Carbon dioxide is a major end product of cellular metabolism. It is delivered to the lungs via the pulmonary circulation and thus capnometry can provide information not only on alveolar ventilation and pulmonary gas exchange, but also on metabolic rate and pulmonary blood flow. Therefore, carbon dioxide (CO2) is related to a patient’s ventilation, perfusion and metabolism.

**End tidal carbon dioxide (EtCO2)**

This is the measurement of CO2 in the exhaled respiratory gases and gives a non-invasive estimate of arterial CO2 (PaCO2). Although arterial blood gas analysis is the gold standard for analyzing a patient’s PaCO2 and therefore assessing ventilation status, obtaining an arterial blood sample is not always easy or possible in small animals. Repetitive blood gas sampling can also be associated with a risk of thrombosis or infection and does not give continuous information. Capnography allows CO2 levels to be monitored in intubated patients and therefore minimizes the need for repetitive arterial blood gas sampling.

Hypoventilation is the primary concern during anesthesia maintenance. A recent review of cardiopulmonary complications during general anesthesia in dogs indicated an incidence of >60% for hypoventilation. Anesthetic drugs, especially inhalant anesthetics cause decreased chemoreceptor responsiveness to PaCO2 which results in decreased respiratory rate and tidal volume, leading to an increase in PaCO2 and EtCO2. Monitoring EtCO2 allows identification of hypercarbia. “Permissive” hypercarbia is when EtCO2 is allowed up to 55 - 57mmHg; mild hypercarbia helps increase heart rate and blood pressure due to mild sympathetic stimulation.

**Elevated PaCO2 is potentially dangerous for several reasons**

1. As PaCO2 increases, pH decreases (although some compensation will occur through increased HCO3-)
2. As PaCO2 increases, PAO2 and PaO2 will decrease causing hypoxemia, unless inspired oxygen is supplemented.**see example below**

During general anesthesia (with oxygen supplementation), PaCO2 should be kept ≤ 60mmHg because this corresponds to ~ 7.2pH (pH Δ .08/ every 10mmHg increase in PaCO2) and cellular enzymes malfunction outside a pH range of 7.2-7.5. Treatment of hypercarbia should consist of lowering the level of inhalant anesthetic and intermittent positive pressure ventilation (IPPV), mechanical or manual if needed.

**How does elevated PaCO2 (as measured by EtCO2) affect PAO2 and PaO2?**

The alveolar gas equation describes the partial pressure of O2 in the alveolus which should be similar to arterial blood in normal patients. It says:

- \( \text{PAO2} = \text{FIO2} \times (760 - 47) - \frac{\text{PaCO2}}{\text{R}} \)
- Where:
  - \( \text{PAO2} \) - alveolar partial pressure of O2 (in mmHg)
  - \( \text{FIO2} \) - fraction of inspired oxygen
  - \( \text{PA} \) - ambient pressure in mmHg (760mmHg)
  - \( 47 \) - saturated vapor pressure of water at 37 degrees celcius
  - \( \text{PaCO2} \) - arterial partial pressure of CO2 (in mmHg)
  - \( \text{R} \) - respiratory quotient (assumed to be 0.8)

**Real case example**

4 year old CM Rottweiller anesthetized with an intra-muscular injectable anesthetic combination of Dexmedetomidine (10mcg/kg), Ketamine (2mg/kg) and Butorphanol (0.3mg/kg). The patient is breathing room air and his PaCO2 = 78mmHg

What will PAO2 be with this patient breathing room air with significant drug induced respiratory depression?

\[ \text{PAO2} = \text{FIO2} \times (760 - 47) - \frac{\text{PaCO2}}{\text{R}} \]

This dog’s estimated pulse oximetry reading (SpO2) would be ~ 82%

PaO2 can be estimated using pulse oximetry: \( \text{PaO2} = \text{SpO2} \times 30 \) (for pulse oximeter readings between 75% and 90%)

This formula ONLY applies to pulse oximeter readings of 75% to 90% because of the linear relationship between PaO2 & SpO2 values on mid portion of hemoglobin disassociation curve. The rule cannot be applied outside of these values. This is the same relationship as the mnemonic: 60 is 30, 90 is 60, 40 is 75.

The patient was then intubated and placed on 100% oxygen:

- \( \text{PAO2} = \text{FIO2} \times (760 - 47) - \frac{\text{PaCO2}}{\text{R}} = 1.0 \times (760 - 47) - \frac{78}{0.8} = 713 – 98 = 615\text{mmHg} \)
It is also important to monitor SpO2 at the time of recovery since the transition from 100% oxygen to room air (21%) can result in hypoxemia in the face of even mild hypoventilation. Monitoring of EtCO2 can give the anesthetist a measure of the level of hypoventilation and therefore the likelihood of hypoxemia at recovery.

Capnography is **most useful for detection of apnea, hypoventilation** but also detects esophageal intubation, airway disconnection, airway obstruction, leak in endotracheal tube cuff, exhaustion of CO2 absorbent, incompetent one-way valve of anesthetic rebreathing circuit, inadequate O2 flow rate for NRB circuit (increased inspired CO2), indirect measure of cardiac output (e.g., sudden, acute drops in cardiac output are associated with decreased ETCO2 due to poor pulmonary circulation), ventilation, perfusion and metabolic status.

When the concentration of CO2 in exhaled gas is displayed graphically overtime, the resulting waveform is known as a capnogram. Capnometers that display a capnogram are typically more expensive than those that just provide end-tidal CO2 (PEtCO2) concentration; however, these devices are extremely useful monitoring tools as analysis of the capnogram waveform frequently can diagnose problems with either the patient or equipment that might go unrecognized otherwise.

