What to Do when They Turn Blue? Pearls of Respiratory Distress

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INTRODUCTION

Respiratory illness is common in small animal medicine and often represents a diagnostic and therapeutic challenge. Common causes of respiratory distress in small animal medicine include pulmonary parenchymal diseases and trauma. Common causes of pulmonary parenchymal disease include infectious (bacterial, fungal, viral, protozoal, parasitic or rickettsial), inflammatory, or neoplastic (primary or metastatic). Examples of common veterinary trauma presentations include motor vehicle accidents (i.e. hit by car) interaction with other animals, interaction with humans, fall from heights, and penetrating trauma such as gunshot wounds, knife wounds, and impalement by sticks. It is essential to determine the underlying cause using physical examination findings and diagnostic tests for rapid patient assessment and treatment.

INITIAL ASSESSMENT

Regardless of the cause, initial assessment of a patient in respiratory distress should include a detailed medical history and visual inspection of the patient's breathing pattern. Before placing your hands and stethoscope on the patient, just observation of the breathing pattern and patient comfort can provide important clues and help localize the origin of the respiratory disease (e.g. upper airway, lower airway, pulmonary parenchyma, pleural space or thoracic wall).

The initial triage evaluation should be rapid, developing a problem list outlining life-threatening conditions. The goals of the initial triage examination are to:

1) Assess / evaluate the ABCD's of triage medicine:
    a. **A**irway: Does the patient have a patent airway? Upper airway or lower airway abnormalities?
    b. **B**reathing: Does the patient have an abnormal breathing pattern? Is the patient dyspneic? Is there a rapid, shallow breathing pattern? Is there a slow, labored breathing pattern? Is there increased stertor or stridor?
    c. **C**irculation: Is there an abnormal heart rate? Are the mucous membranes an abnormal color with evidence of internal or external hemorrhage? Are the pulses weak? Are the extremities cold?
    d. **D**isability: Is there evidence of head trauma or other neurological injury?

Following the history and visual / triage examination, a more detailed examination is performed to further aid in localizing the origin of the respiratory disease. The common origins of respiratory distress seen in small animal medicine include:

Upper airway disease (stertor, stridor, inspiratory effort)

- Brachycephalic airway disease
- Laryngeal paralysis
- Laryngeal neoplasia
- Foreign body

Lower airway disease: (cough, wheezing, increased bronchovesicular sounds)

- Feline asthma
- Chronic bronchitis

Lung (pulmonary parenchyma) disease: (increased bronchovesicular sounds, crackles, rapid shallow breathing)

- Pneumonia
- Pulmonary fibrosis
- Congestive heart failure

Pleural space disease: (dull lung sounds on auscultation, muffled heart sounds, restrictive, shallow respiratory pattern)

- Pleural effusion
- Pneumothorax

Specifically, when a cat is presented for respiratory distress, while the clinician is getting a brief medical history from the owner and observing the patient's breathing pattern, several important questions should be answered:

1. Is the respiratory distress more notable on inspiration, expiration or both?
2. Is the breathing pattern short and shallow (restrictive)? Is there a long inspiratory pattern with rapid, forceful expiration?
3. What is your treatment plan going to be for immediate stabilization?

Using the information obtained from the history, visual examination, triage examination, and signalment will often allow the clinician to make an accurate assessment to better characterize the source of dyspnea:

- Pleural space disease (pleural effusion, pneumothorax, mass)
- Lower airway disease (feline asthma, bronchitis)
- Pulmonary parenchymal disease (pneumonia)
- Congestive heart failure (pulmonary edema, pleural effusion)

Conversely, the most common differential diagnoses in dogs with pulmonary parenchymal disease include infectious causes (i.e. pneumonia), neoplasia, congestive heart failure, and inflammatory, non-infectious causes (NCPE). These should be differentiated from other causes of respiratory distress based on history, examination, and diagnostics. These causes include upper airway disease (laryngeal paralysis, collapsing trachea) and pleural space disease (pleural effusion, neoplasia),

INITIAL STABILIZATION

Initial stabilization of patients in respiratory distress should include intravenous access, supplemental oxygen therapy, and careful monitoring.

Oxygen supplementation should be provided immediately. During the patient assessment, oxygen may be provided via flow-by or facemask. If the patient is too distressed to tolerate an examination, they should be placed in an oxygen cage to prevent decompensation. The goal of oxygen supplementation is to increase the FiO_2 to increase the PaO_2 and hemoglobin saturation, ultimately improving oxygen delivery to the tissues. While the patient is receiving supplemental oxygen, a more complete medical history can be obtained from the owner. Historical information that may help includes smoke inhalation, toxin exposure (rodenticide), trauma, previous cardiac disease, and current medications.

