VOL 1

**Chapter 5
Respiratory distress**

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**Introduction**

Respiratory distress is the second most common chief complaint after minor trauma, making up 13% of adult EMS calls [1]. It is both challenging and rewarding for the EMS provider. Diagnosis depends on often subtle and overlapping signs and symptoms. While incorrect management is potentially detrimental, with correct diagnosis comes the potential for life-saving intervention and rapid improvement. The landmark Ontario Prehospital Advanced Life Support study demonstrated a significant survival benefit in respiratory distress when interventions including nebulized beta-agonists, sublingual nitroglycerin, intubation, and intravenous medications and fluids were added to the EMS systems of the included cities [2].

**Prehospital assessment and diagnosis**

The approach to the dyspneic patient must always begin with a focus on immediate threats to survival, such as airway obstruction. Once this has been dealt with, provisional causes can be considered so as to guide specific therapy. Accurate diagnosis of the cause of dyspnea in prehospital settings remains difficult. Studies have shown that paramedics are able to determine the etiology of dyspnea with only moderate accuracy. In one of the more positive retrospective studies, a prehospital diagnosis of cardiac, pulmonary, or other as the cause of “difficulty breathing” agreed with that of the emergency department (ED) diagnosis 81% of the time [3]. However, Jaronik et al. studied 144 patients given furosemide in the field and noted that it was given appropriately only 58% of the time to patients with a subsequent diagnosis of congestive heart failure or an elevated B-type natriuretic peptide (BNP) level. It was given inappropriately 42% of the time, and for diagnoses in which it was potentially harmful 17% of the time [4]. Almost one-quarter of patients who received furosemide from EMS in this study subsequently required IV fluid therapy in the hospital [4]. Therefore, prehospital treatment must carefully find a balance between disease severity, diagnostic certainty, and the likelihood of harm.

Much of the assessment of disease severity comes from general observation of the patient supplemented by physical examination and close monitoring of vital signs, cardiac rhythm, pulse oximetry (SpO2), and end-tidal carbon dioxide (EtCO2) levels. Some of the useful questions that can be asked by a medical oversight physician over the radio or phone include how many words the patient can speak at a time, whether there is associated diaphoresis, and if the patient appears to be fatiguing. If the initial assessment reveals the possibility of impending respiratory failure, appropriate supplemental ventilation should be considered, including the use of non-invasive positive pressure ventilation (NIPPV) or bag-valve-mask (BVM) ventilation in conjunction with oral/nasopharyngeal airways, supraglottic devices, or endotracheal intubation (ETI). An important early task is to question the family/caregivers and gather available paperwork regarding the patient’s wishes for life-sustaining treatment or end-of-life care.

Once disease severity and the immediate needs have been addressed, the next step is to attempt to categorize the underlying cause. The four most common categories for respiratory distress are upper airway obstruction, small airway obstruction including chronic obstructive pulmonary disease (COPD) and asthma, acute cardiogenic pulmonary edema (ACPE), and pneumonia. In addition, there are a host of other medical conditions that can cause subjective dyspnea and/or objective impairment of oxygenation and ventilation ([Box 5.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0001)).

**Box 5.1 Common causes of respiratory distress in the EMS setting**

* *Pneumonia*
* *Acute decompensated heart failure/acute cardiogenic pulmonary edema*
* *Pulmonary embolus*
* Acute coronary syndrome (ST-segment elevated myocardial infarction, non-ST-segment elevated myocardial infarction, unstable angina)
* Pneumothorax
* *Metabolic acidosis with attempted compensation (e.g. septic shock)*
* Toxic ingestions (e.g. salicylates)
* Pleural effusion
* Pulmonary hypertension
* Upper airway obstruction
* Interstitial pulmonary fibrosis
* *Asthma*
* *Chronic obstructive pulmonary disease*
* Fever
* Physiological dyspnea of pregnancy
* Bronchitis
* *Psychiatric, hyperventilation, panic attack*
* Arrhythmias, especially atrial fibrillation
* Abdominal distension, obesity
* *Italics = Very common*

