JUST BREATHE - RESPIRATORY EMERGENCIES
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Respiratory distress is a very common form of emergency in veterinary medicine. The primary role of the respiratory system is to oxygenate and control CO2 in the blood. Inability for patients to properly oxygenate blood and saturate hemoglobin (hypoxemia) will lead to inadequate delivery of oxygen to the tissues (hypoxia). In a hypoxic state, cellular energy production is shifted primarily to anaerobic metabolism, resulting in lactic acid buildup and acidemia (metabolic acidosis). In addition, insufficient alveolar ventilation will lead to an elevation in the arterial CO2 level, or hypercapnia. Hypercapnia leads to respiratory acidosis, decreases cardiac contractility, and depresses diaphragmatic function. Both of hypoxemia and hypercapnia, when allowed to persist, will lead to the demise of a patient and swift assessment of respiratory compromise is required for appropriate treatment.

Initial Assessment and Treatments

Assessment of a patient starts from external physical signs. Patients presenting with signs such as tachypnea, increased respiratory effort, and open-mouth breathing are clearly in trouble. Exaggerated movement of parts of the body surrounding the physical construct of the airway such as flaring nostrils, lip movement with respiration, sucking in and out of the skin under the chin and thoracic inlets, and paradoxical abdominal movement are all signs of severe respiratory distress. An orthopneic position, characterized by open-mouth breathing, extending of the head and neck, sitting up sternal, and abduction of the elbows in the effort to open up the airway as much as possible, is another common external sign of respiratory distress. If the patient progresses to being unable to hold themselves up, going into lateral recumbency with no improvement in respiratory signs, the patient may be experiencing respiratory fatigue and facing imminent arrest.

Assessment and treatment of a patient in respiratory distress poses a dilemma, as swift determination of the patient's problem is required, yet they may be compromised such that the stress of diagnostics and treatment may push them into respiratory and cardiac arrest. These patients are in a very fragile state, and initial efforts are aimed at improving the patient's ability to breathe while minimizing stress and deterioration in respiratory status. Providing oxygen supplementation through flow-by, mask, induction chamber, or cage would be one of the first lines of therapy to alleviate distress.

Patients in respiratory distress are often very anxious, which often makes the patient even more dyspneic. A light sedation with small doses of benign sedative such as butorphanol may be beneficial to help ease anxiety. The staff working on the patient should conduct themselves in a calm and quiet manner yet maintaining swiftness. A calmer environment will not only benefit the patient, but may benefit a worried owner. The presence of the owner can either be beneficial or detrimental to the patient, and staff directing attention to calming a panicked client (and successfully doing so) may also help the patient.

Diagnostics and treatment such as physical examination, radiographs, blood work and IV catheterization may have to be held off until the patient is more relaxed and breathing better. Evaluation of a patient's respiratory problem begins with external visualization of their breaths. The manner in which a patient breathes is adapted to the method requiring the least work of breathing. An obstructive breathing pattern, involving a prolonged inspiration (upper airway) or expiration (intrathoracic lower airways), will be observed in patients with narrowed

airways. A restrictive breathing pattern, involving shallower but tachypneic breathing, will be observed in pleural disease or reduction of lung compliance. Abdominal effort may be seen with patients with compromised lungs. Certain conditions (anemia, metabolic acidosis, and pain, for example) can cause "non-respiratory look-alikes".

Auscultation is a valuable skill in early detection and detection of change in lung states. Stertor (snoring), wheezes (whistling), and stridor (high pitched noise) can indicate different upper airway issues. Crackles indicate fluid in the alveoli, such as in pneumonia or pulmonary edema. The location lung sounds are present or absent in helps indicate causes as well. Cardiogenic pulmonary edema often begins near the heart (perihilar region), and aspiration pneumonia often originate in the cranioventral lobes. Absence or decrease in lung sounds in the caudal and ventral fields may indicate pleural effusion, while dorsal fields may be due to pneumothorax. While abnormalities in auscultations do not lead to a diagnosis, it serves as an indication for further diagnostics.

If pleural space issues like pleural effusion or pneumothorax is suspected, performing thoracocentesis to evacuate the fluid or air can provide diagnostic information and therapeutic treatment simultaneously. The staff should be prepared to perform endotracheal intubation and provide positive pressure ventilation (PPV) if the patient does not stabilize with initial treatment and progressively gets worse.

