Interpretation of capnography

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The anesthetist will get the most information out of a capnograph if it is examined systematically. First, the anesthetist must determine whether exhaled CO₂ (i.e., a waveform) is present. The differential diagnosis of absent CO₂ includes esophageal intubation, accidental tracheal extubation, disconnection of the breathing circuit, complete obstruction of the endotracheal tube or conducting system (kink, inspissated blood or secretions, extremely severe bronchospasm) or of the breathing circuit, apnea, and cardiac arrest. Second, the shape of the waveform must be analyzed systematically by looking at, and in sequence, phase I (inspiratory baseline, which should be zero); phase II (expiratory upstroke, which should be nearly perpendicular to the inspiratory baseline); phase III (expiratory or alveolar plateau, which should be a straight, nearly horizontal, line); and phase IV (inspiratory downstroke, which should be nearly perpendicular to the inspiratory baseline). This discussion will follow this systematic approach but will emphasize diagnosis that can be obtained from the phase III alveolar plateau.

Key words: Capnogram, end-tidal CO₂ concentration, ventilation.

Monitoring and interpretation of the end-tidal carbon dioxide (ETCO₂) waveform are skills critical to the conduct of a modern anesthetic. This review offers a systematic examination of the ETCO₂ waveform and includes the relevant physiological and pathophysiological considerations.

Basic principles

The best measure of the pressure of end-tidal carbon dioxide (PETCO₂) will be obtained when:

1. Tidal volumes are large enough to displace dead space.
2. Fresh gas flow rates are low enough to prevent dilution or washing out of CO₂.
3. Sample aspiration rates are low enough that they do not interfere with patient ventilation or entrain air that may dilute the CO₂.
4. The sampling site is close to the patient, minimizing the dead space.
5. The waveform is displayed for end-tidal alveolar plateau analysis.¹²

In any situation in which there is doubt about the validity of the PETCO₂ value, PacO₂ should be measured as a guide for tracking and interpretation. A change in the PacO₂-PETCO₂ gradient in itself may indicate an important pathophysiologic change (e.g., change in dead space, see below).

Clinical use

The anesthetist will get the most information out of a capnograph if it is examined systematically. First, the anesthetist must determine whether
exhaled CO₂ (i.e., a waveform) is present. Second, the shape of the waveform must be analyzed systematically by looking at, and in sequence, phase I (inspiratory baseline), phase II (expiratory upstroke), phase III (expiratory plateau), and phase IV (inspiratory downstroke) (Figure 1). This discussion will follow this systematic approach but will emphasize diagnoses that can be obtained from the phase III alveolar plateau. In the future, capnography may display CO₂ concentration as a function of the volume of exhaled gas from each breath (the single breath test or SBT) because the SBT allows online determination of V̇CO₂ (CO₂ concentration × tidal volume) and is a more sensitive test of and reveals more information about gas elimination in the late phase of each breath.¹

Is exhaled CO₂ (waveform) present?

If the presence or persistence of CO₂ is not detected by the capnometer or capnograph, failure to ventilate the patient’s lungs must be assumed. The differential diagnosis of absent CO₂ includes esophageal intubation, accidental tracheal extubation, disconnection of the breathing circuit, complete obstruction of the endotracheal tube or conducting system (kink, inspissated blood or secretions, extremely severe bronchospasm) or of the breathing circuit, apnea, and cardiac arrest (Table 1).² With respect to esophageal intubation, capnometry/capnography can make the diagnosis in one breath and is therefore far superior to pulse oximetry, which usually requires some time for desaturation to occur.³ If the stomach contains exhaled gas from previous mask ventilation attempts or carbonated beverages, a few tidal ventilations through an esophageal tube may contain minimal and progressively diminishing concentrations of carbon dioxide. It should be noted that very severe bronchospasm (enough to cause complete airway obstruction) can prevent carbon dioxide from being registered by the capnograph even though the trachea has been properly intubated.⁴

Only after all of the above life-threatening possibilities have been (quickly) ruled out and ventilation of the patient’s lungs has been confirmed by clinical examination, should failure of the capnometer or capnograph be considered in the differential diagnosis. A rapid qualitative check of the capnograph consists of simply removing the CO₂ sensing or sampling port and exhaling into it. Of all the entities in the differential diagnosis listed above, monitoring cardiac output and cardiopulmonary resuscitation during cardiac arrest is a new application of capnography and therefore will be the only entity further discussed.

