It is essential that patients in respiratory distress be recognized immediately. Observation of the patient and a detailed physical examination of the respiratory system are the most important tools for diagnosis and treatment, often providing clues about the cause of dyspnea when more stressful diagnostics are not possible. The clinician should attempt to localize the disease process to the airways, lungs, or pleural space. Since dyspnea is often a life-threatening emergency, localization helps determine what steps need to be taken to stabilize the patient.

Overview of Approach to the Patient in Respiratory Distress
1. Perform a brief physical exam to try to identify the cause of dyspnea.
2. Stabilize the animal with O2 supplementation and minimize stress.
3. Perform diagnostics as indicated and as the patient’s status allows.
4. Provide definitive treatment for the underlying cause.

When an animal presents with an increased respiratory rate or difficulty breathing, the clinician must determine if it is due to primary respiratory disease or is secondary to another process. Respiratory changes occur frequently in small animal patients. It is important to recognize that not all respiratory changes are caused by diseases of the respiratory system. Changes also occur with other processes, including pain, stress, exercise, fever, hyperthermia, metabolic acidosis, anemia, obesity, abdominal distension, cardiovascular disease, nervous system disease, and musculoskeletal disorders. These are often referred to as “look-alikes” for respiratory disease. Basic physical examination findings such as observation and auscultation are often enough to make this distinction, but occasionally diagnostics such as thoracic radiographs, pulse oximetry, or blood gas analysis may be needed.

Observation of the Patient in Respiratory Distress
Dogs and cats with respiratory distress are recognized by an increase in respiratory rate and effort during respiration. Normal respiratory rate in dogs and cats is 20–30 breaths per minute. Panting is a form of tachypnea characterized by breathing through an open mouth, and is used by dogs as a means of thermoregulation. As air moves over the surface of the tongue, water evaporates and heat energy is dissipated. Some dogs will also pant when they are nervous, stressed, or painful. Because panting primarily involves the movement of air through dead space (portions of the respiratory tract not involved in gas exchange), animals that are panting are not hyperventilating; they have not increased their minute ventilation. If the animal is not panting, tachypnea is usually associated with an increase in respiratory effort and change in pattern. Respiratory rate can be monitored frequently and noninvasively in patients with respiratory disease to assess their progression and response to therapy.

Increased respiratory effort is a manifestation of recruitment of the secondary muscles of respiration, specifically the scalene and sternomastoid muscles of the neck and chest; the alae nasae, which dilate the nostrils; and the muscles of the abdominal wall, which contract when exhalation becomes active rather than passive. Recruitment of the secondary muscles of respiration is a nonspecific response to increased respiratory drive and does not necessarily confirm the presence of hypoxia or respiratory distress. Paradoxical respiration, however, is a specific indication of distress, increased work of breathing, and the presence of respiratory muscle fatigue. Normal respiration is characterized by concurrent outward movement of both the chest and abdomen during inhalation. Paradoxical respiration is recognized by a lack of synchronous movement of the chest and abdominal walls, with a tendency of the diaphragm, caudal intercostal, and abdominal muscles to collapse inward and forward during inhalation.

Postural adaptations are common in patients with respiratory distress, permitting the animal to minimize resistance to air flow. Many patients in severe respiratory distress breathe through an open mouth to remove the resistance to airflow posed by the nasal turbinates. Also, the neck is often extended and the head lifted to straighten the trachea. Most dyspneic patients demonstrate some degree of orthopnea, preferring to remain standing or in sternal recumbency, and abduct their elbows to minimize compression of the chest wall. Lateral recumbency can pose limitations to airflow by reducing the ability to abduct one side of the chest wall during inspiration. This is why many patients decompensate when restrained for diagnostics such as venipuncture or radiography; any restraint that limits postural adaptations can lead to further hypoxemia and decompensation of the patient.
Patterns of respiration can be helpful in determining the type of disease process causing respiratory distress. Animals with restrictive disease (parenchymal disease that stiffens the lungs such as severe pneumonia, fibrosis, or neoplasia or diseases that restrict expansion of the lungs such as air or fluid in the pleural space) cause a rapid, shallow pattern. Animals with obstructive disease (airway obstruction or narrowing) usually have slower, deeper respirations.