Acute coronary syndrome is an important consideration among these disparate causes of shortness of breath. It can present as cardiogenic shock with ACPE but can also cause subjective dyspnea without severe impairment of cardiac function. Dyspnea associated with acute coronary syndrome may not be accompanied by chest discomfort and is more common in women, older individuals, and those with diabetes [5,6]. Dysrhythmias can also cause dyspnea and are readily diagnosed by cardiac monitoring. If time and the patient's condition allow, a 12-lead electrocardiogram (ECG) may be useful in guiding treatment and destination decisions for the dyspneic patient. Severe sepsis can also present with respiratory distress due to increased oxygen consumption. Toxic exposures can cause respiratory distress either through direct irritation of the respiratory tract or secondarily by central nervous system impairment of respiratory function. Tachypnea and subjective dyspnea may also be compensatory for an underlying metabolic acidosis as with diabetic ketoacidosis or salicylate toxicity. If these acidotic patients require ETI and mechanical ventilation, it is important to continue to hyperventilate them to maintain their preexisting respiratory compensation for the underlying metabolic acidosis. This can be facilitated through the use of continuous EtCO2 monitoring. Neuromuscular diseases such as myasthenia gravis and Guillain–Barré syndrome are rare causes of inadequate ventilation and respiratory failure. Although a diagnosis of exclusion, shortness of breath is also a common manifestation of anxiety disorders, panic attacks, and psychogenic hyperventilation. Having a patient breathe into and out of a paper bag, which is sometimes done by the uninformed for hyperventilation, actually decreases inspired oxygen and has no place in EMS.

Although auscultation of breath sounds is an important part of the physical examination for respiratory distress, there can be much overlap in the cause of any one particular finding. Thus breath sounds must be interpreted in the context of the rest of the focused exam. For example, a common mistake is to equate “crackles” with an ACPE exacerbation and “wheezing” with asthma, although both findings can be found in either disease process. Examination should also include a careful auscultation of heart sounds as well as palpation and inspection of the neck for jugular venous distension (JVD), chest for retractions and injury, lower back for sacral edema, and extremities for edema or evidence of deep vein thrombosis (DVT) ([Box 5.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0002)).

**Box 5.2 History and exam findings by disease state**

**Asthma**

Dyspnea with prolonged expiratory phase, tripoding position when severe, decreased breath sounds when very severe to diffuse wheezing, chest tightness

**Chronic obstructive pulmonary disease**

Cough, increased or change in sputum production, dyspnea with prolonged expiratory phase, tripoding position when severe, decreased breath sounds when very severe to diffuse wheezing, barrel chest appearance

**Acute decompensated heart failure with volume overload**

Jugular venous distention, S3 or S4 heart sounds, pulmonary wheezing, pulmonary crackles (rales), lower extremity edema, sacral edema, weight gain with normal or elevated blood pressure

**Acute decompensated heart failure with low cardiac output state**

Cool skin from peripheral vasoconstriction and low blood pressure

**Pneumonia**

Unilateral decreased breath sounds, focal wheezing, unilateral or bilateral crackles (rales), fever, normotensive to hypotensive

**Pneumothorax**

Pleuritic chest pain, unilateral decreased breath sounds, jugular venous distension, hypotension and hypoxia with tension physiology

**Pulmonary embolism with infarction**

Dyspnea, pleuritic chest pain, hemoptysis, possibly decreased oxygen saturations

**Pulmonary embolism with saddle embolism**

Syncope, hypoxemia, jugular venous distension, acute right heart strain on ECG, dilated right ventricle on portable echocardiogram

The physical exam may be enhanced through the use of ultrasound of the chest in the patient with acute respiratory distress. Ultrasound is now commonly used in the ED for evaluation of pneumothorax, pleural effusion, pericardial effusion, large pulmonary embolism, cardiac function and volume status.

