Reading

CEPCP

Professional Development:

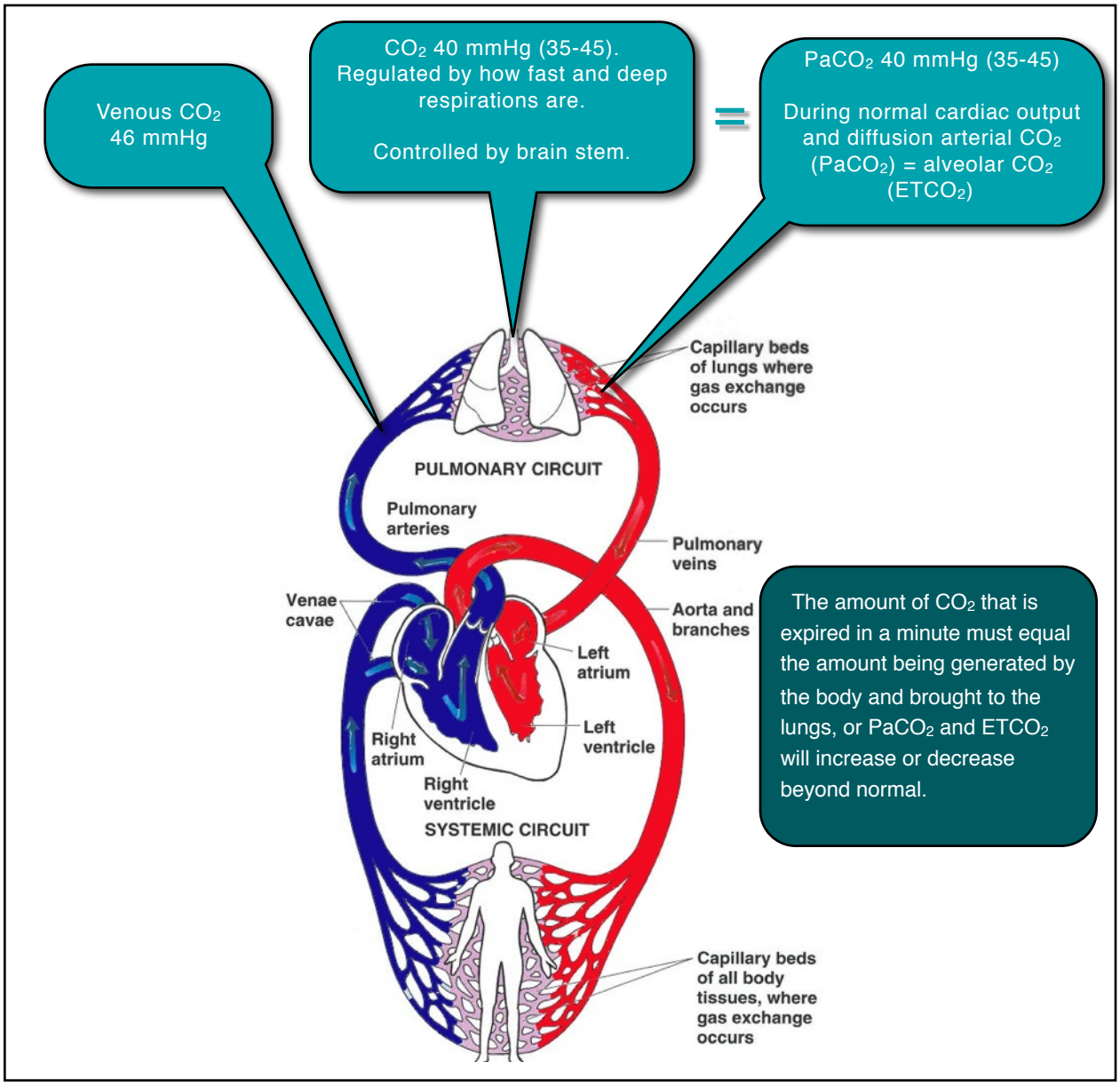
ETCO$_2$ Monitoring

The Virtues of $ETCO_2$

Imagine for a moment that there was a device in your ambulance that had magical powers. The device would take minimal space, would require minimal time to attach to a patient but would be able to give you unprecedented insights into the status and severity of your patient's condition. Imagine it could tell you, within minutes of a cardiac arrest, whether or not the patient will survive. Imagine it could tell you how severe your septic patient is. Imagine it could provide you with a 'picture' of your patient's bronchospasm, giving you an objective before and after snapshot when treating with ventolin. Imagine it could alert you to the often deadly respiratory depression that can occur after midazolam administration, way before the SpO_2 monitor reacts. Imagine also that it could help you detect the often missed pulmonary embolism.

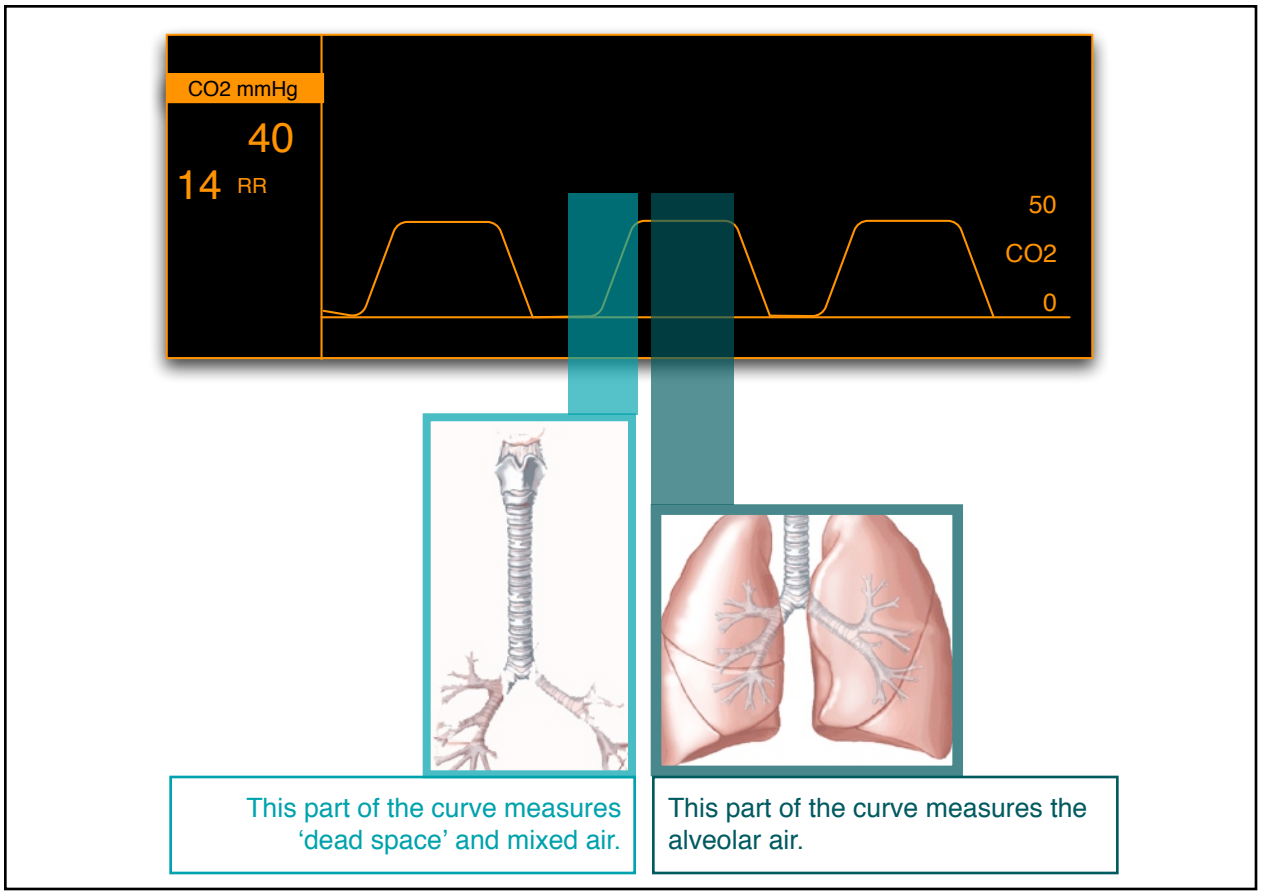
Now stop imagining (before you get too carried away) the device is already in most of the ambulances in the province. Not only is it in the ambulances but it has been considered a gold standard for advanced airway placement confirmation for a long time as it has proven to completely eliminate the issue of unrecognized esophageal intubations. We are speaking, of course, of the $ETCO_2$ detector, capnography, end tidal monitor or whatever you like to call it.