Oxygen Supplementation Techniques.

Supplementation technique	Required flow rate	Maximum inspired oxygen concentration achieved
Flow-by	3-15 l/min	40%
Oxygen cage	15 l/min	45-60%

Oxygen hood (unsealed bag)	5-15 l/min	85-95%
Oxygen collar	1 l/10 kg bodyweight/min	<80%
Nasal cannula	50-100 ml/kg/min	40%
Nasal catheters	50-100 ml/kg/min	40-50%
Nasopharyngeal catheter	50-100 ml/kg/min	60-70%
Nasotracheal catheter	25-50 ml/kg/min	80-90%

Intravenous catheter placement for vascular access is also recommended. Benefits of vascular access include fluid therapy, administration of blood products, parenteral nutrition, procurement of blood samples, and importantly to allow for drug administration in case of patient decompensation.

To limit the number of times phlebotomy is needed, while placing the intravenous catheter, blood should be drawn for anticipated testing, including a minimum database (packed cell volume, total protein, blood glucose, blood urea nitrogen, and blood smear) or more complete testing such as a complete blood count and serum chemistry panel.

Other diagnostics that may aid in patient assessment include pulse oximetry, arterial blood gas analysis, and thoracic radiographs. It is essential to remember that if the patient becomes increasingly stressed; they should be placed back in the oxygen cage or allowed to rest with supplemental oxygen to reduce the risk of rapid decompensation.

Pulse oximetry is a non-invasive way to measure oxygen saturation of hemoglobin. This tool gives real-time monitoring of hemoglobin saturation and can detect desaturation of hemoglobin immediately. This can be used as a single test to get a point of reference or as a constant monitor in critically ill or anesthetized patients.

Thoracic radiographs can also be an invaluable tool for the patient with respiratory illness, but they are not without risk. The author would much prefer to diagnose a pneumothorax or pleural effusion based on auscultation during the examination than with a stressful radiograph procedure. If radiographs are needed for disease classification, lateral views and a dorsoventral radiograph would be preferred over a ventrodorsal radiograph, which can be more stressful for the patient during restraint. Supplemental oxygen as well as chemical restraint, such as an opioid, may be useful to decrease patient stress.

Pulmonary Parenchymal Diseases

Patients with pulmonary parenchymal disease often present in respiratory distress with moderate to severe hypoxemia. Common examination findings in patients with parenchymal disease include an increased respiratory rate and effort, nasal discharge, cough, lethargy, decreased appetite, and fever. A patient that presents with pulmonary parenchymal disease often has a short and shallow, restrictive breathing pattern. In these patients, hypoxia is caused by ventilation/perfusion mismatch due to alveolar disease.

If the patient has a productive cough (often described as a soft moist sound that is followed by swallowing), they should be evaluated for etiologies such as pneumonia.

On auscultation, patients with pulmonary parenchymal disease often have increased bronchovesicular sounds or crackles. Auscultation of the heart should be a priority as pulmonary crackles combined with a significant heart murmur may lead to a diagnosis of congestive heart failure.

Radiographically, pulmonary parenchymal disease is often represented with an alveolar pattern on thoracic radiographs. The clinician should then evaluate the distribution of the alveolar pattern to provide a more accurate assessment on the etiology of respiratory disease.

Alveolar Disease Radiograph Distribution Chart	
Radiographic appearance	**Differential Diagnoses**
Dorsal distribution Perihilar, bilaterally symmetrical	Pulmonary edema Cardiomyopathy
Caudal and dorsal distribution	Non-cardiogenic edema (*Electrocution, seizure, smoke inhalation, upper airway obstruction, choking)*
Cranioventral and lobar	Pneumonia *(Aspiration, inhalation)*
Irregular and patchy (focal)	Hemorrhage, trauma
Primary caudal lung lobes. Metastatic	Neoplasia
Irregular and patchy	Granulomatous, parasitic

BACTERIAL PATHOGENS - PNEUMONIA AND PYOTHORAX

Lower respiratory tract infections result from a variety of bacteria including *Streptococcus* spp. *E. coli,* mycoplasmas, *B. bronchiseptica,* and *Pasteurella* spp. Patients with bacterial pneumonia may have historical findings including fever, a productive cough, tachypnea, lethargy, anorexia, and respiratory distress. On thoracic auscultation, common findings include increased bronchovesicular sounds and crackles. Although coughing and fever are thought to be common with pneumonia, approximately 50% of patients with bacterial pneumonia do not present with a history of cough or fever. Patients with bacterial pneumonia may also have an increased white blood cell count (leukocytosis) with a left shift.

