

AIRWAY MANAGEMENT

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I MECHANICS OF BREATHING

Oxygen provides the energy that every cell in the body needs in order to function. Without it, the body comes to a standstill resulting in death. Our respiratory system, in conjunction with the cardiovascular system assures continuous delivery of oxygen to every cell in the body, as well as the exportation of carbon dioxide out of the cells and our body.

OXYGENATION and VENTILATION are the basic mechanics of oxygen delivery and carbon dioxide exchange. Oxygenation refers to the delivery of oxygen into the body. Ventilation refers to the exchange of carbon dioxide with oxygen in the alveoli of the lungs, along with the expulsion from our body. In the overall mechanics, when we take a breath, oxygen comes in from the atmosphere and enters the lungs. Oxygen traverses all the way into the alveoli where it comes into contact with the red blood cells and the specifically hemoglobin. The oxygen crosses through the alveolar membrane attaching to the hemoglobin, and the carbon dioxide seeps into the alveoli. The oxygenated red cells continue onto the left atrium and ventricle where they are pumped out into the body. The carbon dioxide, which is now in the alveoli, gets expelled from the body when the person exhales. Every time a person breathes, this process is repeated. In so doing, the body is able to oxygenate every cell and remove the poisonous byproduct of energy synthesis; carbon dioxide, from the body.

When the system does not function appropriately, a break down occurs in the oxygenation, the ventilation, or both. A lack of oxygen results in hypoxia, which can cause inefficiency to the cells. At its most severe state, anoxia or complete lack of oxygen occurs. This results in the death of the cells as well as the organ involved. Deprivation of oxygen and the death to the organ varies with each organ. Muscle can survive for hours without oxygen. However, the body's most vital organ, the brain, can only last minutes without oxygen! Ventilation has to do with the exchange of oxygen and carbon dioxide. When the carbon dioxide does not exchange and get exhaled, it builds up in the body and causes acidosis and sedation to the body. The organs become inefficient and the person becomes sedated. At its most severe form, the organs shut down because of the acidosis and the sedation causes apnea.

Ventilation is a better measure of how a person is breathing and of cellular metabolism. Supplemental oxygen can help prevent hypoxia in a near apneic state. The supplemental oxygen does not reverse the hypercapnia (high CO₂) that sets in if the patient is not ventilating and not exchanging the oxygen for the carbon dioxide. Monitoring the

oxygen saturation may provide a false sense of security since the patient could also have elevated carbon dioxide levels that are going undetected.

II RESPIRATORY DRIVE

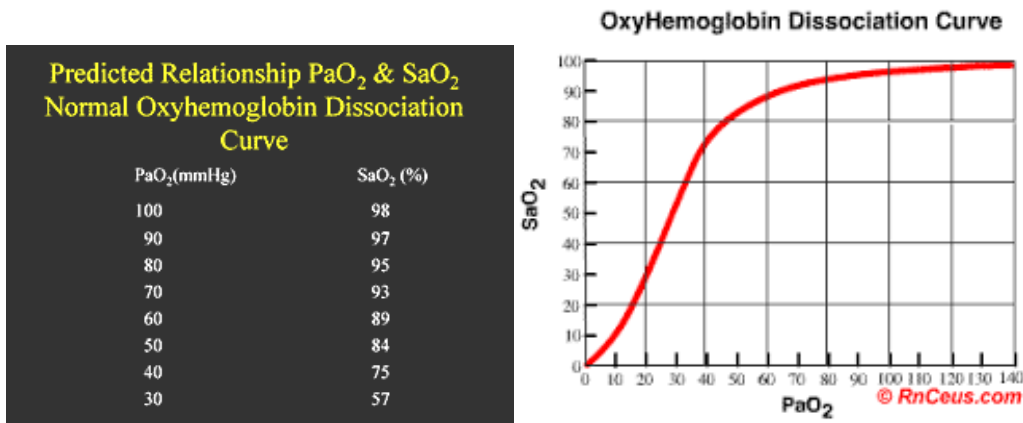
The normal act of breathing or our respiratory drive is not driven by our need for oxygen but primarily by our need to expel the carbon dioxide. The brain has sensors in the medulla oblongata that measures CO₂ levels in the cerebrospinal fluid (CSF). The increase of the carbon dioxide in the CSF triggers the need to exhale the gases.

The actual act of breathing, or ventilating the lungs, has two phases; inhalation and exhalation. With each breath, the diaphragm and intercostal muscles contract causing the diaphragm to push downward and the chest to expand. Exhaling, in a normal lung, is passive and the diaphragm and chest wall muscles relax causing the CO₂ to be expelled. The process repeats itself with each breath. A secondary mechanism which relies on the body's need for oxygen comes into play if the person becomes hypoxic such as in CHF or COPD exacerbations. The increased demand for oxygen along with the need to expel the lactic acid build up in the muscles caused by athletics would also drive the person to breathe faster.

CO₂ RETAINERS comprise a group that have “burned out” the receptors that recognize the increase in CO₂ in the brain. Because of this, their normal mechanism for breathing is impaired. They must rely on their secondary mechanism or their hypoxic drive to breathe. When this group is given too much oxygen, the body senses it and will not take a breath as frequently or as deeply because there is no hypoxia to drive the breathing. Carbon dioxide accumulates and the person now becomes sedated and acidotic. Respiratory rates can diminish until they stop breathing and die.

CO₂ retainers must always be somewhat deprived of oxygen. Saturations of 90% are considered borderline hypoxia and for this population, a saturation of 90% to 92% is where they need to “live”. In the setting of respiratory distress, oxygen should not be restricted, but the patient's ventilation needs fastidious monitoring.

In reality, a saturation of 90% physiologically suffices for everyone. Because of the shape of the oxygen dissociation curve, the amount of benefit gained above 90% is small. However, below 90% there is a steep drop off in the person's oxygenation with rapid desaturation and hypoxia.



As is demonstrated from the oxyhemoglobin dissociation curve, the SaO₂ of 90% provides near total oxygen saturation. Moving along the curve to the right there is only a little bit of gain for the patient in terms of saturating the hemoglobin molecules above 90%. However, there is a steep drop in the saturation and it is almost like diving off a cliff when the saturation moves to the left on the curve.

III RESPIRATORY ASSESSMENT

Whether you are a basic EMT, paramedic, nurse or doctor, the initial assessment of a patient's respiratory status is the same. First visual observation can quickly determine if the person is in respiratory distress. How do they look? Is the patient breathing fast, labored, paradoxical breathing, slow? Is the patient diaphoretic? Is the patient tired, scared, obtunded? Can the patient carry on a conversation; speak only a few words or just nod? A history can add further information. Does the patient have CHF? COPD? Both? What medications does the patient take? Is there a history of a cough or fever? Weight gain? Leg swelling? Recent travel? Was the onset gradual or sudden? Does the patient have other medical issues such as coronary artery disease, renal failure?