Despite its incredible versatility it has been slow to catch on. It is often forgotten on 'busy' calls. Ironically it is exactly on busy calls that the $ETCO_2$ detector can be most useful. Furthermore, it is often misunderstood as to exactly what information it is providing. That is where this reading package comes in. We will show you how to put the magic to use and get the most out of this incredible piece of equipment.



The Basics

Carbon dioxide is a normal by-product of metabolism throughout all of the tissues in the body. The carbon dioxide is transported in the blood back to the lungs where it diffuses from the fine network of capillaries that surround the alveoli into the alveoli. A normal venous CO_2 level is 46 mmHg. As the de-oxygenated blood enters the capillaries it is able to diffuse with the air inside the alveoli which is normally 40 mmHg. By the time the blood exits the capillaries and leaves the lungs it has had time to reach complete equilibrium with the alveolar air and is thus also 40 mmHg. The breathing rate and depth 'sets' the alveolar CO_2 level. Atmospheric air has only minute amounts of CO_2 and by increasing rate and depth of breathing the alveolar air is refreshed at a higher rate and will remove more CO_2 than what is produced by the body. This will decrease the alveolar CO_2 level. The blood will continue to reach equilibrium with this lower level as it passes through the capillaries, consequently the arterial CO_2 level will decrease. As we can see, the rate and depth of breathing is directly responsible for 'setting' the arterial CO_2 level by



setting the alveolar CO_2 level. The respiratory center in the brain works on this principle and will detect minute changes in arterial CO_2 and adjust the breathing appropriately.

Making Sense of the Waveform

In order for us to measure the alveolar CO_2 level we have to wait until close to the end of an exhalation. At the beginning of exhalation air from the trachea and bronchi exits through the mouth and nose. Since this air has been in 'dead space' and is basically the same as atmospheric air, it will only have tiny levels of CO_2 in it. Only at the end of the exhalation does alveolar air pass through our CO_2 sensor, hence the term 'End-Tidal Carbon Dioxide ($ETCO_2$)'. The way CO_2 exits the upper airways is graphically represented on the end-tidal waveform.

The number (40 in this example) is the level of CO_2 measured right at the end of exhalation. The fall from the plateau indicates inhalation where atmospheric air (containing negligible levels of CO_2) passes through the sensor.

The display also shows the respiratory rate (14 in this example).

When cardiac output (blood flow) is normal:

$ETCO_2$ measures ventilation

When cardiac output is decreased:

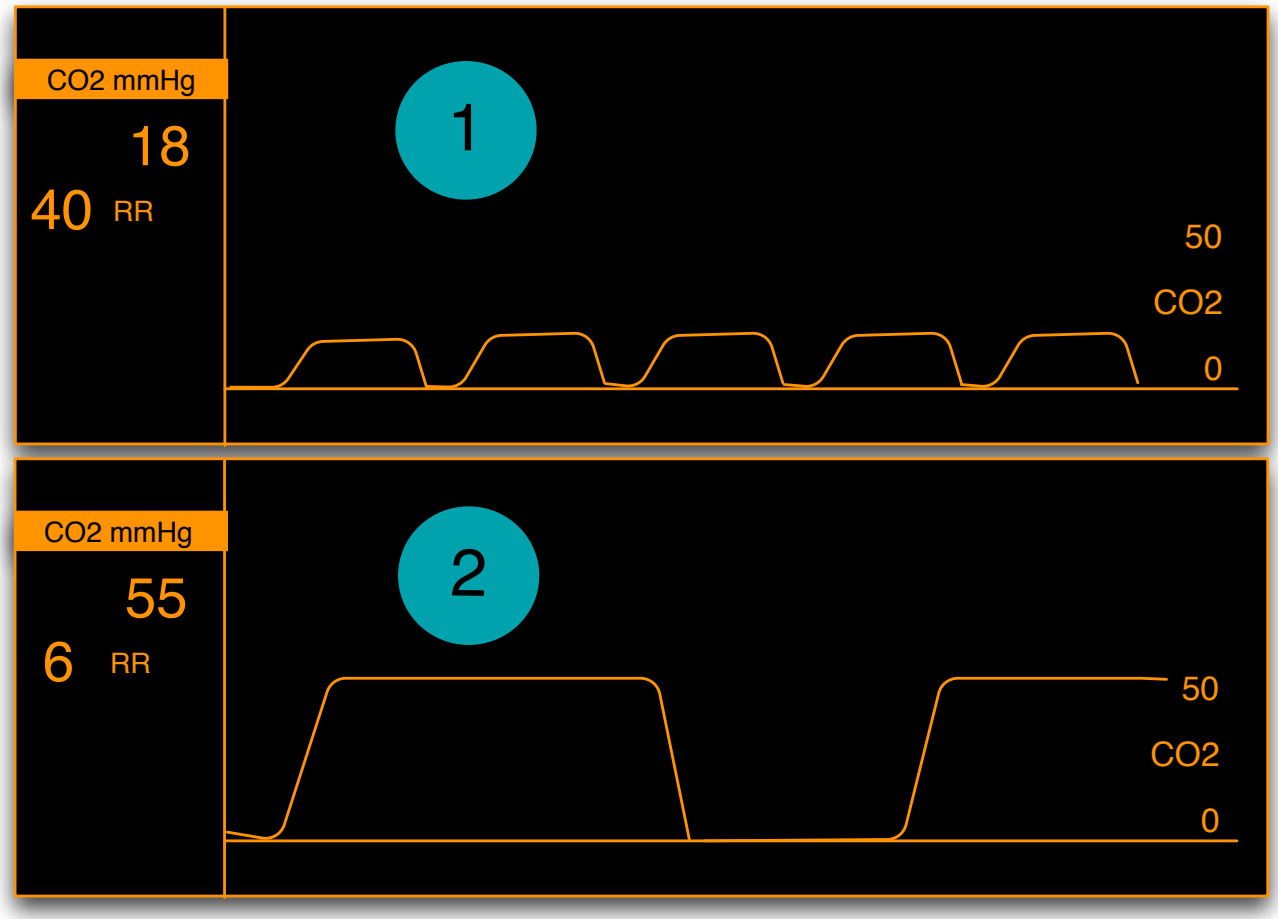
$ETCO_2$ measures Cardiac Output

$ETCO_2$ and its Relation to Cardiac Output and Ventilations

It can be said then that $ETCO_2$ measures the effectiveness of ventilation. And that is true as long as the patient's blood flow (cardiac output) is normal. If the cardiac output falls, less CO_2 is returned to the lungs and if the breathing doesn't change, more CO_2 will be removed than what is brought to the lungs and the $ETCO_2$ will fall (Ornato, Garnett & Glauser 1990; Morton et al. 2007). Consequently if ventilations are kept constant the $ETCO_2$ value can provide a good indication of the cardiac output. Given the extreme case of cardiac arrest, very little metabolism takes place and very little blood flows through the lungs and consequently $ETCO_2$ levels fall dramatically despite little to no ventilation.