Noisy respiration is referred to as stertorous or stridorous and is usually associated with upper airway disease such as laryngeal dysfunction or tracheal obstruction. Wheezes are sometimes heard with lower airway disease.

**Physical Examination of the Dyspneic Patient**

Mucous membrane color and capillary refill time can yield very important information. Mucous membrane color is normally pink, but can range from brick red, white, or gray to purple or blue. Cyanosis only occurs at extreme levels of hypoxemia (\(\text{PaO}_2 < 65 \text{ mm Hg}\)) and indicates the need for immediate O2 supplementation; therefore, a patient can be hypoxemic and still have pink mucous membranes. Many animals in respiratory distress have pale mucous membranes caused by concurrent anemia or shock. Pale mucous membranes pose an additional challenge: with insufficient hemoglobin perfusing the periphery due to anemia or vasoconstriction, cyanosis cannot be appreciated.

A limited physical examination of the respiratory and cardiovascular systems can be performed rapidly and can be very rewarding. The chest and the cervical trachea should be ausculted for increased airway sounds, wheezes, crackles, harshness or increased broncho-vesicular sounds, or areas of dullness (dorsal, ventral, unilateral). Wheezes are musical or squeaky sounds associated with narrowing of the airways by inflammation, mucosal edema, mucus, or masses. Inspiratory wheezes indicate an upper airway problem, while expiratory wheezes indicate disease of the small bronchi or lower airways such as feline asthma. Crackles are discontinuous popping sounds, which indicate the presence of fluid in the alveoli and airways. They are caused by air bubbling through fluid or by the opening and closing of small bronchi and alveoli. Crackles occur with parenchymal disease processes and often indicate pulmonary edema, hemorrhage, or purulent exudate in the alveoli. If the lung or heart sounds are dull, muffled, or difficult to hear, pleural space disease should be considered. The most common pleural abnormalities include pneumothorax, pleural effusion, diaphragmatic hernia, and neoplastic masses. In cats, diminished chest compressibility can be a useful finding when an effusion or mediastinal mass are suspected.

Auscultation of the heart and simultaneous palpation of the pulses help to determine whether the cardiovascular system may be a cause of respiratory distress. In dogs with congestive heart failure, a murmur or arrhythmia is usually ausculted. In the absence of these findings, heart failure is an unlikely cause of the respiratory distress. Cats can be more difficult; murmurs and arrhythmias may be absent or intermittent in cats with heart failure.

**History**

A complete medical history should be obtained from the owner to help complete the initial evaluation of the patient in respiratory distress. This should include information on duration of the clinical signs, the rate of onset, any history of cough (including if it is productive or nonproductive) or collapse, exercise intolerance, the patient’s vaccination status, and any exposure to other animals or toxins. This information can provide valuable clues to the underlying cause of the respiratory distress in a patient in which diagnostics must be carefully chosen and prioritized due to the severity of the respiratory compromise.

**Emergency Stabilization**

Initial stabilization of a patient in severe respiratory distress should consist of O2 therapy while an abbreviated physical exam is performed. Ideally, the patient should be allowed to rest briefly in an O2-enriched environment before further diagnostic testing and manipulation. This is particularly important for cats, as it allows the animal to recover from transport, to calm down and minimize the O2 expenditure required for struggling, and to increase the O2 level in the blood. When and if the animal can tolerate it, a more complete examination should be performed.

