Introduction

Capnography is the non-invasive measurement of exhaled carbon dioxide shown by a waveform on a monitor.

The first use of infrared light to measure carbon dioxide (CO₂) was developed in 1943 and was designed to monitor CO₂ levels in buildings. Since then, capnography has evolved to become an essential piece of anaesthetic equipment. Using capnography provides a breath-by-breath analysis and continuous recording of the patient’s ventilatory status. Due to the versatility of capnography, its utility extends beyond the operating theatre to include x-ray, endoscopic suites and in the transfer of critically ill patients.

This module explains the basic principles of capnography, discusses EtCO₂ waveforms, and how to interpret them in the ventilated patient.

NB: ‘Capno’ is the ancient Greek word for smoke and ‘graphy’ the process of writing or drawing.
What is carbon dioxide?
All cells in the body take in oxygen and glucose to make energy for cellular metabolism and release water and carbon dioxide as by-products. Although CO₂ is a by-product, it is also used by the body to regulate acid–base buffering. Depending on blood pH at any given time, the body can convert CO₂ to either carbonic acid or to bicarbonate, a base.

How is carbon dioxide transported in the body?
As a by-product CO₂ diffuses out of the tissues and into the plasma. While a small proportion of the CO₂ remains in the plasma, most is taken into the red blood cells (erythrocytes). Inside the red blood cell some CO₂ binds to the haemoglobin although most is converted by the cell into bicarbonate. The bicarbonate is transported in the plasma to the lungs. Once it reaches the lungs the process is reversed and the CO₂ is exhaled.

What is capnography?
Capnography is the measurement of the CO₂ concentration in air and when measured at the end of an exhaled breath (end-tidal) the levels correlate with the patient’s arterial pCO₂.

Technically capnography refers to the display of this measurement as a waveform, and capnometry refers to the display of numerical value representing peak CO₂. Most anaesthetic monitors displays a waveform and a value, as well as a respiratory rate.
What are normal etCO$_2$ values?
Numerical values are commonly displayed in kPa or mmHg. In healthy adults with normal lungs, end-tidal CO$_2$ is:

4.0-5.7 kPa (35-45 mmHg)*

* These values differ in various publications

Is the ETCO$_2$ less than arterial CO$_2$?
The end-tidal CO$_2$ is 2-5kPa below the arterial CO$_2$ because the end-tidal CO$_2$ is always diluted with some dead space gas.

What is dead space gas?
Dead space gas is the amount of gas that is involved in ventilation but does not take part in gas exchange. Not all the air in each breath is used for the exchange of oxygen and carbon dioxide. About a third of every resting breath is exhaled exactly as it came into the body.

There are three types of dead space:

- **Anatomic dead space** – This refers to the amount of gas that fills the conducting passages of the airway (i.e. the trachea and upper bronchi) but is not involved in gas exchange. For the normal sized adult, it is usually about 150 mL (2ml/kg). Therefore, if the normal tidal volume is 500 mL, only 350 mL of tidal volume is actually involved in gas exchange.

- **Alveolar dead space** – This is the amount of gas filling the alveoli that does not contribute to gas exchange. This may be caused by gas that reaches alveoli that are not perfused or are poorly perfused although in a patient with healthy lungs this is minimal.

- **Mechanical dead space** – This is the contribution to the patient’s dead space through the addition of respiratory equipment, circuit attachments, filters, etc.

And

- **Physiological / total dead space** – This value is the sum of anatomic and alveolar dead space. It represents the total volume in the airway and alveoli not participating in gas exchange.
How does capnography work?
Capnography uses infrared waves to measure the CO$_2$. Infrared is absorbed by gases that have two or more different atoms. Oxygen gas has two identical atoms; therefore infrared detection cannot be used to measure oxygen levels.

Carbon dioxide, unlike oxygen, has atoms that are different (1x carbon, 2x oxygen). As a result it absorbs infrared waves at a specific wavelength (4.26um). When the patient exhales, a beam of infrared light is passed over the gas sample on a sensor. The presence or lack of CO$_2$ is inversely indicated by the amount of light that passes through the sensor. The CO$_2$ levels are then displayed on a monitor after being adjusted for the presence of nitrous oxide, which can also absorb infrared rays at a similar wavelength and thus can cause some interference if not accounted for.

