.

Diuretics, including furosemide (2–8 mg/kg IV every 2–8 hours) or torsemide (0.2–0.8 mg/kg IV) are the mainstay therapy for patients with pulmonary edema due to CHF or potentially, noncardiogenic pulmonary edema. By removing excessive body water (eg, circulating blood volume and subsequently pulmonary edema), and reducing preload, they will relieve clinical signs of respiratory distress. In dogs, the intermittent use of 2 to 4 mg/kg of furosemide (IV or intramuscularly) every 1 to 2 hours in fulminant CHF, in conjunction with cage rest and supplemental oxygen, should result in improvement in respiratory rate and effort, typically within a few hours. Cats are may be treated similarly, although they be more prone to volume depletion and weakness. Continuous rate infusions (CRI) of diuretics have been used occasionally, but more recent human studies show no better outcomes with a CRI than with intermittent bolus injections. Diuretics are occasionally used without a definitive diagnosis of CHF, particularly in cats considered too unstable for more definitive imaging (radiographs or echocardiogram). Excessive use of diuretics is associated with volume contraction, electrolyte disturbances, including hypokalemia, and metabolic alkalosis. Additionally, prerenal azotemia may develop. Although a single dose of a diuretic is unlikely to be harmful, excessive use in the patient without a definitive diagnosis should be avoided.

Vasodilators are also useful in treatment of CHF and include either topical nitroglycerin (venodilator) or IV nitroprusside (a balanced vasodilator). There is limited evidence of the efficacy of topical nitroglycerin in dogs and cats, although it remains a popular therapeutic option. Nitroprusside is given by continuous-rate infusion and as very effective vasodilator it may be associated with severe hypotension. Nitroprusside must be given by an infusion pump, with a covered IV line to prevent degradation, and in a small volume of 5% dextrose in water (D5W), such as 1 to 2 mL per hour (Fig. 4). As an infusion, the line must not be flushed, as severe hypotension may result. Additionally, long-term therapy is associated with the potential for thiocyanate or cyanide toxicity. Frequent blood pressure monitoring is commonly advocated during use of nitroprusside, and if possible, is ideal. However, in many animals with severe CHF, the amount of time required to noninvasively determine blood pressure may be
counterproductive. Palpation of pulses may be substituted, and any patient receiving nitroprusside should be carefully monitored. The starting dose of nitroprusside is 0.5 to 1.0 $\mu$g/kg per minute; this may be titrated up every 15 minutes by 0.5 to 1.0 $\mu$g/kg per minute until 10.0 $\mu$g/kg per minute is reached, although most animals are improved by 2.0 to 3.0 $\mu$g/kg per minute.

Glucocorticoids are useful for the treatment of inflammatory lower airway disease, and tracheal collapse. In the short term, glucocorticoids also may be useful in laryngeal paralysis with arytenoid inflammation, and with brachycephalic airway syndrome with pharyngeal or laryngeal swelling. Airway inflammation is associated with airway narrowing, which increases airway resistance, worsens cough, and perpetuates inflammation. Cats with suspected lower airway disease will typically respond very quickly to dexamethasone (2–4 mg/cat) or prednisone/prednisolone. Most cats convert prednisone to prednisolone; however, a small subset of cats do not, and, thus, if possible, prednisolone should be substituted for prednisone. Dogs with tracheal collapse and chronic bronchitis will also respond rapidly to glucocorticoids. Long-term high doses of glucocorticoids are associated with side effects, similar to Cushing disease, including lethargy, polyuria/polydipsia, polyphagia, hepatomegaly, muscle weakness, poor fur growth, and diabetes. Although cats are overall more tolerant of glucocorticoid therapy, diabetes and CHF have been reported, particularly in conjunction with reposital products. Glucocorticoids should be tapered to the lowest possible dose, and consideration should be given for the use of inhaled glucocorticoids if low-dose glucocorticoids are inadequate to control clinical signs.

Bronchodilators include theophylline and $\beta$-2 agonists, such as terbutaline or albuterol (salbutamol outside the United States). Bronchodilators are not effective in tracheal collapse, although they may be useful in some lower airway diseases in dogs.