In dogs, alveolar infiltrates with air bronchograms are classic radiographic findings in bacterial pneumonia. In cats, nonspecific infiltrates may also be seen.

Once bacterial pneumonia is suspected, options to consider include empirical antibiotic therapy or further diagnostics, including tracheal wash or bronchoscopy with bronchoalveolar lavage. Less common diagnostics for alveolar disease include fine needle aspiration of the lung or sputum sampling. Regardless, broad-spectrum antibiotic therapy is recommended. If airway sampling is performed, antimicrobial therapy should be adjusted based on the culture results. Ancillary treatment for pneumonia includes intravenous fluid therapy to improve hydration, nebulization for direct airway hydration, and coupage to improve secretion elimination.

MYCOPLASMAS

Mycoplasmas are commensals of the upper respiratory tract (URT) of cats and are an important cause of lower respiratory tract infection in cats. It is unclear if they are primary pathogens, or secondary pathogens colonizing diseased airways. Mycoplasmas have been reported to cause bronchopneumonia, pyothorax, and pulmonary abscessation. *Mycoplasma* spp. are generally susceptible to doxycycline, macrolides and fluoroquinolones. Once diagnosed, a treatment period of six weeks is recommended (10 mg/kg q 24 h PO or 5 mg/kg q 12 h PO).

BORDETELLA BRONCHISEPTICA

Bordetella bronchiseptica (referred to as tracheobronchitis) is a highly contagious respiratory disease in small animal medicine caused by the bacteria *Bordetella bronchiseptica*. It is a small gram-negative coccobacillus shed in oral and nasal secretions and a primary pathogen in dogs and cats. Clinical disease ranges from ocular and nasal discharge and a mild cough to severe lower respiratory tract disease including dyspnea, hypoxemia, and respiratory distress. Diagnosis is often made via culture (ETW, TTW, or BAL fluid) but can also be made via PCR testing. *Bordetella* is usually susceptible to doxycycline, chloramphenicol or fluoroquinolones.

HEARTWORM-ASSOCIATED RESPIRATORY DISEASE (HARD)

Cats and dogs are at risk of *Dirofilaria immitis* wherever disease is endemic. Although cats are believed to have natural resistance to *D. immitis,* a unique syndrome of bronchointerstitial inflammation and pulmonary arteriolar hypertrophy known as HARD, can develop in the absence of adult worms. This is most commonly appreciated three months post-infection. Clinical signs include dyspnea, coughing and wheezing. Cats with HARD are heartworm-antigen negative, usually heartworm-antibody positive and have a bronchointerstitial pattern on thoracic radiographs.

TOXOPLASMOSIS

Toxoplasmosis is caused by the intracellular coccidian *Toxoplasma gondii*. Infection occurs after ingestion of bradyzoite cysts in the tissues of intermediate vertebrate hosts resulting in patent infections in 97% of naïve cats. The most common treatment recommendation is clindamycin 12.5 mg/kg PO or IV q 12 h. Other options for treatment include trimethoprim-sulfonamide 15 mg/kg PO q 12 h or azithromycin 10 mg/kg PO q 24 h for a minimum of 4 weeks.

Airway Trauma

Trauma to the major airways may be seen with penetrating wounds or blunt trauma to the neck and chest. Clinical signs of upper airway trauma include abnormal upper airway noise on inspiration and expiration. Respiratory changes may result from traumatic inflammation, edema, hemorrhage, or even tracheal rupture or avulsion.

Subcutaneous emphysema may also be noted on examination, prompting a thorough airway integrity assessment. Pneumomediastinum and pneumothorax are more severe complications of airway trauma. While subcutaneous emphysema and pneumothorax may be easily found on examination alone, the diagnosis of pneumomediastinum is made radiographically by increased contrast with the mediastinal structures resulting in a clear visualization of the thoracic vena cava, aorta and esophagus.

Pneumothorax

Pneumothorax is defined as the abnormal accumulation of air in the pleural space. Air accumulation is most commonly bilateral, but unilateral pneumothorax can occur. It is the most common complication of blunt trauma to the chest. Studies have shown that animals hit by car with fractures had evidence of pneumothorax 47.1% of the time. Furthermore, 36% of dogs and 63% of cats that fell from high rises had evidence of pneumothorax on examination. Pneumothorax can be further classified as closed, open, and tension pneumothorax.

