Basic Airway & Ventilation Management  
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Airway management is an overlooked skill for all EMS providers. This quarter’s education will review airway management continuum from basic opening maneuvers to ventilation options for smoke inhalation, COPD and Cyanide poisoning. What? Why are we concerned with Cyanide poisoning?

Basic airway maneuvers use two methods. Patients not suspected of cervical spine injuries can use the head tilt-chin lift maneuver.

Doing so extends the neck opening the airway and displacing the tongue from the posterior pharynx.

For patients suspected of having cervical spine injuries the modified jaw thrust method is preferred due to minimizing the potential for additional injuries. Place your thumbs on the cheekbones lifting the jaw forward raising the tongue off the posterior pharynx while maintaining neutral cervical alignment.

Seems basic, right? It is, but is often overlooked by providers who are busy setting up their bag valve mask (BVM), opening a Combi-Tube or nonrebreather oxygen mask.

Your dispatch information:

Family members called for a 19 year old female found unresponsive.

Here’s what you find:

Unfortunately as EMS we see opiate overdoses far too frequently. What’s your first action? Grabbing Narcan? Grabbing a BVM? Securing the syringe?

Opiates kill by suppressing the respiratory drive. Providing an open airway is the most basic
thing we can do to promote adequate breathing. Providing breaths and administering Narcan are the next steps in establishing adequate ventilation, but she will benefit the most by having a patent airway first.

A paramedic friend works with rescue teams for Indy car type racing. His experience with high speed, 180 mph + crashes is extensive. The first thing responders do when they gain access to an unresponsive driver is position the head into neutral alignment. Frequently these drivers are also apneic; and their breathing drive is stimulated by simply opening their airway.

The presence of known or suspected basilar skull fracture has long been one of the absolute contraindications for NPA placement.

A comprehensive review on the use and indications for nasopharyngeal airways was published in a 2005 Journal of Emergency Medicine. This review acknowledged it is widely taught that skull fractures are a contraindication for NPA placement; it went on to identify only two case reports, as of 2005, in all of published medicine. One report was released in 1991 in Anesthesiology, and the other in the Journal of Trauma in 2000. These authors concluded placing an NPA through the skull is extremely rare and is likely associated with improper technique as well as injuries that already have significant morbidity and mortality.

Possibly our concern for placing an NPA in someone with facial trauma may be unfounded. Consider the patient with trismus\(^1\) or clenched jaw. Even in the presence of facial injury, an NPA may be our only means of providing a patent airway. We’re all familiar with the structures and anatomy

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\(^1\) *Trismus,* also called lockjaw, is reduced opening of the jaws (limited jaw range of motion). It may be caused by spasm of the muscles of mastication or a variety of other causes.
of the nose, if your placement attempt seems to deviate from normal, stop and reevaluate. The patient with facial trauma, altered mentation and trismus leaves EMS providers with few options, leaving rapid sequence intubation (RSI) as a prime choice. If Paramedics aren’t available consider placing an NPA into both nostrils, doing so provides the next best option.

What is the correct method for measuring these?

The answer is, it depends on what resource you’re using. For NPA, the American Heart Association says the correct means is the tip of the nose to the earlobe. Others say the tip of the nose to the tragus (the little cartilage appendage at the front of the ear), while others use the tip of the nose to the angle of the jaw. In reality, all these methods will be close to the same.

Remember that there is a preferred side, so which one is correct? You have a 50/50 chance for being RIGHT. Very perceptive of you, the correct answer is the right side, the reason is the bevel slides along the nasal septum and the shape follows the natural anatomic curve. If you meet obstructions while advancing the NPA, be gentle and most times you can turn it back and forth steering it in. Forcing an NPA will cause bleeding, which in turn will cause your patient to swallow the blood. That in turn may cause your patient to vomit as blood is a gastric irritant. We tend to undersize our airway adjuncts. I’m not sure why. I think it starts in EMT school when we learn that the smaller NPAs go in the mannequin head easier. When faced with a real live nare we tend to opt for the smallest reasonable size. Don’t do it. You’ll end up obstructing more usable nasal passage space than you create. Grab the correct size based on the sizing recommendations above.

Moving on let’s take a look at the OPA. OPA measuring is the center of the mouth to the angle of the jaw.

