Respiratory Distress

When our patients have difficulty breathing, it is very important to try and anatomically localize the source of the distress. By localization, one may narrow the list of rule outs for the etiology and also implement strategies that will “rescue” these patients. Generally, these anatomic locations are:

- Airway (either upper, lower, or both)
- Chest Wall
- Pleural Space
- Pulmonary Parenchyma

When evaluating a respiratory distress patient, remember those conditions we call “Nonrespiratory Look Alikes”. These patients can be dyspneic and tachypneic. Nonrespiratory Look Alikes include the following:

1) Compensatory mechanism for metabolic acidosis (hyperventilation). Examples of more common causes of metabolic acidosis include Diabetic Ketoacidosis, Ethylene glycol toxicity, Renal disease and Lactic acidosis. Other causes include salicylate toxicity, paraldehyde toxicity, methanol/ethanol toxicity and starvation.
2) Decreased content of arterial oxygen with subsequent decreased oxygen delivery to tissues due to hypovolemia, decreased cardiac output or anemia.
3) Pain, anxiety or stress
4) Hyperthermia (not fever)
5) Disease of nerves, muscles or neuromuscular junction to intercostal muscles or diaphragm.
6) Brain lesion or drugs affecting medulla or cerebral cortex
7) Severe abdominal distention with pain, decreased venous return and inhibition of inspiratory movement
8) Metabolic diseases Hyperadrenocorticism or excess steroid therapy can weaken respiratory muscles along with fat deposition in chest wall. Hyperthyroidism can result in increased CO2 production from the hypermetabolic state. Severe metabolic derangements involving hypokalemia resulting in muscle weakness and ineffective respirations can be secondary to conditions such as insulin, vomiting, chronic renal disease, DKA and diuretics.

We should also say a word about the panting patient. Panting is defined as rapid shallow respiration with decreased tidal volume. Alveolar ventilation remains unchanged. Panting is a major method of thermoregulation. When panting, the patient uses minimal work of breathing, respiratory muscle fatigue is not a factor. The patient shows no evidence of respiratory distress. Panting is a normal physiologic response to high ambient temperatures, fever or exercise. It can also be associated with anxiety or pain. Other conditions contribute to panting in our patients. These include hyperadrenocorticism, steroid therapy, pheochromocytoma, hyperthyroidism, hypocalcemia, cardiac disease (especially tachycardia), medications (narcotics) and brain disorders.
Using the anatomic localization concept, the following is a reasonable rule out list for patients you may see on your hospice service. Therapeutic options are included.

