PRACTICAL APPROACH TO FELINE RESPIRATORY DISTRESS

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Introduction

“Respiratory distress” is a somewhat generic term that refers to severe difficulty in achieving adequate oxygenation in spite of significant efforts to breathe. The term “hypoxia” refers to a decreased amount of oxygen available to tissues within the body, while “hypoxemia” refers to any measured decrease in PaO2. The clinical manifestation of respiratory distress is some combination of an increase in the rate and depth of breathing. This change in behavior is called “dyspnea”. Interestingly, dyspnea is a somewhat subjective feeling and animal models in general have proven to be poor representations of dyspnea in humans.

The respiratory apparatus does not have a built-in pacemaker, compared for example to the heart. The drive to breathe and activation of respiratory related muscles are the result of impulses from a region in the medulla that has been called the respiratory center or respiratory pacemaker. For breathing to change as physiological conditions change, the respiratory center receives and responds to three general types of information: 1) Chemoreceptors responding to PaO2, PaCO2 and pH, 2) mechanical information from receptors in the lung and chest wall, and 3) behavioral information from higher cortical centers. Thus, a disorder like asthma will trigger changes in breathing due to chemoreceptor input, pleural effusion will trigger changes in breathing due to impulses from the chest wall and fear will trigger changes in breathing due to behavioral information within the higher cortical centers. The task of the clinician faced with a patient in respiratory distress is to quickly determine the anatomic site of pathology and quickly relieve the disorder that is triggering the dyspneic response.

Initial approach and triage

Patients with increased rate and or depth of respiration should be assumed to be in respiratory distress until proven otherwise.

1. The first principle in these cases is to do no harm. That means that the cat should be subjected to minimal physical and environmental stress.
2. The second principle is to administer oxygen as appropriate and realistic for the patient’s state of arousal and stress. A pre-warmed and pre-oxygenated oxygen cage is often a reasonable approach. If the patient is resistant then this approach is counter-productive.
3. The third principle is to determine if the patient is having difficulty primarily during inspiration, expiration or is simply panting during both phases of respiration.

Physical exam and anatomic diagnosis

Patients with abnormal breathing primarily during the inspiratory phase of breathing have either an upper airway obstruction or a disorder of the pleural space. With observation, stertor, stridor or abnormal breathing sounds during inspiration localize the lesion to the upper airway (to the level of the thoracic inlet). In contrast, patients with disease within the pleural space typically open mouth breath without noisy breathing. Patients with abnormal breathing primarily during the expiratory phase of breathing have a lower airway disorder. Finally, patients with abnormal breathing during both phases of breathing more often have disease of the lung parenchyma.

The next step in assessment of the patient is auscultation of the chest. The presence of a heart murmur and or adventitious lung sounds may help increase suspicion of a primary cardiac, airway or parenchymal disorder. However, the absence of a heart murmur and or the absence of adventitious lung sounds does not rule out primary cardiac, airway or lung disease.

If pleural space disease is strongly suspected, the next step is thoracentesis with the appropriate number of persons available to perform the procedure quickly safely and effectively. In practice, this requires one person to safely restrain the patient, a second to place and maintain the centesis needle,
and a third person to manipulate the drainage iv tubing and centesis syringe. In cases of pleural effusion or pneumothorax, drainage of as little as 10cc of air/fluid/kg of body weight can help stabilize the patient and relieve their anxiety.

If pleural space disease is not strongly suspected, the third step is radiographic evaluation of the chest cavity. If the patient is not stable, it makes sense to start with a single view using whatever positioning the patient will tolerate. Many general practices now have ultrasound and “FAST” scanning to determine the presence or absence of pleural fluid. Pulse oximetry used in these awake patients can be extremely valuable in determining the presence of significant hypoxemia (pulse ox <90 roughly correlates with PaO2 60mmHg, >95 is roughly equivalent to PaO2 80 mm Hg). Lastly, n-terminal pro BNP levels can help distinguish cardiac from non-cardiac causes of dyspnea, but this test is not available at the “bedside”.

Diagnoses
The most common causes of respiratory distress in our feline patients include asthma, heart failure, pleural effusion and pneumothorax. Chest radiographs at the time of presentation may demonstrate a classic pattern for each disorder. However, specifically in the feline species, more so than in dogs:
1. Asthma may appear to mimic multilobular metastatic disease or fungal infection. It is still only asthma.
2. Heart failure may appear as multiple patchy infiltrates. There may be a single, triangular infiltrate in a caudo-dorsal position etc. Cats with heart disease additionally may or may not have classic cardiomegaly.
3. Pleural effusion may obscure the nature lines of the diaphragm imitating a rent in the diaphragm. It is still only pleural effusion.

Treatment
Specific treatment of course is dependent on the presumptive diagnosis and cause of the respiratory distress. Emergency treatment of pleural effusion requires removal of fluid. If pneumothorax is present air must be also removed but at a slightly gentler pace to avoid re-expansion pulmonary edema or disturbance of a newly forming fibrin seal over the rent in the parenchyma. If there is no pleural space disorder a thoracentesis done correctly is minimally invasive to the patient. Asthmatic patients should be treated with a bronchodilator such as terbutaline or albuterol given i.m. s.q. or by inhalation. Theoretically, a heart failure patient treated with a bronchodilator may worsen due to increased myocardial oxygen demand as a result of the B1 effects of the (mostly B2 selective) drugs. Similarly, parenteral corticosteroids given to a suspected asthmatic that in reality suffers heart failure can be injurious. Patients with “cardiac disease” causing respiratory distress can usually be safely treated with intravenous furosemide.

Summary
Cats that present in respiratory distress represent a true medical emergency. Accurate triage, minimally invasive approaches and rapid administration of specific drugs and treatments are most often associated with the best outcomes. In the course of the lecture these issues will be approached in much greater detail.