Table I
Differential diagnosis of absent exhaled CO₂

| I. Exhaled CO₂ usually present, then absent |
| 1. Accidental tracheal extubation |
| 2. Disconnection of breathing circuit |
| 3. Monitor failure |
| 4. Complete obstruction of endotracheal tube |
| 5. Cardiac arrest |
| 6. Patient becomes apneic |

| II. Exhaled CO₂ usually absent/minimal initially, remains absent |
| 1. Esophageal intubation |

* Monitoring cardiac output and cardiopulmonary resuscitation by end-tidal carbon dioxide concentration.

During steady state gas exchange equilibrium, the alveolar PCO₂ (PACO₂), tissue CO₂ production (VCO₂), and alveolar ventilation (VA) are uniquely related as given by PACC0₂ = (K) VCO₂/VA. During constant minute ventilation and VCO₂, an abrupt reduction in cardiac output (Qt) reduces P ClienteCO₂ by two mechanisms.⁷ First, a reduction in venous return causes a decrease in CO₂ delivered to the alveolar compartment, resulting in decreased PACO₂. Second, the increase in alveolar dead space, which results from the reduced pulmonary vascular pressures, will dilute the CO₂ from normally perfused alveolar spaces to decrease P ClienteCO₂ below PACC0₂ (see below). During a sustained reduction in Qt, increasing CO₂ accumulation in the peripheral tissues and in venous blood will begin, after 10 to 20 minutes, to restore CO₂ delivery to the lung and P ClienteCO₂ toward baseline levels. Reciprocal changes in P ClienteCO₂ will occur during acute increases in Qt. These Qt versus P ClienteCO₂ observations have been made quantitatively and with very good correlation in both controlled experiments in animals.⁸
and in patients undergoing major vascular and cardiac surgery. With cardiac arrest, there is no pulmonary blood flow and therefore no delivery of carbon dioxide to the lungs. Consequently, PetCO₂ exponentially decreases over a dozen breaths, and there is no steady state exhaled CO₂ (waveform). However, with external cardiac compression, pulmonary blood flow will begin again, and the amount of CO₂ excreted by the lungs (i.e., PetCO₂) will be proportional to the amount of pulmonary blood flow (see above). Indeed, the efficacy of external cardiac compression can be continuously and quantitatively followed by the amount of CO₂ excreted (Figure 2, top panel). Exhaled CO₂ during cardiopulmonary resuscitation can also be used prognostically. In one study, the initial PetCO₂ was 19 mmHg in those who eventually regained spontaneous pulses, but only 5 mmHg in those who did not (P < .0001). Furthermore, a sharp increase in ETCO₂ often heralded and was the first indicator of the resumption of spontaneous circulation. Almost identical data were found in yet another study (Figure 2, bottom panel). Figure 3 shows all of the above, in addition to the effects of sodium bicarbonate infusion, in a patient undergoing ventricular fibrillation, cardiopulmonary resuscitation, and resumption of a spontaneous circulation. Thus, capnography provides an instantaneous and continuous guide to the efficacy of external chest compression and the resumption of spontaneous pulmonary perfusion.

Phase I: The inspiratory baseline

The inspiratory baseline is traced as fresh gas moves over the CO₂ sensing or sampling site (Figure 1). The CO₂ level during this phase should be zero; if it is not, CO₂ is being rebreathed. This may be intentional and/or a characteristic (desirable or undesirable) of the equipment being used. The inspiratory baseline becomes elevated if CO₂ is added to the fresh inspired gas. A CO₂ rebreathing or by-pass valve (which is present on older anesthesia machines) is open, if the CO₂ absorbent is partially exhausted or gas is channeling through the absorbent, if the expiratory valve is missing or incompetent (exhaled gas in the exhalation limb goes back into the patient's lungs during inhalation, thereby pulling CO₂-containing gas by the sampling site during inhalation), or a Bain circuit is being used.

