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\textsubscript{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\textsubscript{2} level. Atmospheric air has only minute amounts of CO\textsubscript{2} and by increasing rate and depth of breathing the alveolar air is refreshed at a higher rate and will remove more CO\textsubscript{2} than what is produced by the body. This will decrease the alveolar CO\textsubscript{2} level. The blood will continue to reach equilibrium with this lower level as it passes through the capillaries, consequently the arterial CO\textsubscript{2} level will decrease. As we can see, the rate and depth of breathing is directly responsible for ‘setting’ the arterial CO\textsubscript{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:

ETCO₂ measures ventilation

When cardiac output is decreased:

ETCO₂ measures Cardiac Output

ETCO₂ and its Relation to Cardiac Output and Ventilations

It can be said then that ETCO₂ 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 CO₂ is returned to the lungs and if the breathing doesn’t change, more CO₂ will be removed than what is brought to the lungs and the ETCO₂ will fall (Ornato, Garnett & Glauser 1990; Morton et al. 2007). Consequently if ventilations are kept constant the ETCO₂ 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 ETCO₂ 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 ETCO₂ and arterial CO₂. In such cases, ETCO₂ 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 ETCO₂ 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 CO₂ 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 continuous 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 ETCO₂ During a VSA

During a cardiac arrest the ETCO₂ 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 ETCO₂ 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 ETCO₂ level is determined by the cardiac output. If a relatively high ETCO₂ 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 ETCO₂ readings. In an arrest with a shorter down time ETCO₂ 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 ETCO₂ 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 ETCO₂ values during the arrest and ROSC (Asplin & White 1995). Another 1995 study found similar results where the average ETCO₂ 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 ETCO₂ 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 CO₂ diffuses into the alveoli. This flood of CO₂ can be observed as a sharp rise in ETCO₂ to levels much higher than normal. If, on the other hand, ETCO₂ 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.
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 patient’s 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$_2$ waveform 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\textsubscript{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\textsubscript{2} level which becomes reflected in the alveolar air (ETCO\textsubscript{2}). However, as the blood exits the lungs the concentrated blood ETCO\textsubscript{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\textsubscript{2} value by a well meaning provider. Note the elevated CO\textsubscript{2} as the blood leaves the lungs though!
Monitoring Ventilations

Another great role for ETCO\textsubscript{2} monitoring is the breath by breath monitoring of an at-risk patient’s ventilation status. ETCO\textsubscript{2} monitoring can detect abnormalities in a patient’s breathing and alert a provider to issues way before the SPO\textsubscript{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\textsubscript{2} increase of 7 mmHg (Krauss & Hess 2007).

ETCO\textsubscript{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\textsubscript{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\textsubscript{2} to accumulate in the alveoli and consequently the blood leaving the lung has the corresponding elevated level of CO\textsubscript{2}(PaCO\textsubscript{2}). The depth of breathing usually doesn’t change much so when the breath occurs the high CO\textsubscript{2} level is reflected in the expired alveolar air. The result is a slow, wide, large waveform with a high ETCO\textsubscript{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\textsubscript{2}. Consequently, the alveoli and the blood leaving the lungs is teeming with CO\textsubscript{2} while the ETCO\textsubscript{2} is showing a lower than normal value. In cases of shallow breathing an ETCO\textsubscript{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\textsubscript{2} values changed well before the SPO\textsubscript{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.

![Blood Pressure Chart]

- **26.5 mmHg**
  Average for febrile patients that died from sepsis. 26 mm Hg Average for intubated trauma patients who died

- **< 28 mmHg**
  With other signs and / or risk factors could indicate a pulmonary embolism

- **< 30 mmHg**
  Could indicate respiratory depression (shallow breathing) if patient is sedated and BP is normal.

- **35-45 mmHg**
  Normal Range
  If ventilating patient with normal or high BP stay in this range.

- **> 70 mmHg**
  Respiratory Failure
  Intervene immediately!

- **< 10 mmHg**
  Confirm CPR quality.
  If CPR is good, the prognosis for ROSC is very poor.

- **50 - 70 mm Hg**
  Could indicate respiratory depression (slow breathing) if patient is sedated and BP is normal.

- **< 30 mmHg**
  Could indicate respiratory depression (shallow breathing) if patient is sedated and BP is normal.

- **< 28 mmHg**
  With other signs and / or risk factors could indicate a pulmonary embolism

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  Could indicate respiratory depression (slow breathing) if patient is sedated and BP is normal.

- **< 30 mmHg**
  Could indicate respiratory depression (shallow breathing) if patient is sedated and BP is normal.

- **< 10 mmHg**
  Confirm CPR quality.
  If CPR is good, the prognosis for ROSC is very poor.

- **Check the tube!**
Reference List

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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


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,


Answers from Page 5:

1:
Hyperventilation. The respiratory rate is fast and the ETCO₂ value is low. The patient is ‘blowing off’ CO₂. This would result in a lower arterial CO₂ 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 CO₂ 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 ETCO₂ value is high. The breathing is too slow to adequately ventilate the CO₂ brought to the lungs. The CO₂ is essentially accumulating in the alveoli faster than it can be removed. This would cause the arterial CO₂ 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 CO₂.