Standard: Airway Management, Respiration, and Artificial Ventilation

Competency: Applies knowledge (fundamental depth, foundational breadth) of additional upper airway anatomy and physiology to patient assessment and management in order to ensure a patent airway, adequate mechanical ventilation, and respiration for patients of all ages.

**INTRODUCTION**

Hypoxia kills. This is a fact that every Advanced EMT should understand. Whether the hypoxia is caused by a primary breathing problem or associated with a larger illness or injury, assessment and treatment must focus on reversing this disorder.

The diagnosis of airway and respiratory dysfunction can be a complicated and difficult process. Many very different illnesses can present with similar signs and symptoms. Thankfully, however, the immediate assessment and treatment of a patient in respiratory failure remain consistent, regardless of the nature of the illness. Although differential diagnosis is important, early recognition and immediate action are more important, especially when the overall cause is unclear.

**EPIDEMIOLOGY**

According to the Centers for Disease Control and Prevention, respiratory distress accounts for roughly 2 percent of all emergency department visits. Although this may seem like a small percentage, the absolute numbers are, in fact, very high, considering how many patients use the EMS system overall. This means that as AEMTs, we see a significant number of respiratory distress patients in the field.

**PATHOPHYSIOLOGY**

Respiratory dysfunctions are typically the result of either an obstruction of air movement, such as something in the way, or changes in the respiratory structures that affect the movement of oxygen and carbon dioxide. Occasionally both issues play a role. Respiratory dysfunctions can be further classified as either upper or lower airway problems. Upper airway issues affect the airway structures above the glottic opening (Figure 13-1), and lower airway disorders affect the structures found from the trachea to the alveoli.

**Upper Airway Dysfunction**

The classic upper airway problem is the obstructed airway. When we think of this, we often picture a child choking on a small toy. However, far more commonly, upper airway obstruction is the result of poor airway muscle tone resulting from altered mental status. When the brain is not functioning properly, the muscles and nerves that protect the airway fail. This failure frequently results in an inability to keep an airway open. For example, when lying supine, a patient with an altered mental status may relax the muscles of the upper airway too much and allow the epiglottis to fall back and cover the glottic opening (Figure 13-2).

The upper airway can also be affected by structural changes. Airflow can be impeded by swelling in and around the larynx. Conditions such as burns, infection, anaphylaxis, and even direct trauma can cause laryngeal edema and inflammation and...
more important in a patient with respiratory distress. It begins by ensuring patency of the airway: “Is it open?” Of course, if the answer is no, you must take steps to open it. Beyond the immediate moment, you must consider the future: “Will it stay open?” Is the ongoing patency of this airway threatened, and if so, what steps are necessary to reverse that course?

When assessing breathing, you also must consider multiple elements. First, you must ensure that the patient actually is breathing; but second, you must further ensure that the patient’s breathing is adequate to meet the needs of his body. “Look, listen, and feel” will quickly provide an answer to the first question, but for the second question you must engage in some critical thinking.

Always keep minute ventilation and alveolar ventilation in mind when assessing breathing. You must ask yourself continually how much air is actually reaching the alveoli each minute. Remember that minute volume is comprised of both rate and volume. Breathing within an acceptable rate is important, but again, you must consider how much volume is being moved down to the alveoli in each breath. In addition, keep in mind the concept of dead space.

In the primary assessment, you need to look at breathing (Figure 13-3). How fast or slow is the patient breathing? Quickly listen to both sides of the patient’s chest. This is the time not for a thorough examination of lung sounds but, rather, for a quick assurance that air is moving in and out on both sides. It takes only seconds to determine that volume is inadequate. Sounds of dysfunction are also rapidly identified with even a quick listen. For example, are there wheezes that interfere with the diffusion of oxygen and carbon dioxide. This pulmonary edema thickens the alveolar membrane, collapses some alveoli, and even (in late stages) can fill the alveoli themselves. Infections such as pneumonia can cause similar dysfunction. In these cases, the alveoli can be obstructed by pus and other byproducts of infection, causing a similar gas exchange issue.