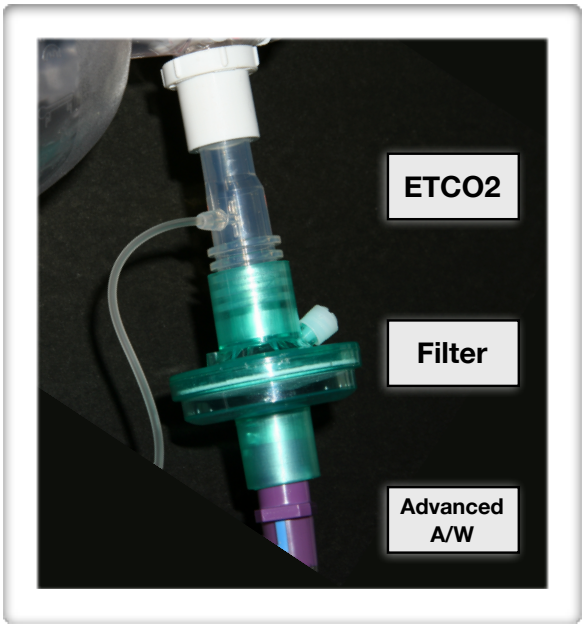
Another consideration is that patients with lung disease may have significant barriers to diffusion between the capillary and the alveoli which will lead to a large difference between $ETCO_2$ and arterial CO_2. In such cases, $ETCO_2$ monitoring can still be useful for observing trends and confirming placement of airway devices etc.

It is important to have a good understanding of these principles when interpreting $ETCO_2$ waveforms and values in the context of patient conditions. Understanding the principles is far more useful than memorizing patterns and values that should be seen with the various patient presentations.



Applying the Knowledge

Let's assume that both of these capnograms (that is what the waveforms are called) come from a patient with normal cardiac output. Applying what has been presented so far, interpret the information and determine if the patient is over- or under-ventilating. What effect would this have on the arterial CO_2 level? Also think about what conditions could cause the abnormality and how it might be treated. The answers can be found at the back of this reading exercise.

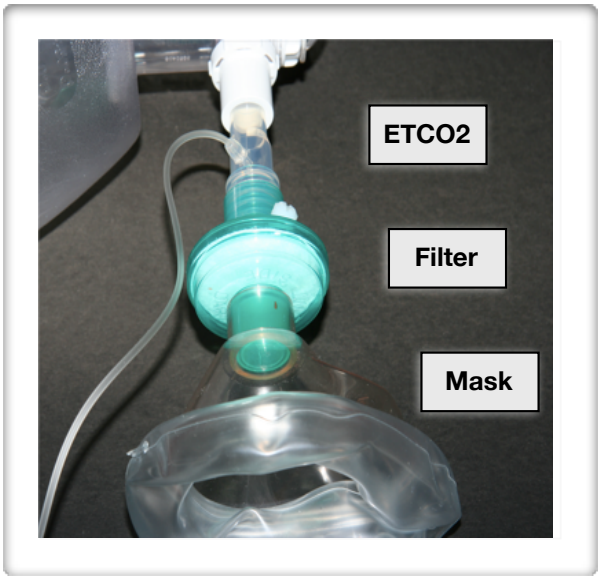


Using the $ETCO_2$ with an Advanced Airway

This is the most typical assembly. A filter is placed directly on the advanced airway. Next the $ETCO_2$ sensor is attached to the BVM (sometimes via a short, flexible extender).

Placing the filter between the airway device and the $ETCO_2$ sensor prevents airway excretions from contaminating the sensor. And protects the paramedic from exposure.

The sensor depicted is the one used with the Medtronic LP12 or LP15. The ZOLL sensor is assembled in exactly the same way.



Using the $ETCO_2$ with a Mask

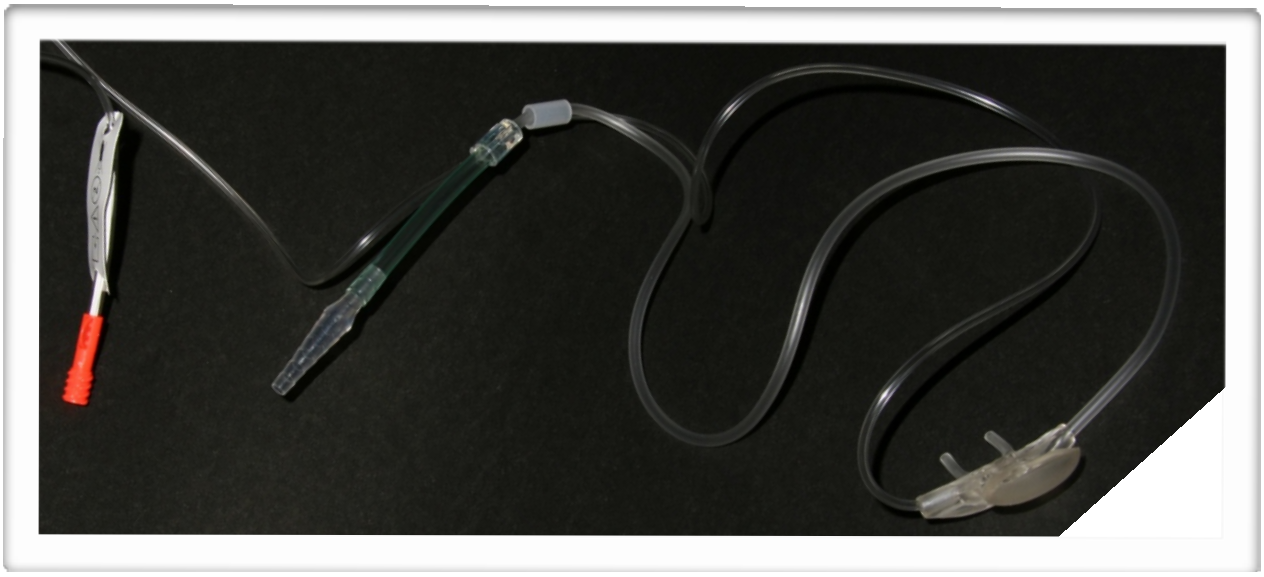
Using the $ETCO_2$ sensor without an advanced airway is not as common but can still provide a great deal of useful information. The assembly is almost identical to that used with an advanced airway. The only difference is that the filter MUST go between the mask and the sensor or the circuit will not fit together.

Using the $ETCO_2$ on a Spontaneously Breathing Patient

Using continuos $ETCO_2$ monitoring on a patient who is spontaneously breathing can be useful in some circumstances (read on to find out more). The Medtronic LP12 and LP15 uses 'sidestream' technology which makes such monitoring possible using the special nasal cannulas pictured below. The orange connector is connected to the $ETCO_2$ port and supplemental oxygen can be attached. as required.

Zoll has sidestream capability but requires a separate module to be purchased (it uses 'mainstream' technology normally). The other option for a 'spot check' of $ETCO_2$ values and waveform shape using the Zoll monitor is to use the same assembly as used when utilizing the device for BVM ventilations but without the BVM attached. The mask will direct all the air through the sensor, and having the patient take a few breaths will give you an idea of the $ETCO_2$ value and waveform shape.

Of course using $ETCO_2$ monitoring with the CPAP circuit is recommended and is assembled in the same way as with the BVM.