The most common method for insertion involves a technique where the mouth is opened using the “crossed or scissors” fingers. The OPA is inserted in the patient's mouth upside down so the tip of the OPA is facing the roof of the patient's mouth. As the airway is inserted it is rotated 180 degrees until the flange comes to rest on the patient's lips and/or teeth. Alternately, a tongue depressor is used to flatten the tongue so the OPA is inserted in the natural anatomic position. Care must be utilized in this method as it can cause the OPA to push the tongue into the pharynx creating an obstruction.

My preferred method is to introduce the OPA sideways at the corner of the mouth allowing it to open as a means for suctioning. As you advance it, you only need to turn it 90 degrees towards the throat for proper placement. This technique helps to prevent trauma to the roof of the mouth from an overly enthusiastic attempt.

The method you choose for insertion is secondary to ensuring that you correctly measured
and that the flange remains outside the lip. An undersized OPA can cause an obstruction due to it migrating into the pharynx.

One of the most significant skills EMS providers need to have is the ability to know how and when to ventilate a patient using a bag valve mask device (BVM). In my EMS career, I have learned several methods: the 4 stair step breath method using increasing volumes with each breath, circa 1983; to squeezing the BVM hard and fast for a pulseless, non-breathing patient; to the current method of one ventilation every 3-5 seconds for pediatrics and one ventilation every 5-6 seconds for adults. The American Heart Association has the scientific research\(^2\) to show why fewer ventilations using less volume is more therapeutic. Preventing excessive ventilation minimizes the risk of air going to the stomach; we have all experienced the end result of that, vomit. Whenever the patient vomits there is risk for aspiration. Patients may be successfully resuscitated in the field only to die from aspiration pneumonia days later.

Your dispatch information:

A restaurant called for an adult female with an airway obstruction while eating. An EMR arrived and successfully performed the Heimlich maneuver expelling a large piece of meat. You find the patient in respiratory arrest.

For EMS, basic BVM ventilation is most often the only option for airway management. In the pediatric population, BVM may be the best option for prehospital airway support. BVM ventilation requires a good seal and a patent airway. Practice with this important skill increases the responder’s ability to provide effective ventilation. Adjuncts such as oral and nasal airways can aid with ventilation by relieving obstruction and by opening up the hypopharynx. Certain factors predict difficult BVM ventilation. These include the presence of facial hair, lack of teeth, a body mass index (BMI) greater than 26, age older than 55 years, and a history of snoring.

The masks come in many sizes, including newborn, infant, child, and adult (small, medium, and large). Most adult model BVM volumes range from 250-1600 milliliters. Choosing the appropriate size helps to create a good seal and, therefore, aids effective ventilation. Bags for BVM ventilation also come in different types. Newer bags are equipped with a pressure valve. Some bags have one-way expiratory valves to prevent the entry of room air; these allow for delivery of greater than 90% oxygen to ventilated and spontaneously breathing patients. Bags lacking this feature deliver a high concentration of oxygen during positive pressure ventilation but only deliver 30% oxygen during spontaneous breaths.

Most average sized adults tidal volume is only 450-550 milliliters per breath or 7ml/kg. For advanced providers who use ventilators, volumes are based on ideal body weight rather than actual weight. The most used method for obtaining a seal while using a BVM for ventilation employs the E-C technique in which the thumb and forefinger form a “C”, while the other three fingers forming the “E” are placed on the bony part of the jaw. Care must be taken to not press on the soft tissue area inside the

\(^2\) [https://www.heart.org/idc/groups/heart-public/@wcm/@ecc/documents/downloadable/ucm_317350.pdf](https://www.heart.org/idc/groups/heart-public/@wcm/@ecc/documents/downloadable/ucm_317350.pdf) Highlights of the 2010 American Heart Association Guidelines for CPR and ECC.
jaw; doing so may cause the tongue to become an obstruction.

Provided there is adequate gas exchange at the alveolar level and adequate circulation to the tissues, artificial ventilation via the BVM in the hands of a skilled practitioner can keep a patient alive indefinitely.

Performed incorrectly, however, BVM ventilation can accelerate hypoxia and exacerbate the airway obstruction that naturally occurs during profoundly depressed levels of consciousness. This can result in serious injury or death.

**Common BVM Pitfalls:**

Here are a few things to avoid:

1. Not properly positioning the airway. Failing to open the airway, or not maintaining an open airway once it has been positioned doesn’t allow air into the lungs.