Main-stream or side-stream analyser?
There are two main methods capnographs use to sample the CO$_2$. These are termed mainstream analysers or side-stream analysers. Most anaesthetic gas analysers use side stream.

**Mainstream** devices require a module containing a sensor be placed in the anaesthetic circuit. This measurement is direct and does not require the removal of any gas from the fresh gas flow so is very quick, but the sensor can be bulky. It must be placed as close to the patient’s airway as possible to provide an accurate measurement, so the additional weight does increase the risk of accidental disconnection or tube kinking.

Moisture and secretions can also be a problem as they interfere with the readings (water also has 2 different atoms so absorbs infrared). This is an issue because the patient’s exhaled breath is humid, so a filter should be placed between the sensor and the patient’s airway. Some main-stream analysers are heated to prevent this happening. Mainstream analysers can only measure CO$_2$, no other gases.

**Side-stream** analysers measure gas drawn from the breathing circuit, usually at a rate of 150-250 mL/min. They have the advantage that they do not require bulky equipment to be placed close to the patient’s airway, and the sensor is often incorporated into the anaesthetic machines gas analyser. The light weight means they can also be used with nasal cannula and facemasks.

Their main disadvantage of side-stream analysis is a slower response time since time is taken to physically conduct the gas from the breathing circuit to the measuring device. There can be a delay of several seconds before changes appear on the monitor.

There is usually a moisture trap and an exhaust port, allowing gas to be returned to the breathing system.
Why is capnography important?

Capnography is a valuable piece of monitoring equipment. Most obviously it confirms airway placement and gives an overview on ventilation adequacy and respiratory rate. As the CO$_2$ concentration is inversely proportional to a patient’s minute volume, a high CO$_2$ usually implies that ventilation is inadequate and vice versa.

The shape of the capnograph waveform also gives vital information as it changes under certain pathological conditions i.e. a slurred upstroke to the wave suggests uneven emptying of alveolar units which might imply bronchoconstriction, asthma or COPD.

AAGBI guidance for United Kingdom:

Continuous capnography should be used in the following patients, regardless of location within the hospital:

- Those whose tracheas are intubated.
- Those whose airways are being maintained with supraglottic or other similar airway devices.

Capnography should be available for use wherever it is possible that a patient’s trachea will be intubated, such as anaesthetic rooms, operating theatres, recovery rooms, other treatment rooms in which general anaesthesia is given, intensive care units, high dependency units, and A & E departments.

It is also recommended that a capnograph be immediately available during the treatment of cardiac arrests in hospital.

Factors that can cause a low EtCO$_2$

- **Disconnectionblocked ET tube** – sensor will no longer be receiving sample gas from lungs
- **Inadvertent extubation** – this will cause a sudden cessation of gas expired from the patient’s lungs reaching the sensor.
- **Oesophageal intubation** – only lungs produce CO$_2$
- **Hyperventilation** – EtCO$_2$ will be low because of excessive speed or depth of ventilation.
- **Hypothermia** – CO$_2$ production is decreased because cell metabolism is reduced.
- **Hypovolaemia** - Drop in cardiac output leads to less CO$_2$ reaching the lungs.
- **Pulmonary embolism** – reduced height on trace
- **Drop in cardiac output** - leads to a drop in CO$_2$ output.
- **Cardiac arrest** – normal cell metabolism is not taking place
Factors that can cause a high EtC02

- **Hypoventilation** - ventilation is inadequate to remove CO₂.
- **Hyperthermia** - cell metabolism is increased and so CO₂ production is increased.
- **Malignant hyperthermia** – cell metabolism is *greatly* increased and so CO₂ production is *greatly* increased. This may be the first sign to present in MH.
- **Sepsis** - temperature increase leads to increase in metabolic rate and so increase in CO₂ production.
- **Increased skeletal muscle activity** (shivering) - leads to increased metabolic rate and so increased CO₂ production.
- **Hypermetabolic states** – as above.
- **Depressed respiratory centre/ lung dysfunction/ weakened respiratory muscles** – leads to CO₂ retention.
- **Rebreathing/ obstructed airways** – poor CO₂ elimination. In rebreathing, the patient may make an inspiratory effort before the expiration is complete.
- **Soda lime is exhausted** – the soda lime will not be able to remove all the CO₂ from the circle and so the patient will rebreathe CO₂ in inspired air.

