End-tidal CO$_2$ monitoring during anesthesia

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End-tidal CO$_2$ monitoring by infrared spectroscopy or mass spectrometry represents an increasingly popular mode of intraoperative patient assessment. This article examines the respiratory physiology and physics principles underlying end tidal CO$_2$ monitoring, and interpretation of the useful data subsequently gained by the anesthetist. Accuracy of endotracheal tube placement, adequacy of ventilation, machine malfunction and metabolic state can all be assessed through this monitoring modality.

The normal capnogram

The ability to monitor the end-tidal CO$_2$ content (P$_{End}$CO$_2$) of expired gas either by infrared analysis or mass spectrometry has become more readily available to the anesthetist in recent years due to technological advances in medical instrumentation. This additional monitor of respiratory and physiological function can enhance patient safety if the underlying scientific principles are understood by the practitioner so that interpretation of the resulting data is facilitated.

Continuous monitoring of tidal exchange with respect to CO$_2$ concentration (in volumes % or torr) will show that CO$_2$ concentration falls sharply on inspiration of ambient air or fresh anesthesia gases to which no CO$_2$ has been added and/or from which CO$_2$ has been effectively removed. As expiration commences, the CO$_2$ concentration will rise in a linear fashion until a constant plateau is reached. This is because the first gas exhaled will be the dead space gas from conducting airways, containing little (or no) CO$_2$, followed by mixing of alveolar gas (from respiratory exchange units) with dead space gas, until only alveolar gas is being exhaled near the end of expiration.  

Thus, in healthy human subjects with normal lung function, the expired CO$_2$ concentration will rise with exhalation and fall with inspiration in a periodic fashion. Plotting the expired CO$_2$ concentration as a function of time results in a curve called a capnogram (Figure 1). Normally, as seen in Figure 1, the curve rises sharply with initial expiration as mixed dead space and alveolar gas is exhaled, then levels off and forms a plateau near the end of expiration. In the absence of significant lung disease, this plateau concentration represents the truest approximation of alveolar CO$_2$ concentration. This is because in this late period of tidal volume exhalation (termed “end-tidal”), the monitor is now “seeing” only alveolar gas. The presence of lung disease, as we shall consider later, can alter the capnogram significantly and can make interpretation difficult. Analysis of the end-tidal portion of the capnogram produced by an accurate, properly calibrated CO$_2$ monitor, in a patient with no lung disease will yield the accurate end-tidal CO$_2$ in torr or volumes %. Normal P$_{End}$CO$_2$ is 37-40 torr or 5.1-5.6 volumes %. To convert from torr to volumes %, divide the P$_{End}$CO$_2$ value in torr by
713 torr (H₂O-saturated barometric pressure, found at the alveolar level).

**Methods of CO₂ analysis**

There are two methods of CO₂ analysis commonly used at present (Table I). The first, and most common, is infrared spectroscopy of expired gases. Spectroscopy measures the vibrational frequency of atoms within the molecule which has resulted from absorption of infrared radiation at frequencies of 10¹³ to 10¹⁴ cycles per second. The mode of vibration is a function of the spatial arrangement, valence forces, intermolecular forces and collision between molecules, all of which may alter the character of the intermolecular vibrations.

Absorption of infrared energy by a given gas such as carbon dioxide produces an infrared spectrum consisting of a number of energy bands, whereby the identity and concentration of carbon dioxide are discerned by the end-tidal gas monitor. Collisions between carbon dioxide and other gases such as nitrous oxide can affect the molecule rotation, and hence the energy absorption at corresponding wave lengths. This effect, commonly termed "collision broadening," results in an increase in the amount of energy absorbed at these wave lengths in the rational bands of carbon dioxide, and may introduce a correctable error in the PETCO₂ monitor. Pendergrast et al. recommend calibrations of the CO₂ monitor with a known concentration of CO₂ in a mixture of either N₂ or N₂O to reduce this error.

The second method of PETCO₂ analysis is mass spectrometry. Until recently, mass spectrometers were too costly and unreliable for continuous respiratory gas monitoring in the operating room. However, with recent technological advances, the monitoring cost per patient can now be reduced to a reasonable level by the use of one central unit to monitor ORs sequentially, and this method of gas monitoring is becoming increasingly popular. Mass spectrometers, unlike infrared monitors, typically monitor multiple different expired gas tensions simultaneously, including CO₂.

