A-P3 CAPNOGRAPHY (ETCO2)		First Responder EMT AEMT Paramedic

Indications

Clinical Symptoms	Clinical Interventions
• Every Cardiac Arrest (excluding DOA's) • Any **Altered Mental Status** • Any **Difficulty Breathing** • Any **Signs of Hypoperfusion** (e.g. hypotension, tachycardia, etc.	• **Any Oxygen Administration or Other Respiratory Interventions** (basic or advanced airways, nebulizers, etc.) • **Any Sedating Medications** (narcotics, benzodiazepines, ketamine, etc.)

End-Tidal CO2 Measurements

Physiologic State	High ETCO2 (>45 mmHg)	Low ETCO2 (<35 mmHg)
High (Venous) Blood pCO_2	**Hypoventilation** (Increased CO_2 in the blood & alveoli)	**No ventilation =** Circuit disruption (obstruction, disconnection, etc.) *– or –* **No perfusion=** Shock/Cardiac Arrest (decreased cardiac output)
Low (Venous) Blood pCO_2	N/A	**Hyperventilation** Metabolic Acidosis with a compensating respiratory alkalosis → hyperventilation blowing off CO2; e.g. DKA
ETCO2 Change	**Sudden Rise** Increased delivery of CO2 to the lungs (e.g. **ROSC**, administration of Sodium Bicarb, etc.)	**Sudden Drop** **Circuit Disconnected** (no ventilation occurring) *– or –* **Cardiac Arrest** (sudden decrease in cardiac output)
	Gradual Rise **Hypoventilation** with resultant respiratory acidosis	**Gradual Drop** **Hyperventilation** **Shock** = worsening cardiac output/perfusion

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End-Tidal CO2 Monitoring Procedure

- Attach the capnography adapter to the BIAD/endotracheal tube, or place EtCO2 cannula with oxygen delivery device.
- The device should remain in place until patient care is transferred.
- Document $EtCO_2$ value and waveform changes along with each pulse oximetry (SpO2) measurement within the Patient Care Report (PCR/ePCR).
- Any loss of CO_2 detection or waveform indicates a potential airway dislodgement or perfusion problem and should be immediately evaluated and documented.

Overview of Clinical Management based on End-Tidal CO2 Measurements

Refer to individual clinical guidelines for more specific approach

Clinical Scenario	*ETCO2 Reading*
Cardiac Arrest	• Generally 0: due to a lack of blood delivery to the lungs • If <10: improve chest compressions • $ETCO_2$ <10 after 20 minutes of CPR likely indicates low CO_2 production (i.e. no cellular metabolism) and is associated with poor outcomes. • Sudden increase: may indicate reperfusion/ROSC (may also be related to Bicarb administration)
General Approach	**Adjust quality of CPR (rate, depth, etc.) to maintain a goal of 20**
Respiratory Distress	• Absolute number not as important as the trend • >45 may be baseline (chronic CO2 retention with COPD) • <35: likely due to hyperventilation (increased work of breathing); but could also be due in part to decreased perfusion (i.e. shock) • *Asthma*: normal/high EtCO2 is concerning for impending respiratory failure (*should be low due to hyperventilation*) • In asthma/COPD exacerbations: patients may be allowed to have higher ETCO2, as lower ventilation rates allow more time for exhalation and prevents auto- PEEP and/or air trapping. This should be balanced with ventilatory needs to maintain oxygenation.
General Approach	**Increasing EtCO2 = worsening respiratory failure and more aggressive ventilatory assistance should be provided (CPAP/BiPAP, intubation, etc.)**

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Post-Intubation/ Paralysis	• Paralysis removes the respiratory drive and will result in the rapid accumulation of CO2 without appropriate assisted ventilation • If the patient is intubated while in a state of metabolic acidosis (e.g. sepsis), their compensatory hyperventilation will be negated potentially leading to a mixed acidosis (both metabolic and respiratory) which may quickly lead to cardiac arrest.
General Approach	• Assist ventilations as soon as a decreasing respiratory drive is noted, and minimize apnea periods during intubation attempts. • **Most patients should have their EtCO2 maintained between 35-45 with a respiratory rate of 8-12 breaths per minute.** • If the patient was hyperventilating prior to sedation/paralysis, it is reasonable to maintain EtCO2 between 30-35 post intubation with a slightly elevated respiratory rate (avoid frank hyperventilation).
Altered Mental Status	• Elevated pCO2 (CO2 narcosis) is one of the many causes of altered mental status. • >45: may indicate CO2 Narcosis due to direct CNS depression (e.g. overdose) or a respiratory cause of hypoventilation (e.g. pneumonia)
General Approach	• **Ventilatory support titrated to the patient's overall clinical condition should be provided, as per protocol (CPAP/BiPAP, intubation, etc.).**