- Closed pneumothorax is seen following trauma due increased intra-thoracic pressure against a closed glottis causing rupture of alveoli or small airways, laceration of lung by a fractured rib, iatrogenic, and/or airway or esophageal rupture causing pneumomediastinum which has progressed to pneumothorax.
- Open pneumothorax may result from gunshots, dog bites, knife wounds, and stick impalement.
- Tension pneumothorax is the third type, resulting when an air leak acts as one-way valve increasing intrathoracic pressure, compressing the lungs and decreasing venous return to the heart.

The astute clinician often makes the diagnosis of a pneumothorax based on history and examination alone. Common examination abnormalities include an increased respiratory rate and effort characterized by a short and shallow breathing pattern, dull lung sounds dorsally, and muffled heart sounds. Less specific examination abnormalities may include pale or cyanotic mucous membranes, poor pulses, and an abnormal posture with the head and neck extended and elbows abducted. While useful in the diagnosis of a pneumothorax, thoracic radiographs risk increased stress on the compromised patient. Radiographic signs of pneumothorax include elevation of the cardiac silhouette from the sternum, collapse of the lung lobes, and absence of vascular markings out to the periphery of the thorax.

Recently, the use of ultrasound has been documented for rapid detection of pleural space disease, specifically the

"TFAST" (thoracic focused assessment with sonography for trauma) procedure. It does, however require practice to be competent in its use.

When radiographs are not suitable, the unstable patient may benefit from thoracocentesis, which can be both diagnostic and therapeutic. The equipment needed for this procedure includes clippers, scrub, sterile gloves, a 10-60ml syringe, 3-way stopcock, butterfly catheter or needle and extension tubing. The site preparation and eventual needle placement for a patient suspected of a pneumothorax is on the dorsal 1/3 of the thorax between the 7th-10th intercostal spaces. The needle is inserted cranial to the rib to avoid the intercostal artery, vein, and nerve located caudal to each rib. Air is aspirated until negative pressure is obtained.

A chest tube is indicated when thoracocentesis needs to be repeated multiple times over a short period of time or when the clinician cannot achieve negative pressure on simple thoracocentesis. Large bore chest tubes require sedation or general anesthesia. Smaller bore chest tubes are also available, placed via the modified Seldinger technique with the patient awake or receiving local analgesia. Equipment required for chest tube placement includes clippers, surgical scrub, surgical blade, local analgesia, suture material, the thoracostomy tube, 3-way stopcock and syringes for initial aspiration. The chest tube can be used intermittently or attached to a suction device for continuous suction. The technique for chest tube placement will depend on the type of tube used, including surgical and trocar methods for the larger bore tubes or the modified Seldinger technique for the smaller bore tubes. Similar to the thoracocentesis, surgical preparation of the site between the 7th-10th intercostal spaces is recommended.

Pulmonary Contusions

Pulmonary contusions result from blunt or crushing trauma and are one of the most common problems associated with thoracic trauma, seen in approximately 50% of all thoracic injuries. Thoracic trauma leads to blood within the alveoli, ventilation/perfusion mismatch, increased pulmonary shunt fraction, and loss of lung compliance. Hypoxemia, increased work of breathing, and hypercarbia are the physiologic results.

Physical examination findings may include tachypnea, hemoptysis, increased respiratory effort, and harsh lung sounds or crackles on auscultation. Radiographically, there may not be evidence of pulmonary contusions on presentation; it may be delayed anywhere from 12 to 48 hours following trauma. When present, contusions appear radiographically as dense patchy, interstitial to alveolar disease.

As discussed above, initial fluid resuscitation must be started with caution as large volumes of rapidly administered fluid can exacerbate the fluid within the alveolar space with increased vascular permeability, worsening the hypoxemia. If radiographs have evidence of pulmonary contusions, the astute clinician should carefully look for concurrent abnormalities including pneumothorax and/or rib fractures. Additional diagnostic findings may include hypoxemia on pulse oximetry or arterial blood, and an increased A-a gradient.

There is no specific medication or reversal therapy for pulmonary contusions. Common supportive care measures include oxygen supplementation, judicious IV fluid therapy, and analgesics. Although evidence is lacking, low dose diuretic therapy has been described anecdotally (furosemide, 0.5 to 1 mg/kg IV intermittently or CRI) in the treatment of pulmonary contusions.