2. Pushing the mask into the face. Pushing the mask down on the face, instead of lifting the jaw into the mask, pushes the tongue against the back of the throat and obstructs the airway. Together with the mask on the face, this practice suffocates, rather than ventilates, the patient.

3. Not maintaining an effective seal. BVM ventilation is recognized as a two-rescuer skill. Only rescuers with exceptionally large hands can effectively maintain an open airway, displace the jaw into the mask and maintain a proper mask seal with a single hand. For most rescuers, two hands are needed on the mask to accomplish all of these tasks simultaneously and effectively.

4. Over-ventilating and hyperventilating. Giving too much volume or going too fast could push air into the stomach, resulting in gastric insufflation. This could lead to vomiting and subsequent airway obstruction or aspiration. Blowing off too much CO2 causes the blood brain vessels to constrict and greatly reduces blood flow to the brain.

The prior standard of care was to automatically attach high flow oxygen to the BVM, at 15 liters per minute the effective oxygen concentration is 75%. The theory being that someone in severe respiratory distress or arrest needed that level of oxygen saturation. Current trends indicate that whenever possible, pulse ox readings should be first obtained to prevent over saturating the patient. AHA recommends post resuscitation oxygen saturations remain between 94% and 99%.

This begs the question, 100% is better than 99%, why don’t I want my patient at 100% saturation?
A range of 94-99% allows a more precise quantification for oxygen diffusion in the blood. Some recent major trials have also shown there was no benefit to giving supplemental oxygen to patients if their pulse ox was 90% or greater. Also, too much oxygen causes vasoconstriction, which leads to other concerns.

In stroke victims, hyperoxia\(^3\) can cause vasoconstriction of the carotid and downstream cerebral arteries. In healthy humans, administration of 100% oxygen during 10 to 15 minutes is associated with a 20% to 33% decrease in cerebral blood flow independently of arterial partial pressure of carbon dioxide.

Oxygen is also a free radical, meaning that it is a highly reactive species owing to its two unpaired electrons. From a physics perspective, free radicals have potential to do harm in the body. The sun, chemicals in the atmosphere, radiation, drugs, viruses and bacteria, dietary fats, and stress all produce free radicals. Cells in the body endure thousands of hits from free radicals daily.

Normally, the body fends off free radical attacks using antioxidants. With aging and in cases of trauma, stroke, heart attack or other tissue injury, the balance of free radicals to antioxidants shifts. Cell damage occurs when free radicals outnumber antioxidants, a condition called oxidative stress. Many disease processes including arthritis, cancer, diabetes, Alzheimer’s and Parkinson’s result from oxidative stress. The concept of free radical damage suggests the old EMS notion that, “high flow oxygen won’t hurt anyone in the initial period of resuscitation” may be dead wrong (with an emphasis on “dead”).

Tissue damage is directly proportionate to the quantity of free radicals present at the site of injury. Supplemental oxygen administration during the initial moments of a stroke, myocardial infarct (MI) or major trauma may well increase tissue injury by flooding the injury site with free radicals.

Finally, consider this: five minutes of supplemental oxygen by non-rebreather decreases coronary blood flow by 30 percent, increases

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\(^3\) Hyperoxia can occur when oxygen levels are higher than normoxic levels (oxygen concentration of 21%). Hyperoxia can damage lung tissue and other vital organs.
coronary resistance by 40 percent due to coronary artery constriction, and blunts the effect of vasodilator medications like nitroglycerin.

As a general rule, any pulse oximeter reading below 90% is considered low. Chronic pulse oximetry levels of 85% or less are associated with increased mortality and the addition of long term oxygen supplementation reduces that increased mortality. Medicare will only pay for home oxygen if a patient’s pulse ox is 88% or less persistently on room air and their pulse ox comes up with oxygen. Two recent large trials, one is suspected MI\textsuperscript{4} and one in stroke\textsuperscript{5}, showed there was no benefit to giving supplemental oxygen to patients with acute cardiac ischemia or stroke if their pulse ox was 90% or greater.