I Airway
The most common underlying causing respiratory compromise from upper airway obstruction are Brachycephalic Syndrome, Laryngeal Paralysis and Collapsing Trachea. In our older hospice patients, space occupying masses in this area are also a concern. Please be aware, these patients may be more fragile than they first appear. In some, added stress can result in rapid decompensation. Clinical signs may occur dynamically on either inspiration or expiration. Static clinical signs usually occur with intra or extraluminal obstruction. Obstruction of the rostral upper airway (nasal passages and nasopharynx) result in a stertorous sound. This classic sound is a low pitch snoring like sound on inspiration or expiration. Obstruction of the larynx or trachea causes stridor, a high pitched sound on inspiration. Note: an upper airway cough is usually dry and nonproductive. Sometimes these patients exhibit gagging like behavior. Lower airway coughing is usually moist. Coughing on expiration is most characteristic of intrathoracic tracheal or mainstem bronchi collapse. The patients may have loud referred sounds on auscultation and hyperthermia (inability to dissipate heat). These upper airway patients are treated emergently with oxygen supplementation, anxiolytics, cough suppressants. In our hospital, we have seen patients in such severe distress that emergent intubation or tracheostomy is required. It is well documented that sometimes a fan blowing cool air in their faces helps the sensation of dyspnea. Restraint may be too much for them and we often administer medications intramuscularly rather than IV. With upper airway obstruction, it is not uncommon to administer one dose of Dex SP for inflammation (0.05-0.2mg/kg IV, IM or SQ. For mild laryngeal paralysis patients, medical management may be able to keep them comfortable for a period of time. This entails walking with a harness, weight loss, avoidance of stressors (heat, exercise and excitement) and anxiolytics. These patients should also receive gastrointestinal support in the form of antiemetics, antacids and promotility drugs to limit episodes of regurgitation and vomiting that could result in aspiration. With our collapsing trachea patients, aggressive cough suppression is the mainstay of therapy, plus steroids if inflammation is suspect. Bronchodilators may be used if there is concurrent bronchoconstriction. These patients should be treated much like Lar Par individuals ie walk with a harness, weight control, avoid stressors, etc. Brachycephalic obstructive airway syndrome patients have increased resistance to inspiratory airflow characterized by stenotic nares (58-75%), elongated thickened soft palate (87-96%), everted laryngeal saccules (55-58%), and hypoplastic trachea (46%). This condition is very similar to apnea-hypopnea syndrome in people where there is repeated airway collapse during sleep. This is a chronic inflammatory condition with an increase in inflammatory markers. Systemic consequences are documented in people and our species of interest. GI disorders have been recognized in brachycephalic dogs. In one study, 97% had abnormal esophagus, stomach or duodenum. The degree of GI disease was associated with the degree of respiratory compromise. These patients should received supplemental oxygen and sedation. Active cooling is provided if refractory to initial management (T> 103). Regardless of the cause, patients with severe acute airway obstruction are at risk for noncardiogenic pulmonary edema.
Allergic Airway Disease causes mucosal edema, airway smooth muscle hypertrophy and constriction plus excess production of airway secretions. Causes in the dog and cat are numerous and often difficult to remove (except infection). Our efforts aim to control and decrease symptoms. These patients have labored, rapid shallow breathing with increased expiratory effort and coughing. Often there is a lack of appreciable wheezes. Steroids, bronchodilators and oxygen supplementation are the rescue therapies of choice. We usually see a predictable response to steroids. Keep in mind that inflammation continues after clinical signs abate. Do not taper the steroids too quickly. You may also consider steroid inhalers such as fluticasone or flunisolide.

Bronchodilators are grouped into two classes: methylxanthines and beta2 agonists. The dose for the former is empiric at this time. The Beta 2 agonists cause increase in heart rate. The patient can also develop tolerance to Beta 2 agonists, therefore they should be reserved as a rescue therapy. Some work has been done with cyclosporine. This drug inhibits T-helper cells which appear to be the primary component in allergic immune respiratory disease. Cyproheptadine has been suggested in cats. Cyproheptadine is a serotonin receptor antagonist that inhibits feline airway smooth muscle contraction. The dose is 2mg q 12 h (experimentally the dose has been up to 8mg q 8 - 12 h).

Medications and dosages used in allergic airway disease:
Dexamethsone SP (long acting glucocorticoid) - parenteral steroid for emergency use, 0.2-0.5mg/kg IV or IM
Prednisone or Prednisolone (short acting glucocorticoid) - appropriate for maintenance use and every other day therapy, 1-2 mg/kg PO for 2 weeks, then taper over 2 - 3 months
Methylprednisone acetate (long acting glucocorticoid), appropriate only for cases with compliance issues, 20mg/cat every 4 - 6 weeks
Aminophyline (bronchodilator -methylxanthine)- not recommended orally due to short half life in dog and cat, 5-10mg/kg PO q 8 h or 5-10mg/kg IV q 6 - 8 h
Theophyline (bronchodilator - methylxanthine)- maintenance therapy - pharmacokinetics unknown, CATS: 5mg/kg PO q 8-12 h, DOGS: 5-10mg/kg PO q 12 h
Terbutaline (Beta2-agonist) - parenteral and oral bronchodilator, 0.01mg/kg IV, SQ or IM q 4 - 8h, CATS 1.25mg PO q 12 h, DOGS: 2.5mg PO q 8h

