Managing dogs and cats in respiratory distress is a multifaceted effort that involves stabilizing patients prior to determining a definitive diagnosis. Fortunately, respiratory distress—no matter what the cause—requires somewhat standardized interventions during initial stabilization.

**INITIAL STABILIZATION**

One of the benefits of initial stabilization is that it provides the practitioner time to consider the appropriate diagnostic and subsequent therapeutic approach.

**Oxygen Supplementation**

Initial stabilization of a patient in respiratory distress generally involves provision of oxygen supplementation, with or without patient sedation.

- The most common type of oxygen supplementation provided is use of an oxygen cage with a high fraction of inspired oxygen ($\text{FiO}_2$) (eg, 40%–60%); a face mask or flow-by oxygen from a hose can also be used.
- In more extreme cases, animals in respiratory distress may require emergency intubation, higher $\text{FiO}_2$ (eg, 100%), and provision of positive pressure ventilation in order to provide adequate respiratory stabilization.
- Particularly in cases of upper airway obstruction, the practitioner may need to ensure a patent airway by intubation or tracheostomy (if oral intubation is not possible).

**Sedation**

Sedation with careful monitoring and, if necessary, intubation and ventilation can be extremely useful in animals that have become anxious due to hypoxemia and/or hypercapnia. In some patients, especially dogs with upper airway obstruction, stabilization may require sedating the animal by administering some form of anesthetic induction agent; then clearing the oral cavity of obstructing material (eg, secretions or foreign material in a choking animal) prior to intubation or tracheostomy.

**Cooling Measures**

Animals with upper airway obstruction, such as those with laryngeal paralysis, may become hyperthermic due to the increased work of breathing. Because of the airway obstruction, these animals are unable to effectively pant, resulting in inability to thermoregulate and dissipate heat. As such, cooling hyperthermic patients in respiratory distress is an important component of initial stabilization, and can be accomplished by:
- Administering room temperature IV fluids
- Covering the patient with wet towels
- Putting a fan on the patient
- Applying alcohol to the axilla, inguinal area, and feet.

Active cooling should stop once the patient’s temperature reaches 103°F to avoid precipitating hypothermia.

**Thoracocentesis**

Initial stabilization may also include thoracocentesis, if severe respiratory distress is secondary to pleural space disease, such as pneumothorax or pleural effusion.

**INITIAL DIAGNOSTIC APPROACH**

Diagnostic approach to a patient in respiratory distress should consider the patient’s signalment and history as well as the broad anatomic differential diagnoses of dyspnea (Table 1, page 54).

**Signalment**

Clues in the patient’s signalment are common. For example:
- Upper airway obstruction due to brachycephalic airway disease is a common cause of respiratory distress in brachycephalic dogs, such as English bulldogs.
- Cardiogenic pulmonary edema is a common cause of respiratory distress in small breed dogs with chronic valvular disease (eg, mitral endocardiosis), such as Cavalier King Charles spaniels.
- Lower airway obstruction associated with asthma is a common cause of respiratory distress in cats, with certain breeds, such as the Siamese, overrepresented.
History

History can also be extremely useful; for example:
- History of blunt trauma (eg, hit by a car) should prompt concern for pulmonary contusions, pneumothorax, diaphragmatic hernia, or flail chest.
- In cats, a history of cough is consistent with asthma, while in dogs, a cough might suggest tracheobronchial disease, interstitial lung disease, or pulmonary edema.

Physical Examination

Examining a patient with respiratory distress should involve:
1. Initial observation: Consider breathing pattern, presence of externally audible noise with breathing, any signs of trauma, or abdominal distension
2. Lung auscultation:
   » Increased adventitial lung sounds (eg, crackles, wheezes, harsh lung sounds) are associated with lower airway and pulmonary parenchymal disease
   » Decreased lung sounds, in an animal with respiratory distress, are associated with pleural space disease.
3. Cardiac auscultation: A murmur, gallop, or other arrhythmia may indicate underlying cardiac disease and the potential for cardiogenic pulmonary edema or pleural effusion.