**General treatment**

As with most potential threats to life, initial therapy should begin with supplemental oxygen, application of monitoring devices, and often IV access. With standard use of SpO2 monitoring, growing information suggests that oxygen therapy should be carefully titrated to a goal between 93% and 96% in patients with general respiratory distress, and to a goal of 88–92% in patients with known COPD [7,8]. A recent randomized controlled prehospital trial by Austin et al. showed decreased mortality among patients who were treated with a titrated oxygen regimen versus those treated with uncontrolled high flow oxygen. Mortality was reduced 58% in patients with any respiratory distress and 78% in patients with known COPD with the titration strategy [7].

Inhaled bronchodilators, including short-acting inhaled beta2-agonists (SABAs) and anticholinergics, are commonly included in protocols for respiratory distress of unclear etiology. Although there is usually little downside to their use, especially if a component of bronchospasm is suspected, SABAs can be potentially harmful in those with ACPE, acute coronary syndrome, and cardiac arrhythmias due to their chronotropic, inotropic, and vasoactive effects on the cardiovascular system. A review of the Acute Decompensated Heart Failure National Registry Emergency Module (ADHERE) database by Singer et al. revealed that 21% of patients ultimately diagnosed with acute decompensated heart failure (ADHF) exacerbation received SABA treatments by EMS or in the ED [9]. The authors also reported an association between bronchodilator use and a subsequent need for IV vasodilators and ETI. It is important to note, however, that no mortality difference was found between patients who did or did not receive SABAs. In addition, patients with combined ADHF and COPD were not studied separately. Fisher et al. reported six cases of acute myocardial infarction precipitated by bronchodilators [10].

Unfortunately, the relationship between cardiac manifestations and bronchodilator use is poorly understood and these cases likely reflect publication bias. SABAs are known to decrease serum potassium concentration by approximately 0.5 meq/L, which could precipitate hypokalemia-associated dysrhythmias. In addition, SABAs may temporarily worsen hypoxemia by increasing the ventilation/perfusion mismatch. Inhaled anticholinergics, such as ipratropium, are not absorbed systemically and have no cardiovascular toxicity. But in the final analysis and in the absence of well-designed trials to better guide the empirical use of bronchodilators in undifferentiated respiratory distress, it seems to make physiological sense to continue to include them in EMS protocols or at the discretion of a medical oversight physician.

Two forms of NIPPV have become standard for treatment of several forms of respiratory distress [11]. A mask is used to deliver ventilation support either at a constant pressure (CPAP) or with a higher pressure during inspiration (BiPAP). The use of NIPPV in the prehospital setting has become accepted as an early intervention, and studies of its use in this setting have demonstrated decreased mortality, reduced intubation rates, shorter intensive care unit (ICU) lengths of stay, and improved vital signs [11,12]. Although NIPPV has been most studied in COPD and ACPE, a recent systematic review and metaanalysis supports its use in all forms of undifferentiated acute respiratory failure [11]. NIPPV may also permit administration of a lower concentration of inspired oxygen, thereby decreasing the potential deleterious effects of hyperoxia [13]. It is important that prehospital providers understand the limitations of this intervention, including patient factors that are specific contraindications to its use. NIPPV is inappropriate for patients who require immediate ETI such as those who are unable to protect their airways, have altered mentation, or cannot tolerate the pressure mask. The patient must have an acceptable respiratory drive prior to application of NIPPV.

Advanced airway management with supraglottic airways or ETI is the final common pathway for most individuals with severe respiratory distress who have failed to respond to the above-mentioned strategies (see Volume 1, [Chapters 2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c01.xhtml)–[4](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c04.xhtml)).

**Asthma**

Asthma is a chronic inflammatory lung disorder characterized by acute attacks of airway hyperresponsiveness with reversible obstruction. Precipitating factors include upper respiratory tract infections, exposure to allergens, high pollution indices, and failure to use preventive and maintenance therapies. The disease affects more than 22 million individuals in the United States [14]. Although there are hallmark features of an acute exacerbation, assessment of asthma exacerbations can be challenging and potentially misleading (see [Box 5.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0002)). For example, absence of wheezing may indicate either severely restricted airflow or clinical improvement following appropriate treatment. A multicomponent guide can be helpful in assessing severity and monitoring the effectiveness of treatment of asthma [14] ([Table 5.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-tbl-0001)).

