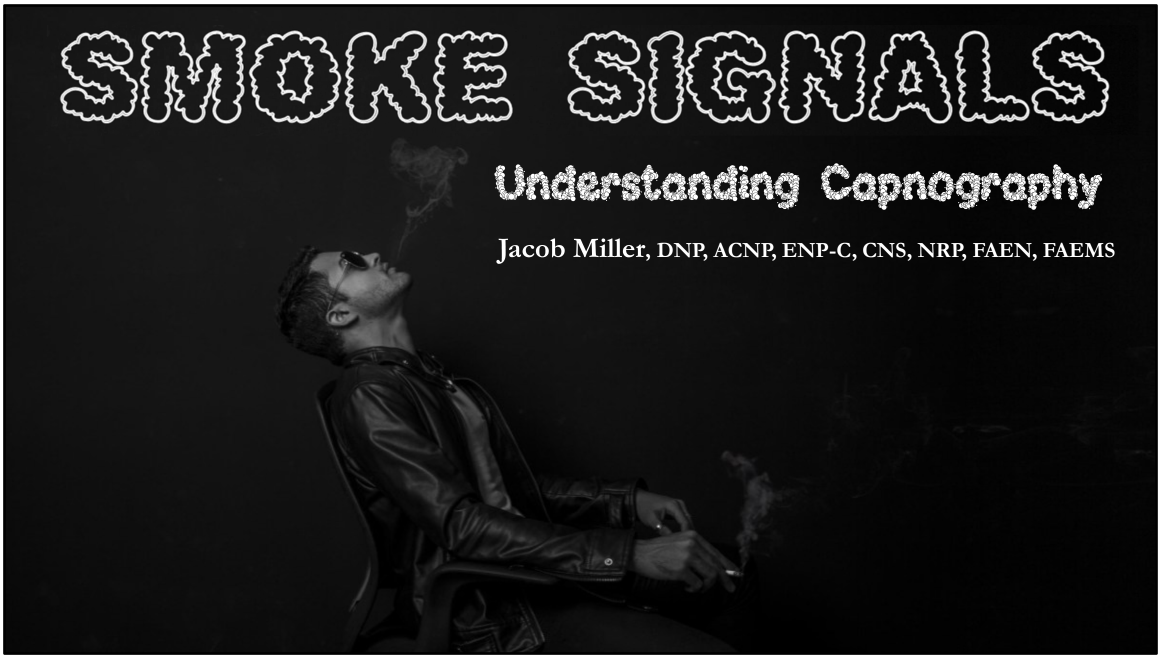
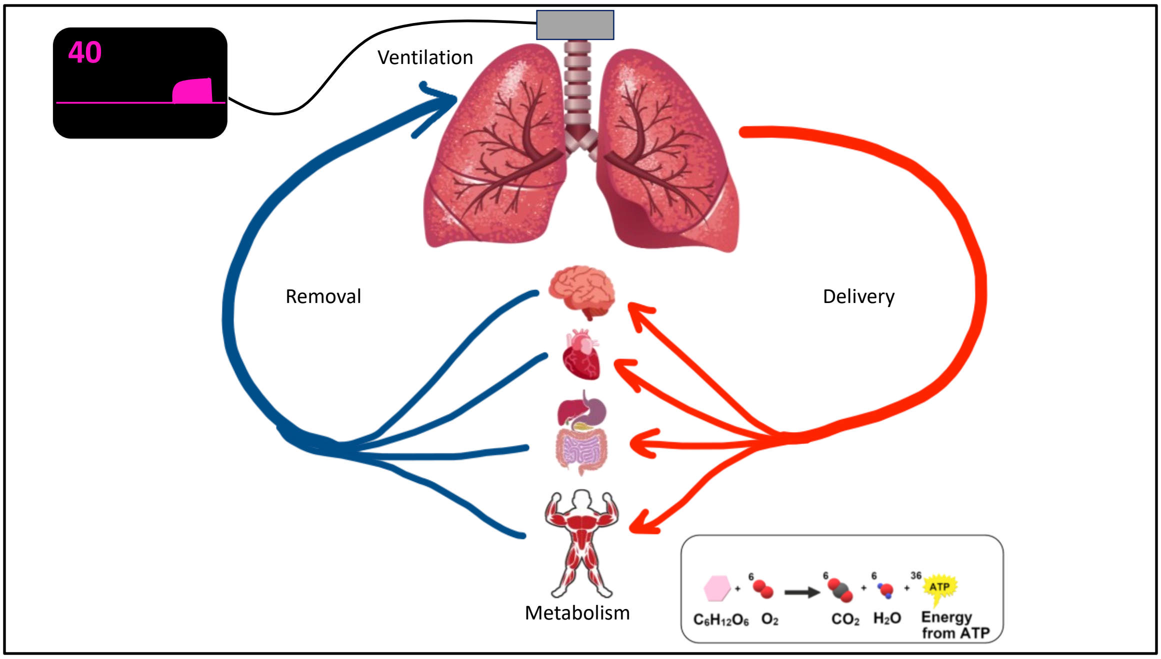