Emergency O2 therapy can consist of an O2 cage for small animals or mask or flow-by for larger dogs. Mask O2 can be used on any patient that is lying still and tolerates the mask. Some agitation patients refuse to accept it, and persistent attempts to place the mask over the muzzle of the animal can create stress. Oxygen cages are one of the easiest methods of O2 administration, but they isolate the patient, making it difficult to examine, monitor, and treat these dynamic cases. Stressed dyspneic cats, however, can benefit from enforced isolation from strange people in a strange environment. The O2 percentage in the cage is determined by the extent of filling with 100% O2. For more
prolonged $O_2$ supplementation, nasal $O_2$ can be provided with nasal prongs that are manufactured for human patients, or by placing a catheter in one or both nostrils and suturing or gluing it in place.

Vascular access with a short peripheral catheter in the cephalic or saphenous vein should be prioritized early during the hospitalization of all dyspneic patients. The catheter should be placed with minimal restraint and stress. Once vascular access is established, intravenous drugs can be administered, and should the animal decompensate, intravenous anesthesia is possible to facilitate rapid control over the airway.

If dullness is suspected on auscultation either dorsally or ventrally, thoracocentesis should be immediately performed to remove pleural air or fluid. In animals with severe distress, this procedure should be performed before radiographs are obtained. Thoracocentesis can be both therapeutic and diagnostic.

**Diagnostics**

**Thoracic Radiographs**

Thoracic radiographs are one of the mainstays for diagnostic work-up of a patient in respiratory distress. No other single diagnostic can give so much information on the anatomical location of the problem and lead to a list of differential diagnoses. However, thoracic radiographs are not without risk, and in severely dyspneic patients may lead to decompensation and the demise of the patient.

**Pulse Oximetry**

Pulse oximetry is used to indirectly determine the arterial hemoglobin saturation with $O_2$. Normal hemoglobin saturation is greater than 95%, and pulse oximetry readings of 93% or higher are acceptable in critically ill patients. $O_2$ supplementation should be considered in patients with hemoglobin saturation less than 93%. Saturation values of 90% correlate with a PaO$_2$ of approximately 60 mm Hg and indicate a serious degree of hypoxemia. Values less than 90% should be addressed immediately by providing supplemental $O_2$. The pulse oximeter is a dual wavelength spectrophotometer that functions by transmitting light through a pulsating arterial vascular bed. Transmission of light through tissue is not constant, but varies with each pulse. The variation in transmitted light is due to arterial blood, with the contribution of venous blood and tissue remaining constant. By using the appropriate transmitted light wavelengths for oxyhemoglobin and deoxyhemoglobin, the microprocessor can continuously calculate $O_2$ saturation.

In cats and dogs, the small probe can be placed on the pinna of the ear, the lip, or a fold of skin at the axilla or the inguinal area. The pulse oximeter is noninvasive and very well tolerated by the majority of animals. It provides a continuous readout of hemoglobin $O_2$ saturation and pulse rate; thus it is a useful tool for continuous monitoring of the hypoxemic patient. When arterial blood gas analysis is unavailable, or when arterial blood cannot be obtained, the pulse oximeter can provide a useful indication of arterial saturation and a means of assessing disease progression.

The major limiting factor in the use of pulse oximetry is tissue perfusion. Any condition that diminishes tissue blood flow, such as hypotension or shock, will prevent the pulse oximeter from accurately reading and measuring hemoglobin saturation. Motion of the probe also can decrease the ability to obtain a signal. Despite these limitations, the pulse oximeter provides a clinically useful and simple measure of hemoglobin saturation.

**End-tidal Capnography**

End-tidal capnography provides a noninvasive, indirect means to estimate CO$_2$ concentrations, and therefore to assess the adequacy of ventilation. The CO$_2$ is measured in exhaled air. Since CO$_2$ diffuses so easily and equilibrates very quickly across the alveolar surface, the CO$_2$ in alveolar air at end-exhalation is a close approximation of plasma CO$_2$ concentration. Normal values should be in the 35–45 mm Hg range. If a value of greater than 50 mm Hg is obtained, urgent measures should be taken to improve the ventilation of the patient. End-tidal capnography is useful for monitoring respiration in intubated patients.