Once the initial visual assessment is done, the patient is examined. What are the vitals? Is the patient wheezing or have rales? Are there diminished breath sounds consistent with a pneumothorax or focal finding consistent with pneumonia? Typical finding are noted with different pathology.

PULMONARY EDEMA: Often sudden onset associated with rales, diaphoresis and hypertension. Pulmonary edema has two types: Cardiogenic which is seen with patients

who have heart conditions and non-cardiogenic edema which may be seen with drug overdoses and head bleeds to name a couple of causes.

CONGESTIVE HEART FAILURE: This is a gradual onset of fluid build up in the lungs. It is usually associated with difficulty lying flat, leg swelling and weight gain. The patients will oftentimes complain of worsening exertional dyspnea. Rales and/or wheezes are heard. The patient may have JVD and usually has lower extremity edema

COPD EXACERBATION: COPD or chronic obstructive pulmonary disease is made up of the triad of chronic bronchitis, emphysema and asthma. These patients, except the asthmatics, have a history of smoking in most cases. They all use inhalers and some underlying event such as a pulmonary infection or allergies triggered the event. Wheezes or diminished breath sounds are common.

PNEUMOTHORAX: A pneumothorax may occur with trauma or spontaneously. They typically cause discomfort with breathing and are associated with diminished breath sounds. A small pneumothorax may not have a significant diminishment in breath sounds. A TENSION PNEUMOTHORAX is a life threatening event. This is always associated with diminished or loss of breath sounds on the affected side. There is an increased respiratory rate, some degree of hypotension and tachycardia. JVD and tracheal deviation may also be present.

PULMONARY EMBOLI: The pulmonary embolus presents in various ways and depends on the size and location. Pleurisy, dyspnea, exertion dyspnea, wheezing all the way to respiratory distress is possible. Tachycardia may or may not occur. Risk factors include women who smoke and use birth control, recent travel, prolonged rest such as hospital stay or surgery, cancer and genetic clotting deficiencies. A recent case illustrates the difficulty of diagnosing pulmonary emboli. This 89 year old female had noted for about a month some trouble breathing on her daily 2 mile walk. She recently noted that on occasion she would become short of breath for a few moments when she was doing nothing. She had not seen a doctor in years and was on no medications. She was noted to be hypertensive and had a normal heart rate and respiratory rate. Her saturation was 89% on room air. Two liters of oxygen caused the saturation to go to 98%. A cat scan of her chest demonstrated multiple pulmonary emboli along with a large one saddling over the left and right pulmonary artery. If this one were to enlarge, it would block off both pulmonary arteries and cause her to die. She was informed of the findings and told she was to be heparinized in order to prevent anymore clotting. Her only question was

whether the medication would interfere with her sex life! Now you understand how difficult pulmonary emboli's are to diagnose and what a bad visual you are left with when an 89 year old female discusses her sex life

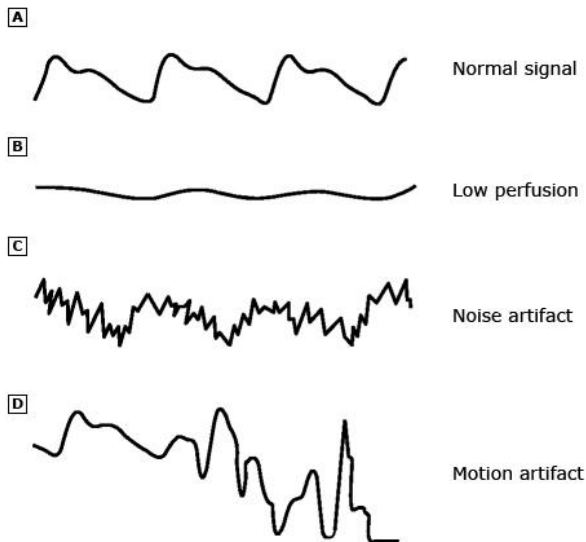
IV. AIRWAY MANAGEMENT

Airway management is the intervention that you perform in order to improve the patients breathing, and thereby improve the oxygenation and ventilation. It may entail nothing more than positioning the patient or calming them down. Medications and forms of oxygen delivery obviously play a role as well.

MONITORING DEVICES such as the pulse oximetry and the end tidal CO₂ device are important adjuncts in assessing and managing the patients breathing. Your eyes however are your best friend in this area. If a patient looks bad you are going to be right in almost all the cases regardless what the numbers tell you. More than once, I have been informed that the patient's saturations are "fine". The only problem was the patient was barely ventilating.

Pulse Oximetry uses infrared spectrometry to measure the percentage of hemoglobin in that person's body that is saturated with oxygen. The measurement does not account for

blood volume. This means that if the person has been bleeding out and has only a third of a normal blood volume, the oximetry will still read 95% if 95% of the remaining intravascular hemoglobin is saturated with oxygen.



The first step in measuring the pulse oximetry is assuring a normal wave form. A normal oximetry waveform mimics an arterial waveform and has a dicrotic arch or two humps. Assuming an appropriate waveform, the rate of the oximetry should be correlated with a manual pulse or observed heartrate from the cardiac monitor.

Common pulsatile signals on a pulse oximeter.

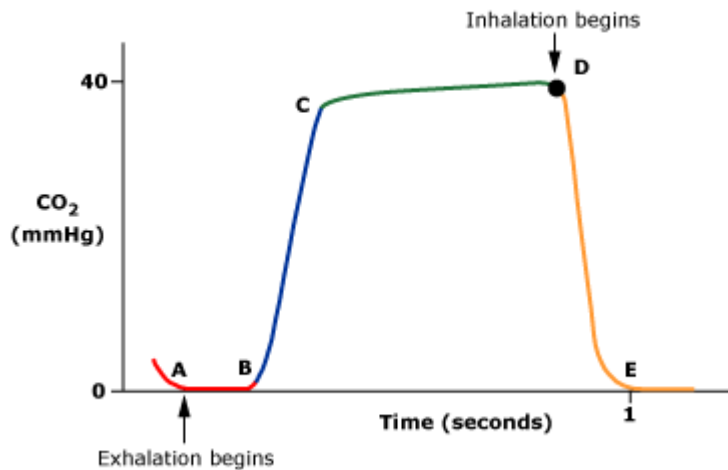
(A) Normal signal showing the sharp waveform with a clear dicrotic notch.