[**Table 5.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#R_c5-tbl-0001) Asthma severity guide

Source: Guidelines for the Diagnosis and Management of Asthma (EPR-3) 2007. National Heart, Lung, and Blood Institute; National Institutes of Health; US Department of Health and Human Services.

| **Parameter** | **Mild** | **Moderate** | **Severe** |
| --- | --- | --- | --- |
| Shortness of breath | Walking | Talking | At rest |
| Ability to speak | Full sentences | Phrases | Words |
| Heart rate | 100 | 100–120 | >120 |
| Respiratory rate | Increased | Increased | >30 |
| Lung sounds | End expiratorywheezing | Full expiratory wheezing | Absent or biphasicwheezing |
| Accessory muscleuse | Rare | Common | Always |
| Mental status | Agitation, variable | Agitated, usually | Agitated to somnolent |
| ETCO2 | 20–30 | 30–40 | >40 |

Oxygen should be provided to relieve hypoxemia and titrated to a SpO2 of 93–96% [7]. The initial drug of choice for treatment is a SABA, which acts by relaxing bronchial smooth muscle and increasing mucociliary clearance. Nebulization is the preferred route of administration in the acute setting with either intermittent or continuous delivery. SABAs can also be administered through the use of metered dose inhalers (MDI). The use of subcutaneous epinephrine (a non-selective beta-agonist) has declined with the availability of SABAs, but epinephrine can be very useful when the patient is critically ill or when the inhaled SABA cannot be delivered effectively. For more severe exacerbations an anticholinergic bronchodilator agent, such as ipratropium, can be added to the SABA.

Patients who fail to respond promptly and completely to inhaled bronchodilators benefit from the administration of systemic corticosteroids. The benefits of prehospital corticosteroid administration have not been proven through randomized controlled clinical trials. Non-randomized observational studies, however, have shown that EMS delivery of corticosteroids is associated with decreased hospital admission rates [15]. It has also been suggested that early use of corticosteroids may enhance the effectiveness of SABAs [16]. Corticosteroid options include prednisone (oral), dexamethasone (oral, IM, IV), and methylprednisolone (IV).

For severe exacerbations that fail to respond to inhaled bronchodilators and systemic corticosteroids, adjunctive therapies, such as IV magnesium sulfate or heliox, if available, should be considered. A meta-analysis of seven randomized controlled trials of magnesium sulfate administered in the ED showed it improved peak expiratory flow rates and reduced hospital admission rates compared with placebo in severe asthma exacerbations [17].

Although NIPPV for acute exacerbations of asthma is traditionally viewed as a last resort due to the fear of worsening air trapping and secondary barotrauma, studies of its use in the ED and ICU settings in children have shown benefit [18,19]. A retrospective study of pediatric patients who were placed on BiPAP and given SABAs in the ED, with initial disposition plans for ICU admission, found that 22% of the patients tolerated BiPAP and were able to be downgraded to ward admission. None required subsequent ICU admission. All of these patients had improved SpO2 levels as well as respiratory rates and there were no BiPAP-related adverse events [19].

If rapid sequence ETI is necessary for an asthma patient, the preferred induction agent is ketamine due to its inherent bronchodilator properties. Once intubated, ventilation should be provided at reduced volumes and rates to prevent air trapping and secondary barotrauma. The inspiratory-to-expiratory (I/E) ratios should be adjusted to provide a prolonged expiratory phase. Permissive hypercarbia is generally well tolerated in these individuals. All NIPPV and intubated asthma patients should be monitored closely for signs of secondary barotrauma, such as tension pneumothorax and pneumomediastinum.