In general, breathing patterns help narrow the list of differential diagnoses (Table 1). For example, upper airway obstruction is associated with inspiratory dyspnea and an externally audible noise. In contrast, lower airway obstruction tends to be associated with expiratory dyspnea and wheeze, with the wheeze generally just audible on thoracic auscultation with a stethoscope, rather than externally audible.

Diagnostic Tests

Extensive diagnostics should not be performed until the patient has been stabilized as much as possible, a brief physical examination has been performed, and the practitioner has localized the disease to the most likely anatomic location (Table 1). Diagnostic tests may subsequently involve:
- Blood analysis: Screening blood tests, blood gases
- Imaging: Thoracic ultrasound, including focused assessment with sonography for trauma, triage, and tracking (tFAST); thoracic radiographs; thoracic computed tomography (CT); or echocardiography
- Respiratory fluid analysis: Bronchoalveolar lavage, thoracocentesis

Dogs and cats with respiratory distress can be classified into 8 disease categories, some of which are associated with distinct breathing patterns observed during physical examination. These categories include both primary respiratory diseases and secondary causes of respiratory difficulty. Diagnostic approach is determined by the category of disease causing respiratory distress.

| TABLE 1. Anatomic Classification: Causes of Respiratory Distress |
|-----------------|-----------------|----------------|
| DISEASE CATEGORY | EXAMPLES | BREATHING PATTERN |
| 1. Upper Airway Obstruction | • Brachycephalic airway disease • Laryngeal paralysis | • Inspiratory dyspnea • Externally audible noise (eg, stertor, stridor) |
| 2. Lower Airway Obstruction | • Asthma | • Expiratory dyspnea • Wheeze (audible with stethoscope) |
| 3. Pulmonary Parenchymal Disease | • Pneumonia • Interstitial lung disease • Pulmonary edema • Pulmonary contusions | • Not consistent; may be rapid, shallow, or both, and have both inspiratory and expiratory components |
| 4. Vascular | • Pulmonary thromboembolism | • Not specific |
| 5. Pleural Space Disease | • Pneumothorax • Pleural effusion | • Inspiratory dyspnea, rapid shallow breathing, or generalized paradoxical breathing • Reduced lung sounds on auscultation |
| 6. Flail Chest | | • Focal paradoxical breathing |
| 7. Abdominal Distension | • Ascites • Organomegaly | • Inspiratory dyspnea |
| 8. Look-alike Diseases | | • Not specific |
• **Airway examination**: Upper airway examination, tracheobronchoscopy  
• **Drug trials**: Such as bronchodilators, diuretics, and corticosteroids.

**UPPER AIRWAY OBSTRUCTION**  
**Etiology**  
Upper respiratory tract obstruction involves a mechanical or functional obstruction of the upper (large) airways (ie, the pharynx, larynx, or trachea). Nasal disorders are not considered in this article as the animal should always be able to open its mouth and breathe, preventing the development of dyspnea even if the nasal cavity is obstructed.

Specific causes of upper airway obstruction include:
• **Naso-oropharyngeal disorders**, including polyps (especially in cats), masses, and foreign bodies  
• **Severe head trauma** that results in bone fractures (especially nasal, jaw, and palatine fractures) and associated hemorrhage and swelling  
• **Laryngeal disorders**, including laryngeal paralysis, laryngeal collapse, laryngeal masses (eg, neoplasia, abscesses, granulomas), and laryngeal inflammation  
• **Tracheal diseases**, including tracheal collapse, tracheal foreign body; tracheal stenosis, stricture, tracheal tear, or tracheal mass (either intra- or extraluminal)  
• **Brachycephalic airway disease**, which involves a combination of primary and secondary anatomic abnormalities of the upper airways, including stenotic nares, an elongated soft palate, everted laryngeal saccules, laryngeal edema and/or collapse and, in some breeds (eg, English bulldog), a hypoplastic trachea.

**Clinical Signs**  
Characteristic signs in patients with an upper airway obstruction include inspiratory distress and an externally audible noise associated with breathing (eg, stertor, stridor). Tracheal disease is usually associated with a cough.

