End Tidal Capnography in the Emergency Department

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Capnography is the measurement of the partial pressure of carbon dioxide ($CO_{2)}$ in exhaled air.[1] It provides real-time information on ventilation (elimination of CO_2), perfusion (CO_2 transportation in vasculature), and metabolism (production of CO_2 via cellular metabolism).[2] The technology was originally developed in the 1970s to monitor general anesthesia patients; however, its reach has since broadened, with numerous applications currently in use and in development for the emergency provider (EP).[3]

Capnography exists in two configurations: a mainstream device that attaches directly to the hub of an endotracheal tube (ETT) and a side-stream device that measure levels via nasal or nasal-oral cannula.[1,3]

Qualitative monitors use a colorimetric device that monitors the end-tidal CO_2 ($EtCO_2$) in exhaled gas and changes color depending on the amount of CO_2 present.[2,4] Expired CO_2 and H20 form carbonic acid, causing the specially treated litmus paper inside the device to change from purple to yellow.[2,4] Quantitative monitors display a capnogram, the waveform of expired CO_2 as a function of time; as well as the capnometer, which depicts the numerical $EtCO_2$ for each breath.[4] In this overview, we will discuss the general interpretation of capnography and its specific uses in the ED.

The Capnogram

Just like the various stages of an electrocardiogram represent different phases of the cardiac cycle, different phases of a capnogram correspond to different phases of the respiratory cycle. Knowing how to analyze and interpret each phase will contribute to the utility of capnography. While there has been considerable ambiguity in the terminology related to the capnogram,[5-7] the most frequently referenced capnogram terminology consists of the following phases (**Figure 1**):

Phase I: represents beginning of exhalation, where the dead space is cleared from the upper airway.[2] This should be zero unless the patient is rebreathing CO_2-laden expired gas from either artificially increased dead space or hypoventilation.[2,8] A precipitous rise in both the baseline and $EtCO_2$ may indicate contamination of the sensor, such as with secretions or water vapor.[2,6]

Phase II: rapid rise in exhaled as the CO_2 from the alveoli reaches the sensor.[4] This rise should be steep, particularly when ventilation to perfusion (V/Q) is well matched. More V/Q heterogeneity, such as with COPD or asthma, leads to a more gradual slope.[9] A more gradual phase 2 slope may also indicate a delay in CO_2 delivery to the sampling site, such as with bronchospasm or ETT kinking.[2]

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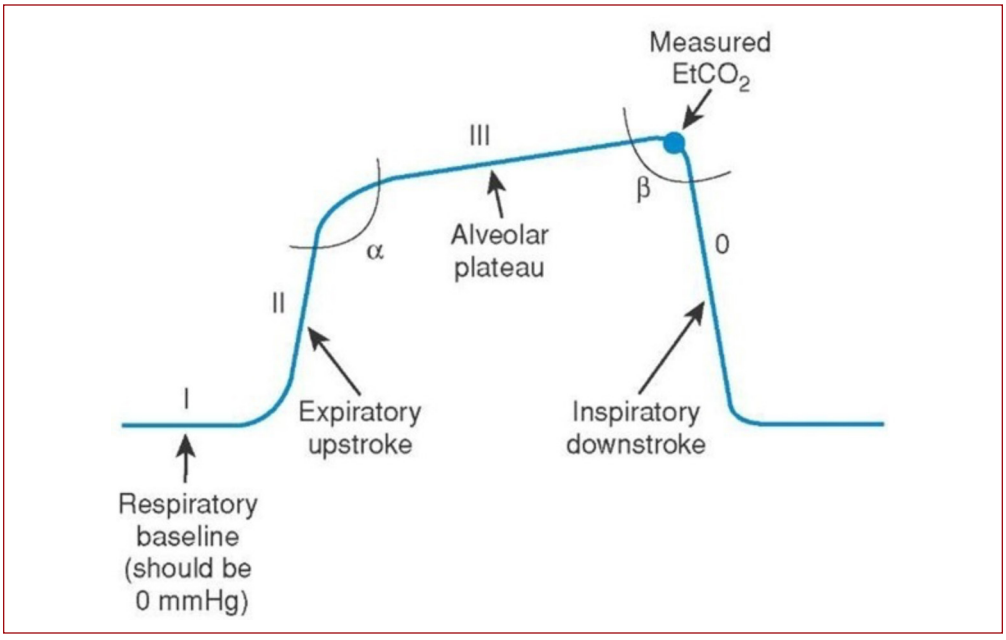

