Dyspneic cats can be challenging to diagnose and manage because of their fragility and level of stress/anxiety when acutely dyspneic. Rapid stabilization, diagnosis and appropriate management can be lifesaving. Although some acutely dyspneic cats have congestive heart failure, other causes of acute tachypnea/dyspnea may include feline asthma, pleural effusions unrelated to heart failure, or trauma of the airways or thorax. The following represents the consensus of the CEG for approach and management of the acutely dyspneic feline patient. For information regarding diagnosis and management of heart failure in cats, please see Recommendations for Diagnosis and Management of Feline Heart Failure.

Approach to the Dyspneic Feline Patient

Dyspnea in cats may result from airway obstruction, pulmonary diseases, pleural space disorders, or congestive heart failure (CHF). Immediate differentiation of these problems may NOT be possible but all acutely dyspneic feline patients can be approached using the following guidelines:

**INITIAL PATIENT MANAGEMENT**

Cats with dyspnea of any etiology are often extremely anxious and may be fractious due to their stressed condition. These patients may deteriorate rapidly; provision of oxygen and minimization of handling are priorities. All unstable patients should be stabilized prior to any imaging.

1. Oxygen may be supplied via oxygen cage, by mask or through “blow-by” techniques (where oxygen via tube is provided near the cat’s face).
2. Sedatives should be administered if restraint is difficult or the patient is distressed; many cats in CHF benefit from anxiolysis. Frequently, butorphanol (0.2 to 0.3 mg/kg) alone will decrease overt anxiety when administered IM in cats. In more fractious patients, butorphanol combined with acepromazine (0.03 to 0.05 mg/kg) and administered IM provides mild to moderate sedation while having minimal cardiovascular depressant effects. Acepromazine should NOT be used in hypotensive or hypothermic cats. The clinical staff should be ready to perform tracheal intubation and positive pressure ventilation should life-threatening dyspnea be evident and respiratory arrest imminent. Obvious laryngeal or tracheal stridor or loud noise associated with breathing may indicate airway obstruction that can be temporarily managed by tracheal intubation.
3. Indirect sources of heat (water circulation pads, warmed fluid bags) may be helpful in cats with subnormal body temperature, but aggressive heating (e.g. use of a Bair Hugger™) should be avoided.

**DISCERNING THE CAUSE OF DYSPNEA/RESPIRATORY DISTRESS**

The clinician should try to determine the anatomic “level” of disease responsible for abnormal ventilation.

1. Airway obstruction should be suspected if the history or physical examination reflects trauma of the neck, if inspiratory (or combined inspiratory-expiratory) stridor is present, if there are loud respiratory noises audible without a stethoscope, or if mucous membranes are cyanotic in a gasping animal. Panting is usually NOT associated with airway obstructions, and pulmonary auscultation often reveals harsh inspiratory sounds reflected from the upper airways.
2. Pulmonary disease as a cause of acute dyspnea in cats is often due to feline asthma; clinical signs may include acute dyspnea, cyanosis, panting, and wheezing. There may be a history of cough and obvious pulmonary crackles if inflammatory pulmonary infiltrates are present. Cats with asthma seldom have jugular distention or evidence of pleural effusion. Other causes of respiratory distress in cats include pneumonia, pulmonary neoplasia, and noninfectious, inflammatory lung diseases. These often have historical clues and a long history of clinical signs. Thoracic trauma should also be considered in acute dyspnea.
3. Pleural effusions including chylothorax are common in cats with CHF. Idiopathic chylothorax, pyothorax, and neoplasia involving the mediastinum, lung, or pleura are other causes of pleural effusion. Clinical signs associated with pleural effusion include rapid ventilation (with or without increased depth), muffled heart sounds, and an audible or percussible fluid line characterized by absence of clear breath sounds in the more ventral lung fields. The presence of jugular distention indicates that the effusion is more likely due to CHF (or excessive volume loading, as in cats receiving fluid therapy). If breath sounds are evident in dorsal lung fields, but heart sounds are muffled, the clinician should consider both impaired heart contractility and pericardial effusion in the differential diagnosis. If an ultrasound unit is available, pleural effusion may be rapidly diagnosed with limited imaging of the thorax (see below).

4. Congestive heart failure should be suspected in a dyspneic feline patient if there is evidence of pleural effusion, jugular distention, or signs of lung parenchymal disease. Cats with pulmonary edema are typically presented with increased bronchial sounds or pulmonary crackles often (but not always) in the presence of a heart murmur, gallop sound or irregular heart rhythm. Although heart disease may be long-standing, cats with CHF can be presented acutely with no premonitory signs of heart disease prior to decompensation.