**Initial Stabilization**  
Initial stabilization and therapy may involve:
• **Oxygen administration/securing an airway**: Generally administered by face mask (if tolerated), flow-by oxygen, or oxygen cage, with intubation or tracheostomy performed if needed  
• **Sedation**: Achieved with anxiolytic drugs, such as acepromazine or dexmedetomidine, or sedative analgesics, such as butorphanol (Table 2)  
• **Cooling**: Many dogs with upper airway obstructions become hyperthermic due to inability to dissipate heat through their upper airways; the goal is to reduce body temperature to at least 103°F, while avoiding hypothermia  
• **Corticosteroids**: Breathing against an obstruction can result in marked edema of the upper airway soft tissue; therefore, anti-inflammatory doses of corticosteroids (eg, dexamethasone SP, 0.15 mg/kg IV single dose or Q 24 H) can be considered.

**Diagnostic Approach**  
Once the patient is stable, diagnostic tests can be pursued.

**Upper airway examination.** Examination is performed after preoxygenation under sedation. At its most basic, examination may involve inspection of the oropharynx and larynx with a laryngoscope; in patients with suspected tracheal disease, it

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In dogs, reasonable choices for sedation are butorphanol, acepromazine, or dexmedetomidine, while butorphanol is the drug of choice in cats.

Choice of drug(s) used for sedation/anxiolysis should be based on the individual drug’s properties, and relative risks versus benefits for the patient. For example:
• **Butorphanol** is a very safe and effective drug at recommended doses; however, it is relatively short acting (often only 1–2 hours) and cannot easily be reversed.  
• **Acepromazine** is also very effective; however, it may be more likely to produce undesirable effects, such as hypotension; has a long duration of action (4–6+ hours); and cannot be reversed.  
• **Dexmedetomidine** has the desirable quality of being reversible (with atipamezole) and titratable (given a short duration of action); however, it can produce undesirable effects, such as bradycardia and hypotension. Regardless of the chosen drug, in potentially unstable patients, lower doses are given initially and later increased as needed and tolerated by the patient.

**Sedation for Patients in Respiratory Distress**

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**TABLE 2. Patients in Respiratory Distress: Sedative Drug Dosages**

<table>
<thead>
<tr>
<th>SEDATIVE DRUG</th>
<th>DOSE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>0.1–0.4 mg/kg IM or IV Q 1–4 H, as needed</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>0.005–0.05 mg/kg IM or IV Q 4–8 H, as needed</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>0.01–0.1 mg/kg/H IV CRI</td>
</tr>
</tbody>
</table>
may involve tracheobronchoscopy with a flexible bronchoscope or endoscope.

When evaluating laryngeal function as part of an upper airway examination:
• Take care to minimize the level of anesthesia to preserve laryngeal function as best as possible
• Consider using the respiratory stimulant doxapram HCl (0.5–1.1 mg/kg IV) to stimulate laryngeal motion
• Carefully observe inspiration versus expiration to ensure that the larynx is abducting (increasing the aperture of the rima glottis) on inspiration (rather than on expiration as might occur with paradoxical motion in patients with laryngeal paralysis).

Cervical and thoracic radiographs are useful for patients with laryngeal or tracheal disease to detect masses and collapse.

Fluoroscopy is useful for detecting dynamic upper airway collapse that may not be visible on standard radiographs.

Management
Definitive management for upper respiratory tract obstruction is extremely varied, depending on the definitive diagnosis, and beyond the scope of this review.

LOWER AIRWAY OBSTRUCTION

Etiology
Lower airway obstruction is associated with a narrowed bronchial lumen, which can be caused by varied pathophysiologic processes, including:
• Bronchial inflammation with edema and hyperemia of bronchial mucosa
• Bronchospasm
• Bronchomalacia
• Mucus accumulation
• Acute anaphylactic reaction (uncommon).
In all of these conditions, the bronchial lumen tends to close early during expiration, while it is opened by radial traction from the lungs during inhalation. Therefore, expiratory dyspnea is a hallmark of lower airway obstruction.