Figure 1. Phases of Capnogram

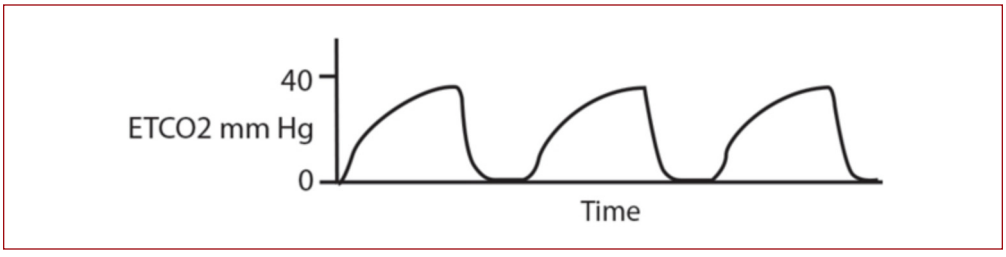

Figure 2. "Shark Fin" Capnogram in Bronchospasm

Phase III: the expiratory plateau, which represents the CO_2 concentration approaching equilibrium from alveoli to nose. The plateau should be nearly horizontal.[2] If all alveoli had the same pCO_2, this plateau would be perfectly flat, but spatial and temporal mismatch in alveolar V/Q ratios result in variable exhaled CO_2. When there is substantial V/Q heterogeneity, the slope of the plateau will increase.[1,2,6]

Phase IV: the initiation of inspiration, which should be a nearly vertical drop to a baseline. If prolonged or bleeding into the expiratory phase, consider a leak in the expiratory portion of the circuit, such as an ETT tube cuff leak.[2]

Phase 0: the inspiratory segment

Another important part of the capnogram is the alpha angle. This is the angle of transition between Phase II and Phase III. The combination of a prolonged phase II and steeper phase III leads to a more obtuse alpha angle and will have a "shark-fin" appearance to the capnogram. This suggests an obstructive process, such as asthma or COPD (**Figure 2**).[1,2,6]

Standard Uses

Intubation

Capnography, along with visualizing ETT placement through the vocal cords, is the standard of care for confirming correct placement during intubation.[4,10,11] Alternative signs of endotracheal intubation, such as chest wall movement, auscultation, condensation of

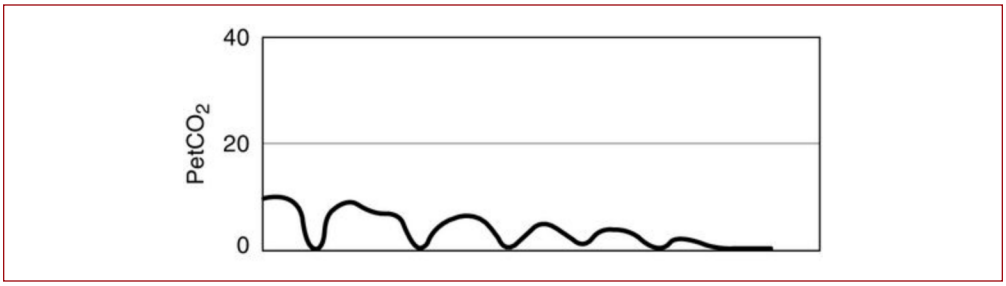

Figure 3. Esophogeal Intubation Capnogram

water vapor in the tube lumen, or pulse oximetry, are less accurate.[12]

While not ideal, correct ETT placement can be confirmed qualitatively using a colorimetric device.[13] Upon correct placement, the resultant exhalation of CO_2 will change the paper color from purple to yellow (indicating $EtCO_2$ values > 15 mm Hg).[2,4] Without this color change, tube placement should be verified to rule out esophageal intubation. Unfortunately, qualitative capnography has false positives and negatives that limit its utility in the ED, and this method should be avoided if quantitative capnography is available.