The “normal” range for a pulse oximeter reading is 95-100%. For many years, we operated under the mistaken assumption that if we made a “number” normal we were doing the right thing but we have found out that is not true in the case of oxygen saturation. Supplemental oxygen is usually not helpful and is harmful but keep in mind that there are factors that can affect pulse oximeter readings. Conditions that can make pulse oximeter readings unreliable include poor peripheral perfusion (i.e. vasoconstriction, shock, and hypotension). Don’t attach the sensing probe onto an injured extremity. Try not to use the sensing probe on the same arm that you’re using to monitor the blood pressure. Be aware that the pulse oximeter reading will go down while the blood pressure cuff is inflated. Remember the blood pressure cuff will occlude the arterial blood flow affecting the reading while the blood pressure is being taken. After the cuff is deflated, the pulse oximeter reading should return to normal.

Hyperventilation: As you may recall, an EtCO\textsubscript{2} less than 35mmHg can lead to alkalosis, and alkalosis causes oxygen to bind tightly to hemoglobin and not releasing it for use at the tissue level. This leads to tissue hypoxia with a falsely high, sometimes even 100%, pulse oximeter reading but the patient is suffering from hypoxia at the tissue level.

Hypoventilation: Remember that an EtCO\textsubscript{2} greater than 45 mmHg can lead to acidosis. Acidosis causes oxygen to bind loosely and reduces the amount carried to the cells. This can lead to a low pulse ox reading that may not respond to O\textsubscript{2} therapy.

Severe anemia or bleeding could lead to falsely high readings because of the lack of red blood cells to carry oxygen. The red blood cells that are present would all be carrying oxygen, leading to high readings unless shock sets in early. In other words, the reading is correct for the little amount of red blood cells that are available but the total

\textsuperscript{5} Effect of Routine Low-Dose Oxygen Supplementation on Death and Disability in Adults with Acute Stroke/The Stroke Oxygen Study Randomized Clinical Trial, Roffe C, et al, JAMA 2017; 318(12): 1125-1135.
oxygen getting to the tissues is too low and imperils survival.

COPD patients often have excess red blood cells, a condition known as secondary polycythemia. Their chronic low oxygen levels make their kidneys produce erythropoietin which makes their bone marrow produce more than normal red blood cells. This can make their blood too thick and make them at risk for blood clots leading to Strokes, MIs and DVTs. The severe COPD patient can have a chronic dusky or blue "cyanotic" color of their skin. This leads to a low pulse oximeter reading that appears out of sorts with the physical exam findings. Frequently “normal” O2 saturations for these patients range from 88-92%.

Hypothermia: Peripheral vasoconstriction causes decreased blood flow to the probe site on the extremities.

Excessive patient movement: This can make it difficult for some pulse oximeter probes to pick up a signal.

High ambient light (i.e., bright sunlight, high-intensity light on area of the sensing probe): can prevent the pulse oximeter from working accurately. Some later generation devices can overcome this problem.

Nail polish or a dirty fingernail when using a fingertip pulse ox: Use acetone to clean the nail before attaching the probe. This is generally accepted practice.

**Your dispatch information:**

Report of a structure fire with occupants trapped inside. Fire has rescued 2 adult males with severe difficulty breathing. What is your treatment? You find both victims with similar signs below.

Carbon monoxide (CO) poisoning: This will give falsely high readings because conventional sensing probes and the oximeters they're attached to can't distinguish between oxyhemoglobin and carboxyhemoglobin. If CO poisoning is suspected, a special monitor and sensor are used to measure levels. CO poisoning can also cause hypoxia because CO binds so tightly with hemoglobin that it takes up the space normally available for oxygen.
A commercially designed device similar to pulse oximetry but specifically designed to determine CO is the Masimo RAD 57. Traditional testing for COHb involves invasive sampling and laboratory analysis. Up to 50% of hospitals do not have on-site laboratory COHb testing ability and Emergency Medical Services personnel cannot measure laboratory COHb levels in the field. Each year, it is estimated that thousands of CO poisoning cases are missed because of a lack of CO testing in emergency department’s devices. They are usually cost prohibitive for EMS agencies as they average $5000.