SMOKE SIGNALS
Understanding Capnography
Jacob Miller, DNP, ACNP, ENP-C, CNS, NRP, FAEN, FAEMS



First, let's discuss what capnography actually measures:

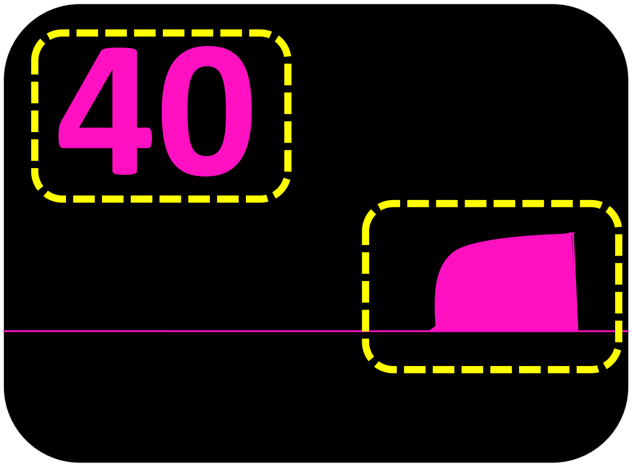
When we breathe in oxygen, oxygen is carried by the arterial system to our tissues. In other words, oxygen is delivered to the body cells.

In the body tissues, oxygen is used for metabolic processes, the byproduct of which is carbon dioxide production. The rate at which CO2 is produced is reflective of the amount of metabolic activity being carried out by those tissues.

Once the CO2 is produced in the body tissues, it is removed via the venous system back to the lungs to be exhaled.

Finally, our lungs exhale the CO2 that is brought back to them through ventilation. It is *this* exhaled CO2—the end-result of the entire cycle—that our EtCO2 monitors are measuring.

This is just the 30,000-foot overview; we'll take a closer look at each of these aspects shortly!

