TRIAGE AND APPROACH TO THE PATIENT WITH RESPIRATORY DISTRESS

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Disorders of the respiratory system can broadly be classified based on the location of the primary pathology. The clinician must first quickly assess the patient from afar, evaluating what respiratory pattern the patient is exhibiting. An obstructive respiratory pattern is associated with inspiratory dyspnea, often with harsh upper airway noise called stridor on inspiration. Diseases associated with upper airway obstruction include laryngeal paralysis, tracheal collapse, oropharyngeal masses including polyps, abscesses, cysts, foreign bodies, pharyngeal collapse and everted saccules, and various forms of neoplasia. In many cases, animals with upper airway obstruction are extremely stressed and may easily become hyperthermic due to anxiety and increased respiratory effort.

In many cases of upper airway obstruction, supplemental oxygen and anxiolytic agents such as acepromazine (0.025 - 0.1 mg/kg IV, IM) should be administered with minimal handling until the anxiolytics take effect. If the obstruction is life-threatening, immediate airway control must be established. Intravenous anesthetic agents such as propofol (4 - 7 mg/kg IV), Thiopental (8.8-13.2 mg/kg), or ketamine 5.5 mg/kg IV with 0.3-0.5 mg/kg diazepam IV) should be administered. The cause of the upper airway obstruction can then be determined, airway control taken through intubation or other supportive measures, and a more complete physical examination be performed. Patient signalment combined with a thorough history is often helpful in guiding your list of differential diagnoses. How quickly have the clinical signs become apparent? Has there been any change in bark or voice? Does the animal have difficulty swallowing? Are the clinical signs worse with exercise? Has there been exercise intolerance? Are there any abnormal sounds associated with breathing? What does it sound like? A goose honk or a harsh wheezing in the animal's throat?

Larger, older breeds such as Labrador and Golden Retrievers are predisposed to the development of laryngeal paralysis, although congenital forms have been documented in Bouvier des Flandres and Siberian Huskies. Small breed dogs such as Pomeranians and Yorkshire Terriers are predisposed to the development of tracheal collapse. Bracycephalic breeds such as pugs, Pekingese and Boston terriers most frequently develop bracycephalic airway syndrome with elongated soft palates, stenotic nares, and hypoplastic tracheas. With time and chronicity, everted saccules and laryngeal collapse may occur.

Diagnostic tests that can cause stress to the patient should be avoided at all costs until the patient's respiratory status is more stable. Then, cervical and thoracic radiographs should be performed, with fluoroscopy if possible to identify pharyngeal or tracheal foreign bodies or the presence of a dynamic airway collapse. In cases where laryngeal paralysis is suspected, evaluation of laryngeal function should be performed with the patient under heavy sedation. Following heavy sedation to induction of a light plane of anesthesia, the larynx should be evaluated using a laryngoscope during all phases of respiration. Doxapram hydrochloride (1 - 5 mg/kg IV) can also be used to assess laryngeal function. Normally, the vocal folds and arytenoid cartilages abduct or open in the airway during inspiration. With laryngeal paralysis, the vocal fold remains adducted in the tracheal lumen during inspiration, causing obstruction. Many cases of unilateral laryngeal paralysis may go clinically unnoticed until bilateral disease is present.

Evaluation of the airway may reveal a tracheal or pharyngeal foreign body. If the foreign body can easily be grasped with the patient under anesthesia, definitive removal can be performed. Alternatively, a Heimlich maneuver can be performed by holding the animal vertically with its rear limbs touching the ground. Grasping or hugging the thoracic cage from behind, compression of the sternum in rapid succession several times may dislodge the foreign body rostrally. Oxygenation of the patient is of paramount importance during any process. Tracheal oxygen insufflation can be performed using a 20-gauge needle connected to a piece of IV extension tubing. The needle or catheter can be inserted into the trachea distal to the point of suspected obstruction, and the tubing attached to an oxygen source connected with a Christmas tree adapter or a 1 mL syringe. Oxygen flow rates can be delivered at 8 – 10 L/minute through the tubing.

In rare cases, a temporary tracheostomy will need to be performed until definitive repair or stabilization occurs. The patient is positioned in dorsal recumbency and the ventral cervical region quickly clipped and scrubbed. Two per cent lidocaine can be injected through the skin to the level of the trachea for local anesthetic block of the region. A small vertical skin incision is made with a number 10 scalpel blade just caudal to the larynx. The fascia is incised then the sternohyoid muscles bluntly separated using the tips of a mettzenbaum scissors or hemostats. The fascia overlying the trachea is grasped with a thumb forceps and dissected away from the trachea, taking care to avoid lacerating the vessels supplying the thyroid glands. A stay suture is placed through the 4^{th} or 5^{th} tracheal ring, taking care to not enter the lumen of the trachea. A horizontal incision is made in between the 3^{rd} and 4^{th} or 4^{th} and 5^{th} tracheal rings, taking care to avoid cutting more than 50% of the circumference of the trachea. The stay suture is used to open the incision, and a tracheostomy tube placed into the trachea. A Shiley tracheostomy tube or a modified endotracheal tube can be placed. The tube is secured around the neck with umbilical tape, and the area loosely wrapped with gauze 4 x 4's and bandage material. Supplemental oxygen can be delivered through the tube. In many cases, bypassing the upper airway obstruction will cause marked improvement in respiratory status and lessen patient anxiety.