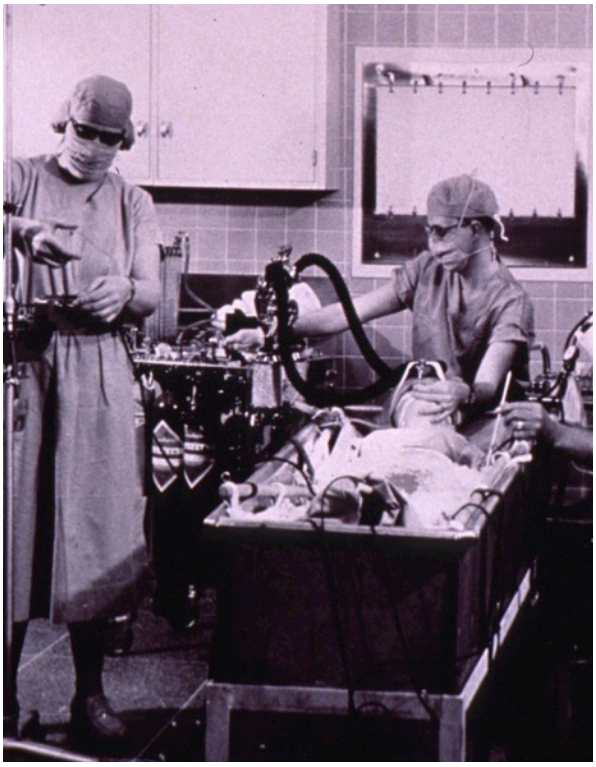
Using $ETCO_2$ Monitoring to Confirm Advanced Airway Placement

When considering the value of using the $ETCO_2$ monitor as tube placement confirmation it might be useful to look to the masters of the art of intubation, the anesthesiologists. Prior to 1999, when $ETCO_2$ monitoring was made mandatory in the operating rooms, there was a high degree of misplaced tubes in the ORs (Silvestri et al. 2005). Clearly a litigation nightmare for the hospitals and doctors, as well as a tragedy for many patients and their families. After $ETCO_2$ monitoring was made mandatory the problem virtually disappeared, a relatively simple solution had been found for a big problem (Silvestri et al. 2005).

To put things in perspective, the best trained intubators with over 1,500 practice intubations during training, working in well lit, well controlled environments, with fully sedated, pre-assessed patients misplaced tubes and did so quite often. These same individuals were able to completely eliminate this issue by the use of $ETCO_2$ monitoring.

It seems strange that we as paramedics with around twenty practice intubations during training, working in all sorts of unfriendly environments, with the most critically ill patients would not utilize this technology with even more gusto.

One prehospital study published in 2005 compared the rate of misplaced endotracheal tubes between those where $ETCO_2$ monitoring was done and those where it was not. The group that had $ETCO_2$ monitoring had zero misplaced tubes. The ones where $ETCO_2$ monitoring had not been used had 23% misplaced tubes (Silvestri et al. 2005). We would like to think we are better at intubating than that and maybe we are, but how many unrecognized misplaced intubations are acceptable when they can be completely eliminated by the use of $ETCO_2$ monitoring? Clearly the usefulness of $ETCO_2$ monitoring



applies to confirming placement of any advanced airway (including the King LT) on patients with or without a pulse.

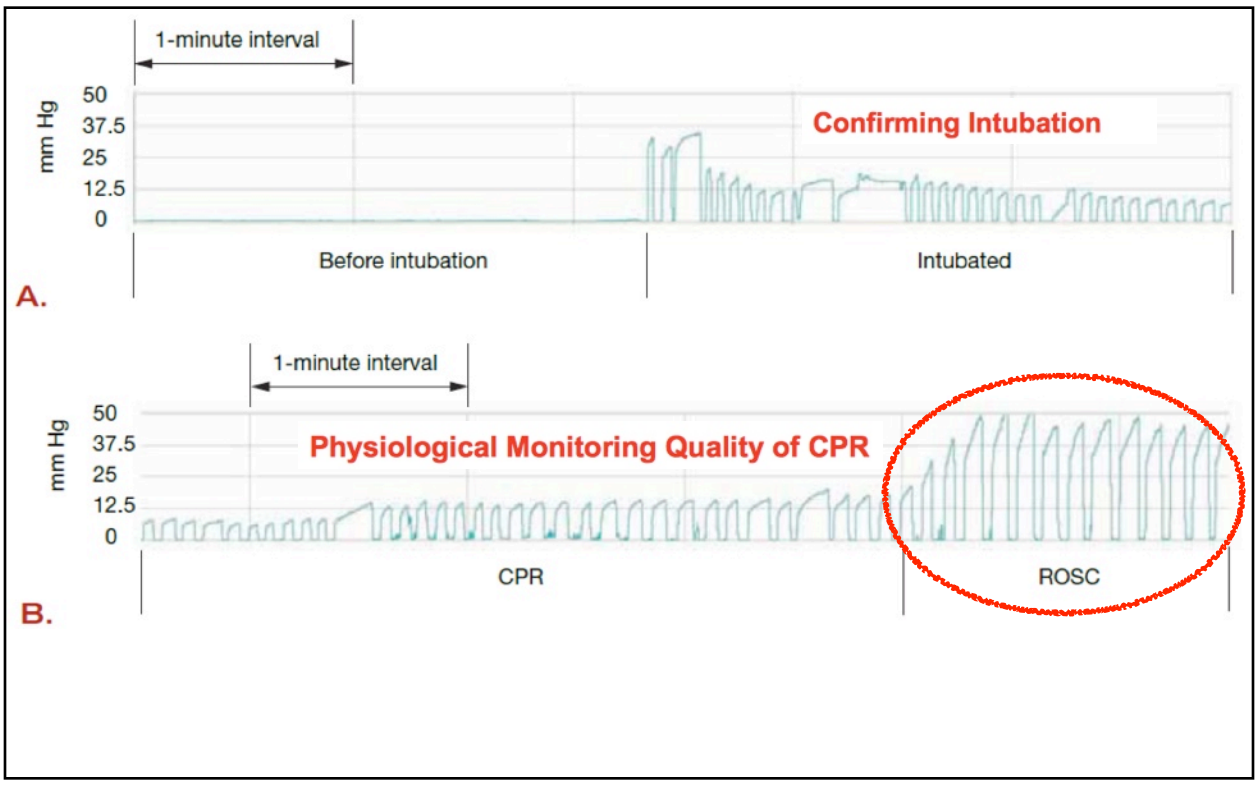
A potential source of error that has received a lot of attention is the potential to get a waveform and $ETCO_2$ value even with an esophageal intubation when a patient has ingested a carbonated beverage just prior to the intubation. Studies have confirmed this potential but have also shown that the values diminish quickly as the carbonation is ventilated off (Garnett, Gervin & Gervin 1989). The $ETCO_2$ waveforms in these cases also do not look normal. In conclusion, the proverbial belly full of beer is not a major source of error when using continuous $ETCO_2$ monitoring with a waveform to confirm tube placement (Garnett, Gervin & Gervin 1989).

Using $ETCO_2$ During a VSA

During a cardiac arrest the $ETCO_2$ monitoring can provide much more than confirmation of tube placement. One use is an accurate prediction of the outcome of the arrest while you are still running it. Several studies have determined that $ETCO_2$ values less than 10 mmHg suggests that the patient will not survive (Asplin & White 1995; Wayne; Levine & Miller 1995; Cantineau et al. 1996). There are a few proposed explanations to this phenomenon. As was explained earlier, when cardiac output is low the $ETCO_2$ level is determined by the cardiac output. If a relatively high $ETCO_2$ can be accomplished by good CPR it can be assumed that the cardiac output is high enough to support a successful resuscitation. If a patient has been VSA for a long time, vasodilation and sluggish blood flow prevents the build-up of significant cardiac output despite good CPR and this would be evident by stubbornly low $ETCO_2$ readings. In an arrest with a shorter down time $ETCO_2$ readings can thus be used to confirm the quality of CPR.

The chart above shows the results of a 1995 study that explored the correlation between average $ETCO_2$ values at 1 and 2 minutes into the arrest, as well as the maximum value reached at any point in the arrest, and their correlation to ROSC. There is clearly a significant correlation between $ETCO_2$ values during the arrest and ROSC (Asplin & White 1995). Another 1995 study found similar results where the average $ETCO_2$ value 20 minutes into the arrest for survivors was 31 mmHg and the average for non-survivors was 4 mmHg (Wayne, Levine & Miller 1995).