Fractured Ribs

Rib fractures result in discomfort and reduced diaphragmatic and chest wall motion. More specifically, the reduced chest wall motion and pulmonary expansion results in decreased oxygenation and ventilation, and atelectasis of the lungs. Rib fractures should be a clue to the astute clinician that severe thoracic trauma occurred, prompting careful evaluation for additional injuries such as pulmonary contusions or a pneumothorax. Physical examination findings may include an increased respiratory rate with shallow respirations, subcutaneous emphysema, palpation of crepitus over the fracture site, and/or conformational changes of the chest wall.

Treatment of rib fractures consists of treating concurrent injuries such as pulmonary contusions, oxygen therapy if hypoxemia exists, and pain management with local or systemic analgesia.

Flail chest

A flail chest is a more severe manifestation of the simple rib fracture. A flail segment occurs when 2 or more ribs are fractured at the junction of ribs and the sternum producing a paradoxical movement of the flail segment. On inspiration, the chest wall normally expands. With a flail segment, the negative intrapleural pressure causes the flail segment to collapse inward during inspiration. On expiration, the chest wall normally collapses. With a flail segment, the intrapleural pressure becomes less negative and the flail segment moves outward on expiration. Abnormal chest movement and the accompanying pain from the fractures themselves result in decreased oxygenation, ventilation, and pulmonary atelectasis.

Treatment consists of placing the patient in lateral recumbency with the flail side down, minimizing movement of the flail segment and reducing the associated fracture discomfort. Pain management includes local nerve blocks and systemic opioid analgesia. Surgical stabilization of the flail segment may also be indicated.

Hemothorax

A hemothorax is defined as an accumulation of blood in the pleural space. This is uncommon following trauma. If present, the amount of blood loss into the pleural space is usually minimal and does not contribute significantly to respiratory compromise. If a large amount of hemorrhage into the pleural space is documented, there should be an increased suspicion for rupture of a large vessel. More common causes for a hemodynamically insignificant hemothorax include laceration of pulmonary or intercostal vessels and/or lung laceration by a fractured rib.

The diagnosis of hemothorax is often be made on physical examination with signs including dyspnea, tachypnea, dull lung sounds ventrally, muffled heart sounds, and signs of hypovolemic or hemorrhagic shock. Thoracocentesis confirms the diagnosis when hemorrhagic fluid is obtained during aspiration with a PCV and TP of the effusion similar to that of the PCV and TP of the peripheral blood.

Treatment of a traumatic hemothorax may include diagnostic and therapeutic thoracocentesis, intravenous crystalloid or synthetic colloid therapy and blood products--notably whole blood or packed red blood cell transfusions. Autotransfusion can be considered if blood products are not available.

Diaphragmatic Hernia

Diaphragmatic hernia is defined as disruption of the diaphragm, allowing displacement of abdominal organs into the thoracic cavity. Diaphragmatic hernia occurs most often as a result of blunt trauma where intra-abdominal pressure is suddenly increased, causing rupture of the diaphragm. The resulting herniation of abdominal contents can range from a single organ or component of an organ (such as a single liver lobe) to almost all the abdominal contents moving cranially through the diaphragmatic rent into the chest cavity. The result is restriction of lung expansion and respiratory distress.

The diagnosis of diaphragmatic hernia can be made with physical examination findings and radiographic abnormalities. Clinical signs of diaphragmatic hernia depend upon the type and number of organs within the chest cavity as well as associated abnormalities such as fluid in the pleural space or pulmonary contusions. Examination findings may be mild and include a slight tachypnea or may result in severe dyspnea, dull lung sounds, muffled heart sounds, borborygmi from the stomach or intestines ausculted in the thorax, abnormal percussion, and a tucked/empty abdomen on palpation. Thoracic radiographs are often diagnostic with the presence of abdominal organs in the thorax.

Treatment for diaphragmatic herniation will depend on the clinical signs of the patient with surgical repair being the definitive therapy. Although there are no recent studies which outline the recommended time from

stabilization to surgical correction, worsening respiratory distress or compromised blood supply to the displaced organs and ischemia would warrant a more rapid surgical correction.

Ultimately, diagnosing and managing respiratory disease in our small animal patients can be a challenge. An accurate history and careful physical examination along with ancillary diagnostic tests are necessary to obtain a clinical diagnosis to form a treatment plan to improve patient outcome and reduce morbidity and mortality.

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