Specific Diseases
Feline asthma and chronic bronchitis in dogs and cats are associated with accumulation of mucus in the lower airways that contributes to obstruction.

Feline asthma. The classic disease in cats that causes lower airway obstruction is feline asthma, the hallmarks of which are eosinophilic airway inflammation, reversible bronchoconstriction and, ultimately, airway remodeling.

Chronic bronchitis. Lower airway disease in cats may also be associated with neutrophilic inflammation (often referred to as chronic bronchitis), or a combination of both eosinophilic and neutrophilic inflammation. In dogs, bronchomalacia—seen in severe, end-stage, chronic bronchitis—can also cause lower airway obstruction.

Clinical Signs
Characteristic signs in an animal with lower airway obstruction usually include expiratory distress and, sometimes, an expiratory grunt or push. These patients may have an expiratory wheeze on thoracic auscultation and, less commonly, an externally audible wheeze.

Initial Stabilization
Initial stabilization and therapy usually involve:
• Oxygen supplementation: See recommendations in the Initial Stabilization section (page 53)
• Bronchodilator trial: Options for an acute bronchodilator trial include either:
  » Inhaled albuterol (1 or 2 puffs from a metered dose inhaler with a spacer)
  » Single dose of terbutaline (0.01 mg/kg IM or SC). Bronchodilator therapy often results in rapid improvement in these patients (eg, within 5–15 minutes).

Diagnostic Approach
Once the patient is stable, the diagnostic approach usually involves:
• Thoracic radiographs: Lower airway disease is classically associated with a bronchial or bronchointerstitial pattern on thoracic radiographs. Additionally, air trapping in cats with asthma may result in pulmonary hyperinflation and a flattened diaphragm.
• Lower airway cytology: Eosinophilic inflammation (> 17% eosinophils) is characteristic of feline asthma, while neutrophilic inflammation is evident in dogs and cats with chronic bronchitis.
• Heartworm testing (ideally both antigen and antibody tests): Determines if heartworm associated respiratory disease is present in cats.
• Baermann fecal test: Evaluates for lungworm disease.

Management
PULMONARY PARENCHYMAL DISEASE

Etiology
Pulmonary parenchymal diseases affect the terminal and respiratory bronchioles, interstitium, alveoli, and vasculature. These diseases include pneumonia, pulmonary edema, interstitial lung disease, pulmonary neoplasia, and others. Examples of pulmonary parenchymal diseases are listed in Table 3.

Clinical Signs
Characteristic signs in an animal with pulmonary parenchymal disease often include abnormally loud breathing sounds on thoracic auscultation, such as harsh lung sounds, crackles, and wheezes. Patients with cardiogenic pulmonary edema may also have obvious cardiac abnormalities on auscultation, such as a murmur or arrhythmia.5

Animals with infectious causes of pulmonary parenchymal disease (eg, pneumonia) may have a fever; however, fever has only been reported in about 1/8 of dogs and 1/4 of cats with pneumonia, making it an unreliable abnormality.6

Initial Stabilization
Initial stabilization and therapy usually involve:

• Oxygen supplementation: See recommendations in the Initial Stabilization section (page 53)
• Diuretic: Depending on index of suspicion for cardiogenic pulmonary edema, a furosemide trial dose may be administered (typically, 2–4 mg/kg IV, IM)
• Antibiotics: If there is a high index of suspicion for pneumonia (eg, history of vomiting, regurgitation, fever), the patient should begin receiving broad spectrum empiric antibiotics as soon as possible.7

Diagnostic Approach
Once the patient is stable, if cardiogenic pulmonary edema is suspected, firstline diagnostics should include:

• Thoracic radiographs
• Echocardiography

When it is unclear whether the etiology is primary cardiac versus primary respiratory disease, other diagnostics can be performed, including:

• Measurement of serum NT-proBNP (aminoterminal pro B-type natriuretic peptide)—a biomarker associated with atrial stretch, which is increased in dogs and cats with clinically significant heart disease; in cats, this test can be performed in a point-of-care fashion but, in dogs, is only available as a reference laboratory test at this time.
• Airway cytology (depending on radiographic abnormalities identified).