The second pattern of respiration is a restrictive respiratory pattern, characterized by rapid shallow breaths with or without an abdominal push on expiration. A restrictive respiratory pattern may be observed with feline lower airway disease, pneumonia, pulmonary edema, and diseases of the pleuritis or thoracic cage, including pleural effusion, diaphragmatic hernia, and rib fractures. Like the patient with upper airway obstruction, patients exhibiting signs of restrictive respiratory pattern are very fragile and require minimal handling. Allowing the patient to relax in an oxygen cage or hood may be necessary before a complete physical examination can be performed. Once the patient is less stressed, the thorax should carefully be ausculted. Lungs may sound harsh with crackles if pulmonary edema, pneumonia, or pulmonary contusions are present. Conversely, lung and heart sounds may be muffled if air or fluid is present within the pleural space. Restrictive respiration may also be present due to pain of rib fractures. In some cases, paradoxical chest wall motion may be observed if a flail segment is present. With paradoxical chest wall motion, the flail segment moves inward during inspiration and out during exhalation.

Once a respiratory pattern has been identified, the next step is to determine whether an animal is oxygenating and ventilating adequately. Oxygenation refers to the process by which oxygen is brought into the lungs, travels across the alveolar membrane, and binds to hemoglobin to be delivered to the tissues throughout the body. A noninvasive means of determining oxygenation is through the use of pulse oximetry. A pulse oximeter uses the science of spectrophotometry with two wavelengths of light to distinguish between oxygenated (oxyhemoglobin) and deoxygenated hemoglobin (deoxyhemoglobin) in pulsatile blood. In most cases, pulse oximetry or arterial oxygen saturation (S_aO_2) corresponds reliably to the oxyhemoglobin dissociation curve. Oxygen saturation greater than 90% corresponds to a $P_aO_2 > 60$ mm Hg. Above this value, large changes in P_aO_2 are reflected in relatively small changes in S_aO_2, making pulse oximetry a relatively insensitive method of determining oxygenation status when P_aO_2 is normal. Pulse oximetry is also unreliable when severe vasoconstriction, hypothermia, excessive ambient lighting, shivering or trembling, methemoglobinemia or carbon monoxide intoxication are present. A more reliable indicator of oxygenation is to obtain an arterial blood sample from a dorsal pedal or femoral artery. When an arterial sample is obtained, one must first make sure that the sample truly is arterial versus venous. Percent saturation (S_aO_2) > 80% is characteristic of an arterial sample, while S_aO_2 < 80% may be venous or mixed venous.

Causes of low P_aO_2 or hypoxemia include decreased fractional inspired oxygen (F_iO_2), hypoventilation, ventilation-perfusion mismatch, physiologic shunting, or diffusion impairment. Numerous causes of hypoxemia exist. The severity of the hypoxemia relative to F_iO_2 by calculating an alveolar to arterial oxygen gradient (A-a gradient) or an oxygenation fraction (P_aO_2/F_iO_2) if the patient is receiving supplemental oxygen. An A-a gradient can be calculated using the formula: $A = (BP-47)F_iO_2 - P_aCO_2/0.8$
where BP = barometric pressure, 47 = water vapor pressure, and P_aCO_2 = arterial carbon dioxide tension in mm Hg. Arterial oxygen (P_aO_2) is then subtracted from the alveolar oxygen (A), to yield an A-a gradient. Values < 10 are normal. 10 – 20 may be normal in aged patients, and > 30 are suggestive of acute respiratory distress, or ARDS. In animals receiving supplemental oxygen, an oxygenation fraction can be calculated by dividing PaO_2 by F_iO_2. Values > 400 are normal, 200 – 300 are suggestive of acute lung injury, and values < 200 are characteristic of severe hypoxemia and ARDS. Improvement in oxygenation is often observed in cases of pulmonary edema, hypoventilation, physiologic shunting, ventilation-perfusion mismatch, and pulmonary thromboembolism. The most dramatic improvement in oxygenation with supplemental oxygen is often observed in PTE cases, and is known colloquially as an "oxygen step-up". Venous blood gas samples serve little useful information when assessing patient oxygenation. However, comparing arterial to venous oxygen P_aO_2 to P_vO_2 can be useful in determining oxygen extraction. Normally, venous O_2 levels are 35 – 50 mm Hg when the patient is breathing room air. If oxygen extraction is impaired, as with sepsis, venous O_2 will be elevated, sometimes > 60 mmHg. Conversely, low venous O_2 levels < 35 mm Hg indicate large consumption of oxygen, and possible poor perfusion. Levels < 24 mm Hg are suggestive of anaerobic metabolism, which can be confirmed by measuring venous lactate.