A newer problem for victims entrapped in structure or vehicle fires is when modern construction materials are burned they produce smoke that contains cyanide. A victim trapped is forced to breathe this smoke, resulting in chemical asphyxiation. Cyanide poisons at the cellular level by preventing cells from using oxygen to make energy. Because the body isn't using any oxygen, the circulating blood will usually be 95-100% saturated, but the patient will still be dying because they can’t use the oxygen that is present inside the cells. Intentional cyanide poisonings are rare, but victims of smoke inhalation may benefit from the same medication used to treat traditional cyanide poisoning. The treatment for cyanide poisoning consists of a single-vial dose of CYANOKIT® (hydroxocobalamin for injection) 5g. CYANOKIT® is approved by the U.S. Food and Drug Administration for the treatment of either known or suspected cyanide poisoning.

“Cyanide may be found within the smoke of some closed-space fires and is an important factor in some deaths caused by smoke inhalation. Cyanide poisoning is a life-threatening situation requiring immediate medical attention. The ability of first responders to recognize symptoms and treat victims of known or suspected cyanide poisoning quickly and efficiently—meaning, on the scene—could mean the difference between life and death,” said Rob Schnepf, chief of special operations with the Alameda County (Calif.) Fire Department.

**Waveform capnography** can now be used at all provider levels to better assess patients in respiratory distress, cardiac arrest and shock.
Capnography offers reliable feedback about the severity of a patient’s condition and how they respond to treatment. Here are five things you should know about waveform capnography.

1. CAPNOGRAPHY PROVIDES BREATH-TO-BREATH VENTILATION DATA

   Waveform capnography represents the amount of carbon dioxide (CO2) in exhaled air, which assesses ventilation. It consists of a number and a graph. The number is capnometry, which is the partial pressure of CO2 detected at the end of exhalation. This is end-tidal CO2 (ETCO2) which is normally 35-45 mm Hg.

   The capnograph is the waveform that shows how much CO2 is present at each phase of the respiratory cycle, and it normally has a rectangular shape. Capnography also measures and displays respiratory rate. Changes in respiratory rate and tidal volume are displayed immediately as changes in the waveform and ETCO2.

   Two sensors can be used to measure capnography. In patients who are breathing, nasal prongs can be applied that capture exhaled air. Those prongs can also be used to administer a small amount of oxygen, or applied underneath a non-rebreather or CPAP mask. In patients who require assisted ventilation, another adapter can be attached to a BVM and advanced airway device.

   Capnography assesses ventilation, which is different from oxygenation. Ventilation is the air movement in and out of the lungs, while oxygenation is the amount of oxygen inhaled by the lungs that reaches the bloodstream. Pulse-oximetry assess oxygenation, and works by measuring the how much of each red blood cell is bound with oxygen. It is expressed as a percent, or SPO2.

2. ETCO2 PROVIDES CLUES ABOUT RESPIRATORY EFFORT

   In people with healthy lungs, the brain responds to changes in CO2 levels in the bloodstream to control ventilation. We assess this by observing chest rise and fall, assessing respiratory effort, counting respiratory rate, and listening to breath sounds. ETCO2 adds an objective measurement to those findings. The patient’s respiratory rate should increase as CO2 rises and decrease as CO2 falls.

   If a patient has slow or shallow respirations, and a high ETCO2 reading, this tells us that ventilation is not effectively eliminating CO2 (hypercarbia), and that the brain is not responding appropriately to CO2 changes. This may be caused by an overdose, head injury, respiratory insufficiency or seizure. Pulse oximetry helps determine how much oxygen should be administered, and capnography helps determine when ventilation should be assisted with a bag valve mask. Conversely, if an unresponsive patient has a normal ETCO2, a conservative approach with close monitoring can be taken.

   While a rise in CO2 should stimulate someone to breathe, in a normal person no effort
should be needed to exhale it. Patients with asthma, COPD, CHF, and pneumonia must often exert themselves to exhale with accessory muscles. It is important to understand that patients in respiratory distress may inhale enough oxygen and have a normal pulse-ox reading, but still struggle to get air out. An elevated capnograph in this group of patients means that their effort is not effectively eliminating CO2 (hypercarbia). They may be progressing to respiratory failure from hypercarbia and fatigue, not hypoxia, and need assisted ventilation.

3. CAPNOGRAPHY HELPS DIAGNOSE THE CAUSE OF RESPIRATORY DISTRESS

Correctly diagnosing the cause of respiratory distress can be difficult, and treating the wrong condition may cause harm. A number of conditions can cause diminished breath sounds, wheezing may be heard with both asthma and pulmonary edema, and crackles may be heard with pulmonary edema and pneumonia. Adding waveform capnography to history and physical exam findings can help with treatment decisions.