Further diagnostics for interstitial lung disease may include:

• Thoracic CT
• Lung biopsy

If a solitary lung mass is identified close to the chest wall, percutaneous fine needle aspiration or biopsy may be an ideal diagnostic modality. Additionally, surgical removal via lung lobectomy may be both diagnostic and therapeutic.

Management
Treatment for pulmonary parenchymal diseases depends entirely on the underlying disease. However, regardless of the underlying cause, judicious fluid therapy is usually appropriate to prevent exacerbation of extravascular lung water and potential diffusion impairment. Intravenous fluid therapy is generally absolutely contraindicated in animals with heart failure; rather, diuretic therapy is a mainstay of treatment.

Specific therapeutic approaches include:

• Cardiogenic pulmonary edema: Diuretic therapy and other cardiac drugs
• Microbial pneumonia: Antimicrobial administration and supportive care; adjunct therapies, such as nebulization and coupage, may be considered. Empirical antimicrobial drug choices depend somewhat on patient stability.

Animals that present in respiratory distress generally warrant broad spectrum coverage with parenterally administered antibiotics, such as:

• Monotherapy with a potentiated aminopenicillin, such as ampicillin + sulbactam

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TABLE 3.
Classification & Examples of Pulmonary Parenchymal Diseases

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>EXAMPLES</th>
</tr>
</thead>
</table>
| Pneumonia                      | • Infectious (viral, bacterial, parasitic, fungal)  
• Aspiration                     |
| Pulmonary edema                | • Cardiogenic                                 |
|                                | • Noncardiogenic                               |
| Interstitial lung diseases     | • Idiopathic pulmonary fibrosis                |
|                                | • Eosinophilic bronchopneumopathy             |
|                                | • Heartworm disease                            |
| Pulmonary neoplasia            | • Primary                                      |
|                                | • Metastatic                                   |
| Traumatic pulmonary            | • Pulmonary contusions                         |
| parenchymal injury             |                                               |
(30–50 mg/kg IV Q 6 H) or ticarcillin + clavulanate (50 mg/kg IV Q 6 H)
» Dual therapy with a beta-lactam antimicrobial (eg, ampicillin, 30–50 mg/kg IV Q 6 H) and enrofloxacin (5 mg/kg IV Q 24 H in cats; 10–20 mg/kg IV Q 24 H in dogs)
» Other antibiotic choices may also be appropriate but are beyond the scope of this article.

- **Interstitial lung disease**: These conditions are challenging to treat; some are steroid responsive
- **Pulmonary neoplasia**: Management depends on type, location, and whether neoplasia is primary versus metastatic; surgery, chemotherapy, and radiation therapy are all considerations.

### PULMONARY THROMBOEMBOLISM

#### Etiology

Causes of PTE are the same as for any thromboembolic disease—essentially abnormalities in Virchow’s triad, which include abnormalities of blood flow (turbulence or stasis), endothelial damage, and hypercoagulability.

With PTE, it is critical to identify and treat the underlying disease if it is not immediately apparent, so as to reduce the risk of further thromboembolic events. Theoretically, any systemic inflammatory state can result in a systemic pro-coagulant state that predisposes the patient to PTE. Table 4 lists diseases and conditions known to predispose veterinary patients to hypercoagulability. 8,9

#### Clinical Signs & Diagnostic Approach

Diagnosis of PTE can be challenging. While thoracic radiographs may be normal, indications of PTE include (Figure):

- Degree of respiratory distress out of proportion with changes on radiographs (ie, a patient in severe respiratory distress with minimal abnormalities on thoracic radiographs)
- Demonstration of focal hypolucency or vessel truncation
- Evidence of main pulmonary artery and/or right heart enlargement due to pulmonary hypertension, a result of significant PTE.

Echocardiography is also a useful diagnostic modality in cases of suspected PTE as it can document pulmonary hypertension that often occurs secondary to PTE; detect right-sided cardiomegaly and main pulmonary artery dilation; and may allow visualization of a thrombus in the main pulmonary artery.