- (B) Pulsatile signal during low perfusion showing a typical sine wave.
- (C) Pulsatile signal with superimposed noise artifact giving a jagged appearance.
- (D) Pulsatile signal during motion artifact showing an erratic waveform.

Certain physical and medical conditions may result in false oximetry readings. Hypoperfusion and hypothermia which results in peripheral vascular constriction may result in low peripheral perfusion and falsely low oxygen levels. In carbon monoxide poisoning, there is a falsely higher oximetry due to the fact that carbon monoxide has a much higher affinity for hemoglobin than oxygen. Carboxyhemoglobin (carbon monoxide and hemoglobin) is formed instead of oxyhemoglobin (oxygen and hemoglobin). Both of these end products are interpreted the same with a pulse oximetry resulting in this case a falsely high reading. In these cases, a co-oximetry should be used.

Capnography is a noninvasive measurement of end-tidal carbon dioxide and an invaluable tool in both respiratory distress and during CPR. **End-tidal CO₂** (EtCO₂) which is the measurement of carbon dioxide concentration at the end of each tidal breath can assist in the diagnosis of disease, tube placement and assessing changes in the patient's overall condition. As stated previously end-tidal CO₂ measurements should occur in any patient with respiratory distress and during CPR. It should also be checked when sepsis is considered.

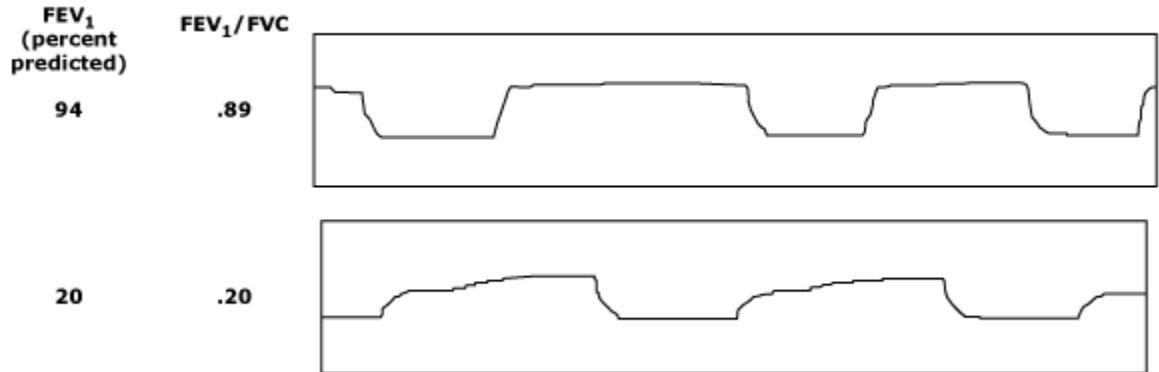
CO₂ WAVEFORM — The capnography will form a wave that has four distinct phases. Normal lungs have a rectangular waveform while COPD has an upsloping shape.



- Phase 1 (dead space ventilation, A-B) represents the beginning of exhalation where the dead space is cleared from the upper airway.
- Phase 2 (ascending phase, B-C) represents the rapid rise in carbon dioxide (CO₂) concentration in the breath stream as the CO₂ from the alveoli reaches the upper airway.
- Phase 3 (alveolar plateau, C-D) represents the CO₂ concentration reaching a uniform level in the entire breath stream from alveolus to nose. Point D, occurring at the end of the alveolar plateau, represents the maximum CO₂ concentration at the end of the tidal breath and is appropriately named the end-tidal CO₂ (EtCO₂). This is the number that appears on the monitor display.
- Phase 4 (D-E) represents the inspiratory cycle.

In cases of COPD, the waveform has a sloping, or "shark fin" upstroke.

CO₂ waveform in obstructive lung disease



The top waveform is from a patient with normal lung function and has a characteristic rectangular appearance. The bottom waveform is from a patient with severe chronic obstructive pulmonary disease and has a characteristic curved appearance and up-sloping of the alveolar plateau.

FEV₁: forced expiratory volume in one second; FVC: forced vital capacity.

Both graphs taken from Uptodate and reproduced with permission from: Krauss B, Deykin A, Lam A, et al. Capnogram shape in obstructive lung disease. Anesth Analg 2005; 100:884. Copyright © 2005 Lippincott Williams & Wilkins.

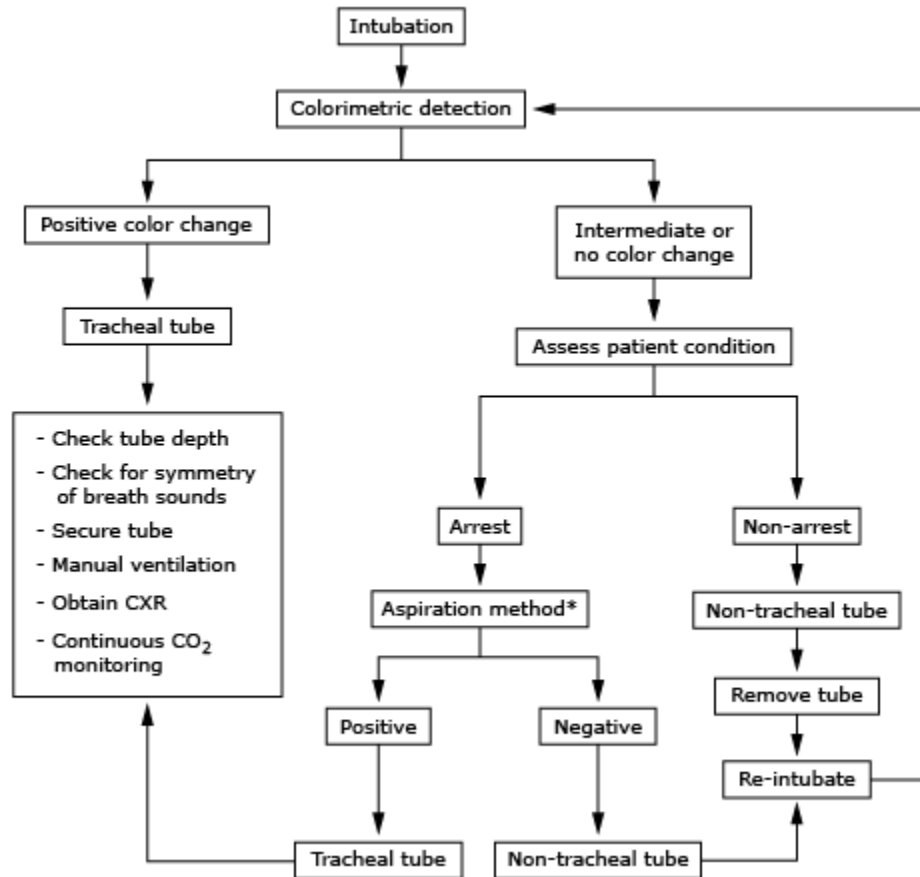
Graphic 64586 Version 10.0

Normal end-tidal CO₂ has a range of 35 to 45 mmHg. In patients hyperventilating the CO₂ goes down and if the patient were to have an esophageal intubation, the end-tidal would range typically be less than five.