Phase II: The expiratory upstroke

Soon after exhalation begins, CO₂-containing gas arrives at the CO₂ sampling site, and it quickly washes away the fresh gas from the previous inspiration. Thus, the expiratory upstroke is steep (Figure 1). When the expiratory upstroke phase of the capnogram becomes prolonged (i.e., the upstroke becomes less steep), delivery of CO₂ from the lungs to the CO₂ sampling site is delayed. Possible causes include mechanical obstruction in the equipment, such as a kinked endotracheal tube, or slow emptying of the lungs, such as with chronic obstructive pulmonary disease or bronchospasm. The expiratory upstroke also becomes prolonged when a side-stream capnograph samples gas too slowly or when the capnograph has a slow response time and the respiratory rate is fast.

Phase III: The expiratory (alveolar) plateau

The expiratory plateau should detect mixed alveolar gas at the CO₂ sampling site. During initial exhalation, elimination of gas from the anatomical dead space (infinite V/Q, zero CO₂ concentration) is followed by elimination of gas from the well-ventilated, low resistance regions of the lung (relatively high V/Q, low CO₂ concentration). Later, gas from poorly ventilated, high resistance regions of the lung (relatively low V/Q, high CO₂ concentration) is eliminated. The continuum of V/Q ratios between high and low V/Q areas creates a positive slope (upward to the right) to the alveolar plateau part of the CO₂ elimination waveform, with the result that the ETCO₂ concentration is the last and
highest (the peak) concentration on the alveolar or the expiratory plateau (Figures 1 and 4). In addition, continued production and evolution of CO₂ into the alveolar space during exhalation contributes to the rise in CO₂ concentration during exhalation. Theoretically, the slope is such that the end-tidal value should be about 2% higher than the time-weighted mean in normal resting subjects and about 4% higher in exercising subjects, assuming tidal volumes are large enough to displace dead space.

Since the alveolar plateau expresses the V/Q continuum in the lung, analysis of the alveolar plateau may result in a wealth of diagnostic information. First, the steepness of the alveolar plateau is directly related to the degree of airway resistance. Second, biphasic waveforms may reveal the presence of a two-compartment lung. Third, leaks in the sampling system may alter the alveolar plateau in a characteristic, and at first glance, peculiar way. Fourth, the $P_{acO_2}$-$P_{etCO_2}$ gradient is directly related to alveolar dead space.

The steepness of the slope of the alveolar plateau is a function of expiratory resistance. When the lungs of a patient without lung disease are being mechanically ventilated, the V/Q units in the lung are relatively uniform and homogeneous (have the same CO₂ concentration), and the expiratory plateau is smooth and nearly horizontal. However, when there is significant lung disease and a wide spread in V/Q ratios within the lungs, very well-ventilated, high V/Q, low CO₂ concentration areas will empty first, causing the alveolar plateau to be relatively low. Following this, very poorly ventilated, low V/Q, high CO₂ concentration areas empty causing the alveolar plateau to be relatively high. Thus, with a wide spread in the V/Q ratios, the upward positive slope of the alveolar plateau will be very steep to the right (it is possible, but unusual, for there to be simultaneous emptying of alveoli with very different V/Q ratios and therefore a minimal slope to the expiratory plateau). Thus, it is not surprising that the increase in airway resistance that is associated with bronchospasm,
which causes a wide spread in the V/Q ratios and emptying times, to be tightly correlated with an increase in the slope of the alveolar plateau (see Figure 4).18

**Figure 4**

*V/Q ratio shifts as a result of bronchospasm*

Bronchospasm spreads the distribution of $R_{airw}$, V/Q ratios, and $[CO_2] \rightarrow \uparrow$ slope phase III

![Diagram showing V/Q ratio shifts as a result of bronchospasm](Reprinted with permission from Nichols K, Benumof JL.19)

The phase III alveolar plateau slopes upward to the right due to a spread in V/Q ratios from high (early phase III) to low (late phase III). Bronchospasm spreads the distribution of V/Q ratios and increases the slope of phase III.