Advanced imaging, such as CT angiography or, less commonly, a ventilation/perfusion lung scan with nuclear scintigraphy, are required to confirm the diagnosis. 8,9

#### Stabilization & Management

Stabilization involves oxygen supplementation, and treatment requires anticoagulant drugs as well as addressing the underlying disease. Therapies that can be used include:

- **Anticoagulants** (unfractionated or low-molecular-weight heparin) and/or **antiplatelet drugs** (eg, clopidogrel) reduce risk of further thrombus formation. Although the ideal antithrombotic strategy for dogs and cats with PTE is unknown, it is reasonable to combine low-molecular-weight heparin (eg, dalteparin, 150 U/kg SC Q 12 H) with clopidogrel (approximately 2 mg/kg PO Q 24 H in dogs; 18.75 mg/day in cats). Dalteparin dosing should ideally be monitored by assessment of anti-Xa activity.
- **Thrombolytic therapies**, such as tissue plasminogen activator (tPA), can also be administered; however, systemic administration of tPA is limited by adverse effects.
- **Sildenafil** is often beneficial

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TABLE 4.

### Diseases & Conditions That Predispose Veterinary Patients to Hypercoagulability

<table>
<thead>
<tr>
<th>DISEASES</th>
<th>CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac disease</td>
<td>Exogenous corticosteroid administration</td>
</tr>
<tr>
<td>Disseminated intravascular coagulation</td>
<td>Indwelling IV catheters</td>
</tr>
<tr>
<td>Heartworm disease</td>
<td></td>
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<tr>
<td>Hyperadrenocorticism</td>
<td></td>
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<tr>
<td>Immune-mediated hemolytic anemia</td>
<td></td>
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<tr>
<td>Neoplasia</td>
<td></td>
</tr>
<tr>
<td>Protein-losing enteropathy</td>
<td></td>
</tr>
<tr>
<td>Protein-losing nephropathy</td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td></td>
</tr>
</tbody>
</table>

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*FIGURE. Thoracic radiograph demonstrating focal hypolucency in the right middle and caudal lung lobes associated with pulmonary thromboembolism; main pulmonary artery enlargement is also evident. Courtesy Dr. Carol Reinero*
for reducing moderate to severe pulmonary hypertension if documented on echocardiography.

**PLEURAL SPACE DISEASE**

**Etiology**

Pleural space disease refers to abnormal accumulations within the pleural space that impair lung expansion on inhalation. These accumulations can be associated with fluid (ie, pleural effusion), air (ie, pneumothorax), masses, or organs (ie, diaphragmatic hernia).

**Clinical Signs**

Animals with pleural space disease may have:
- Inspiratory distress
- Rapid shallow breathing
- Paradoxical breathing pattern in which the chest falls on inspiration and the abdomen expands rather than the chest rising with inspiration
- Decreased lung sounds on thoracic auscultation.

**Diagnostic Approach**

Thoracic imaging is the mainstay of diagnosis. In unstable patients, point-of-care ultrasound is particularly useful to confirm the presence of pleural fluid or air. Radiographs can also confirm diagnosis but ideally, in unstable patients, thoracocentesis should be performed after ultrasound and prior to radiographs. If ultrasound is not available, thoracocentesis should be performed based on clinical suspicion, in order to stabilize the patient prior to obtaining radiographs.

**Stabilization & Management**

In patients with pleural effusion or pneumothorax, therapeutic thoracocentesis should result in immediate improvement. Pleural fluid can then be submitted for analysis/cytology and, in cases of pyothorax, bacterial culture (both aerobic and anaerobic culture).

Once therapeutic thoracocentesis has been performed, the next step is addressing the underlying disease. Specific discussion of treatment of underlying diseases is beyond the scope of this article.

**FLAIL CHEST**

**Etiology**

Flail chest refers to destabilization of a portion of the rib cage, which occurs if there are rib fractures in 2 different locations (proximal and distal) on the same rib(s). This condition often affects multiple ribs (at least 2 consecutive ribs), creating a flail segment.

Concurrent injuries, such as pulmonary contusions and pneumothorax, are common in dogs with flail chest and are generally the cause of respiratory compromise, rather than the flail chest itself.