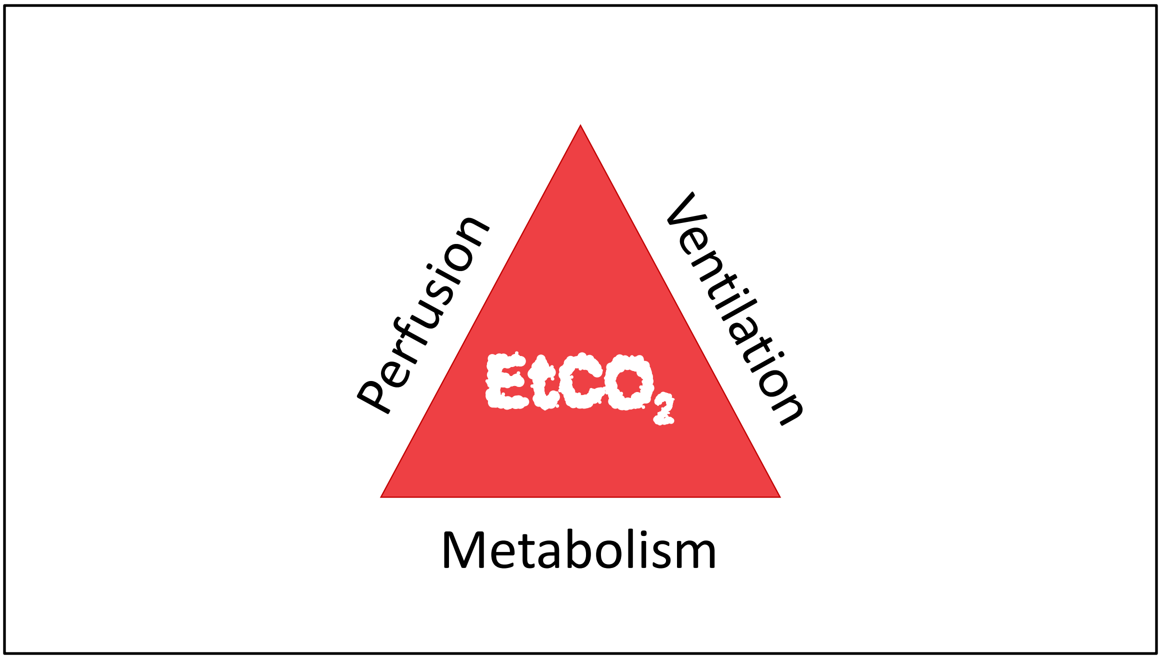


Additionally, waveform capnography provides us with two pieces of information:

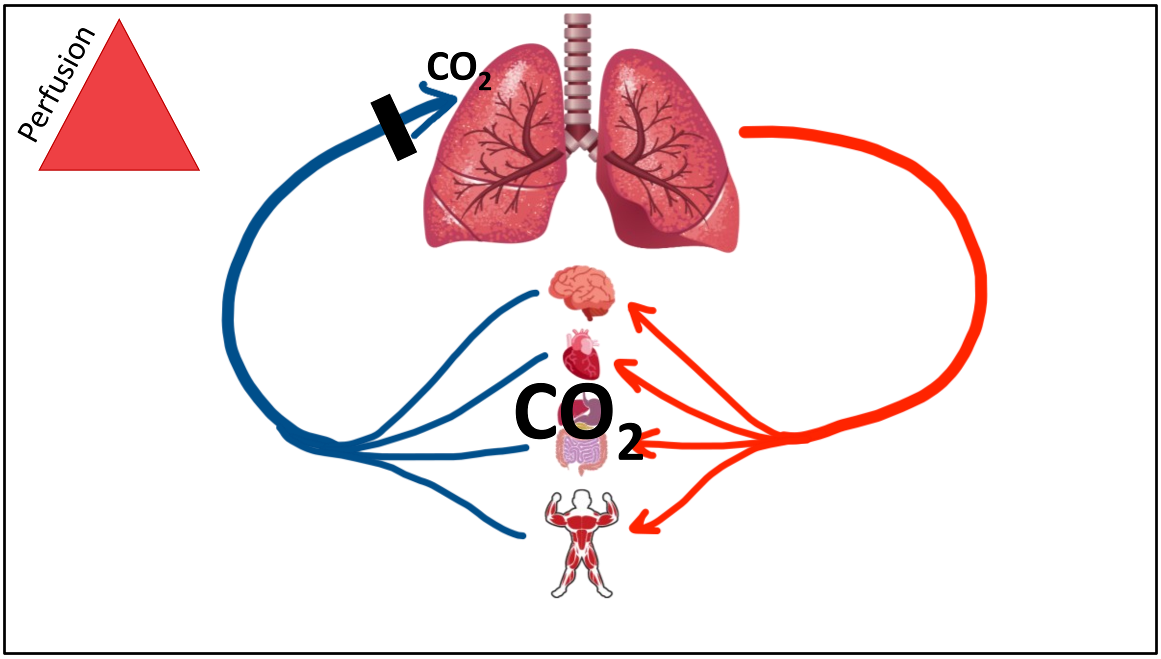
A numerical end-tidal CO2 value,

And a graphical waveform of the exhalation process.