**Arterial Blood Gas Analysis**

Arterial blood gas analysis is the gold standard for evaluation of oxygenation and ventilation. PaO$_2$ is the partial pressure of oxygen dissolved in the arterial blood. It is a measure of oxygenation, not ventilation. Normal PaO$_2$ is 90–100 mm Hg. A PaO$_2$ of less than 80 mm Hg is considered hypoxemia, and less than 60 mm Hg represents a severe compromise to tissue oxygenation. PaCO$_2$ is the partial pressure of carbon dioxide dissolved in the arterial blood. It gives the best measure of the patient’s ability to ventilate. It is important to remember that CO$_2$ is approximately 20 times more diffusible than $O_2$, making it much easier for a patient to maintain normal CO$_2$.  

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concentrations. The PaCO\textsubscript{2} is evaluated to determine if there is a respiratory acidosis or alkalosis. A PaCO\textsubscript{2} of greater than 45 mm Hg is a respiratory acidosis, and a PaCO\textsubscript{2} of less than 35 mm Hg is a respiratory alkalosis.

**Additional Diagnostics**
Complete evaluation of the patient, including a complete physical examination (once stabilized), complete blood count (CBC), chemistry screen, and urinalysis should also be obtained. These may help in identifying underlying or concurrent disease processes. The CBC is useful in evaluating the patient’s immune response. Fecal analysis for parasites including lungworm may be indicated in some patients. Tracheal wash, either trans-tracheal or endotracheal, is indicated in evaluation of patients with either lower airway disease or pulmonary parenchymal disease. Laryngoscopy and bronchoscopy are useful in evaluation of the larynx, pharynx, trachea, and bronchi. Additional imaging including ultrasound and CT is useful in select cases.

**Ventilation**
Ventilation is the movement of air in and out of the lungs, allowing gas exchange to occur. It requires that the chest wall and diaphragm be working adequately to maintain oxygenation and CO\textsubscript{2} removal. For normal ventilation to occur, the animal must have a normal brain stem respiratory control center, normal spinal cord function to the level of C4/C5, normal spinal and phrenic nerve function and neuromuscular transmission, normal muscle and chest wall integrity, absence of pleural space disease, and a patent airway. If there is an abnormality of any of these functions, inadequate tidal volume may occur, resulting in hypoventilation. Hypoventilation can occur if there is depression of the respiratory center in the brain stem from edema or respiratory depressant drugs such as anesthetics; cervical spinal trauma; IVDD; FCE or neoplasia; neuromuscular disease affecting the chest wall and/or diaphragm; restrictive disorders preventing lung expansion such as pleural space disease; or lesions such as flail chest, which affect the ability of the animal to move adequate quantities of air. Severe pulmonary disease such as pneumonia can also occasionally lead to hypoventilation, but this is unusual.

Because CO\textsubscript{2} diffuses through tissues very easily, the amount of CO\textsubscript{2} in arterial blood (PaCO\textsubscript{2}) is used as a measure of ventilation. Normal dogs and cats have a PaCO\textsubscript{2} of 35–45 mm Hg. Hypoventilation causes an increase in CO\textsubscript{2} and hypoxia, since there is less O\textsubscript{2} in the alveoli for gas exchange. If hypoventilation occurs, CO\textsubscript{2} increases, leading to respiratory acidosis. Hypoventilation can often be managed by treatment of the underlying problem such as reversing anesthetic drugs or relieving an airway obstruction. Providing a higher inspired concentration of O\textsubscript{2} will not improve the movement of air into the chest and therefore is ineffective for the treatment of hypoventilation. If conservative methods are not adequate for the management of the hypoventilating patient, then positive pressure ventilation is the only effective option.

**Suggested Reading**