TYPES OF RESPIRATORY EMERGENCIES

Upper Airway Problems

Upper airway issues leading to respiratory distress are common in veterinary medicine, and can involve various causes. Laryngeal paralysis is a common in older, larger breed dogs, with a higher prevalence in males. A loss in innervation of the cricoarytenoideus dorsalis muscle leads to atrophy, preventing the arytenoid cartilage from being abducted. This narrows the laryngeal opening, leading to an increase in airway resistance. Causes may be congenital, or be due to trauma, neuromuscular disease, neoplasia, hypothyroidism, or idiopathic. Affected dogs exhibit inspiratory stridor, exercise intolerance, ptyalism, and a change in their bark. Laryngeal paralysis can cause severe respiratory distress and collapse depending on severity. Medical management may be possible with the goals of minimizing stress, excitement, and exposure to high environmental temperature. Surgical treatment is most effective, involving unilateral lateralization of the arytenoid cartilage, or laryngeal tie-back. Chances of aspiration pneumonia are increased in dogs undergoing this procedure. Cats rarely present with laryngeal paralysis, though a study suggests laryngeal paralysis a differential for cats with dyspnea, inspiratory stridor, coughing/gagging, or a change in voice, with complete resolution through unilateral lateralization.

Brachycephalic syndrome results from anatomical abnormalities seen in brachycephalic breeds leading to upper airway narrowing or obstruction. Stenotic nares, elongated soft palate, and a hypoplastic trachea provide a narrowed airway and force the animals to create larger negative pressure to breath adequately. A further increase in negative pressure may evert the laryngeal saccules and collapse the larynx or trachea. Pulling of air through the narrowed airways can further cause inflammation and edema, placing the patient in further respiratory dysfunction. Surgical intervention through widening of the nares, resection of the soft palate and everted laryngeal saccules is the recommended treatment. Upper airway obstruction can also occur due to lodging of a foreign body, neoplasia, or the formation of nasopharyngeal polyps. Any upper airway dysfunction or obstruction can lead a patient to present with

respiratory distress. Secondary complications such as non-cardiogenic pulmonary edema, heat stroke, and aspiration pneumonia may be seen.

Pulmonary Edema
Pulmonary edema is a common cause of respiratory distress in dogs and cats. Accumulation of extravascular fluid occurs in the alveoli and pulmonary parenchyma due to increased hydrostatic pressure or increased permeability in the pulmonary vasculature. Patients present with respiratory distress and have poor oxygenation. The reduced oxygenation is due to a ventilation-perfusion mismatch (V/Q mismatch) because the presence of fluid in the alveoli leads to compromised ventilation.

Left sided heart failure can lead to pulmonary hypertension, causing cardiogenic pulmonary edema. In cardiac disease, fluid retention and an increase in blood volume is seen as a compensatory mechanism for lowered cardiac ouput. The chronic increase in blood volume leads to an increased hydrostatic pressure (because of congestion) in the pulmonary vasculature, resulting in pulmonary edema. Patient with cardiogenic pulmonary edema may show signs of coughing, exercise intolerance, and may have a heart murmur. An echocardiogram may be performed to confirm cardiac disease and pulmonary hypertension. Fluid volume overload through fluid therapy is a possible cause of cardiogenic pulmonary edema, especially in patients with cardiac or kidney disease. Both cardiac and kidney disease can be asymptomatic, so patients on fluid therapy should be closely monitored for signs of fluid overload.

Non-cardiogenic pulmonary edema can occur because of increased permeability within the lung tissue through damage to the microvasculature or alveolar epithelium. Electrocution, seizures, strangulation, pulmonary thromboembolism, and chemical exposure are all potential causes. Illnesses associated with systemic vasculitis such as sepsis and systemic inflammatory response syndrome (SIRS) are also associated with non-cardiogenic pulmonary edema.

Patients with pulmonary edema are treated with oxygen supplementation to alleviate hypoxemia and improve oxygen delivery. Patients that are unable to maintain an arterial partial pressure of oxygen (PaO2) greater than 60mmHg despite oxygen supplementation may require endotracheal intubation and provided PPV (manual or mechanical). An arterial blood sample and a blood gas analyzer are required to obtain a PaO2 measurement. Placement of an arterial catheter, if possible from a patient stress level and staff technical skill level standpoint, is beneficial in serial monitoring of PaO2. PPV is also indicated if the arterial partial pressure of CO2 (PaCO2) is greater than 60mmHg. A venous sample is acceptable in measuring CO2 levels (PvCO2 for venous) and is typically within 5mmHg from arterial values. Some patients may have a positional "preference" in their ability to oxygenate and ventilate, with sternal recumbency usually being most beneficial.

Medical management of the cause of pulmonary edema is warranted in conjunction with respiratory support. Diuretics are administered to reduce pulmonary capillary pressure and reduce preload through reduction of blood volume. Furosemide is a commonly used diuretic due to its rapid onset. In addition to its diuretic effect, furosemide may have further beneficial effects of pulmonary vasodilation and bronchodilation. Hemoconcentration resulting from reduced intravascular volume increases the plasma colloid osmotic pressure, helping the removal of fluid from the alveoli. Nitroprusside and glycerol trinitrate are vasodilators used as additional methods in reducing hydrostatic pressure. Bronchodilators such as terbutaline may

also be used, and fluid therapy restricted. Chances of resolution depend heavily on the cause, and treatment for the specific underlying disease is required.