Yet another useful quality of $ETCO_2$ monitoring during a VSA is the ability to provide an immediate indication of ROSC without stopping compressions to palpate a pulse. When a patient has a ROSC, large amounts of acidic blood is suddenly returned to the lungs and high amounts of CO_2 diffuses into the alveoli. This flood of CO_2 can be observed as a sharp rise in $ETCO_2$, to levels much higher than normal. If, on the other hand, $ETCO_2$ levels remain the same or rise slightly but remain below normal, it is very unlikely that there has been a return of spontaneous circulation and pulse checks can be kept very brief.



Here is a great example of $ETCO_2$ monitoring during an arrest. Initially it is used to confirm the intubation. Throughout the call it provides the ability to monitor the CPR and finally it shows the return of pulse.

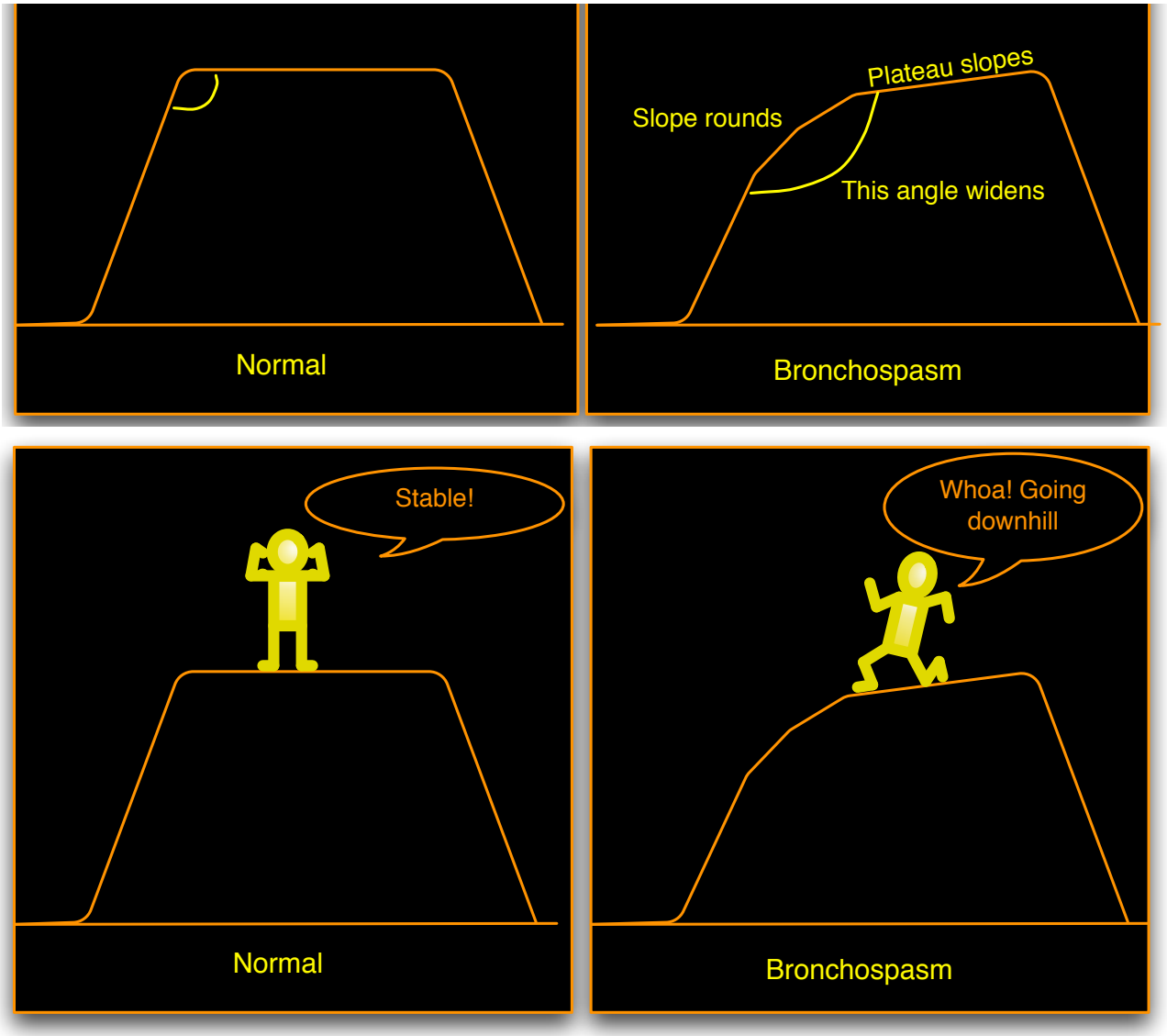
In Summary:

During a cardiac arrest $ETCO_2$ numbers below 10-15 mmHg should prompt a close evaluation of the CPR quality. If the CPR is adequate but the numbers stay low the prognosis for getting a ROSC is very poor. If the numbers rise into the high 20's and 30's the prognosis for ROSC is good. If the values rise to normal (35-45) or above, the patient probably has had a return of pulse so check the rhythm and pulse carefully at the next rhythm check. Keep in mind this monitoring can be accomplished even if no advanced airway is inserted.

Measuring Bronchospasm

Bronchospasm can often be detected by auscultating for wheezing or observing the patients respiratory effort. A forced, prolonged expiration often indicates a lower airway obstruction such as that caused by bronchospasm. However, sometimes those methods are unreliable and measuring whether or not a patient has improved after treatment is often difficult. The use of $ETCO_2$ monitoring will provide an accurate respiratory rate count, which can on its own be very useful for determining if a patient is improving. Furthermore, observing the $ETCO_2$ wave form will reveal bronchospasm to a trained observer (Yaron et al. 1996). Because the emptying of the alveoli is delayed in the person with bronchospasm the rise to the plateau is more gradual and the plateau itself becomes sloped. (Yaron et al. 1996).

In the asthma patient specifically, $ETCO_2$ values can also be used as important prognostic markers. Interestingly the exacerbated asthmatic can present with either abnormally high (> 50 mmHg) or low (< 28 mmHg) values (Lamba et al. 2009). Patients with such abnormal values are more likely to require intubation and/or be admitted to ICU than those with $ETCO_2$ values within the normal range (Lamba et al. 2009).



Determining Severity of Sepsis

This might be a somewhat surprising relationship but $ETCO_2$ values and mortality from sepsis are strongly linked (Hunter et al. 2010). In the case of sepsis, low $ETCO_2$ levels are associated with higher mortality. One recent study from Orlando found that the average $ETCO_2$ level on arrival at the ER of febrile patients who died from sepsis was 26.5 mmHg versus 32.6 for those who survived (Hunter et al. 2010). Exploring exactly why this is the case is beyond the scope of this text but knowing that lower numbers indicate more severe sepsis can certainly be useful.

Detecting Pulmonary Embolisms

When a pulmonary embolism obstructs blood flow to the lungs less CO_2 is delivered to the alveoli and consequently $ETCO_2$ levels decrease below normal levels. Several studies have confirmed that $ETCO_2$ monitoring can be used to detect pulmonary embolisms with good accuracy (Rumpf, Krizmaric & Grmec 2009; Smelt et al 1987). $ETCO_2$ values less than 28 mmHg along with other signs and/or risk factors of pulmonary embolisms make the diagnosis very likely (Rumpf, Krizmaric & Grmec 2009).

Predicting Trauma Mortality

The ability of $ETCO_2$ monitoring to provide a relatively accurate indication of cardiac output, given a decreased cardiac output and normal ventilation, also makes it a useful monitoring tool in the severely injured trauma patient. Using the $ETCO_2$ value to guide ventilations for these patients is not a good idea though since the $ETCO_2$ and arterial CO_2 levels do not correlate well (see illustrations on the following page) (Warner et al. 2009).