ACUTE MANAGEMENT OF THE DYSPNEIC CAT

1. Airway obstruction: acute intubation or (rarely) tracheostomy may be required to stabilize these patients. Supplemental oxygen should be provided at all times. If mild sedation is contemplated to decrease patient stress, preparations should be made for tracheal intubation prior to administration of the sedative.

2. For suspected feline asthma, use a pediatric spacer and a standard albuterol inhaler; administer two “puffs” of albuterol into the spacer and allow the cat to breathe through the mask for 10 to 15 seconds. A positive response should provide a presumptive diagnosis of reactive bronchospasm. If the response is incomplete, but reactive airway disease is strongly suspected, a single dose of terbutaline sulfate (0.1 mg/kg SQ) can be administered. If response to airway dilators is positive, administer short-acting glucocorticoids and oral or subcutaneous airway dilators (and continue using inhaled albuterol 2 to 3 times daily until stabilized). Typical doses include: dexamethasone sodium phosphate (0.1 – 0.25 mg/kg IV or IM); sustained release oral theophylline (25 mg/cat PO once daily); terbutaline sulphate (0.1 mg dose SQ or IV, or 1/4 to 1/2 of a 2.5 mg tablet PO q 12 hrs).

3. Thoracocentesis should be performed if pleural effusion or pneumothorax is strongly suspected or confirmed. Thoracocentesis should be performed prior to radiographic imaging as a method of stabilization in the severely dyspneic patient; centesis of one or both sides of the thorax should be attempted and a maximal amount of fluid removed. When CHF is suspected, special care should be taken when introducing a centesis needle into the left thorax, as an enlarged left auricle can be perforated. In patients with CHF as a cause of pleural effusion, furosemide (1-2 mg/kg IV or IM) can be administered for additional therapy of CHF after thoracocentesis has been performed but is NOT a substitute for thoracocentesis in severely affected patients. Removed pleural fluid does not need to be replaced with fluids.

Thoracic radiographs are needed for all patients with dyspnea once clinical stabilization has been achieved.

4. For suspected pulmonary edema (when the patient cannot be examined by radiography) administer parenteral furosemide (2 mg/kg IV or IM), and oxygen. Additional doses of furosemide (1-2 mg/kg IM) may be administered every 2 hours, as needed to relieve acute pulmonary edema, but the risk of prerenal azotemia and hypokalemia increases with repeated dosing of furosemide. Careful observation should be used to monitor response to therapy so that excessive furosemide dosing can be avoided. Nitroglycerine paste (¼ inch of 2% ointment applied to inside of pinna) may be useful but has not been studied. Close monitoring of respiratory rate is noninvasive and extremely helpful in judging response to acute therapy; the patient’s respiratory rate should progressive decline with therapy. If no change in respiratory rate is noted after 1-2 doses of furosemide and provision of oxygen, re-evaluation for causes of dyspnea other than CHF should be considered.

IMAGING

1. Thoracic radiographs are needed for all patients with dyspnea once clinical stabilization has been achieved, and can be used to assess heart size, vascular size and assess for pulmonary infiltrates or pleural effusion. Note: the absence of radiographic cardiomegaly does not rule out a diagnosis of
CHF but the presence of cardiomegaly is strongly supportive of a diagnosis of CHF.

2. Echocardiography is recommended in stable patients if CHF has been diagnosed and in stable patients in which the cause of pulmonary infiltrates or pleural effusion remains unclear. In well-equipped emergency rooms with capabilities for a cavitary fluid assessment thoracic scan of feline patients, a rapid ultrasonographic examination can proceed while the cat is gently restrained in sternal recumbency and receiving oxygen. This study can be used to quickly identify or exclude these key findings: 1) pleural effusion; 2) pericardial effusion (usually caused by CHF in cats); 3) left atrial dilatation (expected finding in cats with CHF); and 4) an air/fluid interface typical of intrapulmonary fluid (comet tails and related findings).

Rapid stabilization of the acutely dyspneic cat typically includes oxygen supplementation and anxiolysis, which may include light sedation. Clinicians should always be prepared to intubate the patient if needed in the acute situation. Parenteral drugs (e.g. furosemide or steroids) can be given and therapeutic thoracocentesis performed as soon as the need is determined, but other diagnostic tests, such as thoracic radiography and echocardiography, may need to be delayed until the patient’s condition is more stable.