Pleural Space Disease

When the pleural space, which normally serve as a potential space to create negative intrathoracic pressure during breathing, is filled with material which normally do not exist, normal breathing is compromised. The material may be various types of fluid, air, or even organs. The pleural space being occupied by these abnormal substances will cause the lunges to collapse and prevent adequate inflation, leading to a decrease in tidal volume, total vital capacity, and functional residual capacity. The lung volumes lead to hypoventilation, which can result in hypoxemia and hypercapnia.

Accumulation of fluid in the pleural space is called, pleural effusion. There are numerous types of fluid which can accumulate in the pleural space. Hydrothorax, or accumulation of transudate can be a result of reduced plasma colloid osmotic pressure, increased hydrostatic pressure, increased vascular permeability, or neoplasia. Transudate is defined as effusion containing TP < 2.5g/dl and total nucleated cell count (TNCC) < 1500/µl. Effusion with TP between 2.5 and 7.5 g/dl and TNCC between 1000-7000/µl is considered to be modified transudate. Effusion with TP > 3.0g/dl and TNCC > 7000/µl is defined to be exudate.

Feline infectious peritonitis (FIP), caused by a coronavirus can cause exudative or modified transudate effusion with yellow to straw-colored, viscous fluid with a high protein but low TNCC. Pyothorax is an accumulation of purulent exudate in the pleural space. Causes include bacterial infection due to migrating inhaled foreign objects, penetrating trauma to the chest wall, pneumonia, migrating plant material, and iatrogenic causes. Patients with pyothorax are typically treated with supportive care, antimicrobial therapy, and chest tube placement for intermittent lavaging with physiologic saline. In some cases, surgical exploration of the chest cavity to remove the source of the infection may be chosen. Accumulation of pink or white, milky, chylous effusion is termed chylothorax. The opaqueness is a result of a high triglyceride concentration. Potential causes include cardiomyopathy, congestive heart failure, pericardial disease, thoracic duct obstruction or rupture, lymphosarcoma, thymoma, and lung lobe torsion. Patients with chylothorax are typically managed by removal of the effusion through thoracocentesis. Hemothorax can result from coagulopathy, trauma, neoplasia, lung lobe torsion, pulmonary thromboembolism, and thymic hemorrhage. Iagtrogenic causes are also possible, from procedures such as thoracocentesis, thoracostomy, and intrathoracic biopsy.

An open pneumothorax can result from penetrating thoracic trauma. Closed pneumothorax can occur due to damaged lung parenchyma, trachea, airway, esophagus, mediastinum, or diaphragm. Traumatic pneumothorax is the most common type of pneumothorax, caused by blunt force trauma such as automobile accidents or falling from heights. Gradual accumulation of air and pressure due to the lesion acting as a one-way valve results in a tension pneumothorax. A tension pneumothorax is life-threatening; increased intrathoracic pressure causes cardiovascular depression through reduction of venous return, leading to shock. Immediate thoracocentesis, and in persistent tension pneumothorax, a thoracostomy tube may be placed to continuously evacuate air out of the chest cavity. This is accomplished with a continuous suction device, or a one way valve. The patient is monitored for subsequent occurrences of dyspnea, indicating a return of pneumothorax. If a closed pneumothorax does not resolve in 3-5 days, surgical exploratory is warranted. Autologous

blood patch, or instilling of fresh whole blood collected from the patient into the pleural space encouraging sealing of the source of the air leak is performed for persistent pneumothorax.

Diaphragmatic hernia may occur due to trauma or could be congenital. The degree of dyspnea varies depending on the degree of herniation, presence of concurrent pleural effusion, and presence of thoracic injuries. Surgical treatment is warranted when diaphragmatic hernias are seen, and should be performed immediately if any organ torsion or strangulation is suspected. Prognosis is good in patients receiving surgical intervention within 24 hours.

Pneumonia

Pneumonia is the inflammation of the pulmonary parenchyma caused typically by an infectious agent which enters the airway. In an emergency and critical care setting, aspiration pneumonia, caused by inhalation of contaminated material leading to an infection. Patients with aspiration pneumonia may present in respiratory distress and exhibit signs like coughing, weakness and collapse, pyrexia, cyanosis, and purulent nasal discharge. Lung sounds will be loud, and crackles may be heard. Abnormal sounds more often than not can be localized in the cranioventral areas. Treatment will consist of antimicrobial therapy, oxygen therapy and mechanical ventilation if necessary. Nursing interventions such a nebulization and coupaging may be instituted, though human evidence relating to a faster recovery from pneumonia has not been seen.