We will focus, at first, on the numerical value.



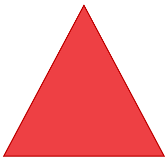
In essence, capnography provides us with an evaluation of the patient's perfusion (both O2 delivery & CO2 removal), metabolism, and ventilation



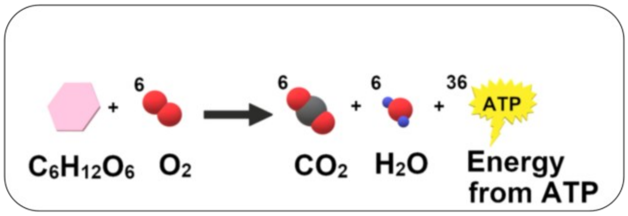
With respect to perfusion, recall that oxygen must be delivered to tissues, then CO2 must be removed from the tissues and moved to the lungs for exhalation.

In general, as long as they are receiving *some* degree of oxygen, tissues can largely perform aerobic metabolism and produce CO2.

However, in states of poor perfusion, the ability to remove the CO2 and/or circulate it through the lungs becomes impaired. As a result, less CO2 is exhaled (and, therefore, a lower EtCO2 value is seen on capnography). This can be the result of global hypoperfusion (e.g., in shock states), or localized perfusion defect in the lung (e.g., as would occur with a large pulmonary embolism).



Metabolism

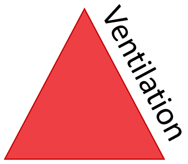


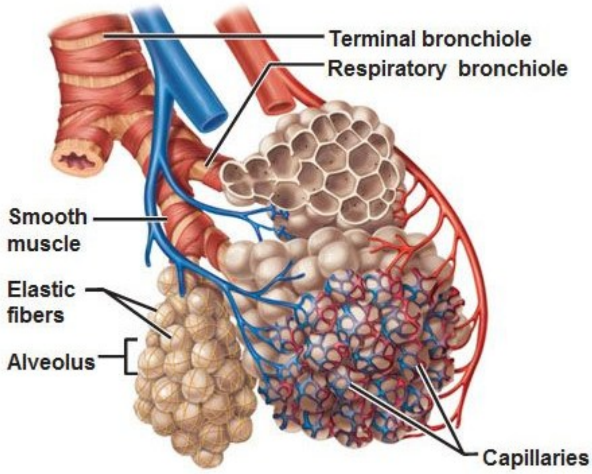
From a metabolic standpoint, CO2 is produced through aerobic metabolism as a byproduct of ATP production.

In cases of HIGH metabolic demands where tissues are required to produce high amounts of ATP (think, the excess workload on your skeletal muscles during exercise), this results in a HIGH degree of CO2 production, and may manifest with high EtCO2 values seen on exhalation.*

Conversely, during LOW metabolic demands, less ATP (and, therefore, less CO2) is produced.

**Note*: *that, in general, the body will recognize elevated CO2 levels and adjust ventilation to compensate – as a result the actual* <u>*measured*</u> *EtCO2 may be within normal limits due to respiratory compensation; if the patient did NOT adjust their respiratory rate and ONLY their metabolic activity increased, you would see an increase in CO2.*





When we breath deeply and/or quickly, their *minute ventilation* is increased.

The removal of CO_2 from the lungs is proportional to the patient's alveolar minute ventilation (assuming an unobstructed airway and intact pulmonary perfusion). Thus, as minute ventilation increases, so does CO_2 removal; and since our $EtCO_2$ is proportional to the amount of CO_2 in the blood, as more CO_2 is removed (via increased ventilation), the CO_2 dissolved in the blood falls. As the CO_2 level in the blood falls, so does the exhaled CO_2.

Essentially, increased minute ventilation will "blow off," or lower, CO_2 levels.