Inhaled Therapy

In human medicine, inhaled corticosteroids (ICs) are the mainstay for people with asthma and other inflammatory airway diseases. Advantages of ICs include avoidance of systemic side effects of oral glucocorticoids. ICs have been proposed in both cats and dogs with moderate to severe lower airway disease that are steroid responsive. The appeal of ICs includes targeting therapy to the lungs, and avoidance of the bulk of the systemic side effects. The “Internet presence” of the use of ICs in
dogs and particularly cats is strong, including the site www.fritzthebrave.com, which provides guidelines for the cat with lower airway disease. It is important that the clinician is familiar with ICs, and is able to discuss both pros and cons logically with clients.

Because of the lack of voluntary cooperation in cats and dogs, administration of inhaled medication requires the use of a face mask and spacer. Although several models are available, [Trudell Medical, London, Ontario Canada (http://www.trudellmed.com/animal-health)] has the most widely used products with the Aerokat and Aerodawg chambers.

The nasal cavities of both dogs and cats are much more complex than the human nasal cavity. This permits more aerosolized drug to settle out in the nasal passage rather than being delivered specifically to the lungs. A study by Schulman and colleagues,7 using a similar device with aerosolized radiopharmaceuticals, demonstrated that there was deposition of the radiopharmaceutical to the lung, although in three-fourths of the cats there was also evidence that some of the radiopharmaceutical was present in the stomach, suggesting that the aerosol had been subsequently swallowed.

ICs have also been found to affect the hypothalamic pituitary axis in a study by Cohn and colleagues,8 although there were limited effects on the immune system. The diabetogenic effect of ICs has not been evaluated to date in cats, although systemic glucocorticoids are well known to facilitate the development of diabetes mellitus. As mentioned earlier, administration of ICs requires the use of a face mask and spacer. Although some animals, particularly dogs, may be trained quite easily to accept the facemask, in other pets it may take a period of acclimation to have the acceptance of the mask and treatment.

There are several IC preparations available, including fluticasone (Flovent), beclomethasone (QVAR), budensonide (Pulmicort), ciclesonide (Alvesco), and mometasone (Asmanex). There is no generic product available yet. All ICs are expensive, with beclomethasone dipropionate HFA (QVAR) usually the least expensive. Most descriptions of small animal use have focused on fluticasone. The optimal dosing of ICs is unknown in dogs and cats. One study in cats supported a starting fluticasone dose of 44 mg per cat twice a day, whereas some anecdotal studies suggest 110 μg per cat twice daily up to a maximum of 440 μg twice daily. Most commonly, oral glucocorticoid therapy is continued for about 10 to 14 days with a tapering overlap. In dogs, a starting dose of 110 μg (up to 10 kg), 220 μg (10–25 kg), and 440 μg (>25 kg) may be considered. ICs were initially available in metered dose inhalers (MDIs) with the propellant chlorofluorocarbons (CFCs). However, because of evolving concerns about the effect on the ozone layer, CFCs have been phased out. MDIs are available using other ozone-safe propellants, or as a dry powder. The dry powder preparations are also available but are NOT useful in animals because of the voluntary effort required to use the product. There are also products (eg, Advair) that combine fluticasone and the long-acting β-2 agonist salmeterol; one recent study by Leemans and colleagues9 showed improved efficacy in an experimental model of feline asthma.

To date, there have been no studies specifically evaluating any advantages of ICs over oral prednisone in dogs or cats with naturally occurring airway disease; however, it is prudent for the advanced practitioner to be familiar with inhaled medications. The advantages of inhaled medications may include fewer systemic effects, and limitations of potential complications. It is rare for a cat or dog to respond well to ICs if they have not shown an improvement with oral steroids. Thus, if the pet is not clearly improved with oral prednisone (or prednisolone in cats), it is quite unlikely they will do well on ICs. On the other hand, if a pet does well on steroids,
but the side effects are poorly tolerated, or if another risk factor, such as diabetes or CHF is present, it is a very reasonable plan to transition to ICs. Clients should learn of the options of inhaled medication from their primary care veterinarian, who can effectively counsel them on their specific pet. Inhaled glucocorticoids are rarely started as an emergency therapy.

Other medications may also be administered by aerosol; these include bronchodilators, antibiotics, and saline. Older textbooks suggest the use of N-acetylcysteine (NAC) as a nebulized agent to decrease the viscosity of mucus; however, recent evidence supports that nebulized NAC may cause bronchoconstriction or even death.10

**Tracheostomy**

Although not commonly performed in the emergency setting, a tracheostomy can be life-saving in patients with upper airway obstruction that fail to respond to medical management (eg, sedation, supplemental oxygen therapy, inability to intubate orally) or in cases of complete airway occlusion. With complete occlusion that cannot be relieved (eg, a ball in the pharynx), an emergent (“slash”) tracheostomy may be performed to provide the patient with an airway. Slash tracheostomies are also occasionally required in association with an upper airway examination in cats with laryngeal masses that are unable to be intubated. Ideally, a more controlled, surgical approach to tracheostomy is preferred, to minimize stress on both the patient and the surgeon.