Shock States (Metabolic Acidosis)	• In states of shock, EtCO2 may be used as a surrogate for cardiac output.
	• A low ETCO2 should prompt appropriate interventions to relieve the shock state:
	  ○ Hemorrhagic Shock → TXA/Blood Products
	  ○ Hypovolemic Shock (e.g. Septic/Distributive) → IV Fluid Bolus(es) & Vasopressors

General Approach	• **Cardiovascular support titrated to the patient's overall clinical condition should be provided, as per protocol (IV fluid resuscitation, blood product administration, vasopressors, etc.).**

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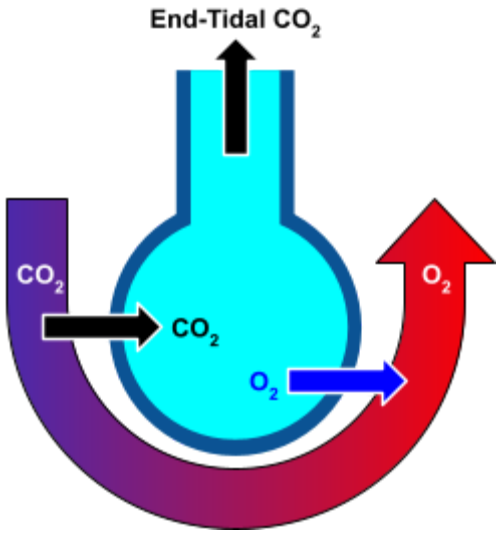
Notes on ETCO2

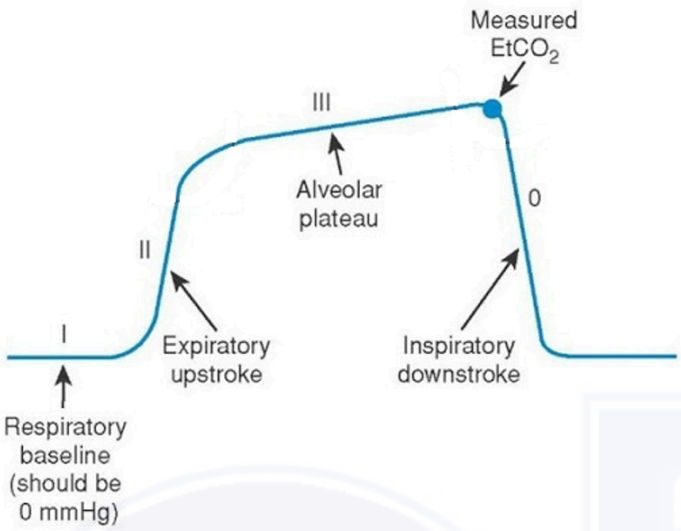
CO2 Physiology

- CO2 rapidly diffuses from blood entering the lungs into the alveolar space.
- End-tidal CO2 is a direct measurement of the pressure (in mmHg) of the CO2 in the alveoli (i.e. the pCO2).
- ETCO2 is thus an INDIRECT measurement of the CO2 in the venous blood entering the lungs (i.e. the paCO2 - as measured in an venous blood gas).
- Therefore, ETCO2 can be affected by either ventilation or perfusion.



Capnogram

- Baseline (I): Dead Space Air = same O_2 and CO_2 concentration of inhaled air
- Expiration (II) & Inspiration (0): rapid change from dead-space air (trachea, bronchioles, etc.) to alveolar air
- Plateau (III): mostly pure expired air
- **End-Tidal CO_2: Maximum CO_2 = approximate venous pCO_2**



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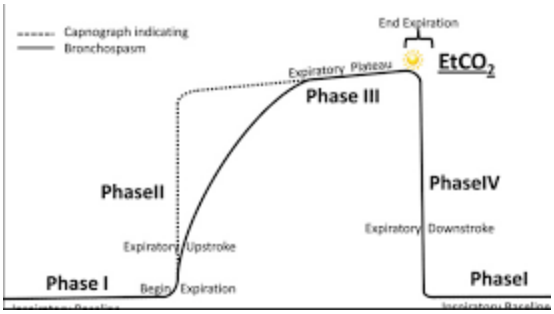
Capnogram: Shark Fin Appearance

- Seen in: Obstructive lung disease (asthma, COPD, etc.)
- Cause: air trapping (due to bronchospasm) in the alveoli cause a delay in expiration, making the Expiratory Upstroke (II) more prolonged (less vertical)



QI Review Parameters:

1. Was ETCO2 used appropriately (based on indications)?
2. Was ETCO2 documented appropriately (i.e. with all SpO2 readings)?
3. Was the ETCO2 level used to guide appropriate clinical management?