**Lower Airway Dysfunction**

The most common cause of lower airway dysfunction is bronchoconstriction. A number of diseases and disorders, such as asthma and anaphylaxis, can cause the bronchiole passages to spasm and constrict. Even small changes in the diameter of these tubes can cause tremendous resistance to airflow that can seriously decrease the movement of air.

In addition to bronchoconstriction, other disorders can structurally change how gas is exchanged in the alveoli. Problems such as congestive heart failure, near drowning, and even altitude sickness can cause the fluid portion of the blood to cross the alveolar membrane and interfere with the diffusion of oxygen and carbon dioxide.

**ASSESSMENT FINDINGS**

When assessing a patient with respiratory distress, the most important goal may not be to determine the exact nature of the disorder. Far more important may be the need to recognize respiratory failure and support ventilation.

The primary assessment is a critical component of the assessment in any patient. It is even more important in a patient with respiratory distress. It begins by ensuring patency of the airway: “Is it open?” Of course, if the answer is no, you must take steps to open it. Beyond the immediate moment, you must consider the future: “Will it stay open?” Is the ongoing patency of this airway threatened, and if so, what steps are necessary to reverse that course?
The sympathetic nervous system tells the heart to beat faster and stronger. These compensatory mechanisms can be easily identified in your assessment of breathing. What is the respiratory rate? How hard is the patient working to breathe? Look at the patient’s chest: Is the patient using accessory muscles? All of these findings indicate the body’s effort to compensate. As with compensated shock, these measures can temporarily sustain normal body function and hold off the challenge.

When respiratory compensation works, we deem the patient to be in respiratory distress—that is, he is challenged, but the compensatory efforts are sustaining normal function despite the problem. A patient in respiratory distress will exhibit signs of the challenge, such as tripod positioning (see Figure 13-3), accessory muscle use, and increased respiratory rate, yet he should also be showing signs that these measures are allowing normal function. The patient’s brain should be oxygenated, and therefore he should have a normal mental status. He should not be showing signs of profound hypoxia, such as cyanosis. The key to differentiating respiratory distress from respiratory failure is identifying that normal function.

Respiratory Failure

Unfortunately, the body’s compensation is limited. Some respiratory challenges exceed the body’s ability to compensate. Other times, compensation simply fails over time. Keep in mind that when we ask muscles to do more work, more oxygen is required. If hypoxia is already a challenge, the muscles of compensation can help for only a short time. Respiratory failure occurs when compensation fails. At this point, the challenge continues and the body may be attempting to compensate, but function has been affected. Oxygen may not be getting distributed, carbon dioxide is being retained, and the muscles of respiration tire. As an AEMT, you must be ever vigilant to recognize respiratory failure because it demonstrates that what the patient is doing on his own is not enough.

A patient in respiratory failure exhibits signs and symptoms similar to those of a patient in respiratory distress. He will have a challenge, he will be compensating, but he will not be meeting his needs. Look for signs that compensation has failed. Altered mental status is a key indicator. Anxiety, combativeness, somnolence, and even unconsciousness all point to hypoxia and hypercapnia. Look for additional signs of hypoxia, such as cyanosis and low oxygen saturation. These findings are especially worrisome if they occur despite supplemental oxygen. Look for other signs of failure, including signs of respiratory fatigue. Respiratory muscles need oxygen and eventually fail as they become hypoxic. Look for slowing rates, irregular patterns, and gasping as indicators of respiratory failure (Figure 13-5).

Finally, as an advanced provider, you may be tempted to move on to advanced modalities such as airway and intravenous lines. Resist this temptation until the primary assessment is complete and all immediate life threats have been addressed.

Respiratory Distress

When a person experiences a challenge to respiratory system function, the body responds in a predictable manner: It compensates. As in shock, the brain takes specific steps to help overcome the deficit caused by the offending issue. When the brain senses increasing carbon dioxide and low oxygen, the respiratory center in the medulla increases the respiratory rate. Additional muscles in the neck, chest, and abdomen (accessory muscles) are engaged to assist with breathing (Figure 13-4).

Figure 13-3 Patient suffering respiratory distress, indicated by his tripod position.