However, it is preferable for a tracheostomy to be performed more as an elective procedure, with first oral intubation, and then subsequent surgical approach to the airway. The incision into the trachea may be transverse, longitudinal, or T-shaped, but ideally the incision should not be more than 50% of the diameter of the trachea. The tracheostomy tube should NOT be sutured to the patient, as the clinician needs to be able to remove the tube quickly in case of occlusion. Following placement of a temporary tracheostomy tube, the patient should be supervised continuously to ensure that occlusion of the tube does not occur. The high-volume, low-pressure cuff on the tracheostomy tube should not be insufflated unless the pet is undergoing mechanical ventilation or if general anesthesia is necessary (eg, for a procedure). This will minimize pressure necrosis to the trachea, and also allow limited ventilation if the lumen of the tracheostomy tube were to occlude unobserved.

Sterile technique should be observed when suctioning the tracheostomy tube. If supplemental oxygen is provided via the tracheostomy, it should be humidified, if possible. A “trach kit” should be placed near the patient, with readily available supplies for changing the tube, suction, and, if needed, sedation. When the tracheostomy tube is no longer needed, the site should be allowed to close by second intention. Closure of the skin before closure of the tracheostomy site may result in marked subcutaneous emphysema.

**Intubation and PPV**

For animals with severe respiratory distress that fail to respond to oxygen therapy or therapeutic intervention, intubation and intermittent PPV is an important option to consider. Ventilation removes the work of breathing from the patient, and provides humane relief for patients with respiratory distress. Ventilation may be associated with a good prognosis in animals with reversible underlying diseases; however, ventilation is labor-intensive and expensive, and important considerations must be considered (see the article “Analgesia, Anesthesia, and Chemical Restraint in the Emergent Small Animal Patient” elsewhere in this issue, for more information).
Thoracic Radiographs, Ultrasound, or Echocardiography

Thoracic radiographs are a mainstay of diagnostics for identification of intrathoracic sources of respiratory distress. Radiographs in patients with respiratory disease are beneficial, as they will help identify pulmonary infiltrates, mediastinal masses, cardiomegaly, pleural space disease, and trauma (eg, rib fractures, diaphragmatic hernias). Readers are directed to a radiology resource for additional information.

Thoracic ultrasound is also useful in characterizing the cause of respiratory distress. TFAST is a diagnostic test that is rapidly gaining popularity, relying on the use of brief ultrasound to evaluate a patient for pleural space disease. This technique can be used to identify the presence of pleural effusion and pneumothorax. Advantages of TFAST include rapidity of the procedure, convenience, radiation sparing, and allowance for repeat evaluation; also this diagnostic tool can be performed with less patient stress and restraint than radiography. It can be performed in either sternal or lateral recumbency, further contributing to minimal patient stress. In a study of TFAST evaluation of dogs after trauma, it was found to have a high specificity but poor sensitivity for pneumothorax detection versus conventional thoracic radiographs. TFAST was found to have a higher sensitivity and specificity in dogs with penetrating trauma than those with blunt trauma (see the article “Emergency Management and Treatment of the Poisoned Small Animal Patient” elsewhere in this issue, for more information).

Last, echocardiography is useful for assessment of chamber size and myocardial function, and can be performed in a cursory, limited fashion in the emergency setting to rapidly support a diagnosis of cardiac disease. In the emergency room, echocardiography can be used to assess the left atrial-to-aorta size ratio (LA:AO), which normally should be approximately 1:1. Increased left atrial dimensions are consistent with a cardiac cause of pulmonary infiltrates and respiratory distress (cardiogenic pulmonary edema). Short training courses for the use of echocardiography by the emergency room clinician have been shown reasonably effective to teach evaluation of left atrial enlargement. In some cases, diuretic therapy may result in a decrease in the size of the left atrium, which may make the subsequent determination of cardiac disease challenging. In all cases in which cardiac disease is suspected, a complete echocardiogram and cardiac evaluation, ideally by a cardiologist, should be performed as soon as the patient is stable.

