INITIAL APPROACH TO THE EMERGENT RESPIRATORY PATIENT
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Introduction

Respiratory distress is a commonly encountered, and truly life-threatening, emergency presentation. Successful management of the emergent respiratory patient is contingent upon rapid assessment and stabilization, and action taken during the first minutes to hours often has a major impact on patient outcome. While diagnostic imaging is undoubtedly a crucial part of the workup, patients at presentation may be too unstable to safely achieve imaging and clinicians may be called upon to institute empiric therapy based primarily on history, physical exam and limited diagnostics. This lecture will cover the initial evaluation and stabilization of the emergent respiratory patient, with a particular emphasis on clues from the physical exam that may help localize the cause of respiratory distress. Additionally, we will discuss 'cage-side' diagnostics, including ultrasound and cardiac biomarkers, which may be useful in the working up these patients.

Establishing an airway

The first priority in the dyspneic patient is ensuring a patent airway. Signs of an obstructed airway can include stertorous or stridorous breathing or increased respiratory effort with minimal air movement heard when auscultating over the trachea. If an airway obstruction is present efforts should be made to either remove or bypass the obstruction. Clinicians should be prepared to anesthetize and intubate patients if necessary to provide a patent airway. Supplies to have on hand for difficult intubations include a variety of endotracheal tube sizes, stylets for small endotracheal tubes, a laryngoscope with both small and large blades, and instruments for suctioning the oropharynx. Emergent tracheostomy is indicated when orotracheal intubation is not feasible, although in the author's experience this is a rare occurrence.

Physical exam and stabilization

Respiratory distress may be a manifestation of an abnormality in any one or a combination of the following areas: the upper airway, the lower airway, the pulmonary parenchyma, the pleural space, or the chest wall/diaphragm. Classically patients in respiratory distress will present with overt tachypnea or dyspnea; with severe distress they may adopt an orthopneic posture with elbows abducted and head and neck extended in an effort to minimize resistance to airflow into the lungs. Cats may present with less overt signs of distress than dogs. It should be noted that cyanosis is a specific but insensitive marker of hypoxia as it requires an arterial partial pressure of oxygen less than 50mmHg and greater than 5g/dL of deoxygenated hemoglobin. Thus, cyanotic mucous membranes always indicate severe hypoxemia but the absence of cyanosis cannot rule out severe respiratory compromise.

Careful attention to the physical exam may help to better localize the cause of distress (Table 1). It should be noted, however, that patients in respiratory distress can be quite fragile and may decompensate quickly particularly if stressed. Efforts should be made to minimize stress where possible. In some cases this may limit the initial physical exam to a brief cardiopulmonary auscultation and assessment of the cardiovascular status (mucous membrane color, capillary refill time, heart rate and rhythm, presence of a murmur, pulse palpation). A full physical exam is performed once the patient is more stable.

Upper Airway Disease

Patients with disease of the upper airway frequently have audible upper airway noise which is occasionally loud enough to make accurate auscultation of the heart and lungs challenging. Stertor is generally associated with obstruction in the nasopharynx while stridor more commonly points toward laryngeal obstruction. A harsh, honking cough is frequently noted with tracheal collapse. Evaluation of the respiratory pattern in patients with upper airway obstruction often reveals a long, shallow breathing pattern. A long, shallow inspiratory phase generally suggests obstruction of the extrathoracic airway while intrathoracic airway obstruction more commonly results in expiratory difficulty. In animals with severe respiratory distress these patterns

may be difficult to distinguish and in some dogs with both extrathoracic and intrathoracic tracheal collapse both of these patterns may be present. Patients with upper airway obstruction often present in a very agitated state, may be quite dyspneic and very frequently are hyperthermic.