As with all new technology, new modalities for its use are constantly being added.

Endotracheal Tube Placement: Capnography is the gold standard for tube placement. When used, it assures appropriate tube placement and also provides documentation of the appropriate location. It also alerts medics in real time if a tube becomes dislodged.

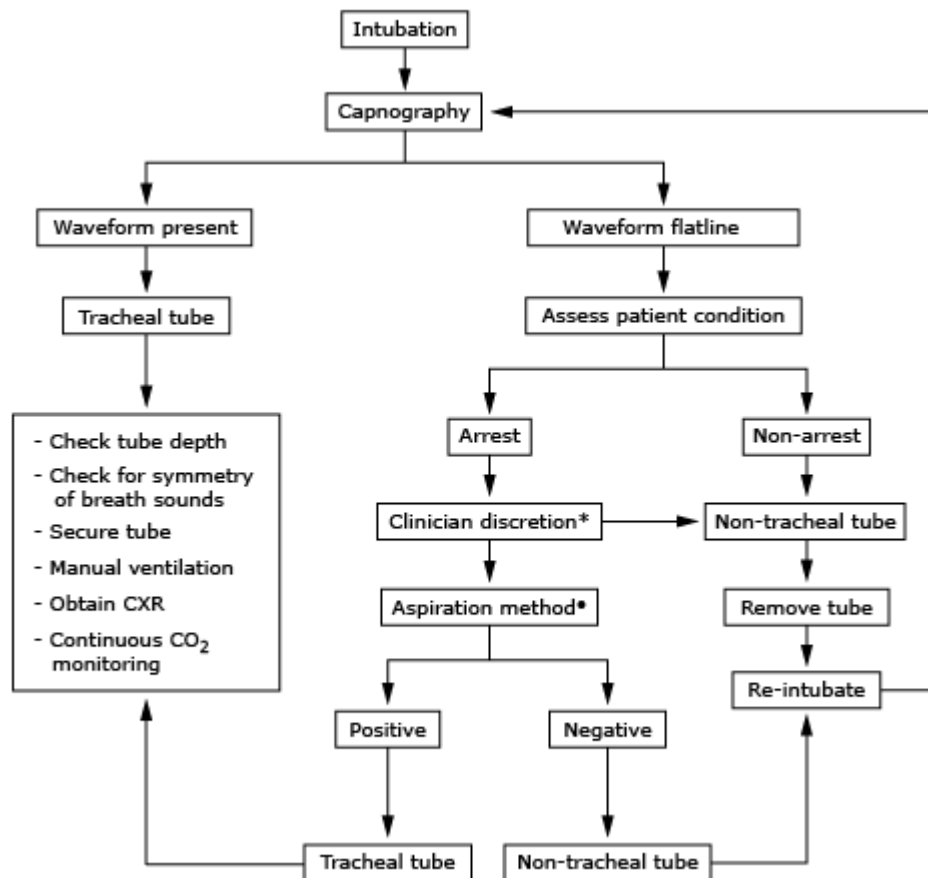
Universal tube placement confirmation with Colorimetric CO₂ detectors



* Clinical maneuvers, such as auscultation (chest, epigastric) and direct laryngoscopy may be utilized at physician discretion, but clinicians must be aware of their limitations in discriminating between esophageal and tracheal intubation.

Graphic 71442 Version 2.0

Universal tube placement confirmation with capnography



* Based on the clinical situation, provider may choose to remove the tube or obtain a secondary confirmation measure.

• Clinical maneuvers, such as auscultation (chest, epigastric) and direct laryngoscopy may be utilized at physician discretion, but clinicians must be aware of their limitations in discriminating between esophageal and tracheal intubation.

Effectiveness and monitoring CPR: During CPR, EtCO₂ levels can reflect the effectiveness of cardiac output and compressions. With effective CPR, cardiac output should improve and the EtCO₂ will increase, which reflects improved perfusion. So what are the levels we are talking about? EtCO₂ levels were found to typically be less

than 3 mmHg at the time of intubation during a cardiac arrest. Levels that reach 7.5 mmHg or greater were seen just prior to spontaneous return of circulation. When ROSC occurs, a large bump in EtCO₂ will occur. Therefore, the use of EtCO₂ can eliminate the need for withholding compressions to check for pulses. This in turn assures optimal cerebral perfusion during CPR. Finally, studies have demonstrated that EtCO₂ of less than or equal to 10 mmHg lasting for 20 minutes after commencing ACLS accurately predicted death in adult patients who presented with cardiac arrest.

Monitoring Ventilation: Hyperventilation results in vasoconstriction which can interfere with optimal cerebral perfusion. Ventilating a patient at a rate of 10 to 12 is actually very difficult to perform especially during a high adrenaline event such as CPR or resuscitating a major trauma or ill patient. Monitoring the EtCO₂ has been shown in several studies to improve patient outcome. *“In a prospective observational study of blunt trauma patients requiring prehospital intubation, EtCO₂ values obtained 20 minutes after intubation distinguished the great majority of survivors from nonsurvivors [55]. Median EtCO₂ among survivors was 30.8 mmHg and among nonsurvivors 26.3 mmHg (95 percent CI of difference between medians 3 to 6.75 mmHg).” Uptodate End Tidal CO₂ Monitoring in Adults*

Clinical Assessment of Clinically Ill Patients: EtCO₂ monitoring can effectively assist in monitoring patients who present clinically ill but are still spontaneously breathing and not intubated. Changes in the CO₂ may determine if the patient is improving or even worsening. Using the end tidal CO₂ monitor is just another adjunct in the management of the airway that can help alert you to changes, both good and bad, in the patients breathing. Let's take a COPDer who is found to be very tachypneic. Early on, because of the tachypnea, they may be blowing off the CO₂ and have a low level. You supply supplemental oxygen and start a duoneb. Now you note the breathing is slowing down and the CO₂ is rising. One scenario is the patient is improving and the airways are opening. Another scenario is that the patient is tiring and the breathing is slowing because of the fatigue. Possibly, the patient is a CO₂ retainer and is not breathing because of the supplemental oxygen. In the first scenario, we would expect the CO₂ to normalize but go no higher. In the other two, the expectation would be for a continued rise in the CO₂.