The normal capnography Waveform

The output of both the main stream and side stream capnograph is a graphical plot of CO₂ partial pressure versus time.

Although reading EtCO₂ waveforms is not difficult, interpreting what you see requires an understanding of how the waveforms and numbers are produced.

This is the normal capnography waveform of the respiratory cycle.
Each normal capnography wave goes through four phases.

**Phase 1:**

Phase I (the inspiratory baseline) represents the end of inspiration and the start of expiration, and therefore no \( \text{CO}_2 \) is detected. The start of expiration has no \( \text{CO}_2 \) because the initial gases expired originate from the dead space of the upper airway and the capnography trace remains at zero.

**Phase 2:**

Phase 2 is the continuation of exhalation. Phase 2 is characterised by a rapid rise in \( \text{CO}_2 \) concentration as dead space gas is replaced with alveolar gas.

**Phase 3:**

Phase 3, all of the gas passing by the \( \text{CO}_2 \) sensor is alveolar gas which causes the capnograph to flatten out. This is called the Alveolar plateau. The peak measurement at the end of phase 3 is the Et\( \text{CO}_2 \) reading.
Phase 4:

Phase four is inspiration and marked by a rapid downward direction of the capnograph. This downward stroke corresponds to the inspiratory fresh gas which is free of carbon dioxide.

Some abnormal capnography Traces

The waveform analysis of abnormal capnography can identify a variety of pulmonary and airway issues:

**Hyperventilation** or overventilation leads to a lessening of the EtCO₂ value

![Hyperventilation waveform](image)

**Hypoventilation**, although it can also be caused by other events such as tourniquet release or very occasionally early malignant hyperthermia.

![Hypoventilation waveform](image)

**Malignant hyperthermia** is seen as a dramatic increase in end-tidal carbon dioxide in a ventilated patient and/or respiratory rate in a spontaneously breathing patient.

![Malignant Hyperthermia waveform](image)
**Oesophageal intubation**, no or very little carbon dioxide is detected although some carbon dioxide might be present in the stomach. This may result in up to three or four waveforms with an abnormal shape and decreasing in amplitude.

**No trace, wrong place! Confirm the location of the ETT.**

Even during cardiac arrest there will be some CO$_2$ (for up to 15 minutes after loss of circulation)

![Oesophageal intubation](image)

**Cardiac arrest after a normal trace**, even with cardiac arrest there will be some detectable CO$_2$ trace for up to 15 minutes and will continue as long as effective chest compressions are done.

![Cardiac Arrest](image)

**A flat trace after a normal capnography** may include the following:

- Ventilator or circuit disconnection/ accidental extubation
- Airway obstruction (e.g. patient suddenly bit down on the tube or it is kinked- check the airway pressures)
- Capnograph sample line disconnection or obstruction

![Normal followed by flatline trace](image)

**Rebreathing** can be seen when the baseline rises above zero which can be because of exhausted soda lime, inadequate fresh gas flows (e.g. with Bain’s circuit) or faulty valves.

![Rebreathing](image)
**Loss of neuromuscular blockade or curare clefts** appear when the action of the muscle relaxant begins to subside and spontaneous ventilation starts to return (curare was the very first muscle relaxant used in medicine).

![Curare cleft](image)

**Cardiogenic oscillations** are caused by. They may be observed in paediatric patients who are mechanically ventilated at low respiratory rates with prolonged expiratory times.

![Cardiogenic oscillations](image)

**COPD or asthma**, the waveform shows a sawtooth slope and does not accurately reflect the end-tidal CO₂. Because areas of the affected lung empty more slowly than areas of lung with normal bronchi, there is no regular separation between dead space air and alveolar air during exhalation, resulting in a more gradual rise of phase 2.

![COPD or Asthma](image)

In **emphysema**, the alveolar slope will be reversed. The gas exchange surface is so poor, and the compliance of the lungs so abnormally increased, that the alveolar gas exchanges very rapidly. Thus, the part of the curve which represents arterial CO₂ is the early peak, not the end-tidal value.

![Emphysema](image)
References:


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