Common differentials for upper airway disease include brachycephalic obstructive airway syndrome, laryngeal paralysis or collapse, tracheal collapse, nasopharyngeal collapse, nasopharyngeal polyp, laryngeal or nasopharyngeal edema, foreign body obstruction, or obstruction by a benign or malignant mass. Laryngeal paralysis is the most common cause of upper airway obstruction in large dogs while collapsing trachea more commonly occurs in small breed dogs. If a foreign body obstruction is suspected, an abdominal thrust maneuver may be employed in an attempt to clear the airway.

Patients with a dynamic airway obstruction, for example, dogs with laryngeal paralysis, tracheal collapse or brachycephalic obstructive airway syndrome, often benefit from sedation with one or a combination of butorphanol (0.1-0.3 mg/kg IM) and acepromazine (0.01-0.02 mg/kg IM). This helps to relieve the anxiety associated with increased work of breathing that often exacerbates their degree of distress. A low dose of a corticosteroid (dexamethasone sodium phosphate, 0.1 mg/kg IM or IV) may be provided to reduce laryngeal edema or tracheal inflammation. Following sedation, placing these patients in a cool and quiet environment with oxygen supplementation may help alleviate their dyspnea. If available, placing the patient into an oxygen cage is helpful as this will allow for delivery of supplemental oxygen and frequently the white noise effect inside the cage may help to calm some stressed patients. Active cooling should be employed for patients with a rectal temperature greater than 103 degrees Fahrenheit.

Intubation is required for patients with dynamic upper airway obstruction that do not respond to sedation. Laryngeal function can be assessed at the time of intubation although this may be difficult to interpret in the setting of moderate or severe laryngeal edema – this may necessitate performing a laryngeal exam in a more controlled setting once the inflammation has resolved. Although definitive therapy for dynamic upper airway obstruction frequently requires a surgical or interventional approach, many patients may respond to medical management with a combination of weight control, cough suppression and sedation as needed.

Lower Airway Disease

Lower airway disease is a common cause for respiratory distress in cats. In fact, lower airway disease/asthma ranks among the top causes for feline respiratory distress along with congestive heart failure and pleural effusions. Typically cats will have a history of cough although owners often don't recognize this as coughing and rather suspect that the cat is attempting to vomit a hairball.

On presentation cats may present in variable degrees of respiratory distress, ranging from moderate tachypnea to orthopnea with open-mouth breathing. Cats are notorious for masking signs of respiratory compromise; often by the time distress is recognized by owners their disease is quite severe. The breathing pattern in these patients most commonly involves expiratory dyspnea and pulmonary auscultation may reveal wheezes or occasionally harsh crackles.

When an asthmatic crisis is suspected, oxygen is provided and a trial dose of a glucocorticoid (dexamethasone sodium phosphate, 0.15-0.2 mg/kg IM) and rapidly acting bronchodilator may be administered. At our facility, terbutaline, a selective B2 agonist (0.01 mg/kg, IM or SQ), is the preferred bronchodilator. Albuterol, an alternative B2 agonist, can be administered via inhaler; however, these cats are frequently quite stressed and may not tolerate the use of inhaled medication. Typically we expect to see some improvement within 30 minutes of administration. B2 agonists may cause transient tachycardia so these drugs must be administered with caution in cats with myocardial disease.

Pulmonary Parenchymal Disease

Pulmonary parenchymal disease can lead to a variable degree of dyspnea dependent on disease severity and distribution. Typically dyspnea is more inspiratory in nature and patients will often develop a restrictive breathing pattern due to decreased pulmonary compliance, leading to short, shallow but fast respirations. Auscultation often will reveal either increased bronchovesicular sounds or inspiratory crackles which may be focal or diffuse in distribution;

however, breath sounds may be diminished when auscultating over a severely consolidated lung lobe. Dogs with pulmonary parenchymal disease will commonly cough but this is less frequently seen in cats.