The capnography waveform represents air movement in the lungs, similar to how complexes on an ECG represent electrical conduction through the heart. The waveform starts at the beginning of exhalation, and senses air from dead space in the upper airway and bronchi. There is normally no CO2 present in dead space, and the graph should be at baseline. A sharp spike is normally seen when exhaled air from the alveoli reaches the sensor, and plateau’s when all of the exhaled air detected came from the alveoli. A sharp downward spike is then seen during inhalation. The height of the waveform depends on the amount of CO2 detected, and the length of the waveform depends on the time of exhalation.

In cases of bronchospasm, air is trapped in the alveoli and inconsistently released. This creates a curve in the initial spike and plateau, or “shark fin” appearance. The worse the bronchoconstriction, the more pronounced the curve on the waveform, and the higher the ETCO2 is likely to be. If the waveform is upright and “crisp,” there is no bronchospasm and respiratory distress must be from another cause.

Increased work of breathing from pulmonary edema may lead to fatigue and respiratory failure. This would cause a rise in ETCO2, but the waveform will remain upright. Hyperventilation causes excess CO2 to be exhaled, which would present with a crisp waveform and low ETCO2, or hypocapnea. Causes of hyperventilation include diabetic ketoacidosis, pulmonary embolism, and anxiety.

4. CAPNOGRAPHY PROVIDES REAL-TIME FEEDBACK ON HOW WELL TREATMENT IS WORKING

Imagine a wheezing patient whose respiratory rate and work of breathing decrease after receiving albuterol. If ETCO2 also decreases, and their shark-finned capnograph shifts upright after receiving albuterol, this means the patient is responding well to treatment. If their ETCO2 increases and shark fin waveform becomes more pronounced, they are progressing to respiratory failure. Treatment plans can be quickly adjusted when capnography is used to monitor trends.
When providing positive pressure ventilation with a bag valve mask, it can be difficult to track how often the bag is squeezed and how much air reaches the lungs. When capnography is used to assist ventilating patients with a pulse, a waveform will be seen after each squeeze when air reaches the lungs. Ventilation is not effective if there is no waveform, and troubleshooting is needed. Consider repositioning the head, suctioning the mouth, placing an adjunct, having a second person hold the mask, and reassess. Capnography can also help guide how fast to ventilate the patient. Harm is associated with hypo and hyperoxia, as well as hypo and hyperventilation. Oxygenation should be titrated to achieve an adequate SPO2 (in severe chronic lung 88-92%) and ventilation should be titrated to achieve ETCO2 between 35 and 45 mm Hg (unless the patient has a metabolic acidosis like DKA-diabetic ketoacidosis- where the patient hyperventilates to partially correct for acidosis- in this rare case they should be allowed to hyperventilate).

Capnography is the most reliable method to confirm correct advanced airway placement, and provides documentable proof. If an ET tube is outside the trachea, or if air from a supraglottic device is not directed into the glottic opening, no waveform or end-tidal reading will appear. If a correctly placed airway device is dislodged, the capnography waveform will immediately be lost.

5. CAPNOGRAPHY ALSO DETECTS SHOCK

Capnography has a ventilatory and circulatory component. Cells use oxygen and glucose to make energy, and release CO2 into the bloodstream to be carried to the lungs. The amount of exhaled CO2 depends on the adequacy of circulation to the lungs, which provides clues about circulation to the rest of the body. Low ETCO2 with other signs of shock indicates poor systemic perfusion leading to metabolic acidosis, which can be caused by hypovolemia, sepsis or dysrhythmias.

Cardiac arrest is the ultimate shock state; there is no circulation or metabolism and no CO2 production unless effective chest compressions are performed. Capnography provides feedback on the quality of compressions and when a compressor change is needed. An ETCO2 less than 10 mm Hg indicates that compressions are not fast or deep enough, or the patient has been dead for too long. If circulation is restored, a spike in ETCO2 often appears before a pulse is detected. Sometimes it can be difficult to determine if a patient has a pulse, but circulation must be present if ventilation produces a waveform without compressions.

In conclusion, the EMS provider has many tools to help in the assessment and treatment of patients with respiratory disorders. To best serve your patients, EMS providers should maintain a thorough working knowledge of those tools and use them on a regular and consistent basis.