**Chronic obstructive pulmonary disease**

Chronic obstructive pulmonary disease is a chronic disease characterized by expiratory lung flow obstruction that is only partially reversible. The underlying pathophysiology involves a complex process of chronic inflammation, remodeling of the small airways with destruction of alveoli, and increase in extracellular matrix production. COPD is usually a response to noxious particles and gases including cigarette smoke and environmental pollutants, though genetic factors also play a part [20]. It is the fourth leading cause of death in the United States. COPD patients who continue smoking will eventually require permanent oxygen and/or ventilator assistance. EMS is often called to evaluate a COPD patient during an acute exacerbation of the condition precipitated by respiratory tract infections, exposure to pollutants or allergens, or medication non-compliance. The clinical presentation is similar to asthma (see [Box 5.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0002)). COPD patients should receive oxygen with a goal to maintain SpO2 between 88% and 92%, which was associated with reduced mortality by twofold in the study by Austin et al. [7] Impending respiratory failure can be detected by continuous EtCO2 monitoring. Increasing EtCO2 levels indicate a deteriorating condition.

As with asthma, the primary treatments during acute exacerbations are directed toward reversing airway obstruction through the use of SABAs and anticholinergic agents. The latter are much more effective in COPD and should be used early. Corticosteroids and antibiotics are also important adjuvant therapies. Use of NIPPV has become established as a life-saving therapy in the treatment of COPD exacerbations [11,12]. If it is necessary to intubate a COPD patient, appropriate settings for mechanical ventilation include decreased respiratory rates, lower tidal volumes, and increased expiratory phase. As with asthma, these patients must be monitored closely for evidence of secondary barotrauma [21].

**Acute decompensated heart failure/acute cardiogenic pulmonary edema**

No widely accepted guidelines exist for the treatment of ADHF either in the prehospital or ED setting. One pathway proposed by DiDomenico et al. for the ED bases the initial management strategy on whether the problem appears to be a state of volume overload versus one of inadequate cardiac output, which can often be differentiated on exam [22] (see [Box 5.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0002)). If the patient is volume overloaded, positioning in an upright posture is the first step. It allows pleural effusions and edema to localize at the lung bases and venous blood to pool in the lower extremities, thereby reducing cardiac preload. If immediate prehospital pharmacotherapy is required, nitrates are the preferred option. If the patient has poor cardiac output, which is far less common, field treatment is largely supportive, although this category of diagnosis should prompt EMS providers to consider causes such as acute myocardial infarction (MI) as well as hypovolemic, distributive, or obstructive causes for shock. If the cause is truly low output cardiac failure, specific treatment might include the use of inotropic and vasopressor medications such as dobutamine, milrinone and dopamine.

If the patient has volume overload with an adequate or high blood pressure and is in acute distress, nitrates should be administered. Diuretics are rarely indicated in prehospital settings. Nitroglycerin acts rapidly to dilate veins, allowing blood to distribute to the periphery, thereby decreasing cardiac preload. At higher doses, typically above 30 μg/min intravenously, nitroglycerin also acts as an arterial vasodilator, decreasing cardiac afterload [23]. Sublingual nitroglycerin is 50% bioavailable. A dose of 400 μg given every 5 minutes, with frequent reassessment to ensure maintenance of a systolic blood pressure of at least 100 mmHg, is often effective. However, for patients *in extremis* in terms of respiratory distress, and with an adequate blood pressure, sublingual nitroglycerin spray can be safely administered more frequently, as often as every 2 minutes in some cases, to rescue the patient from invasive airway intervention and ventilation. Sublingual nitroglycerin also has the advantage of a rapid time to peak effect of 5–15 minutes and duration of action of less than 1 hour. Transdermal nitroglycerin paste is not recommended since its effectiveness is limited by slow absorption, which is further worsened by the presence of decreased skin perfusion during ADHF.

Intravenous access should ideally be obtained before the administration of nitroglycerin, as it has the rare potential to produce hypotension and bradycardia [24]. However, inability to obtain IV access should not preclude or delay its use. Observational studies of nitroglycerin use have shown relatively low rates of serious adverse effects ranging from 0.3% to 3.6% [24]. EMS providers must also remember the potential interaction with all antierectile dysfunction phosphodiesterase-inhibiting drugs (e.g. sildenafil), which are contraindications to the use of nitroglycerin. Notably, however, NIPPV is not a contraindication to concomitant nitroglycerin use.