Conversely, when we breath slowly or shallowly, *minute ventilation* is decreased.

As minute ventilation decreases, so does CO_2 removal; as less CO_2 is removed, the CO_2 dissolved in the blood rises.

Essentially, decreased minute ventilation will "retain," or raise, CO_2 levels.

SPECIAL SITUATION!

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$$

There is one very important special situation that needs to be called out – because this will directly impact whether or not it is safe to “fix” a patient’s CO2 values.

Carbon dioxide functions as an **acid** in the blood.
In fact, excessive CO2 can be referred to as “hypercarbic acidosis” (because of the “carbon” portion of CO2) or “respiratory acidosis” (because the respiratory system is responsible for regulating CO2).

Thus, when confronted with a severe **<u>metabolic</u>** acidosis, the body will often cause an increase in respiratory minute ventilation to remove “respiratory acids” (CO2) to compensate for the metabolic acids it cannot remove, thus attempting to maintain a normal pH.

For this reason, a **low EtCO2** value **<u>might</u>** be in response to a metabolic acidosis (and not necessarily because of a problem with perfusion, metabolism, or ventilation).

If this is the case, “fixing” the low EtCO2 (for example, by lowering the set respiratory rate on the ventilator and allowing the EtCO2 value to return to “normal” ranges) can have catastrophic consequences by allowing the pH to fall.

For this reason, **<u>DO NOT</u>** correct a LOW EtCO2 until you KNOW the underlying cause!

		$EtCO_2$
Perfusion	↓	↓
Perfusion	↑	↑
Metabolism	↓	↓
Metabolism	↑	↑
Ventilation	↓	↑
Ventilation	↑	↓

This chart summarizes the expected changes in EtCO2 values based on changes in perfusion, metabolism, or ventilation.

Importantly, this is reflective of <u>*isolated*</u> *changes in the respective area; often (especially in healthy individuals), the body can compensate for an increase or decrease in one area to maintain a "normal" CO2 value, as was noted before.*

↓ $EtCO_2$

- Decreasing cardiac output / shock states
  - **Cardiac arrest!** (actual or imminent)
  - Poor CPR / fatigue / mCPR migration
- Hyperventilation
  - Anxiety / psychogenic
  - Iatrogenic (overzealous medic!)
- Decreased metabolic demand
  - Hypothyroidism
  - Hypothermia
- V/Q Mismatching
  - Interstitial lung disease, ARDS, etc.
  - Pulmonary embolism
- **Compensation for metabolic acidosis**
- *Dilution from constant oxygen (NRB, CPAP)*

But let's get down to the actual **practical** application of EtCO2 monitoring:

What does it ACTUALLY mean when my patient has a LOW EtCO2 value?

The above list identifies potential causes of **decreased** EtCO2. Obviously, this should be used in concert with other aspects of patient assessment, including history and physical exam, vital signs, and other available diagnostic data.

↑ $EtCO_2$

- Increased cardiac output
  - ROSC!
  - Positive pacemaker capture
- Hypoventilation
  - Opioid intoxication
  - CNS impairment
  - Iatrogenic (excessive analgesia/sedation)
- Expiratory compromise
  - e.g., Asthma/COPD
- Increased metabolic activity*
  - Hyperthyroidism
  - Exercise
  - Fever
- Exogenous CO_2 load (bicarb!)

Assuming no compensatory increase in respiratory rate!

Likewise, this list identifies potential causes of **increased** EtCO2.

Same disclaimer: this should be used in concert with other aspects of patient assessment, including history and physical exam, vital signs, and other available diagnostic data.

⊘ $EtCO_2$

- Apnea
- (Very) dead
- **Loss of ET Tube!**

Most importantly, there is really nothing good that comes from having NO EtCO2 production!

KEY POINTS

- TRENDS are probably most important
- ABSENT $EtCO_2$ almost never good
- HIGH $EtCO_2$ generally safe to fix
- LOW $EtCO_2$ might be compensatory (think hard before “fixing”!)

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