Metabolic Acidosis and Sepsis: The loss of bicarbonate results in a loss of a base in the body. This causes the body to have a metabolic acidosis. In order to return to a normal pH, the body has to dump an acid and this is done by blowing off CO₂. In these cases the respiratory rate usually increases and the CO₂ goes down. EtCO₂ has been shown to

assist in the diagnosis of several different illnesses including diabetic ketoacidosis and severe gastroenteritis especially in children. **Studies have also shown that EtCO₂'s less than or equal to 24 correlate to a lactate of 4 and a higher rate of sepsis.**

OXYGEN DELIVERY

OXYGEN DELIVERY can come in many forms. What is used depends on the level of distress and also what the patient can tolerate. CPAP may suffice but if the patient cannot tolerate the mask, it is worthless.

NASAL CANNULA delivers oxygen directly into the nose. It is for lower flows and is no longer considered necessary for chest pain patients who have no respiratory issues.

NON REBREATHING MASK delivers high flow 100% oxygen. It is used in respiratory distress and has fenestrations so that the CO₂ disburses.

VENTURI MASK allows for adjustments in the percentage of oxygen delivered. Different valves when attached deliver varying amounts of oxygen.

CPAP or CONTINUOUS POSITIVE AIRWAY PRESSURE mask maintains continuous airway pressure so that the alveoli and bronchus do not collapse. This reduces the work of inhaling by reducing the resistance the patient is experiencing. This in turn allows the intercostal muscles to relax and recover from the fatigue.

BAG VALVE MASK is best performed with two people. When done appropriately, it is an effective airway even in resuscitations. Positioning of the patient and the seal of the mask are most important. The ear should align with the sternal notch except in the case of a cervical spine immobilization. A tight seal needs to be maintained and small slow breaths prevent over ventilation and regurgitation.

MASK VENTILATION 2 PERSON TECHNIQUE

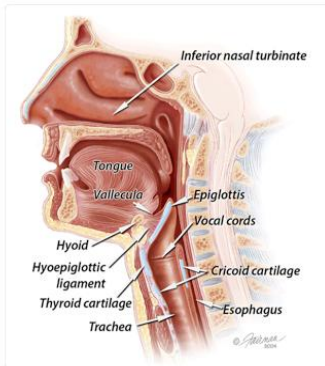
- Proper Positioning – Ear-To-Sternal Notch Alignment, Unless Contraindicated By Cervical Spine Immobilization.
- Jaw Thrust – Lift Up On Mandible And Submandibular Tissues.
- Press Mask Down On The Face And Make A Tight Seal.
- Fully Insert Oral And/Or Nasal Airways.
- Small, Slow, Easy Squeeze Of Bag: 6-7 Cc/Kg, Over 1-2 Seconds, Using Low Pressure. Rate Should Not Exceed 12 Breaths Per Minute In Adults.
- Over-Ventilation, High Volume, And High Pressure Increase Regurgitation.



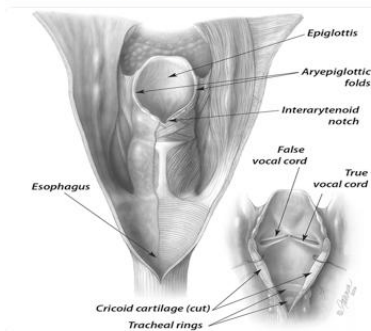
INTUBATION is done both to breathe for the patient and also protect the airway. Protection may be from regurgitation in an overdose or swelling from an infection, trauma or anaphylaxis. You are given two choices or "holes" in intubations. One "hole" is the trachea which leads to maintaining the airway. The other "hole" is the esophagus which if intubated can lead to hypoxia and death. Therefore, in order to intubate, you need to know the anatomy of the airway. Landmarks are extremely important and the most important is the epiglottis. This is called the 'BEACON' to the vocal cords. It is at the base of the tongue and at the midline of the airway. Right behind it is the vocal cords and the "hole" you want to intubate.

ANATOMY OF THE AIRWAY

- EPIGLOTTIS IS THE “BEACON” TO THE VOCAL CORDS.
- IT IS AT THE BASE OF THE TONGUE, CONNECTED TO THE TONGUE AND AT THE MIDLINE OF THE AIRWAY.



ANATOMY OF THE AIRWAY



NOTE THE LOCATION OF THE ESOPHAGUS BEHIND THE TRACHEA

ANATOMY OF THE AIRWAY

- **FIRST ROW**
 - UVULA
 - TONSILS
 - EPIGLOTTIS
- **SECOND ROW**
 - MOVING BEYOND THE EPIGLOTTIS TO THE GLOTTIC OPENING
- **THIRD ROW**
 - VOCAL CORDS..OPEN, CLOSED AND OPEN AGAIN



V. INTUBATION

The intubation has several indications which are changing as we learn more about resuscitation and as we devise new tools for use in airway management. For instance, in CPR, the airway was considered the most important aspect of the resuscitation even in cardiac arrests. The philosophy has changed and now all evidence points to compressions as the focus. In fact, breathing may not need any adjunct beyond a 100% non-rebreather mask! This is not to say intubations are not needed. Respiratory distress, overdoses and airway obstruction all may require intubations. Technique and understanding the airway anatomy will assure success. The goal is always intubating on the first attempt. And always remember that if the airway cannot be secured with a traditional endotracheal tube, our escape valve is the supraglottic tube.

INDICATIONS FOR INTUBATION

- CPR
- Respiratory Distress
- Altered Mental Status With Hypoventilation / Airway Protection
- Facial Trauma
- Angioedema Of The Airway
- Airway Infection i.e. Epiglottitis

PRE-OXYGENATION

- Pre-Oxygenation Helps Prevent Hypoxia During The Intubation.
- BVM 15 LPM
- Non Rebreather Face Mask 15 LPM
- Nasal Cannula 15 LPM in addition to the face mask
- Use Nasal Cannula 15 Lpm In Conjunction With The Face Mask At 15 LPM
- Leave The Nasal Cannula On During Intubation
- Nasal Cannula Will Expel CO2 Build Up In The Nasopharynx
- It Will Also Maintain O2 Saturations During The Intubation

FIRST PASS SUCCESS

- Goal Is To Intubate On First Pass
- Studies Indicate That Complications Increase With Increasing Attempts.

EPIGLOTTIS

- Epiglottis Is The “Beacon” Leading To The Glottis And Trachea
- It Is Midline In The Posterior Pharynx And Attached To The Base Of The Tongue.