Loop diuretics, including furosemide, have also been used in the prehospital setting for patients with ADHF, especially those presenting with ACPE. However, intravenously, peak response time is 30 minutes, and this is even more delayed in patients with decreased cardiac output and renal vasoconstriction. The duration of action of furosemide is 2 hours, and up to 6–8 hours in renal failure. Further, because of its effects on plasma electrolytes, which in general are not assessed in most field situations, its use is discouraged. Because furosemide has less of an immediate benefit than nitroglycerin, a long duration of action, and unforeseen potential side-effects, it probably has limited utility in the prehospital setting. Many EMS systems have eliminated its use in favor of nitroglycerin alone.

Morphine, once a staple of therapy for ADHF, has also been largely supplanted by other therapies. A review of the large ADHERE database found a significant association between receiving morphine and death, as well as several other adverse outcomes [25]. One explanation may be that as morphine causes hypotension, it takes away the therapeutic room available for other medications used to reduce preload and afterload. In addition, as a respiratory depressant, morphine may decrease the respiratory drive of an already struggling patient.

Non-invasive positive pressure ventilation is very useful in the immediate treatment of ACPE. Continuous pressure at a level of 5–10 cmH2O improves oxygenation by recruiting atelectatic alveoli and decreasing the work of breathing. The increase in intrathoracic pressure also alters hemodynamics by decreasing the transmural wall tension of the heart [26]. Small EMS case series have demonstrated decreased intubation rates and shorter ICU lengths of stay, although effect on mortality is less clear [27,28]. As mentioned, sublingual nitrate therapy should be continued in conjunction with NIPPV.

An emerging modality for diagnosis of ADHF and ACPE in the ED, which may also be useful in the prehospital setting, is focused ultrasonography. In a prospective prehospital study, Prosen et al. reported that the combined use of NT-prBNP (a marker of cardiac atrial stretch) and chest ultrasound had a sensitivity, specificity, negative predictive value (NPV), and positive predictive value (PPV) of 100% for the diagnosis of ADHF and ACPE [29]. More recently, Neesse et al. reported that the presence of a pleural effusion on the prehospital chest ultrasound appears to be a novel marker for ADHF [30]. Portable chest ultrasonography may therefore hold promise as a tool in the differentiation of COPD and ACPE in the prehospital setting.

**Pneumonia and infectious respiratory disease**

In general, there are few specific field interventions for patients who are determined to have pulmonary infectious causes of their shortness of breath. Pneumonia treatment guidelines are universally focused on prompt diagnosis and early treatment with antibiotics.

Pneumonia should be considered in patients with cough and/or fever. Specific etiologies of these diseases and issues regarding crew protection are discussed in [Chapter 25](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c25.xhtml) of both Volumes 1 and 2. It is important to note that some of these diseases can be highly contagious, and it is recommended that some type of respiratory precaution (e.g. an N95 mask to protect against novel viruses as well as tuberculosis) is maintained when evaluating a respiratory distress patient who is presumed to have an infectious etiology.

Treatment of these patients will typically consist of oxygen, NIPPV if ventilation support is needed to further improve oxygenation, IV fluids if hypotensive, and transport. Patients with known asthma or COPD who have reactive airways in response to the inflammation may also benefit from bronchodilators. Many pneumonia patients may also wheeze from infectious inflammatory processes within the small airways, and hence may respond to inhaled bronchodilators.

**Pulmonary embolus**

Pulmonary embolus (PE) is another clinical condition that can present with respiratory distress. Classic risk factors for venous thromboembolism (VTE) include the Virchow triad of venous stasis, trauma, and hypercoagulability. There are many risk factors for VTE but the ones that have been clinically validated by Wells criteria and the PERC rule for risk stratification include recent surgery or immobilization of an extremity, malignancy, exogenous estrogen use, and prior DVT [31]. Other notable risk factors include genetic deficiency of anticlotting factors, pregnancy, obesity, and extended travel.