Common causes of pulmonary parenchymal disease include pneumonia, left-sided congestive heart failure, neurogenic pulmonary edema, pulmonary contusions or hemorrhage, pulmonary thromboembolism, pulmonary fibrosis, acute respiratory distress syndrome, and neoplasia. Chest radiographs are typically required for diagnosis but frequently history and clinical signs may point to one differential over others. For example, dogs with pneumonia may be febrile or have mucopurulent nasal discharge. Evidence of trauma or petechiae/ecchymoses may indicate pulmonary contusion or pulmonary hemorrhage, respectively. The presence of a heart murmur coupled with increased bronchovesicular sounds or inspiratory crackles, arrhythmia or pulse deficits might suggest congestive heart failure as the cause of respiratory distress and a trial dose of furosemide (2-4 mg/kg IM for dogs, 1-2 mg/kg IM in cats) could be considered. For patients with congestive heart failure we generally expect to see some (although at times very minimal) improvement within 20-30 minutes of furosemide administration.

Pleural Space Disease

Patients with pleural space disease are unable to fully expand their lungs and, as a result, frequently develop a restrictive breathing pattern. A paradoxic or inverse respiratory pattern, that is, inward movement of the thoracic wall and outward movement of the abdomen during inspiration, may be seen with severe cases of pleural space disease. This respiratory pattern may also be seen with flail chest and severe upper airway obstruction. Auscultation commonly reveals muffled lung sounds. Pneumothorax typically results in diminished lung sounds dorsally while pleural effusion and diaphragmatic hernia more often cause diminished ventral lung sounds. Tension pneumothorax is rapidly life threatening; these patients are generally markedly dyspneic, may develop cardiovascular collapse as a result of inhibition to venous return, and their chest may have a 'barrel shaped' appearance. Patients with acute hemothorax may manifest signs of hypovolemic shock prior to the development of respiratory compromise.

Common causes of pleural space disease include pneumothorax, hemothorax, chylothorax, right-sided heart failure, pyothorax, neoplastic effusions, and diaphragmatic hernia. If pleural effusion or pneumothorax are suspected, thoracocentesis should be performed as this procedure can be both diagnostic and therapeutic. Thoracocentesis does carry some minimal risk of hemorrhage and iatrogenic pneumothorax but in the vast majority of cases the benefit to performing thoracocentesis far outweighs these risks. Thoracostomy tubes are placed in patients that have persistent pneumothorax despite thoracocentesis and may be considered for medically managed pyothorax.

Chest Wall and Diaphragm Disease

Poor chest excursions may be seen with diseases that affect the neuromuscular control of the chest wall. Patients with a flail chest will have a free-floating segment of the rib cage that moves paradoxically inward on inspiration and outward on expiration. When an open pneumothorax occurs clinicians will typically note either a palpable defect in the chest wall or the presence of a sucking chest wound. Care should be taken in the exploration of traumatic chest wounds as occasionally manipulation of tissue can lead to an open pneumothorax.

Common causes of chest wall and diaphragm disease include central respiratory depression (for example, due to sedation or intracranial disease), cervical myelopathy, lower motor neuron disorders (for example, myasthenia gravis, botulism, polyradiculoneuritis, and tick paralysis), and severe hypokalemia leading to profound respiratory muscle weakness. An arterial or venous blood gas should be performed in these patients to evaluate for hypercapnea. Severe hypercapnea ($PaCO_2$ > 60mmHg) may warrant intubation and positive pressure ventilation. Open chest wounds should be occluded with Vaseline impregnated gauze and thoracocentesis is performed to address the pneumothorax. For larger chest wounds a thoracostomy tube is inserted through the wound and the wound is subsequently occluded with Vaseline impregnated gauze. Ultimately these wounds should be explored and closed surgically once the patient is stable. Patients with flail chest often respond well to analgesia and may not require surgical

stabilization. Placing them in lateral recumbency with the flail segment down may help alleviate discomfort.

The "Look Alikes"

Many non-respiratory diseases present with signs suggestive of respiratory compromise. Some examples include pain, anxiety, hyperthermia, severe hypovolemia and metabolic acidosis. Pulse oximetry or arterial blood gas analysis may be useful to rule out hypoxemia in these patients. However, when in doubt, oxygen therapy should be provided until a definitive diagnosis is reached.