II Chest Wall Disease
This is characterized by decrease in chest wall excursions. When there is a component of muscle fatigue or paralysis one will observe paradoxical breathing pattern which is characterized by inward movement of the diaphragm on inspiration instead of the usual outward movement.
Chest wall disease may be due to neoplasia (usually secondary to excision), trauma, penetrating wounds, cervical spinal disease (fracture, IVDD), neuromuscular disease, fulminant myasthenia gravis and coral snake envenomation.
In our population of hospice patients, I would suspect that neuromuscular disease due to botulism (rare), coral snake envenomation (certain parts of the country), cervical IVDD or nontraumatic rib fractures would be the types of chest wall disease we would encounter.
Nontraumatic rib fractures are usually seen in cats secondary to chronic respiratory disease, chronic renal disease and neoplasia. These fractures usually occur mid-rib in the caudal aspect of the rib cage.

III Pleural Space Disease
These patients usually present tachypneic characterized by short shallow breathing. They may be compromised enough to present open mouth breathing with head and neck extended, crouched sternal recumbency with abducted elbows (orthopnea). Heart sounds may be muffled, absent or amplified.

Pleural space disease may be due to effusions (transudates, exudates, blood, chyle), pneumothoraces or space occupying soft tissue.

Thoracocentesis is diagnostic and therapeutic. Care needs to be taken when aspirating a chronic effusion. Changes to the visceral pleura overlying the lungs may result in a friable state with pneumothorax post centesis or re-expansion injury. The recommendation with chronic effusions is to first “tap to comfort”. Assess how the patient manages after the procedure, analyze the fluid, then consider removing more. For hospice patients with chronic effusions, pleuroports may be placed via a surgical intervention.

Paradoxical breathing has been strongly associated with pleural space disease in cats.

IV Pulmonary Parenchyma
Atelectasis - advance hospice patients are, quite often, relatively immobile. Atelectasis can be an important cause of tachypnea, dyspnea and cyanosis. It occurs secondary to prolonged recumbency, high fraction of inspired oxygen (supplementation) or a recent anesthetic episode. Frequent positioning (left, sternal, right, sternal, etc.) makes a significant difference in these patients.

Pneumonia - Can be challenging to diagnose without radiography. Many patients with bacterial pneumonia will be afebrile (40% have a fever). The CBC is neither sensitive nor specific. Respiratory signs are rarely sensitive or specific. Coughing is usually absent in cats but dyspnea is common. Some dogs will have crackles or wheezes but most cases (70-75%) will simply have “loud” lung sounds. In mild cases, patients may not be dyspneic at all. It is important to remember that in certain areas of our country, fungal pneumonia may be a real threat. Fungal titers early will be advantageous. Patients with pneumonia almost always have an underlying cause. These predisposing causes may include advanced age, impaired patient mobility, upper airway disorders, decreased mentation, regurgitation syndromes, immune compromise, metabolic disease, seizures, among others. Patients that are breathing comfortably may be managed with long term oral antibiotics and supportive care. Those that are hypoxemic need humidified oxygen supplementation at the lowest concentration that stabilizes the patient. The use of bronchodilators are controversial. The patient may be nebulized with saline and then coupaged q 4-6 hours. Coupage is not necessary if the patient is coughing.

Antimicrobial therapy is always best when guided with culture and sensitivities. In the hospice patient, this may not be feasible. Therefore a good first choice for antibiotics is a betalactam and fluorquinolone. Patients that have moderate to severe pneumonia
should receive parenteral antibiotics for a prolonged period of time (6 weeks to 6 months). NOTE: antibiotics that penetrate lung tissue well are chloramphenicol, doxycycline, enrofloxacin, trimethoprin-sulfa and clindamycin.

Mucolytic therapy is used commonly but scientific proof of its benefit is lacking. Atelectasis can contribute to hypoxemia. Recumbant patients should be turned every 1-2 hours and supported in an upright position every 12 hours. Response to appropriate antibiotics is observed in most dogs (69-88%)

Aspiration pneumonitis and pneumonia - The incidence of aspiration pneumonia may be decreased by attentiveness to risk factors. Trying to control or minimize these risk factors is important. Gastrointestinal disorders are the most common. This is present in > 60% of aspiration cases. Incidence can be decreased by use of antiemetics, prokinetics and gastric alkalization. Enteral feedings and recent anesthesia are also risk factors.