With quantitative capnography, obtaining the typical box-waveform on the capnogram reflects endotracheal intubation. In comparison, a flat capnogram is more indicative of an esophageal intubation (**Figure 3**).[10] While other things may cause this waveform, such as technical malfunction or complete airway obstruction distal to the tube, tube placement confirmation to rule out esophageal intubation would be the first step to troubleshooting this waveform. In addition, if the ETT is placed in the hypopharynx above the vocal cords, the waveform may initially appear appropriate but will likely become erratic appearing over time.[10]

Quantitative capnography does have some limitations. For example, a main-stem bronchus intubation would still likely demonstrate normal-appearing capnography, so secondary strategies and a confirmatory chest x-ray are still indicated. False-negative $ETCO_2$ readings can occur in low CO_2 elimination states, such as cardiac arrest, pulmonary embolus, or pulmonary edema, while false-positives can theoretically occur after ingestion of large amounts of carbonated liquids or contamination of the sensor with stomach contents or acidic drugs.[10] However, many of these misleading results can be caught by simply checking for an appropriate waveform.

Cardiac Arrest

Capnography has numerous uses in the monitoring, management, and prognostication of intubated patients in cardiac arrest.[1,3,4,10,14] Under normal conditions, $EtCO_2$ is 35-40 mm Hg. While the body still makes CO_2 during cardiac arrest, it will not reach the alveoli without circulating blood.[10] Without CPR, CO_2 accumulates peripherally and won't reach the lungs, causing $EtCO_2$ to approach zero. This means that $EtCO_2$ correlates directly with cardiac output during CPR, as long as ventilation remains constant.

This means the effectiveness of cardiac chest compression can be assessed in intubated patients using $EtCO_2$, with higher values during CPR correlated with increased return of spontaneous circulation (ROSC) and survival.[14-18] Using $EtCO_2$ monitoring during cardiac arrest may improve outcomes,[19] and the American Heart Association (AHA) recommends monitoring capnography during cardiac arrest to assess compression efficacy.[10,20]

$EtCO_2$ >20 mm Hg is considered optimal, while $EtCO_2$ <10-15 mm Hg is considered suboptimal.[4,10,16] In a recent meta-analysis, the average $EtCO_2$ was 13.1 mm Hg in those who did not obtain ROSC, compared to 25.8 mm Hg in those who did.[21] As such, goal $EtCO_2$ for effective compressions may be even higher in future recommendations. If $EtCO_2$ is low, either compression technique should be improved or a different operator should do compressions. Every 1 cm increase in depth will increase $EtCO_2$ by approximately 1.4 mm Hg.[16] Interestingly, compression rate is not a significant predictor of $EtCO_2$ over the dynamic range of chest compression delivery.[16]

An abrupt increase in $EtCO_2$ is an early indicator of ROSC.[10,14-16,22,23] A return of a perfusing rhythm will increase cardiac output. This allows for accumulated peripheral CO_2 to reach the lungs, subsequently causing a rapid rise in $EtCO_2$.[24] It is important to note that when it comes to evaluating for ROSC, the actual numbers are less important than the change from pre- to post-ROSC. Providers should look for a jump of at least 10 mm Hg on capnometry.[4] Nevertheless, an abrupt rise in $EtCO_2$ is a non-sensitive marker for ROSC (33%, 95% CI 22-47% in one multicenter cross-sectional study), meaning that the lack of an abrupt rise of $EtCO_2$ may not necessarily mean a lack of ROSC.[23]