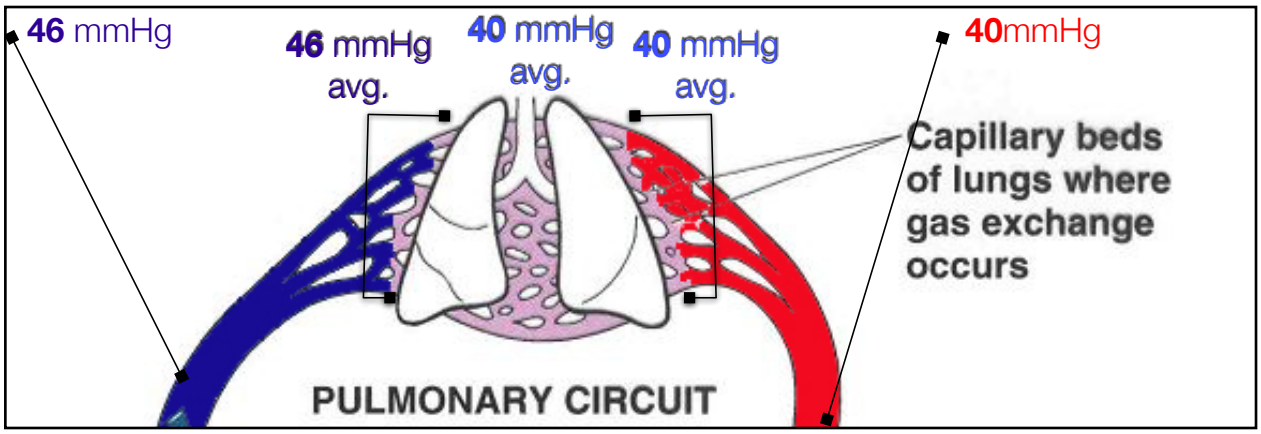
The lower $ETCO_2$ readings are (as eluded to earlier) not caused by a lower CO_2 level in the blood but rather less blood flow to the lungs . If a Paramedic slowed the ventilations in an effort to increase the $ETCO_2$ levels to normal, the arterial CO_2 level would become elevated, leaving the patient acidic. $ETCO_2$ monitoring is better used as a predictive tool in these patients. As with the septic patient, a low number is a bad sign. One prehospital study from London England published in 2004 found that the average initial $ETCO_2$ reading on intubated trauma patients was 31 mmHg for the patients who ultimately survived but only 26mmHg for those who died (Deakin et al. 2004). Furthermore, only 5% of those with an initial reading less that 24 mmHg survived (Deakin et al. 2004). That leaves us with two important take-home points to consider when monitoring $ETCO_2$ in a severe trauma patient; do not adjust ventilations to 'normalize' $ETCO_2$ readings and stay alert for low $ETCO_2$ readings.

In Summary:

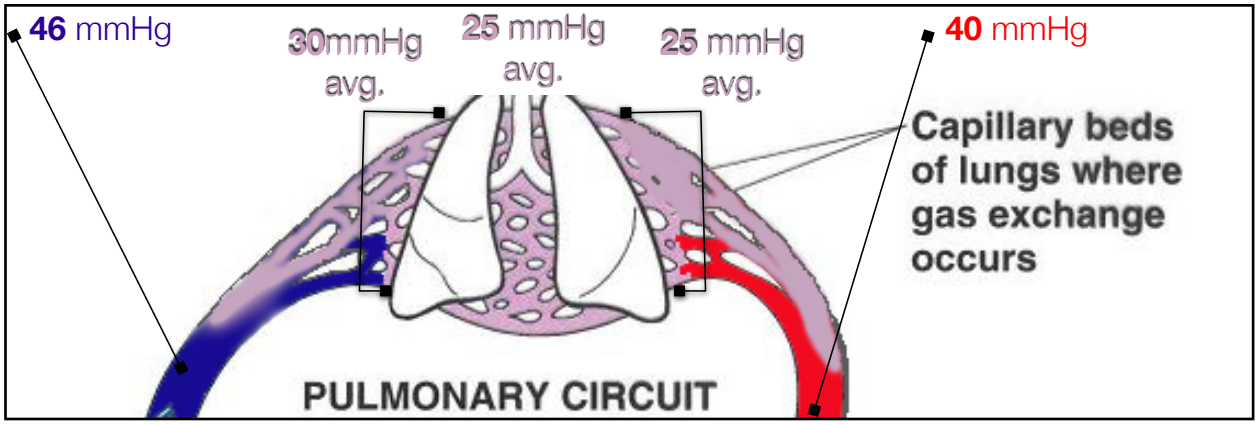
When patients are seriously ill or injured a low $ETCO_2$ reading is a bad sign.

The $ETCO_2$ reading is often much lower than the blood CO_2 level in patient who are seriously ill or injured.

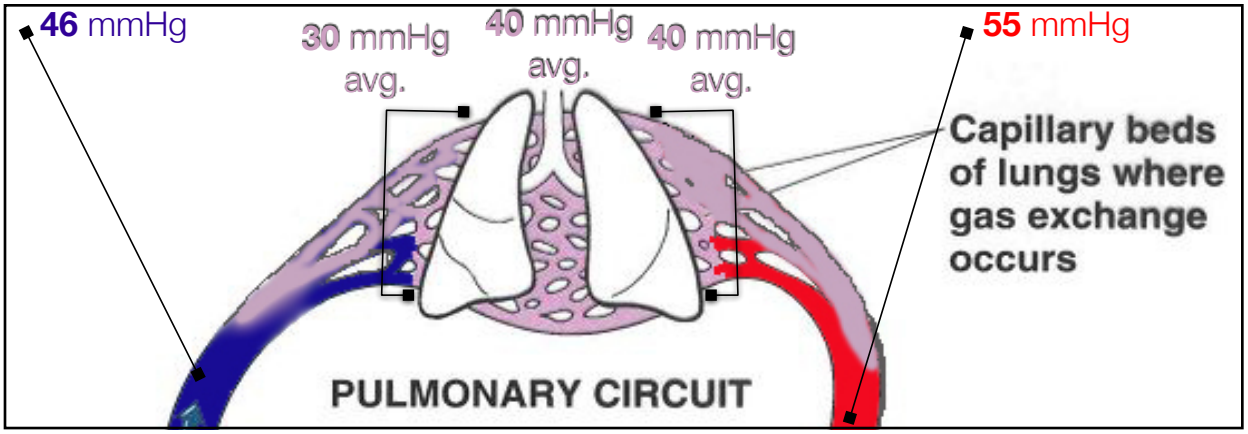
Do not attempt to normalize the $ETCO_2$ in a seriously ill or injured patient by slowing the ventilatory rate below normal.



Normal ventilation, normal circulation. blood enters the lung with an average CO_2 level of 46 mmHg. The alveolar air is regulated to an average of 40 mmHg by the brain stem's regulation of breathing. The blood undergoes diffusion so the blood matches the alveolar air as it leaves the lungs.



Normal ventilation, decreased circulation. The blood that enters the lungs is still 46 mmHg. Because the lungs are under-filled, an averaging of the entire pulmonary capillary bed would reveal a much lower CO_2 level which becomes reflected in the alveolar air ($ETCO_2$). However, as the blood exits the lungs the concentrated blood $ETCO_2$ level is still normal.



Compensated ventilation, decreased circulation. This is the same situation as above only the ventilations have been slowed down to 'normalize' the $ETCO_2$ value by a well meaning provider. Note the elevated CO_2 as the blood leaves the lungs though!

Monitoring Ventilations

Another great role for $ETCO_2$ monitoring is the breath by breath monitoring of an at-risk patient's ventilation status. $ETCO_2$ monitoring can detect abnormalities in a patient's breathing and alert a provider to issues way before the SPO_2 will detect an issue. This type of monitoring is useful for patient's who have overdosed and is imperative when the patient is given sedatives or high doses of analgesic by the medic. During sedation or under the influence of opioids a patient risks developing respiratory depression, failure or complete apnea. One study found that patients given sedation with fentanyl and midazolam had an average $ETCO_2$ increase of 7 mmHg (Krauss & Hess 2007).