Figure 13-4 Barrel chest in an emphysema patient.
PATIENT’S CONDITION     WHEN AND HOW TO INTERVENE

**Adequate breathing:**
Speaks full sentences; alert and calm

**Increasing respiratory distress:**
Visibly short of breath; Speaking 3–4 word sentences; increasing anxiety

**Severe respiratory distress:**
Speaking only 1–2 word sentences; Very diaphoretic (sweaty); Severe anxiety

**Continues to deteriorate:**
Sleepy with head-bobbing; Becomes unarousable

**Respiratory arrest:**
No breathing

**Key decision-making point:**
Recognize inadequate breathing before respiratory arrest develops.

**Assisted ventilations**
Pocket face mask (PFM), bag-valve mask (BVM), or flow-restricted, oxygen-powered ventilation device (FROPVD)
Assist the patient’s own ventilations, adjusting the rate for rapid or slow breathing

**Artificial ventilation**
Pocket face mask (PFM), bag-valve mask (BVM), or flow-restricted, oxygen-powered ventilation device (FROPVD)
Assisted ventilations at 12/minute for an adult or 20/minute for a child or infant

**Nonrebreather mask or nasal cannula**

**Nonrebreather mask**

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Figure 13-5 The continuum of breathing ranges from normal, adequate breathing to no breathing at all. It is essential to recognize the need for assisted ventilations even before severe respiratory distress develops.
TOPIC 13  ■  Issues in Airway Management, Oxygenation, and Ventilation

Remember that a patient in respiratory failure may indeed be breathing. Consider the following example:

You are assessing a 14-year-old asthma patient. He has been having an attack for roughly an hour now. You assess his airway and find that he has a patent airway, but he has difficulty speaking. He is breathing at a rate of 54 breaths per minute. You find him in tripod position; when you look at his chest you note retractions and other accessory muscle use. When you listen to his chest you can barely hear any air movement at all. His fingernails are blue. When you attempt to engage him with questions, you note that he has a very sleepy affect.

This young man is in respiratory failure. He is compensating for his asthma, as evidenced by his respiratory rate, his use of accessory muscles, and his position, but those compensatory mechanisms are not working, as demonstrated by his altered mental status and cyanosis. This patient needs immediate help.

It is important to also remember that identifying respiratory distress does not require diagnosing a specific respiratory dysfunction. You may identify failure (and take action) long before you have time to accurately complete a differential diagnosis. Luckily, the treatment for respiratory failure is, for the most part, the same regardless of what disorder is causing it.

EMERGENCY MEDICAL CARE

Different respiratory dysfunctions will require different treatments. Discussion of all the specific treatments for individual respiratory dysfunctions is beyond the scope of this topic. However, some general treatment philosophies are common despite the etiology of the respiratory dysfunction. Those common treatments are presented in the following sections.

Airway

Ensuring an open airway is essential to all other respiratory treatments. If the patient does not have an open airway, give him one. If that airway is threatened, take steps to protect it. When appropriate, consider oropharyngeal and nasopharyngeal airway adjuncts to help keep an airway open.

Respiratory Distress

The treatment goal when dealing with a patient in respiratory distress is to support the compensatory efforts of the patient and work on reversing the challenge. Remember, a patient in respiratory distress is compensating for a respiratory challenge and—at least so far—this compensation is successful. Supplemental oxygen to increase saturation is important. After you ensure that the status is distress and not failure, you should use your assessment to better determine the nature of the dysfunction. In this case, you have time to investigate and focus treatment.

For example, you might listen more carefully to lung sounds in the secondary assessment. You might hear wheezes and recognize them as the sound of bronchoconstriction. Knowing this, you might discuss with medical control the option of assisting the patient with his home nebulizer treatment. Overall, the key purpose of treating respiratory distress is preventing it from becoming respiratory failure. A non-rebreather mask delivering high concentrations of oxygen is one option for treating respiratory distress (Figure 13-6; also see Figure 13-5).

Respiratory Failure

When you recognize respiratory failure, you no longer have time for lengthy assessment and investigation. Remember that, by nature, respiratory failure means that what the patient is doing alone is not working. Respiratory failure requires immediate outside intervention to prevent the next stage: respiratory arrest.