INTUBATION FIRST PASS TECHNIQUE

Initial blade insertion is with the laryngoscope handle pointed at the patient's feet. The tongue and jaw are distracted downward to insert the blade. Minimal force is required for downward jaw distraction, assuming the head is not overextended. AIRWAYCAM.COM



INTUBATION FIRST PASS TECHNIQUE

The tip of the blade gets around base of tongue, permitting change in angle of lifting and better mechanical advantage. Epiglottis edge is lifted off the pharyngeal wall. The epiglottis is often camouflaged against the mucosa of the posterior pharynx. AIRWAYCAM.COM



INTUBATION

FIRST PASS TECHNIQUE

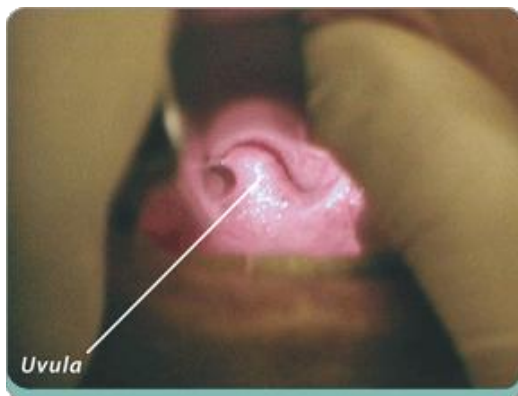
With full insertion of the curved blade into the vallecula the angle of lifting changes to ~40 degrees from the horizontal. Once the tip is fully in the vallecula, the lifting force can be increased as needed. **Tip position (not force) is the main determinant of glottic exposure**

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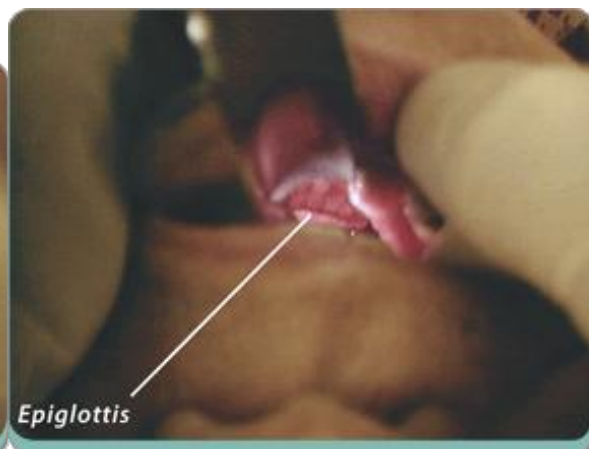


PROGRESSION OF LANDMARKS IN INTUBATION

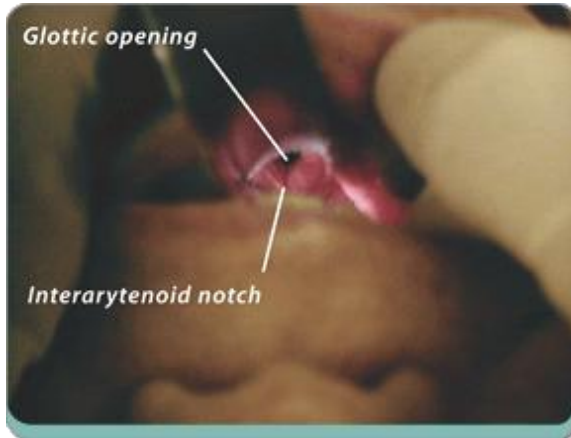
(1) TONGUE AND UVULA



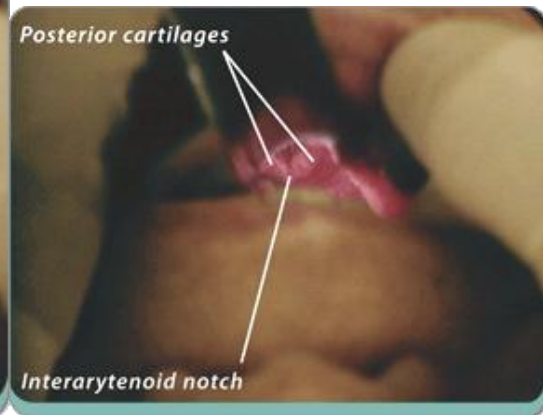
(2) EPIGLOTTIS



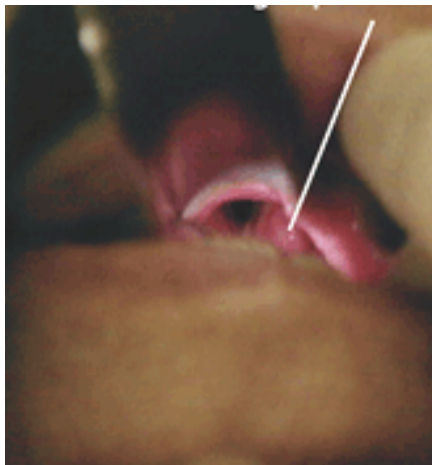
(3) POSTERIOR CARTILAGES



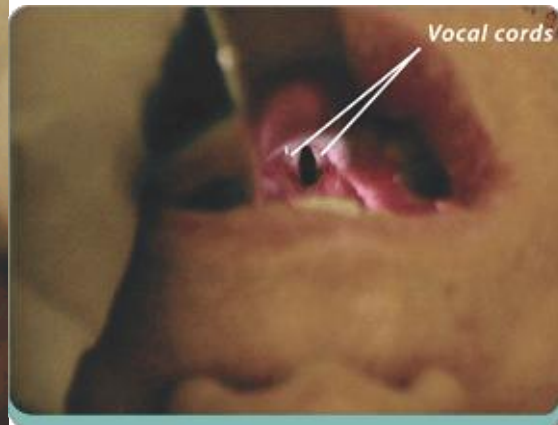
(4) INTERARYTENOID NOTCH



(5) GLOTTIC OPENING



(6) VOCAL CORDS



EPIGLOTTIS CAMOUFLAGE

- In The Supine Position, Poor Muscle Tone And Sedation Can Cause The Epiglottis To Flop Back And Rest Against The Posterior Pharyngeal Wall
- It Is Easy In This Position To Go Past The Epiglottis And Overshoot The Glottis
- Maintain The Head At The Ear-Sternal Notch Position.

- Extending The Head At The Base Of The Skull Will Only Cause The Epiglottis To Push Further Against The Posterior Pharynx
- Lifting The Head Can Better Expose The Epiglottis

BIMANUAL LARYNGOSCOPY

- Allows For The Curved Blade To Slip Completely Into The Vallecula.
- When Only Partially Inserted, The Epiglottis Cannot Be Adequately Lifted.
- Using The Right Hand And Pushing Down On The Thyroid Cartilage Will Allow For The Curved Blade To Slip Fully Into The Vallecula
- This Will Cause The Epiglottis To “Pop Up” And Expose The Vocal Cords.