Laboratory Evaluation

Several clinicopathologic tests are important in the patient with respiratory disease. The use of arterial blood gas (ABG) or venous blood gas (VBG) analysis will allow the clinician to assess both oxygenation and ventilation. In patients, in whom primary respiratory disease cannot be definitively ruled out from cardiac disease, the use of tests such as N-terminal prohormone of brain natriuretic peptide (NT pro-BNP) or possibly troponin can be considered.

Blood Gas Analysis

ABG analysis is considered the gold standard in assessing oxygenation and ventilation. The use of blood gases may assist the clinician in confirming the severity of respiratory dysfunction; however, it is unlikely to be able to definitively provide a diagnosis.

Hypoxemia, or decreased oxygen content of the blood, is confirmed by establishing a low partial pressure of oxygen dissolved in plasma (Pao2), typically lower than 80 mm Hg at sea level. The partial pressure of carbon dioxide (Paco2) on an ABG or
VBG may be used to assess adequacy of ventilation. A PaCO₂ greater than 40 mm Hg or partial pressure of CO₂ in mixed venous blood (PVCO₂) greater than 45 mm Hg is supportive of hypoventilation. In severe hypoperfusion, venous CO₂ is not an appropriate surrogate for arterial CO₂.

Historically, blood gas analysis has been available only at large referral or university hospitals, but in the past decade a variety of portable point-of-care machines have become affordable and available in practices. The I-STAT (Abbott Laboratories, Abbott Park, IL) model has been widely used and is generally considered accurate and user-friendly; the IRMA (ITC, Edison, NJ) is also popular. These devices (and other similar products) are able to measure blood gas values and other blood chemistries on small volumes of blood. Disposable cartridges make each test relatively economical and the turn-around time is excellent. Larger veterinary hospitals often used the Nova Biomedical Analyzer (Nova Biomedical, Waltham, MA). Other alternatives may include local human hospitals or regional referral hospitals. Guidelines for interpretation of blood gases are shown in Table 3.

Collecting an ABG may be challenging to obtain technically, particularly in a dyspneic patient or small patient. For example, obtaining an ABG in a distressed, open-mouth-breathing cat is not a viable option. Therapy should not be delayed while attempting an ABG, and a patient showing respiratory distress should not be removed from supplemental oxygen for assessment of oxygen levels (pulse oximetry or ABG analysis) while breathing room air, as the treatment plan should include supplemental oxygen.

As mentioned previously, blood gas analysis is useful in evaluating oxygenation and ventilation. Oxygen levels should always be interpreted in light of the PCO₂ level and the inspired oxygen concentration (FiO₂). It is also important to remember that even though an ABG analysis may look “normal,” it does not always imply normal oxygen delivery to tissues. The PaO₂ is an indicator of arterial oxygen tension and measures the dissolved oxygen in the blood (or whatever fluid is analyzed). Oxygen content of blood

| Table 3 |
The following guidelines have been adapted from the references and serve as the recommendation for interpretation from the American College of Veterinary Emergency and Critical Care |

<table>
<thead>
<tr>
<th>At Sea Level</th>
<th>Normal Value (Arterial)</th>
<th>Normal Value (Venous)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.36–7.44</td>
<td>7.32–7.36</td>
</tr>
<tr>
<td>PCO₂</td>
<td>36–40 mm Hg</td>
<td>40–45 mm Hg</td>
</tr>
<tr>
<td>PaO₂</td>
<td>90–100 mm Hg</td>
<td>40–50 mm Hg</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>20–24 mEq/L</td>
<td>24–28 mEq/L</td>
</tr>
</tbody>
</table>

**Clinical Guidelines for Compensation**

- **Metabolic acidosis**: Each 1 mEq/L decrease in HCO₃ will decrease PCO₂ by 0.7 mm Hg
- **Metabolic alkalosis**: Each 1 mEq/L increase in HCO₃ will increase PCO₂ by 0.7 mm Hg

**Respiratory acidosis**

- **Acute**: Each 1 mm Hg increase in PCO₂ will increase HCO₃ by 0.15 mEq/L
- **Chronic**: Each 1 mm Hg increase in PCO₂ will increase HCO₃ by 0.35 mEq/L

**Respiratory alkalosis**

- **Acute**: Each 1 mm Hg decrease in PCO₂ will decrease HCO₃ by 0.25 mEq/L
- **Chronic**: Each 1 mm Hg decrease in PCO₂ will decrease HCO₃ by 0.55 mEq/L
is defined as \((1.34 \times \text{hemoglobin concentration} \times \% \text{saturation}) + 0.003 \text{ PaO}_2\). Adequate oxygen delivery is dependent on normal cardiac function, normal hemoglobin concentrations, and the affinity that exists between oxygen and hemoglobin. Methemoglobin, and carboxyhemoglobin are 2 examples of altered hemoglobins that are markedly less effective in oxygen delivery.