With few exceptions, respiratory failure requires assisting the patient with a bag-valve mask. You must now take over for where the patient’s own respiratory system has failed. There are two goals for this assisted ventilation: improving oxygenation and improving ventilation. Once again, consider minute volume. In a patient breathing exceptionally fast, your goal is not to slow down the rate but rather to increase a diminished tidal volume. Every third, fourth, or even fifth breath, you will deliver a positive pressure breath with a tidal volume greater than the patient is able to achieve on his own.

A bag-valve mask delivers high-concentration oxygen, and in a state of fatigued respiratory muscles, it may be the only way oxygen will enter the system effectively (Figure 13-7).

Choosing to ventilate a breathing patient is a very difficult decision. Keep in mind that it is better to be aggressive in this situation than to allow prolonged hypoxia. Although positive pressure
Positive Pressure Ventilation

The body normally uses negative pressure to bring air into the chest. The diaphragm contracts, the intercostal muscles flex, and negative pressure is created in the chest cavity to pull air in through the glottis opening. With a bag-valve mask, an opposite mechanism is used to move air into the lungs: Positive pressure is applied externally to force air in.

Positive pressure ventilation sometimes disrupts normal body functions. For example, the heart uses the negative pressure of breathing to assist with filling. When positive pressure is applied, the heart can no longer rely on the negative pressure, and often filling is decreased. This can drop cardiac output. The esophagus is also not designed for positive pressure air entry. Because it is an expandable tube, positive pressure ventilation often drives air into the stomach. This gastric insufflation can lead to pressure on the diaphragm and decreased lung capacity.

Despite these difficulties, positive pressure ventilation is essential for a patient who is not breathing or is in respiratory failure. Keeping the following side effects in mind will help you improve your positive pressure ventilation technique.

- **Minimize the effect of positive pressure.** Ventilate with only enough volume to raise the chest wall. Doing this helps minimize the effects of positive pressure on the heart and can increase cardiac filling.
- **Keep gastric insufflation in mind.** Always use an airway adjunct (when possible). Airway adjuncts help create better channels for air and help avoid forcing air into the esophagus. Although you may consider also using cricoid pressure (the Sellick maneuver) to compress the esophagus during ventilation (Figure 13-8), recent evidence has cast some doubt on the value of this procedure, and the American Heart Association (AHA) has significantly limited its recommendations for cricoid pressure in its 2010 guidelines.
- **Hyperventilation kills.** Ventilate at appropriate rates (12–20/minute for children and 10–12/minute for adults). This helps prevent gastric insufflation as well as preventing the unnecessary removal of too much carbon dioxide, which can lead to cerebral vasoconstriction and reduced blood flow to the brain.

The cost–benefit analysis of positive pressure ventilation is an important one. Your ability to effectively identify respiratory failure is essential. Even more essential, however, is your decision to act on that problem. Far too often, respiratory failure is identified but allowed to worsen because of indecision. Always remember that your assessment is more than just a gathering of information; it is a very important element of making critical decisions.

**Continuous Positive Airway Pressure**

Continuous positive airway pressure (CPAP) is a technology that uses positive pressure in a different manner than a bag-valve mask. The positive pressure created by a CPAP system does not force air in but, rather, creates a constant, slight flow of air against which the patient will breathe. This “wall of resistance” will often make the work of breathing easier, keep alveoli open, and make breathing more effective (Figure 13-9).

A variety of CPAP systems are available. In general, CPAP systems create a higher flow of air by mixing oxygen with room air (although some systems use just room air). For years, sleep apnea patients used positive pressure to keep open the soft tissues of the hypopharynx and prevent snoring. In EMS, that pressure is used to “pneumatically splint” open lower airways and the alveoli.

**USES OF CPAP** CPAP is most commonly used to treat acute pulmonary edema (APE). By pressurizing the inside of the alveoli, the fluid of pulmonary edema is prevented from crossing the alveolar membrane. It also helps prevent the collapse of alveoli under the weight of the edema. CPAP has been proven to rapidly improve APE in some patients and, in many cases, to prevent the need for intubation.