The $EtCO_2$ level may help guide decision-making in assessing whether continued resuscitation in cardiac arrest is futile. Values <10 mm Hg after 20 minutes of active resuscitation have consistently demonstrated minimal chance of survival.[17,25,26] In one study, an $EtCO_2$ of <10 mm Hg at 20 minutes had a sensitivity, specificity, PPV, and NPV of 100% for death in PEA arrest.17 However, determination of the specific $EtCO_2$ cutoff and the timing is still an area of research with a final consensus pending.[17,18,25-30] One recent study suggested that even 3 min with $EtCO_2$ <10 mm Hg could be an appropriate cutoff to cease resuscitation efforts.[27]

Unfortunately, there is a large amount of heterogeneity in the available literature using capnography to assess for ROSC and in guiding resuscitation efforts. $EtCO_2$ should not be used as the only factor in the determination to cease resuscitation. In addition, the AHA recommends that $EtCO_2$ for prognostication should be limited to intubated patients only.[20]

It is important to note that while cardiac output is the largest factor for $EtCO_2$ in arrest, other physiologic and iatrogenic causes may affect $EtCO_2$ during resuscitation. For example, there is considerable variation in $EtCO_2$ with changes in ventilation rate.[4] Measured CO_2 may be significantly lower with manual instead of mechanical ventilation, likely due to over-ventilation that not only reduces alveolar CO_2 but also causes excess intrathoracic pressure, reducing venous return.[21] For these reasons, use caution when using $EtCO_2$ during manual ventilation of an intubated patient in cardiac arrest. In addition, administration of epinephrine may cause a small decrease in $EtCO_2$, although the effect may vary for each individual.[10,31] Sodium bicarbonate can also cause a transient increase in CO_2 due to its conversion into CO_2 and H_2O.[10]

Procedural Sedation

Capnography is being used with increasing frequency to monitor patients during procedural sedation; it is now considered standard of care in many settings.[32] Although rare, hypoventilation is a risk of procedural sedation.[33] Typically, respiratory depression during procedural sedation is diagnosed with non-invasive pulse oximetry and visual inspection.[34] However, capnography has been shown to identify respiratory depression, airway obstruction, apnea, and laryngospasm earlier than pulse oximetry, allowing the provider to intervene quicker.[34,35] Unlike pulse oximetry, the capnogram also remains stable during patient motion and is reliable in low-perfusion states.[36]

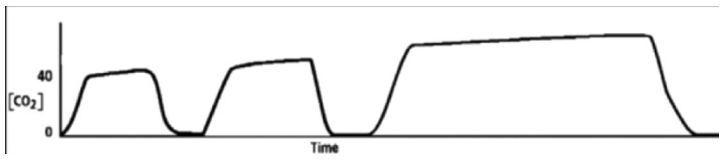

Figure 4. Bradypneic Hypoventilation Capnogram

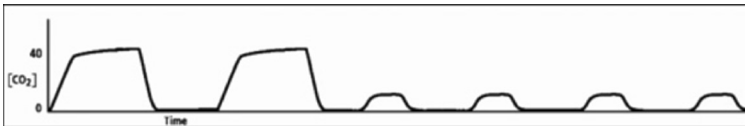

Figure 5. Hypopneic Hypoventilation Capnogram

There are two distinct types of hypoventilation detected by capnography. Bradypneic hypoventilation (type 1), which is characterized by a decreased respiratory rate, results in a decreased expiratory time and a subsequent rise in $EtCO_2$.[36] This is depicted on capnography by a high $EtCO_2$ and longer waveform, and is commonly observed after oversedation with opioids (**Figure 4**).[36] In contrast, hypopneic hypoventilation (type 2) occurs with low tidal volumes but a normal respiratory rate.[36] Type 2 is graphically represented by a suddenly lower $ETCO_2$ with otherwise normal waveform and occurs most commonly with sedative-hypnotic drugs (**Figure 5**).[36] Seeing either type during procedural sedation should alert the clinician to assess for airway obstruction, consider supplemental oxygen, cease drug administration or reduce dosing, and consider reversal if appropriate.[36]