Cage-side Diagnostics

Thoracic FAST (Focused Assessment with Sonography for Trauma) ultrasound is a quick, non-invasive cage-side test that, used in conjunction with the physical exam, can help guide the initial treatment of the respiratory distress patient. A T-FAST exam can be performed rapidly during the initial patient evaluation and may help to identify the presence of pericardial or pleural effusion, pneumothorax, abnormalities in cardiac structure or function, and, in some cases, evidence of a "wet" lung (for example, pulmonary edema or contusions).

In addition to T-FAST, a variety of cardiac biomarkers have been investigated as a means to differentiate cardiac (i.e. congestive heart failure) from non-cardiac causes of dyspnea. The natriuretic peptides, in particular NT-proBNP, have shown promise in this regard. Natriuretic peptides function to modulate intravascular volume and arterial blood pressure by inducing diuresis and natriuresis, altering vascular tone, and antagonizing the renin-angiotensin-aldosterone system. Production of B-type natriuretic peptide (BNP) pro-hormone by cardiac myocytes is enhanced by cardiac wall stretch, such as would occur in the setting of congestive heart failure. Pro-BNP is cleaved into both a biologically active hormone (C-BNP) and an inert prohormone (NT-proBNP). The NT-proBNP molecule has both greater stability and a longer half-life than C-BNP; consequently, it has been investigated more extensively in small animal medicine. A number of studies have demonstrated higher concentrations of NT-proBNP in dogs and cats with dyspnea due to CHF compared to those with dyspnea due to other causes and to healthy controls. Recently, a qualitative point of care feline NT-proBNP ELISA test has become available. This test has been found to have both a high sensitivity and negative predictive value; thus, a negative test suggests that moderate-severe heart disease is unlikely.

Summary

In summary, respiratory distress may present a challenge as patients are often too unstable to allow for a comprehensive diagnostic workup. However, close attention to the physical exam and history often will narrow the differential list and allow for the initiation of empiric stabilizing therapy. Once the patient is stable, a definitive diagnosis and specific therapy can be pursued.

References Available by request.

Table 1: Localizing respiratory distress

Localization	Physical Exam Findings	Common Differentials	Initial Stabilization
Upper airway	• Stertor • Stridor • Long shallow respiratory pattern • Lack of air movement with complete obstruction	• Brachycephalic obstructive airway syndrome • Laryngeal paralysis • Tracheal collapse • Laryngeal edema • Benign or neoplastic mass • Foreign body	• Oxygen • +/- Sedation • +/- Intubation
Lower airway	• Expiratory dyspnea • +/- Crackles or wheezes • +/- History of cough (cats)	• Chronic bronchitis • Asthma	• Oxygen • +/- Steroid • +/- Bronchodilator
Pulmonary parenchyma	• Inspiratory dyspnea • +/- Increased bronchovesicular sounds or crackles • +/- Murmur, arrhythmia • Restrictive or paradoxic respiratory pattern	• Congestive heart failure • Pneumonia • Neurogenic pulmonary edema • ARDS • Pulmonary hemorrhage • Pulmonary thromboembolism • Pulmonary fibrosis • Neoplasia	• Oxygen • +/- Furosemide if congestive heart failure suspected
Pleural space	• Muffled breath sounds • Restrictive or paradoxic respiratory pattern	• Pneumothorax • Right-sided heart failure • Chylothorax • Pyothorax • Hemothorax • Neoplasia • Diaphragmatic hernia	• Oxygen • Thoracocentesis • +/- Chest tube
Chest wall and diaphragm	• Poor chest wall excursions • Paradoxic chest wall movement with flail chest • Palpable chest wall defect or sucking wound	• Flail chest • Open pneumothorax • Central respiratory depression • Cervical myelopathy • Lower motor neuron disease • Severe hypokalemia	• Oxygen • Arterial or venous blood gas to assess ventilation • +/- Intubation, positive pressure ventilation • Cover open wounds, thoracocentesis