**Mainstream vs side stream capnometry**
The two types of capnometers based on the sensing device location, are the mainstream and sidestream. In sidestream capnometers, a sampling tube is placed between the endotracheal tube and the breathing circuit. A sample of the exhaled gases is transmitted to the measurement device which is located away from the breathing circuit. The rate at which respiratory gases are aspirated from the breathing circuit varies from 50 to greater than 400ml/min and can be adjusted in some models. This is an important consideration when setting the oxygen flow rates for both circle and non-rebreathing circuits in veterinary patients to ensure that adequate oxygen is supplied to the breathing circuit. In mainstream capnometers, the measurement device itself is placed between the endotracheal tube and breathing circuit. Infrared light within the sensor traverse the respiratory gases in the breathing circuit and are detected by a photodetector within a cuvette. Mainstream sensors are heated to prevent condensation of water vapor, which can lead to falsely elevated CO2 readings.

**The advantages of sidestream analysis include**
- lightweight sampler near the patient
- smaller sample chamber volume
- ability to sample other gases such as inhalant anesthetics and nitrous oxide.

**The disadvantages of sidestream analysis include**
- plugging of the sample line by secretions and condensation
- a 2- to 3-second delay in CO2 measurement
- need to scavenge sampled gasses
- aspiration of extraneous air from leaks in the breathing circuit that dilute the sample.

**The advantages of mainstream analysis include**
- gives a real-time measurement
- no scavenging of sampled gases needed

**The disadvantages of mainstream analysis include**
- excessive dead space in the patient breathing circuit produced by the sensing chamber can lead to false readings
- the weight and bulk of the device near the patient.
- the sensing chamber may be contaminated by secretions and condensation
- patients may be burned by the heated cuvette

**Normal capnogram (thefreedictionary.com)**
Normal capnogram. *(A)*, Carbon dioxide cleared from the anatomic dead space; *(B)*, dead space and alveolar carbon dioxide; *(C)*, alveolar plateau; *(D)*, end-tidal carbon dioxide tension (PEtCO2).

A normal capnogram waveform (above) is generated by the measurement of CO2 (PCO2) at various points during the respiratory cycle. Phase I occurs during the initial part of exhalation; gas measured at this point typically contains no or an unmeasurable concentration of CO2 because it is coming from dead space—from the circuit as well as anatomic from the large airways (trachea, bronchioles) that do not participate in gas exchange. Phase II represents the transition between gas from the airway dead space to alveolar gas, which contains CO2. Phase III, known as the alveolar plateau, occurs when all the gas is coming from the alveoli. PEtCO2 occurs at the end of the alveolar plateau and represents the concentration of CO2 at the end of expiration. Phase IV, the inspiratory down stroke, occurs at the beginning of the next inspiration.
Common causes of increased EtCO2 values
- Metabolism: Fever, malignant hyperthermia, sodium bicarbonate administration, tourniquet release, seizures
- Pulmonary Perfusion: Increased cardiac output or blood pressure
- Alveolar Ventilation: **hypoventilation, rebreathing
- Technical Errors*: Exhausted soda lime, inadequate fresh gas flow (NRB), faulty valves, leaks

Common causes of decreased EtCO2 values
- Metabolism: Hypothermia, hypothyroidism, muscle relaxants
- Pulmonary Perfusion: * decreased cardiac output, blood pressure, hypovolemia, pulmonary embolism, *cardiac arrest
- Alveolar Ventilation: *hyperventilation, *apnea, partial airway obstruction, asthma, pulmonary edema
- Technical Errors: patient disconnect, sampling line leak

Capnometry in CPR
During CPR, EtCO2 provides extremely useful information about the efficacy of the resuscitative effort as delivery of CO2 to the lungs requires adequate pulmonary perfusion. EtCO2 is often zero or very low at the start of CPR due to poor pulmonary perfusion. An increase in EtCO2 (EtCO2 = 10 – 20 mmHg) during CPR suggests that chest compression are generating sufficient cardiac output to perfuse the lungs or that return of spontaneous circulation (ROSC) has occurred. In human patients, an increase of EtCO2 greater than 10mmHg in the first 20 minutes of CPR has been shown to be a positive predictor of ROSC. Ineffective CPR, resulting in EtCO2 < 10 mmHg is an indication of poor outcome and the resuscitation strategy should be re-evaluated.

Limitations of Capnometry
EtCO2 is usually slightly less than PaCO2 due to dilution of alveolar gas by gas in the dead space. When pulmonary perfusion and gas exchange are normal (good V/Q matching), EtCO2 correlates well with PaCO2 and, in most patients, the PaCO2-EtCO2 gradient is 3 – 5mmHg. Therefore, EtCO2 may be a useful for a continuous estimate of PaCO2. When gas exchange is compromised by pulmonary pathology, pulmonary embolism, poor cardiac output, or excessive dead space (physiologic or mechanical), the PaCO2-EtCO2 gradient will increase and can be as high as 20 mmHg or more in severe pulmonary disease. In this situation, an elevated EtCO2 does indicate increased PaCO2 (but may not reveal the extent of hypercarbia) but a normal or low EtCO2 does not rule out hypercarbia. For this reason, it is recommended to obtain a PaCO2 reading from arterial blood gas analysis to verify the PaCO2–EtCO2 gradient.

Blood gas analysis
- pH = acidosis or alkalosis
- PaCO2 - measure of ventilatory status of patient
- ↓ PaCO2 – hyperventilation, respiratory alkalosis
- ↑ PaCO2 – hypoventilation, respiratory acidosis
- Normal awake 35-45mmHg

References