Aspiration pneumonitis is caused by direct injury and characterized by localized inflammatory mediator cascades producing chemotaxis of neutrophils followed by permeability edema. Aspiration pneumonia is an infectious process either caused by aspiration of contaminated material or by bacterial colonization of damaged lungs after a sterile aspiration event. Clinical signs include acute onset of respiratory distress, lung sounds that are louder than normal. Sometimes one will auscult fine crackles on inspiration, especially cranioventrally. Treatment includes oxygen supplementation when indicated, bronchodilators, judicious fluid therapy, nebulization and coupage plus antibiotics. The most common empiric antibiotic choices are a beta-lactam and florquinolone. Cytology, culture and sensitivity cannot be overstated.

Oxygen supplementation: A word of caution is needed here. Studies suggest that prolonged high concentrations of oxygen may be detrimental to the patient. Oxygen toxicity causes increased lung permeability, protein extravasation and impaired pulmonary compliance. Oxygen supplementation longer than 12 - 24 hours should be no greater than 60% FiO2.

There are many other pulmonary parenchymal diseases conditions that exist but have not been addressed here as they are beyond the scope of this discussion. Conditions such as Acute Lung Injury (ALI), Acute Respiratory Distress Syndrome (ARDS) and Pulmonary Thromboembolism may be life threatening and cannot be appropriately managed in the home setting.

The problem of suffering from dyspnea is one that is rapidly gaining attention in the veterinary community. In human medicine, studies show that over 40% of chronically critically ill people report pain at the highest level on the assessment scale. Other physical symptoms include unsatisfied thirst and dyspnea. Several tools to assess pain and dyspnea in nonverbal human patients are available such as Behavioral Pain Scale, Critical Care Pain Observationl Tool, Assume Pain Present approach and Respiratory Distress Observation Scale.
Owners often do not realize how important this clinical sign is and the degree of suffering it causes. When it comes to the problem of dyspnea, it is often helpful to ask your pet parents how well their pet is sleeping. Patients who can’t breath don’t sleep well.

Recently, advances have been made in alleviating the feeling of dyspnea. One such therapy is nebulization with furosemide. This common diuretic has effects on the slow and rapid acting receptors in the pulmonary parenchyma. Slow reacting receptors (SARs) are stimulated by inflation of the lung and typically found in airway smooth muscle. Reflex actions mediated by this receptor’s activation include shortening of inspiration, prolongation of expiration and reflex bronchodilation. Agents that increase SAR firing have been found to alleviate some sensations of dyspnea. The rapid adapting receptors (RARs) stop firing quickly after lung inflation. Stimulation results in effects on bronchomotor tone and breathing pattern opposite to those seen with SARs. Interventions that decrease RAR firing can decrease sensations of dyspnea. When the patient is nebulized with 5mg/kg furosemide, they only systemically absorb 20% which usually does not cause a diuretic effect. The furosemide will speed up the slow adapting receptors and slow down the rapid adapting receptors. The result is alleviation of the feeling of breathlessness (the feeling of dyspnea). Often one treatment will palliate for 4 - 6 hours. In conjunction with this, use of MgSO4 as a nebulized bronchodilator is quite helpful. MgSO4 has no contraindications for use and no significant side effects. To use this as a bronchodilator, dilute one mL of MgSO4 with 6 mL of sterile water (makes it isotonic with plasma). Then use enough of the mixture to nebulize the patient for 10min. Do not mix the furosemide and MgSO4, you will have to nebulize them individually.

Don’t forget the benefit of opioids in the hospice dyspneic patient. In human medicine they are the first, second and third choice of drugs for relief of suffering from breathlessness. There are multiple studies that show opioids in the dyspneic human patient do not hasten death.

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