- **Biphasic waveform in patients who have markedly different individual lungs.** Theoretically, if the continuum of the V/Q ratios is broken into two distinctly different lung regions (a low-resistance, high V/Q, low CO$_2$ concentration region and a high-resistance, low V/Q, high CO$_2$ concentration region), a biphasic CO$_2$ excretion waveform might be expected. Situations in which such a biphasic CO$_2$ waveform have been described are the lateral decubitus position in which the nondependent lung has relatively low airway resistance, high V/Q ratio, and low CO$_2$ concentration, compared with the dependent lung, in a patient with severe rotary kyphoscoliosis causing severe compression of one lung (Figure 5),19 and a major mainstem bronchial intubation (Figure 6).20

Some patients with chronic obstructive pulmonary disease may also display a slight biphasic expiratory plateau if they have, throughout both lungs, two distinct populations of alveoli with very different time constants. In this situation, rapidly exchanging alveolar spaces are over-inflated during inspiration (their compliance is high) so that their CO$_2$ concentration is low, whereas slower exchanging alveoli empty only during the latter part of exhalation, releasing a higher CO$_2$ content.21 In patients with active expiratory efforts, a similar pattern may also be precipitated by airway closure due to increased intrapleural pressure during expiration.21 Finally, spontaneous breathing effort during a mechanical positive-pressure breath will create a cleft in the expiratory plateau and therefore a biphasic appearance.

**Figure 5**

*Biphasic CO$_2$ excretion waveform during manual intermittent positive-pressure breathing*

[Graph showing CO$_2$ waveform](Reprinted with permission from Gilbert D, Benumof JL.20)

Left panel (read right to left): Biphasic capnogram during right mainstem bronchial intubation. Each horizontal line indicates 10 mmHg CO$_2$ concentration. The first CO$_2$ concentration peak ranged from 21 to 23 mmHg, and the second CO$_2$ concentration peak ranged from 26 to 29 mmHg. No spontaneous ventilation was apparent during this period of controlled ventilation.

Right panel (read right to left): Normal appearing capnogram after the tip of the endotracheal tube was pulled back above the tracheal carina. Each horizontal line indicates 10 mmHg CO$_2$ concentration; the end-tidal CO$_2$ concentration at the end of this breath was 28 mmHg.
Sampling line leak = peculiar waveform. When there is a leak in the sampling line and the sampling line can entrain room air, the alveolar plateau will be artificially low. In addition, when the next inspiration forces gas through the sampling line at a faster rate so that room air is no longer entrained, a peak will follow the low plateau and the peak will be equal to the true ET-CO₂ (undiluted end-tidal gas being pushed through the sampling line) (Figure 7).  

The PaCO₂-PETCO₂ gradient = alveolar dead space. The PaCO₂-PETCO₂ gradient is a function of the temporal sequence of alveolar emptying (i.e., the slope of the phase III of the single breath test and how high the CO₂ concentration rises) and the total dead space in the lung.  

The alveolar concentration of CO₂ (PaCO₂) is ordinarily just slightly less than the PVCO₂ but slightly higher than Paco₂ (Figure 8). However, the alveolar concentration of CO₂ is ordinarily diluted by alveolar and anatomical dead space gas that has no CO₂ in it so that the ET-CO₂ ordinarily is less than the Paco₂ (Figure 8). If the lungs are small (reduced functional residual capacity) and homogenous (normal V/Q relationship and no alveolar dead space), and the anatomical dead space is small (these conditions occur in healthy, supine, term, pregnant women about to undergo cesarean section, patients having postpartum tubal ligations, and women in early pregnancy) and in exercise, then Paco₂ is only diluted to a small extent by CO₂ free gas and PETCO₂ may be greater than PaCO₂. The existence of negative arterial to end-tidal Paco₂ gradients is best understood if it remembered that Paco₂ represents the temporal and spatial mean alveolar Pco₂ (i.e., physiologic integrator), whereas the ET-CO₂ is the highest (peak) alveolar Pco₂ coming from slow and low V/Q areas (especially when the tidal-volume is large and the respiratory rate is low). However, normally the amount of anatomical dead space is large enough so that the PaCO₂-PETCO₂ is slightly positive (by 2 to 4 mmHg).