Most PEs are the result of DVTs in the pelvic or lower extremity veins, though a DVT from any location can result in a PE. PE is a challenging clinical diagnosis because the manifestations can be subtle. The most common symptom is dyspnea and the most common clinical signs are tachycardia and tachypnea. The pulmonary exam is usually unremarkable, though examination of the extremities, particularly the legs, may reveal swelling, erythema, and/or pain in a limb with a DVT. With the increased use of peripherally inserted central venous catheters, PEs are also reported more frequently as a result of upper extremity DVTs [32]. Small PEs often present with respiratory distress. Larger emboli that cause lung infarction can present with more severe findings, and those with saddle embolism cause findings suggestive of obstructive shock (see [Box 5.2](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c05.xhtml#c5-fea-0002)). The latter can be detected by findings such as right axis deviation, right ventricle strain, and right bundle branch block on a 12-lead ECG. Additional useful ECG features are the presence of T-wave inversions in both V1 and lead III as well as the presence of an S-wave in lead I and a Q wave and inverted T wave in lead III (S1Q3T3) [33]. Acute right ventricular dysfunction can also be visualized using portable cardiac ultrasonography. In some severe cases, the embolus can even be visualized directly within the heart.

Emergency medical services treatment priorities include high-flow oxygen, vascular access, and cardiac monitoring. A fluid bolus is indicated in the patient who presents with a suspected massive PE and perfusion failure. In some patients, the presentation can take the form of a cardiac arrest with a narrow complex pulseless electrical activity (PEA) rhythm. The presence of prearrest respiratory distress, altered mental status, and shock, along with a presenting rhythm of PEA, has been shown to be predictive of PE as a cause of cardiac arrest [34]. Although the use of prehospital thrombolysis in these instances has been reported to be effective in selected cases, a randomized controlled clinical trial failed to show improved outcomes during cardiac arrest when tissue plasminogen activator (t-PA) was administered compared to placebo for patients with refractory PEA [35,36].

**Pneumothorax**

Spontaneous pneumothorax is an uncommon condition that can present as acute respiratory distress. It is caused by rupture of the alveolar air sacs into the pleural space, followed by variable collapse of the lung. Symptoms include dyspnea as well as pleuritic chest pain. The exam may reveal decreased breath sounds on the affected side. Typically, spontaneous pneumothorax occurs in male patients who are taller than average with a slim build. There are also secondary causes of spontaneous pneumothorax, most notably COPD. Other underlying causes include tumor, infection, or a connective tissue disorder. Spontaneous pneumothorax rarely progresses to tension pneumothorax. Patients with a suspected simple pneumothorax should be monitored closely for evidence of tension physiology such as worsening respiratory distress, hypoxia, hypotension, JVD, and tracheal deviation, in which case immediate chest needle decompression is indicated. In the future, the clinical diagnosis of pneumothorax will be supplemented by objective evidence through the use of portable chest ultrasound.

**Conclusion**

Respiratory distress is a very common complaint in the prehospital setting. Initial evaluation should be focused on identifying immediate threats to life and determining needs for an immediate intervention such as NIPPV, BVM ventilation, or ETI. Once this evaluation is completed, efforts should be focused on attempting to determine the provisional underlying cause of the respiratory distress. Respiratory distress may be caused by a primary pulmonary problem, a cardiac problem, an infectious problem, or as part of compensation for another non-pulmonary problem.

A vast number of these patients will remain “undifferentiated” through their EMS and possibly even their ED courses. In general, treatment should include titrated oxygen and monitoring of cardiac rhythm, SpO2, and ETCO2 while ensuring timely transport. In stable situations, the emphasis should focus on avoiding overtreatment and resisting the urge to give multiple medications in an undirected fashion. However, little harm comes to the patient with an initial trial of inhaled bronchodilator therapy, and nitrates should be considered as first-line therapy in the patient with findings consistent with ADHF or ACPE.