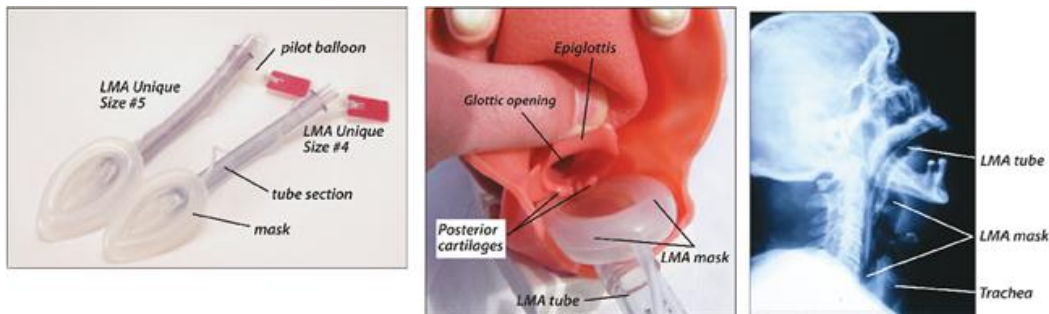


VI. SUPRAGLOTTIC AIRWAY

SUPRAGLOTTIC AIRWAYS have changed the way intubations are performed and have also provided an escape valve for the patient where the vocal cords are not visualized. The tubes work in the majority of the cases by blocking off the esophagus and sealing off the airway above the vocal cords. When a breath is given, the only place for the air to go is into the lungs. On a rare occasion, the tube may slip into the trachea and direct ventilation occurs. The LMA is the exception to this technique since it has only one port and does not enter the esophagus. Supraglottic tubes facilitate an airway rapidly and are secure.

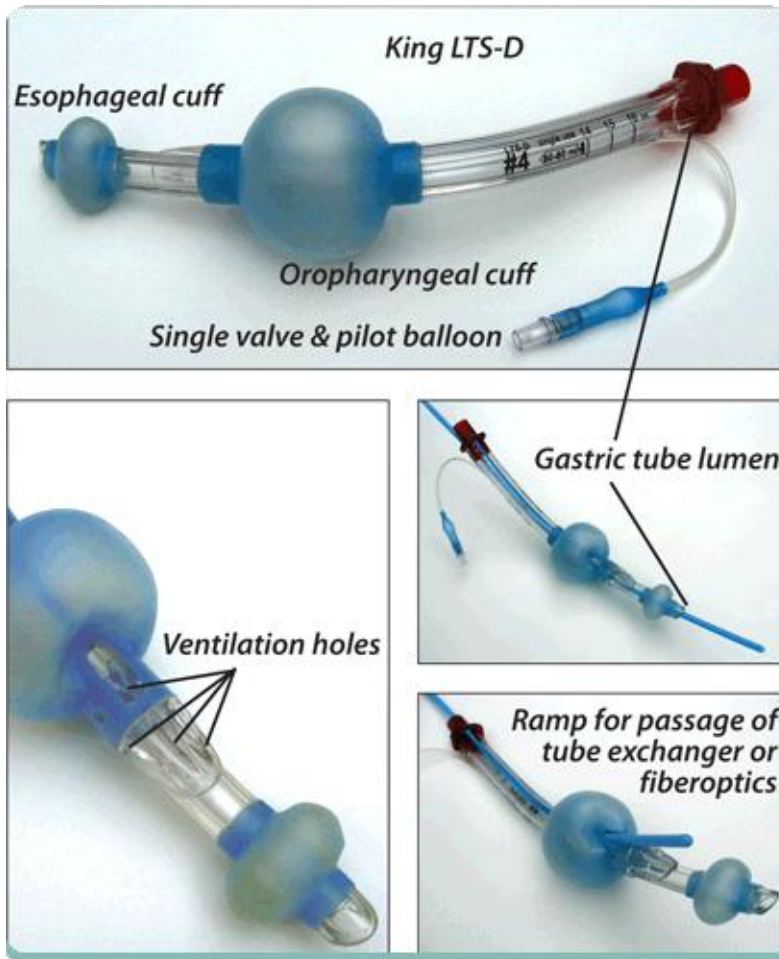
LMA

- 1) Wedge-shaped inflatable mask fits over larynx, creating seal within upper airway. Patient must have absent gag reflex.
- 2) Tip of wedge fits into upper esophagus and bowl of mask covers laryngeal inlet.
- 3) Numerous sizes from pediatric to adult; typical adult female requires #4 LMA, typical adult male #5 LMA.
- 4) Recommended insertion technique (LMA Unique, Classic): With mask deflated, lubricate undersurface, using fingers at base of bowl, push mask down throat, up against palate, and backward to posterior hypopharynx, until bowl is around curvature of tongue.
- 5) Best seal pressure is with under-inflation of mask; recommended volume is 30 cc for #4 , and 40 cc for #5--many users start with half this volume and listen over the neck for a cuff leak. If cuff leak, try smaller mask volume or larger LMA.
- 6) Cricoid pressure prevents optimal placement and pushes tip out of upper esophagus



KING TUBE

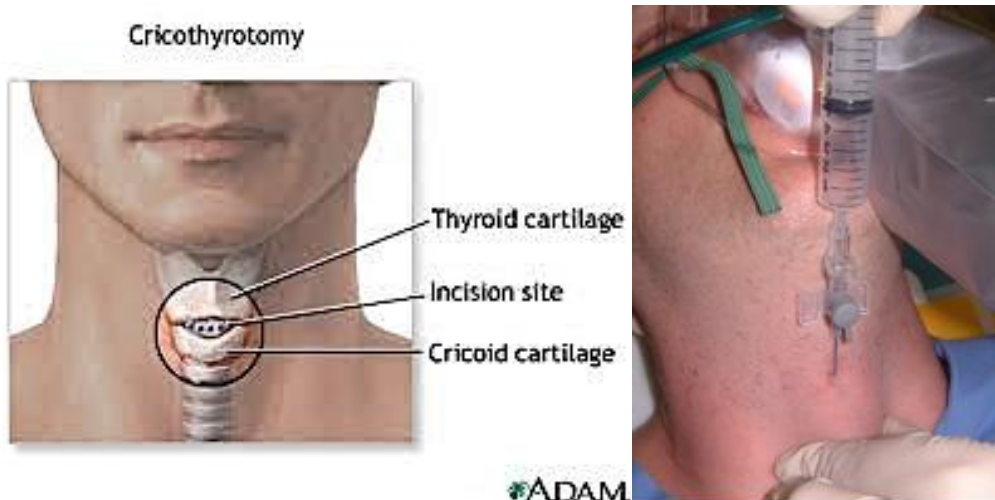
- The KING supraglottic tube that has only one ventilation port.
- Inflation fills up both the supraglottic and esophageal balloons.
- It is possible, but unlikely for the esophageal tip to go into the trachea.
- Always check placement both clinically and with end tidal Co2