PATIENT MONITORING

During the treatment of patients with respiratory compromise, the patient should be closely monitored on three different aspects; oxygenation, ventilation (carbon dioxide elimination), and the degree of respiratory effort. In addition to the visible respiratory effort and auscultation, different instrumentation and blood analysis can give insight to the progression of the patient's recovery.

Oxygenation

A physical sign seen in patients with severe hypoxemia is cyanosis, or a blue color to the mucous membranes. Cyanosis becomes apparent when there is more than 5 g/dL of deoxyhemoglobin present in the blood. An average hemoglobin level in dogs is approximately 13-17 g/dL, and in cats is approximately 10-14 g/dL. This means the oxygen saturation of hemoglobin will be a significantly decreased level on average of 61-70% for dogs and 50-64% for a cat before cyanosis is seen. Patients presenting with cyanosis is severely compromised in their DO_2 and requires immediate attention.

Oxygenation can be better gauged through measurement of PaO_2, serving as an indicator of pulmonary function. PaO_2 can be measured through blood gas analysis of arterial blood, requiring arterial blood sampling and a blood gas analyzer. A patient with normal respiratory function breathing room air will have a PaO_2 of 80-100mmHg. PaO_2 of less than 80mmHg qualifies as hypoxemia, and 60mmHg is considered severe hypoxemia.

Pulse oximetry allows non-invasive measurements of the percentage of oxygenated functional hemoglobin in the arterial bloodstream, utilizing the concept of light absorption. The saturation of oxygen measured by pulse oximetry (SpO_2) closely reflects SaO_2 and can be used to estimate the PaO_2 level. The oxygen-hemoglobin dissociation curve expresses the relationship between SaO_2 and PaO_2. A SaO_2 of 95-98% corresponds to a PaO_2 of 80-100mmHg. A SaO_2 below 90% indicates a PaO_2 of less than 60mmHg. Pulse oximetry has

its limitations, including false reading in the presence of significant levels of dysfunctional hemoglobin species (methemoglobin, carboxyhemoglobin), inconsistent readings with movement, poor perfusion, anemia, and pigmented skin. Interpretation of oxygenation and pulmonary function can performed by calculated values called the $PaO_2:FiO_2$ Ratio (PF ratio) and alveolar-arterial (A-a) gradient.

Carbon Dioxide Elimination

CO_2 is produced by tissues as a metabolic byproduct of energy production. The body normally maintains control of CO_2 levels in order to control the pH level of the body. An accumulation of CO_2 causes an increase in levels of carbonic acid, leading to higher levels of dissociated hydrogen ions, leading to a more acidic environment (lower pH). A reduction in CO_2 level will lead to a decrease in hydrogen ions, leading to a more basic environment. This effect is called respiratory acidosis and respiratory alkalosis, respectively.

The amount of CO_2 eliminated by the body depends on the movement of air in and out of the alveoli to perform gas exchange, or ventilation. Room air contains about 0.04% CO_2 (0.3mmHg), and the replacement of gas within the alveoli with fresh room air will promote diffusion of CO_2 out of the blood stream into the gas within the alveoli, which in turn gets expired out of the lungs and airway. A normal CO_2 levels within the blood is approximately 35-45mmHg in dogs, and 30-40mmHg in cats, and can be measured by blood gas analysis ($PaCO_2$ if arterial or $PvCO_2$ if venous). The difference in PCO_2 in the pulmonary capillaries and alveoli create a pressure gradient required for gas exchange (high to low; high in the capillary, low in the alveoli).

PCO_2 is largely influenced by the amount of air that can be moved in and out of the alveoli, or alveolar ventilation. The value will increase with hypoventilation (>45mmHg) in cases of respiratory depression (suppression of respiration due to drugs, neuromuscular disease, CNS disease), inability to expand the lungs (pleural space disease, compromise to chest walls), or increased resistance to breathing (narrowed airway). Hyperventilation and subsequent low $PaCO_2$ can be seen in patients with increased RR due to anemia and hypoxia. In metabolic acidosis, compensatory increase in respiratory effort and hyperventilation is often seen, countering the metabolic acidosis effect with respiratory alkalosis. This occurs because the presence of hydrogen ions will stimulate the respiratory center of the brain to increase respiratory efforts.

The $PaCO_2$ can be estimated by measurement of End-tidal CO_2 (ETCO2). The CO_2 content in the gas present at the probe at the end of expiration is measured to obtain this value. The ETCO2 in normal cardiovascular and respiratory situation, is within 5mmHg of the $PaCO_2$. The ETCO2 is most easily measured when an endotracheal tube is placed in a patient (anesthetic procedure or mechanically ventilated patients, for example). There are nasal tubes and masks available allowing for less invasive ETCO2 measurement.