There is some debate as to the utility of capnography for procedural sedation. While it is clear that capnography decreases the incidence of hypoxia, some studies suggest that it may not reduce patient-centered outcomes such as adverse respiratory events, neurologic injury, aspiration, or death compared to standard monitoring.[35,37,38] However, pulse oximetry alone can suffer response delay, while $EtCO_2$ can rapidly detect hypoventilation.[39]

Potential Uses/Applications

Respiratory Distress

Capnography can provide dynamic monitoring in patients with acute respiratory distress. Measuring $EtCO_2$ with each breath provides instantaneous feedback on the clinical status of the patient and has numerous specific uses.[1,3,4]

Determining the etiology of respiratory distress in either the obtunded patient or those with multiple comorbidities can be a challenge. Vital sign abnormalities and physical exam findings can overlap in numerous conditions, which may only further obscure the diagnosis. Since different etiologies for respiratory disease require different management modalities, anything that can help clue in to the specific cause can be beneficial. As discussed above, obstructive diseases such as COPD or asthma demonstrate a "shark-fin appearance" on capnogram due to both V/Q heterogeneity and a prolonged expiratory phase due to airway constriction, which will contrast to the typical box-waveform in other conditions (Figure 2).[1,2,6] Some studies have been able differentiate COPD from

congestive heart failure (CHF) by waveform analysis alone, though this was primarily done via computer algorithms.[40] Seeing the shark-fin (or the lack thereof) can help guide management of respiratory distress in conjunction with the remainder of the initial assessment.

Monitoring capnography can help with management and disposition in those with COPD or asthma. During exacerbations, $EtCO_2$ levels may initially drop as the patient hyperventilates to compensate.[1] It is not until ventilation becomes less effective that $EtCO_2$ levels begin to rise. This may occur before hypoxia sets in and can prompt the clinician to escalate ventilation strategies. In addition, the normalization of the "shark-fin" obstructive pattern towards the more typical box-form wave may indicate effective treatment, though more data is needed before it can be recommended.[41] One of the advantages of this technique would be that it is independent of patient effort, unlike peak-flow monitoring.

$EtCO_2$ can be beneficial even before patients get to the ED. In one study, prehospital patients presenting with asthma or COPD who were found to have $EtCO_2$ of >50 mm Hg or <28 mm Hg, representing the upper and lower limits in the study, had greater rates of intubation, critical care admission, and mortality.[42] The patients in this cohort with higher $EtCO_2$ were likely tiring after prolonged hyperventilation and therefore would be more likely to need ventilatory support. Those on the lower end were likely hyperventilating and had not yet tired out. It is important to note that while arrival $EtCO_2$ levels may aid in determining the more critically ill, post-treatment levels were not found to have a statistical difference in determining disposition in patients with asthma or COPD.[43]

Caution is advised when attempting to use $EtCO_2$ to approximate an arterial blood gas CO_2 ($PaCO_2$). While $EtCO_2$ can correlate with $PaCO_2$ within 5 mm Hg in greater than 80% of patients with dyspnea,[44] large discrepancies are common depending on the disease state.[45] In general, the $EtCO_2$ should always be lower than the $PaCO_2$ due to the contribution to the $ETCO_2$ from dead space, which has a low CO_2 content due to lack of perfusion.

Sepsis

$EtCO_2$ may help identify septic patients given its inverse relationship with lactate levels.[46-49] In conditions of poor tissue perfusion, lactate builds up. This begins to make the blood acidotic in the form of newly acquired anions, with a resultant anion gap metabolic acidosis. The body then tries to acutely compensate for this by hyperventilating, resulting in the observed lowering of $EtCO_2$. Since lactate is a predictor of mortality in sepsis,[50] and monitoring lactate clearance to evaluate resuscitation efforts in sepsis is recommended,[51] $EtCO_2$ could play a similar role. One group in particular has demonstrated that, when used with SIRS criteria, abnormally low prehospital $EtCO_2$ levels is predictive of sepsis and inhospital mortality, and is more predictive than SIRS criteria alone.[48,50] That said, $EtCO_2$ was not associated with lactate temporally at 3 and 6 hours,[51] so it should not be used to guide resuscitation like a lactate clearance. It appears that $EtCO_2$ may be helpful for triage in sepsis, but more study is needed to determine the exact role particularly given most of the available research involves multiple studies from one group.[47,48,52]