VII. SURGICAL AIRWAY

With the addition of supraglottic tubes, the need for a surgical airway has become less likely. However, there are times, such as severe facial trauma and airway obstruction, cricothyroid when a surgical airway is the only alternative. It has and should always be considered an airway of last resort. On the other hand, never second guess yourself for doing it. The alternative to not doing it is a dead patient. The technique is to place a needle through the cricothyroid membrane. In reality, this may be difficult because of swelling or loss of landmarks. If the needle end up going through the trachea and the patient is ventilated, the airway is a success. A 14 gauge needle would suffice but most departments have kits. The technique is the same for all of them.

- After identifying the cricothyroid membrane, insert the needle angling 45 degrees toward the feet.
- Placement is confirmed when air comes back in the syringe.
- If a dilator is used, the guide wire is inserted through the needle, the needle is removed and the dilator is then inserted over the wire so as to enlarge the opening.
- The breathing tube is then placed over the wire and into the trachea.



COMPLICATIONS OF NEEDLE CRICOTHYROTOMY

1. Asphyxia
2. Aspiration
3. Cellulitis
4. Esophageal perforation
5. Exsanguinating hematoma
6. Hematoma
7. Posterior tracheal wall perforation
8. Subcutaneous and/or mediastinal emphysema
9. Thyroid perforation
10. Inadequate ventilations leading to hypoxia and death

VIII. CPR

Changes in CPR have resulted on emphasis on circulation. In part this was done because medical personal would spend all the time on securing the airway and not circulating the blood. Compressions probably cause some passive exchange with the outside by changing the difference between the pressure in the lungs and the atmospheric pressure. Certainly ventilating the patient with a bag valve mask has been found to be as effective as an endotracheal intubation. Applying 100% oxygen non rebreather mask may also turn out be be as effective.

IT IS IMPORTANT IN ALL THESE CASES NOT TO HYPERVENTILATE. Studies have demonstrated that hyperventilation will result in alkalosis and constriction of blood vessels. This constriction can result in poor cerebral perfusion which in turn reduces the amount of oxygen getting to the brain. Our goal now in CPR ventilation is 8-10 breaths per minute.

End-Tidal Carbon Dioxide and Outcome of Out-of-Hospital Cardiac Arrest

Robert L. Levine, M.D., Marvin A. Wayne, M.D., and Charles C. Miller, Ph.D.

N Engl J Med 1997; 337:301-306 [July 31, 1997](#) DOI: 10.1056/NEJM199707313370503

BACKGROUND

Survival after cardiac arrest occurring outside the hospital averages less than 3 percent. Unfortunately, the outcome of prolonged resuscitative attempts cannot be predicted. End-tidal carbon dioxide levels reflect cardiac output during cardiopulmonary resuscitation. We prospectively determined whether death could be predicted by monitoring end-tidal carbon dioxide during resuscitation after cardiac arrest.

METHODS

We performed a prospective observational study in 150 consecutive victims of cardiac arrest outside the hospital who had electrical activity but no pulse. The patients were intubated and evaluated by mainstream end-tidal carbon dioxide monitoring. Our hypothesis was that an end-tidal carbon dioxide level of 10 mm Hg or less after 20 minutes of standard advanced cardiac life support would predict death.

RESULTS

There was no difference in the mean age or initial end-tidal carbon dioxide level between patients who survived to hospital admission (survivors) and those who did not (nonsurvivors). After 20 minutes of advanced cardiac life support, end-tidal carbon dioxide (\pm SD) averaged 4.4 ± 2.9 mm Hg in nonsurvivors and 32.8 ± 7.4 mm Hg in survivors ($P < 0.001$). A 20-minute end-tidal carbon dioxide value of 10 mm Hg or less successfully discriminated between the 35 patients who survived to hospital admission and the 115 nonsurvivors. When a 20-minute end-tidal carbon dioxide value of 10 mm Hg or less was used as a

screening test to predict death, the sensitivity, specificity, positive predictive value, and negative predictive value were all 100 percent.

CONCLUSIONS

An end-tidal carbon dioxide level of 10 mm Hg or less measured 20 minutes after the initiation of advanced cardiac life support accurately predicts death in patients with cardiac arrest associated with electrical activity but no pulse. Cardiopulmonary resuscitation may reasonably be terminated in such patients.

[Resuscitation](#). 2012 Jul;83(7):813-8. doi: 10.1016/j.resuscitation.2012.02.021. Epub 2012 Feb 25.

Factors complicating interpretation of capnography during advanced life support in cardiac arrest--a clinical retrospective study in 575 patients.

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Abstract

BACKGROUND:

End tidal carbon dioxide (ETCO₂) monitoring during advanced life support (ALS) using capnography, is recommended in the latest international guidelines. However, several factors might complicate capnography interpretation during ALS. How the cause of cardiac arrest, initial rhythm, bystander cardiopulmonary resuscitation (CPR) and time impact on the ETCO₂ values are not completely clear. Thus, we wanted to explore this in out-of-hospital cardiac arrested (OHCA) patients.

METHODS:

The study was carried out by the Emergency Medical Service of Haukeland University Hospital, Bergen, Norway. All non-traumatic OHCA treated by our service between January 2004 and December 2009 were included. Capnography was routinely used in the study, and these data were retrospectively reviewed together with Utstein data and other clinical information.

RESULTS:

Our service treated 918 OHCA patients, and capnography data were present in 575 patients. Capnography distinguished well between patients with or without return of spontaneous circulation (ROSC) for any initial rhythm and cause of the arrest ($p < 0.001$). Cardiac arrests with a respiratory cause had significantly higher levels of ETCO₂ compared to primary cardiac causes ($p < 0.001$). Bystander CPR affected ETCO₂-recordings, and the ETCO₂ levels declined with time.

CONCLUSIONS:

Capnography is a useful tool to optimise and individualise ALS in cardiac arrested patients. Confounding factors including cause of cardiac arrest, initial rhythm, bystander CPR and time from cardiac arrest until quantitative capnography had an impact on the ETCO₂ values, thereby complicating and limiting prognostic interpretation of capnography during ALS.

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