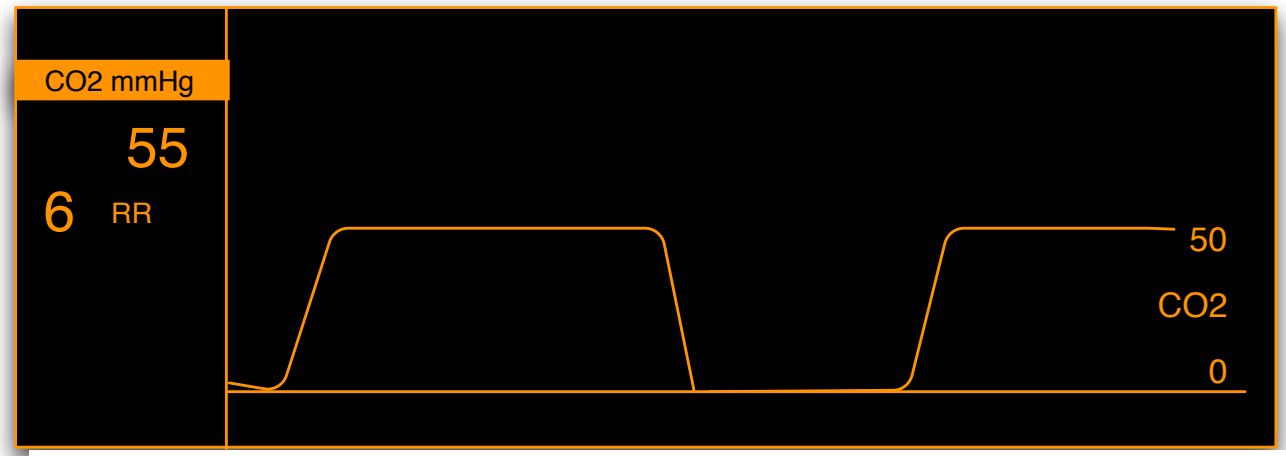
$ETCO_2$ values exceeding 50 mmHg is considered respiratory depression, greater than 70 mmHg is categorized as respiratory failure and at values greater than 80 mmHg the patient is at significant risk of developing complete apnea (Krauss & Hess 2007). A paramedic monitoring a patient can easily detect the development of respiratory depression and intervene before it further deteriorates to respiratory failure by using $ETCO_2$ monitoring.

Two types of abnormal patterns are critical for the medic to recognize and understand when monitoring a patient for breathing abnormalities; slow breathing and shallow breathing.

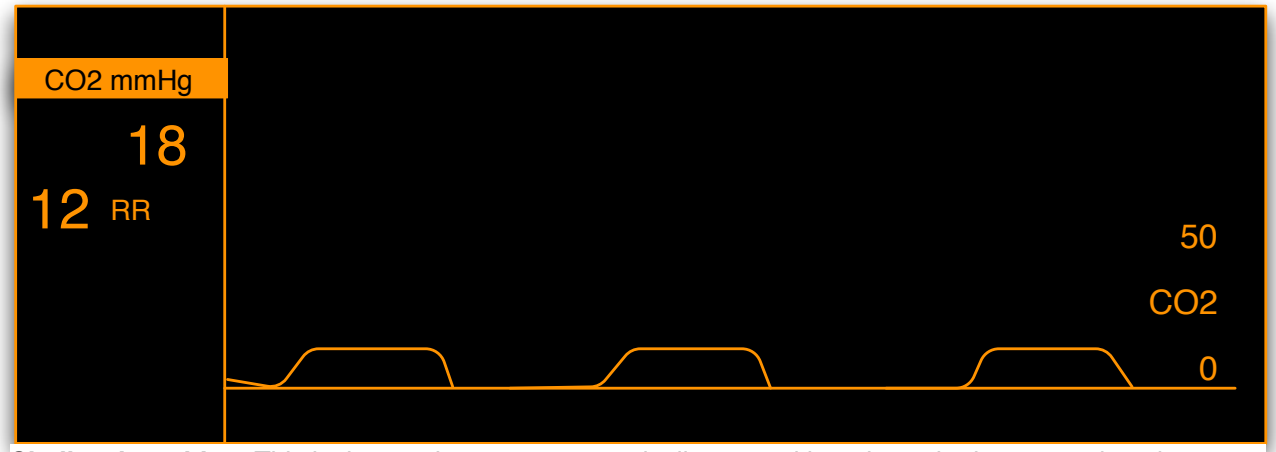
Slow breathing is often seen when a patient has overdosed on opioids and is characterized by a slowing of the respiratory rate. In some cases the rate gets extremely slow or even stops all together for periods of time. The slow rate causes CO_2 to accumulate in the alveoli and consequently the blood leaving the lung has the corresponding elevated level of CO_2($PaCO_2$). The depth of breathing usually doesn't change much so when the breath occurs the high CO_2 level is reflected in the expired alveolar air. The result is a slow, wide, large waveform with a high $ETCO_2$ value (Krauss & Hess 2007).

In cases of shallow breathing the respiratory rate may not fall as much as the depth. The shallow breathing means that mostly the upper airways (dead space) are being ventilated. The alveolar air is stagnant, accumulating ever higher levels of CO_2. Because this air is not exhaled, the air that passes through the CO_2 sensor has low levels of CO_2. Consequently, the alveoli and the blood leaving the lungs is teeming with CO_2 while the $ETCO_2$ is showing a lower than normal value. In cases of shallow breathing an $ETCO_2$ value less than 30 mmHg is considered respiratory depression (Krauss & Hess 2007). This type of respiratory depression is commonly seen with sedative-hypnotic drugs as well as with midazolam and diazepam and happens in roughly half of the cases of sedation-induced respiratory depression (Krauss & Hess 2007).

In many cases of sedation induced respiratory depression the $ETCO_2$ values changed well before the SPO_2, alerting clinicians to a potential developing problem (Krauss & Hess 2007).



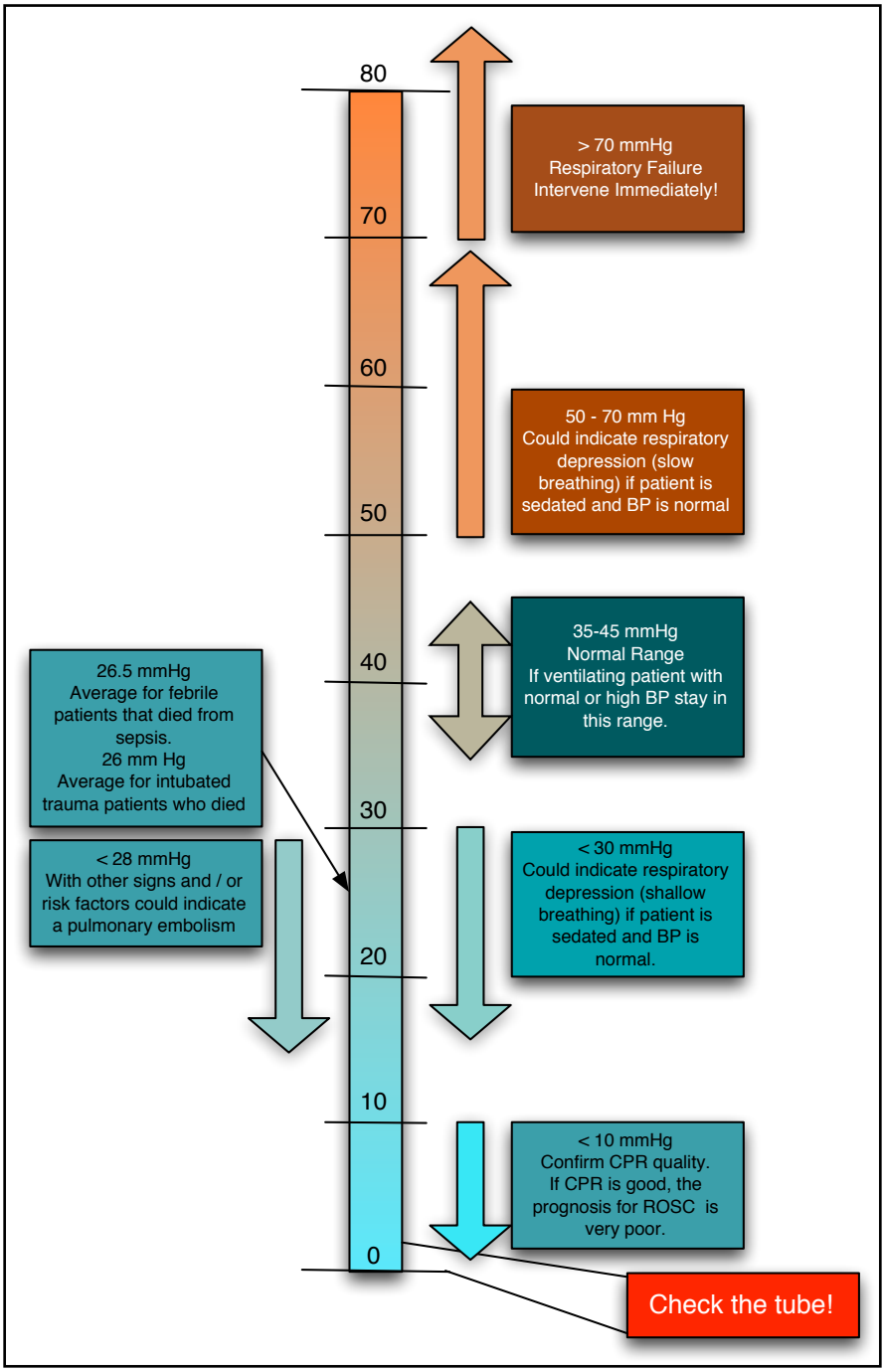
Slow breathing. This is the respiratory pattern typically seen with patient who have overdosed on opioids. The respirations are slow and deep, and the ETCO2 level becomes elevated. The arterial CO2 level is the same as the ETCO2 value since the lungs are well ventilated, just not frequently enough.