Diabetic Ketoacidosis

Initial bicarbonate levels and venous pH are associated with low $EtCO_2$ readings in diabetic ketoacidosis (DKA).[54,55] This could have many practical uses, in particular for patients presenting with hyperglycemia to rule out DKA. One study demonstrated that a blood glucose >250 mg/dL and capnography of >24.5 mm Hg had 90% sensitivity for

excluding DKA.[55] A value of 35 mm Hg or greater demonstrated 100% sensitivity for excluding DKA in patients with initial glucose >550 mg/dL,[56] though this blood glucose is not practical, as this excludes many patients the EP would seek to rule out DKA (recall that blood glucose only has to be >250 mg/dL for the diagnosis). Smaller studies focused on the pediatric population found a 100% sensitivity marker for DKA varied from >30 to >36 mm Hg.[57,58] Clearly a role exists, but no study has demonstrated sufficient sensitivity for ruling out DKA with $EtCO_2$ and blood glucose alone within the framework of clinically relevant values.

Trauma

As described above, low $EtCO_2$ is inversely correlated with lactate.[46] Because of this, it could theoretically be a marker of hypoperfusion in trauma. Initial $EtCO_2$ values <25 mm Hg have been associated with mortality and hemorrhage in intubated trauma patients,[59] as well as mortality prior to discharge in nonintubated trauma patients.[60] However, it did not demonstrate added clinical utility when combined with Glasgow Coma Scale (GCS) score, systolic blood pressure, and age in predicting severe injury.[61]

Pulmonary Embolism

A pulmonary embolism (PE) causes a blockage in blood flow to alveoli, which results in a decrease in CO_2 transportation to the alveoli and thus lower $EtCO_2$, while also widening the gradient between $PaCO_2$ and $EtCO_2$.[37] Because of this, it has a theoretical role in the diagnosis of PE, though numerous studies have demonstrated that $EtCO_2$ alone is not sensitive nor specific enough for this role.[62-66] In a recent meta-analysis, a pretest probability of 10% could lead to a posttest probability of 3% using capnography.[62] While further study is needed before recommendation, this indicates that capnography could obviate the need for imaging in low to intermediate risk patients either after a positive D-Dimer or instead of obtaining a D-dimer.[62-64]

Triage

Simply measuring an initial $EtCO_2$ as a triage vital sign may have added benefit to the EP, and consideration could be made for making this a policy in your ED. One study demonstrated that abnormal initial $EtCO_2$ (outside of 35-45 mm Hg) was predictive of admisison (RR 2.5, 95% CI 1.5-4.0).[67] An abnormal $EtCO_2$ (outside of 31-41 mm Hg for this study) was 93% sensitive (95% CI 79-98%), with expectedly low specificity of 44% (95% CI 41-48%) for mortality prior to discharge.[47] This potential vital sign may be treated similarly to tachycardia; while an abnormal heart rate should increase a clinician's concern for a pathological condition, it needs to be taken in context of the situation to accurately interpret it.

Summary

Capnography has numerous uses in the ED in both intubated and spontaneously breathing patients. Quantitative capnography is the standard of care for confirming endotracheal intubation. It is recommended as an aide in maximizing chest compressions during cardiac arrest and can assist in prognostication. It rapidly identifies hypoventilation during procedural sedation. It also has many more potential applications that continue to be explored in areas such as respiratory distress, sepsis, trauma, DKA, and PE. Ultimately, capnography should always be used in association with the remainder of the clinical assessment.

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