CPAP is also used to treat other forms of respiratory distress. Keeping small airways open via the pneumatic splint tends to increase oxygenation and decrease the sensation of difficulty breathing. Keeping the alveoli from collapsing also leads to...
increased surface area for gas exchange. These effects can help a variety of respiratory disorders, including bronchospasm and pneumonia. Indications for CPAP vary from system to system, so always follow local protocol.

**APPLYING CPAP** CPAP is not artificial ventilation. If the patient cannot maintain an airway or breathe on his own, he is not a candidate for CPAP. Many patients will need more aggressive treatments. Always use a thorough patient assessment to make the correct treatment choice.

Just as with a bag-valve mask, the positive pressure of CPAP can also drop cardiac output by counteracting the negative filling pressure of the heart. Therefore, you should never apply CPAP to a hypotensive patient. Follow local guidelines for minimum systolic blood pressure values.

CPAP can also be difficult psychologically for a patient. A mask is strapped to the face of a patient who is already having difficulty breathing. Often a patient will not tolerate this treatment. CPAP should never be forced. Consider allowing the patient to hold the mask on his face before strapping it on. Often, when the patient feels the effects, he will be more likely to allow the strap.

CPAP can be rapidly beneficial, but not every patient will get better after its application. Reassessment is critical. Many times CPAP will be applied to patients who are close to respiratory failure. These patients sometimes progress to respiratory failure and will need more aggressive treatment. Remember also that hypotension can be a side effect.

**REVIEW ITEMS**

1. Which of the following problems would be designated as an upper airway disorder?
   a. laryngeal edema caused by anaphylaxis
   b. bronchoconstriction caused by asthma
   c. destruction of the alveoli caused by emphysema
   d. pulmonary edema caused by congestive heart failure

2. You are assessing a patient who has a hoarse voice after being struck in the throat by a baseball. Given the primary assessment, which of the following would be your most important next step?
   a. Request ALS for fear of a potentially threatened airway.
   b. Apply a cold pack to the neck for pain management.
   c. Ask the patient to sip cool water.
   d. Continue on to your secondary assessment.

3. Which of the following signs would help you differentiate respiratory distress from respiratory failure?
   a. altered mental status
   b. increased respiratory rate
   c. accessory muscle use
   d. increased heart rate

4. You are treating a patient in respiratory failure. Which of the following treatments should you complete first?
   a. Deliver positive pressure ventilations with a bag-valve mask.
   b. Deliver supplemental oxygen with a nasal cannula.
   c. Begin chest compressions.
   d. Complete a thorough secondary assessment.

5. Which of the following vital signs would rule out the use of CPAP?
   a. respiratory rate of 4/min
   b. respiratory rate of 40/min
   c. blood pressure of 198/100 mmHg
   d. heart rate of 140

**APPLIED PATHOPHYSIOLOGY**

1. List three ways in which the body compensates for a respiratory challenge.

2. Explain how these compensatory methods may be recognized in an assessment.

3. Discuss why compensation might fail in a hypoxic respiratory distress patient.

4. Describe how positive pressure ventilation is different from the way a person normally breathes.
CLINICAL DECISION MAKING

You are assessing a 65-year-old female patient with COPD. Her family says that she has had shortness of breath for three days, but “she is much worse today.” You find the patient sitting upright, looking scared, and unable to speak.

1. What indications does your general impression give you that this woman’s condition may be critical?

Your patient has a patent airway and is breathing 48 times per minute. She has lung sounds bilaterally but is moving little air, with a prominent wheeze. Her radial pulse is 128, her skin is wet, and she is confused (this is not her normal state).

2. Is this patient in respiratory distress or respiratory failure? Please discuss why you feel this way.

3. Based on your primary assessment, are any immediate interventions necessary? If so, what are they?

4. Explain the pathophysiologic cause for the following:
   a. Confusion
   b. Wheezing
   c. Increased heart rate

The family reports to you that the patient also has a history of acute pulmonary edema secondary to CHF.

5. Based on your primary survey, is it important to determine whether this patient is currently in APE or exacerbated COPD? Why or why not?

6. If you determined that this patient was in APE, how would it change your immediate interventions?