**Clinical Signs & Diagnostic Approach**

Flail chest is usually visually obvious on examination, but radiographs are indicated to confirm the nature of the rib fractures and allow assessment of severity of the underlying pulmonary parenchymal damage. Rib fractures are extremely painful and may cause rapid, shallow breathing because big chest excursions cause more pain than little breaths.

**Stabilization & Management**

Management of flail chest is often supportive; the following should be provided:
- **Oxygen supplementation**, given the high likelihood of underlying pulmonary contusions
- **Appropriate analgesia**:
  - Usually in the form of systemic analgesia (eg, pure mu-opioid agonists, such as hydromorphone or fentanyl) ± local nerve blocks
  - Intercostal nerve blocks can be performed in dogs using 0.5% bupivacaine, with a total of 1 to 4 mg/kg divided between sites
  - If local anesthetic nerve blocks are used in cats, dose reduction to prevent toxicity is important; generally, the total local anesthetic dose should not exceed 0.2 to 0.5 mg/kg in cats; particular care should be taken to avoid inadvertent IV administration
  - Although use of nonsteroidal anti-inflammatory drugs (NSAIDS) should be avoided in the initial stabilization and management of trauma patients, NSAIDs can be considered later in the course of hospitalization once the patient is hemodynamically stable.
  - Additional supportive care may include:
    - **Patient positioning in lateral recumbency**, with the flail segment facing downwards
    - **Bandaging the chest** to reduce movement of the flail segment, although, extreme care must be taken to avoid further impeding inspiration
  - Surgery is not indicated unless penetrating thoracic wounds are present, in which case an exploratory thoracotomy should be performed. Assuming unilateral penetrating thoracic wounds, a lateral thoracotomy is performed to allow visualization of the affected thorax, a lung lobectomy if necessary, and thoracic lavage, prior to closure with placement of a chest tube.
ABDOMINAL DISTENSION

Etiology

Significant abdominal enlargement (Table 5) can result in respiratory distress because it impedes thoracic expansion during inspiration. Dyspnea is rarely a presenting sign, but tachypnea is common in these patients.

Clinical Signs & Diagnostic Approach

Respiratory distress due to abdominal distension is usually visually obvious. Abdominal palpation and imaging (ie, abdominal radiographs and/or ultrasound) can help determine the underlying cause.

Stabilization & Management

Supplemental oxygen may provide some relief, but treatment should be aimed at reducing the degree of abdominal enlargement. Reducing the abdominal distension may be straightforward, such as with abdominocentesis in the case of ascites, or more complicated. For example, in the case of severe hepatosplenomegaly in dogs with immune-mediated hemolytic anemia, nothing can be immediately done other than treating the underlying cause and giving the patient time to recover.

LOOK-ALIKE SYNDROMES

Apparent breathing difficulty caused by non-respiratory conditions can occur in association with severe pain, acidosis (eg, Kussmaul respiration associated with diabetic ketoacidosis), anemia, drug administration (eg, opioids), shock, and hypotension. These diseases or conditions can generally be identified based on a complete history, physical examination, and screening laboratory tests. Thoracic radiographs can help definitively rule out underlying respiratory disease.

Since the increased respiratory effort associated with these conditions is not usually oxygen responsive, management is aimed at treating the underlying disease.

IN SUMMARY

The mainstays of management of a patient in respiratory distress are:
1. Initial stabilization, including oxygen supplementation and potentially sedation
2. Characterization of the breathing pattern to localize the disease
3. Systematic approach to diagnostics and therapy based on identifying the anatomic location of the cause of respiratory distress.

CT = computed tomography; FiO₂ = fraction of inspired oxygen; NSAID = nonsteroidal anti-inflammatory drug; PTE = pulmonary thromboembolism

References


### TABLE 5.

**Intra-abdominal Pathology That Results in Significant Abdominal Distension**

- Abdominal masses
- Ascites
- Gastric dilatation +/- volvulus
- Hepatomegaly
- Late-term pregnancy
- Splenomegaly

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