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**Figure 7**

**CO₂ excretion waveforms**

Top panel: Photograph of a CO₂ excretion waveform when the CO₂ sampling line was loosely connected to the CO₂ analyzer sample port. The CO₂ excretion waveform consists of a long low plateau followed by a brief peak. This patient was being ventilated with an inspired O₂ concentration of 30% and an inspired N₂O concentration of 70% (as indicated by flowmeter settings).

Bottom panel: Photograph of the CO₂ excretion waveform from the same patient as in the top panel, but when the connection between the CO₂ sample line and CO₂ analyzer sample port was made tight. The CO₂ excretion waveform is now almost square wave or rectangular. Note that the mean exhaled O₂ and N₂O concentrations are now near the inspiratory settings.

(Reprinted with permission from Zupan J, Martin M, Benumof JL.)

**Figure 8**

Diagram of PaCO₂-PETCO₂ gradient

The PaCO₂-PETCO₂ gradient is directly proportional to the amount of alveolar dead space because CO₂-free gas from alveolar dead space dilutes CO₂-containing gas from gas exchanging alveolar ventilation (PaCO₂). The origin of PACO₂ is CO₂ in the mixed venous blood (PVCO₂) and decreases the concentration, usually below the arterial level (PaCO₂).
As alveolar dead space progressively increases, the $\text{PaCO}_2 - \text{PETCO}_2$ gradient progressively increases. In patients with pulmonary disease, the $\text{PaCO}_2 - \text{PETCO}_2$ gradient increases unpredictably by 10 to 20 mmHg or more with the result that $\text{PETCO}_2$ is no longer a reliable reflection of the effectiveness of ventilation and $\text{PaCO}_2$. 

Although trends in $\text{PETCO}_2$ may still be useful, one author found that the trends were not useful in patients with severe lung disease. The reasons trends in $\text{Paco}_2 - \text{PETCO}_2$ may not follow $\text{Paco}_2$ in patients with severe lung disease is because a fall in $\text{PETCO}_2$ may be associated with an equal fall in $\text{Paco}_2$ (no change in distribution of V/Q), with a greater fall in $\text{Paco}_2$ (due to better ventilation and improvement in V/Q matching), or with a constant or increased $\text{Paco}_2$ (due to increased dead space and worsening of V/Q matching). All authors agree that in patients with severe respiratory failure, the $\text{Paco}_2 - \text{PETCO}_2$ gradient is usually a good index of efficiency of ventilation and VD/VT. Causes of increased alveolar dead space include decreased pulmonary blood flow (decreased cardiac output, and/or pulmonary artery pressure), pulmonary embolization, kinking of pulmonary vessels, thrombosis in the pulmonary circulation, application of positive end-expiratory pressure, pulmonary artery pressure causes a decrease in upper lung has more volume to empty (i.e., it makes a greater contribution to late expiration). Incision of the chest wall produced an increase in mean pulmonary artery pressure that was associated with an increase in $\text{CO}_2$ elimination by the upper lung and a decrease in dead space. Consequently, a surgical stimulation-induced increase in mean pulmonary artery pressure causes a decrease in upper lung $\text{PaCO}_2 - \text{PETCO}_2$.

**Phase IV: The inspiratory downstroke**

Soon after inspiration begins, fresh gas from the breathing circuit washes $\text{CO}_2$ from the previous exhalation away from the $\text{CO}_2$ sampling site. Because the volume of gas at the sampling site is small, the inspiratory downstroke (like the expiratory upstroke) is steep. A missing inspiratory valve or an inspiratory valve that is incompetent during exhalation allows $\text{CO}_2$-containing gas to go up the inspiratory limb. In this case, during inspiration the $\text{CO}_2$ previously exhaled into the inspiratory hose is pushed back into the patient's lung and the inspiratory downstroke becomes prolonged and slanted.

**Summary**

The capnogram is an extremely useful breath-by-breath monitor of $\text{CO}_2$ exhalation. As such, it should be considered by anesthetists as a vital sign. Furthermore, the diagnosis of significant physiology and pathophysiology is contained within the shape of the capnogram. The diagnosis of this pathophysiology is best made by a systematic analysis of the four phases of the capnogram.

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