Shallow breathing. This is the respiratory pattern typically seen with patient who have overdosed on benzodiazapines or other sedative hypnotics. The respirations are slow and shallow. Because mostly dead space is being ventilated the ETCO2 value is low. However, this low value is not reflective of the deeper alveolar air and the arterial CO2 level which becomes dangerously elevated.

In closing

Hopefully this reading has provided insight into how versatile $ETCO_2$ monitoring can be. Clearly there is a lot more to learn about the information this device can deliver. The purpose of this reading was simply to establish the basic principles and provide a brief overview of what information can be gleaned. Hopefully the $ETCO_2$ will work its way into your pre-hospital assessment arsenal and become as natural as getting a blood pressure reading.



Reference List

Ornato, JP, Garnett AR & Glauser, FL 1990 'Relationship Between Cardiac Output and the End-Tidal Carbon Dioxide Tension', *Annals of Emergency Medicine*, vol. 19, no. 10, pp. 1104-06

Morton, MJ, McManus, Jr. JG, Ryan, KL, Rickards, CA, Cooke, WH & Convertino, VA 2007 'End-Tidal CO2 Correlates With, But May Not be an Early Predictor of, Central Hypovolemia in Humans', *Annals of Emergency Medicine*, vol. 50, no. 3, p. S63

Silvestri, S, Ralls, GA, Krauss, B, Thundiyil, J, Rothrock, SG, Senn, A, Carter, E & Falk, J 2005 'The Effectiveness of Out-of-Hospital Use of Continuous End-Tidal Carbon Dioxide Monitoring on the Rate of Unrecognized Misplaced Intubation Within a Regional Emergency Medical Services System', *Annals of Emergency Medicine*, vol. 45, no. 5, pp. 497-503

Garnett AR, Gervin, CA & Gervin, AS 1989 'Capnographic Waveforms in Esophageal Intubation: Effect of Carbonated Beverages', *Annals of Emergency Medicine*, vol. 18, no. 4, pp. 387-390

Asplin, BR & White, RD 1995 'Prognostic Value of End Tidal Carbon Dioxide Pressures During Out-of-Hospital Cardiac Arrest', *Annals of Emergency Medicine*, vol. 25, no. 6, pp. 756-761

Wayne, MA, Levine, RL & Miller, CC 1995 'Use of End-Tidal Carbon Dioxide to Predict Outcome in Prehospital Cardiac Arrest', *Annals of Emergency Medicine*, vol. 25, no. 6, pp. 762-767

Cantineau, JP, Lambert, Y, Merckx, P, Reynaud, P, Porte, F, Bertrand, C & Duvaldestin, P 1996 'End-tidal carbon dioxide during cardiopulmonary resuscitation in humans presenting mostly with asystole: A predictor of outcome', *Critical Care Medicine*, vol. 24, no. 5, pp. 791-796

Yaron, M, Padyk, P, Hutsinpiller, M & Cairns, CB 1996 'Utility of the Expiratory Capnogram in the Assessment of Bronchospasm', *Annals of Emergency Medicine,* vol. 28, no. 4, pp. 403-407

Lamba, S, Gluckman, W, Nagurka, R, Rosania, A, Bechmann, S, Langley, DJ, Scott, S & Compton, S 2009 'Initial Out-of-Hospital End-Tidal Carbon Dioxide Measurements in Adult Asthmatic Patients', *Annals of Emergency Medicine*, vol. 54, no. 3, p. S51

Rumpf TH, Krizmaric, M & Grmec, S 2009 'Capnometry in suspected pulmonary embolism with positive D-dimer in the field', *Critical Care*, vol. 13, no. 6,

Warner KJ, Cuschieri J, Garland, B, Carlbom, D, Baker, D, Copass, MK, Jurkovich, GJ & Bulger EM 2009 'The Utility of Early End-Tidal Capnography in Monitoring Ventilation Status After Severe Injury', The *Journal of Trauma Injury, Infection and Critical Care*, no. 66, pp. 26-31

Hunter, CL, Silvestri, S, Dean, M, Falk, J & Papa, L 2010 'End-Tidal Carbon Dioxide Levels Are Associated With Mortality In Emergency Department Patients With Suspected Sepsis', *Annals of Emergency Care*, no. 56, no. 3, p. S151

Deakin, CD, Sado, DM, Coats, TJ, & Devries Gareth 2004 'Prehospital End-Tidal Carbon Dioxide Concentration and Outcome in Major Trauma', *The Journal of Trauma Injury, Infection and Critical Care*, vol. 57, no. 1, pp. 65-68

Krauss, B & Hess DR 2007 'Capnography for Procedural Sedation and Analgesia in the Emergency Department', *Annals of Emergency Medicine*, vol. 50, no. 2, pp. 172 - 181

Smelt, WLH, DeLange, JJ, Baerts, WDM & Booij, LHDJ 1987 'The Capnograph, a Reliable Non-invasive Monitor for the Detection of Pulmonary Embolism of Various Origin', *Acta Aneasthesiologica Belgica*, vol. 38, no. 3, pp. 217-224

Answers from Page 5:

1:

Hyperventilation. The respiratory rate is fast and the $ETCO_2$ value is low. The patient is 'blowing off' CO_2. This would result in a lower arterial CO_2 level as well. This might be caused from anxiety but other causes should be explored as well. A metabolic acidosis may cause the respiratory system to compensate by dropping the CO_2 level and thus normalizing the pH. The treatment will be directed at coaching breathing in the setting of anxiety or treatment of underlying causes if caused by anything else.

2:

Hypoventilation. The respiratory rate is slow and the $ETCO_2$ value is high. The breathing is too slow to adequately ventilate the CO_2 brought to the lungs. The CO_2 is essentially accumulating in the alveoli faster than it can be removed. This would cause the arterial CO_2 level to increase as well leading to acidosis. This may be caused by opioid overdoses, head injuries or anything else that directly affects the respiratory center. The treatment goal is to increase the depth and rate of respirations to ventilate the excess CO_2.