The efficiency of ventilation is evaluated using the P_aCO_2 value on an arterial blood sample. Increases in P_aCO_2 is known as hypercapnia, and is caused by hypoventilation. Decreases in P_aCO_2 is known as hypocapnia, and is caused by hyperventilation. Potential causes of hypoventilation include anesthesia, pain, central nervous system disorders, central depression of respiratory drive, peripheral neuromuscular disease including Coonhound Paralysis and Botulism, and administration of supplemental oxygen in a chronically hypoxemic patient. Hyperventilation can be caused by pain, rib fractures, anxiety, sepsis, hyperthermia, and central nervous system disease. In intubated patients, an end-tidal CO_2 monitor can be placed on the end of the endotracheal tube. The science of capnometry uses a spectrophotometer to measure carbon dioxide levels of exhaled gas. When placed in graphic form, a waveform known as a capnograph is displayed. Normally, at the onset of exhalation, the gas exhaled into the expiratory limb of the tubing comes from the upper airway or physiologic dead space, and contains relatively little CO_2. As exhalation continues, a steep uphill slope occurs as more CO_2 is exhaled from the bronchial tree. Near the and of exhalation, the capnogram reaches a plateau, which most accurately reflects the CO_2 level at the level of the alveolus. Because CO_2 diffuses across the alveolar basement membrane so rapidly, this reflects arterial CO_2 levels. If a plateau is not reached and notching of the waveform occurs, the system should be checked for leaks. If the baseline waveform does not reach zero, the patient may be rebreathing CO_2, or may be tachypneic, causing physiologic positive end-expiratory pressure (PEEP). The soda-sorb in the system should be replaced if it has expired. Conversely, low end-tidal CO_2 may be associated with a decrease in perfusion or blood flow. Carbon dioxide is transported from the periphery to the lungs, where it normally is exhaled. Decreased perfusion can be associated with low $ETCO_2$ values, particularly during cardio-pulmonary-cerebral resuscitation. End-tidal CO_2 levels are one of the most accurate predictors of the efficacy of cardiopulmonary-cerebral resuscitation and patient outcome.

Arterial blood gas analysis, pulse oximetry, and capnometry can be very useful in determining the efficacy of patient response to therapy and when mechanical ventilation is necessary. A useful guideline for using mechanical ventilation in a patient is known as the "Rule of 60". If $P_aO_2 < 60$ mm Hg on 40% or greater supplemental oxygen, if $P_aCO_2 > 60$, or if the patient is experiencing severe respiratory distress or fatigue, mechanical ventilation is necessary. Mechanical ventilation can improve hypoxemia caused by hypoventilation by increasing respiratory rate and tidal volume. Positive end-expiratory pressure (PEEP) can prevent airway closure and atelectasis and further improve oxygenation. Improvements in ventilation can also be measured using P_aCO_2 levels and $ETCO_2$ monitoring.

Acid-base status also can be evaluated using an arterial blood sample. First, looking at the pH of the sample, the clinician can determine whether an acid-base disturbance exists. Normal pH is 7.40 +/- 0.04. Values < 7.36 indicate acidosis, whereas values > 7.44 indicate alkalosis. Next, looking at the acid-base excess (ABE), the clinician can tell if the problem is primarily metabolic or respiratory in nature. Normal values for ABE are –1 to +1. Values < -1 indicate that bicarbonate (HCO_3^-) levels are decreased or that other unmeasured anions are present in excessive quantities, and that a metabolic acidosis is present. Conversely, values > +1 indicate that HCO_3^- is present in excess, and that a metabolic alkalosis is present. Causes of metabolic acidosis are numerous, and include ingestion of salicylates (aspirin), ethylene glycol, methanol, uremia, diabetic ketoacidosis, and lactic acidosis. Lactate can easily be measured using hand-held monitors purchased from sporting-goods stores. Normal lactate values for dogs are < 2.0. Lactate values > 6.0 have been negatively correlated with survival in dogs with critical illness, including gastric dilatation-volvulus syndrome. Metabolic alkalosis is observed less commonly in veterinary medicine, but can be caused by excessive vomiting of upper gastrointestinal contents, as with a high duodenal or pyloric outflow obstruction, or due to excessive use of loop diuretic agents such as Furosemide that prevent bicarbonate reabsorption. If blood pH is < 7.1, supplementation with HCO_3^- should be considered, as severe acidosis can result in decreased enzymatic activities, poor cardiac contractility, and ventricular arrhythmias. The bicarbonate deficit, or the amount of bicarbonate to supplement can be calculated using the formula: HCO_3^- (mEq) = 0.3 x Body Weight in kg x base excess (ABE). This author only supplements ¼ of this calculated value over 15 minutes, then replaces ¼ in intravenous fluid over several hours. Oversupplementation of HCO_3^- can create a metabolic alkalosis that may be difficult to treat.

Evaluation of a patient's respiratory status, administration of supplemental oxygen, and monitoring patient response to therapy is a necessary tool both in emergency medicine and day-to-day practices, including administering general anesthesia. Pulse oximetry and capnometry are non-invasive, relatively inexpensive means of monitoring respiratory status on a breath-by-breath or moment-to-moment basis. Arterial and venous blood samples, too, are useful in measuring patient oxygenation, ventilation, and acid-base status, and can be used to guide both oxygen and fluid therapy.

References available upon request.