Anemic animals will have lower oxygen content in the blood, and oxygen delivery in these patients is improved by transfusion of red blood cells rather than supplemental oxygen.

It is crucial that the clinician does not just look at the \(\text{PaO}_2\) values in interpreting blood gases, but rather to look as well at the \(\text{PCO}_2\) values. Low partial pressure of CO\(_2\) supports that the patient is hyperventilating to maintain oxygen values, whereas high partial pressure of CO\(_2\) supports respiratory fatigue or neuromuscular failure. The clinician should recall that providing supplemental oxygen to a patient with respiratory failure (high \(\text{PaCO}_2\)) will improve oxygen level, but often worsen hypercarbia.

When an ABG sample is obtained, an alveolar-arterial (A-a) gradient should be calculated to assess if pulmonary dysfunction exists. For example, a dog may be able to maintain a near normal oxygen concentration (\(\text{PaO}_2\) 80 mm Hg) by hyperventilating (as detected by a low \(\text{PCO}_2\) of 20 mm Hg), but have moderate to severe pulmonary dysfunction, A-a gradient equal to 45. Similarly, a dog with laryngeal paralysis may have severe hypoventilation (\(\text{PCO}_2\) of 70 mm Hg) and appear to be hypoxemic (\(\text{PaO}_2\) of 60 mm Hg), which in actuality, his A-a gradient is normal (3), indicating normal lung function. This can be detected by calculating the A-a gradient (Box 2) or by using the nonogram shown in Fig. 5.

Another useful screening tool to differentiate primary respiratory disease versus cardiac causes for respiratory distress is the NT pro-BNP. Chronic pressure or volume overload results in increased synthesis of pre-proBNP (a precursor molecule of BNP) by the ventricles, which is then processed to active BNP and inactive NT-proBNP. Multiple studies have evaluated the clinical utility of NT-proBNP and found that it can be used to detect occult cardiomyopathy in cats and to distinguish cardiac and noncardiac causes for respiratory signs in dogs.\(^{14,15}\) Species-specific assays must be used for NT-proBNP evaluation, because of variation in the protein structure between species.

Last, troponin is a biomarker that is released from ischemic myocardium. Troponin elevations have been appreciated with a variety of cardiovascular diseases, including hypertrophic cardiomyopathy and pericardial effusion. However, troponin increases are unlikely to be helpful in evaluation of the patient with respiratory distress.\(^{16}\)

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**Box 2**

**Calculation of the Alveolar to arterial gradient (A-a gradient)**

- **Alveolar oxygen content** is calculated using the Alveolar gas equation: 
  \[ \text{Alveolar oxygen content} = \left( \text{FiO}_2 \times \frac{\text{Barometric pressure - water vapor pressure}}{\text{PCO}_2/R} \right) \]

- \(R = \text{respiratory quotient, usually set at 0.8 or 0.9}\)
- \(\text{Barometric pressure is in mm Hg, typically 760 at sea level}\)
- \(\text{Water vapor pressure is 53 mm Hg at dog/cat body temperature, classically given at 47 for people}\)
- **Arterial content = measured using blood gas analyzer**
- **Normal <15 mm Hg**
Troponin is well-preserved across species, so human analyzers are acceptable for dogs and cats.

SUMMARY

Respiratory distress is a common presenting complaint for animals brought to the emergency room, and it is important for clinicians to feel comfortable diagnosing and treating these animals. Prompt recognition of the localization of the source of respiratory distress based on history, pattern recognition, and physical examination findings will help to determine the underlying cause and is key to determining an appropriate therapeutic course. Careful handling, minimizing stress, and rapid and focused treatment are crucial in the management of all patients in respiratory distress.

REFERENCES


Fig. 5. Arterial oxygen to arterial carbon dioxide nonogram. Match the patient’s PaCO₂ and PaO₂ values, and determine if the value is normal or abnormal. Abnormal values support pulmonary dysfunction. Elevated PCO₂ values alone may reflect simple ventilation failure.