In a typical mass spectrometry system for operating room use, the gas is drawn into an analyzer chamber through a needle valve, where it is subjected to a "high-vacuum" of approximately 10⁻⁶ mmHg, resulting in intermolecular distances of approximately 1 meter. The molecules are then bombarded by an electron beam creating a charged ion, which is accelerated by an electrostatic field. The ion then passes through a powerful magnetic field producing a curved path dependent upon the mass:charge ratio of the ion. A small metal collecting plate is placed in the path of ion species of interest; the composition of the sampled gas is determined by dividing the total number of "hits" by the number of "hits" per unit time recorded electronically within the spectrometer. Unfortunately, several gases of interest in the anesthetized patient overlap in their mass:charge

<table>
<thead>
<tr>
<th>Table I</th>
<th>PETCO₂ monitors</th>
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<td><strong>Method</strong></td>
<td><strong>Advantages</strong></td>
</tr>
<tr>
<td>Infrared spectroscopy</td>
<td>Low cost, Portable, Non-invasive, Reliable</td>
</tr>
<tr>
<td>Mass spectrometry</td>
<td>Monitors all gases, Non-invasive</td>
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ratio, in particular, CO₂ and N₂O, both with an atomic mass of 44 units. Manufacturers have devised methods of differentiating between these two ubiquitous gases. When N₂O is bombarded, some nitric oxide (NO, mass 30) is generated; when CO₂ is bombarded, some carbon (C, mass 12) is created. Thus, looking for N₂O at mass 30 and CO₂ at mass 12 will differentiate between these two gases.⁶

The P₂₆CO₂ can be monitored by observing a gauge or dial indicating intermittent end-tidal values of CO₂ either in volumes % or torr, or through the generation of a capnogram. Some P₂₆CO₂ monitors have a write-out attachment while others (usually mass spectrometers) will produce an image of the capnogram on an oscilloscope screen.

In both types of monitoring systems, gases are aspirated for analysis from a location in the ventilatory circuit as close to the patient’s endotracheal tube as possible to enhance accuracy of end-tidal values (Figure 2). The gases are transported down a narrow-bore tubing to the monitor itself, where the analysis process is undertaken.

As with any monitor, potential problems exist within the mechanism itself. The narrow-bore aspiration tubing must be kept free of obstruction by mucus, condensed water or kinking, otherwise the end-tidal valves and waveform may be distorted and lead to inaccurate patient assessment. Proper calibration is essential, as indicated by Pendergrast and colleagues, to eliminate problems of inaccuracy associated with nitrous oxide use. The presence of abnormal values of P₂₆CO₂ should lead the clinician to assess all possible explanations, including monitor malfunction.

Another potential source of problems is the aspiration mechanism by which gases are drawn into the CO₂ monitor. Gases are generally aspirated at rates varying from 100-250 cc/minute, and if these rates vary due to pump malfunction, distorted values and signals may result. In very small neonates, aspiration of even this small amount of gas might cause loss of tidal volume to the patient; this should be borne in mind when use is contemplated.

### Physiological principles and end-tidal CO₂ analysis

Gaining an understanding of the basic respiratory physiology and factors influencing the P₂₆CO₂ concentration will assist the anesthetist in effective interpretation of this monitoring modality.

First, it is important to realize that a gradient will usually exist in anesthetized patients such that arterial CO₂ levels (PaCO₂) are higher than P₂₆CO₂ levels, due to dilution of exhaled alveolar gases with dead space gases from the conducting airways and non-perfused alveoli.³ Most studies have shown this gradient to be quite small, on the order of 0.5-6 torr.⁸-¹⁰ Investigators have also shown that, though a gradient exists, changes in PaCO₂ will be paralleled by similar changes in P₂₆CO₂, allowing trends to be observed and followed.⁸

Thus, any factor which affects dead space (Vₚ) will also affect P₂₆CO₂. Nunn and his colleagues¹⁰ demonstrated that in fit, anesthetized subjects, Vₚ increased in direct proportion to tidal volume, so that larger tidal volumes may yield an increased PaCO₂ to P₂₆CO₂ gradient. This classical study also demonstrated an increase in physiological dead space during anesthesia, probably due to alterations in ventilation to perfusion ratios (Vₚ/Q) of alveoli throughout the lungs.

Vₚ can also be increased by lung disease. Pulmonary emphysema can result in increased dead space through destruction of alveolar walls and lung parenchyma with resultant loss of radial traction and lung recoil.¹¹ Diffusion difficulties from thickened alveolar walls or alveolar fluids may also contribute to the widened PaCO₂-P₂₆CO₂ gradient sometimes seen in lung disease. Takki et al.⁸ demonstrated that even in patients with chronic lung disease, P₂₆CO₂ bears a constant relationship to PaCO₂ and can be used as an assessment of adequate ventilation.

The relative PaCO₂-P₂₆CO₂ gradient may be reduced somewhat from the reduction in anatomical dead space resulting from endotracheal intuba-
tion and exclusion of upper airway structures from ventila-
tory exchange.\textsuperscript{1,10} This fact points to in-
creased accuracy of $P_{ET}CO_2$ in estimating ventila-
tory adequacy.

Decreases in pulmonary perfusion will result in in-
creased physiological dead space, and thus reduced $P_{ET}CO_2$. Pulmonary embolus may cause this to occur. Reductions in cardiac output due to deep anesthesia, hypovolemia or shock may reduce pulmonary perfusion and $P_{ET}CO_2$ values. Conversely, increased cardiac output, which may be seen with heightened sympathetic nervous system tone from surgical stimulation or lightening anes-
thesia, will result in elevated $P_{ET}CO_2$ values.

Finally, $P_{ET}CO_2$ is also affected by pulmonary
ventilation and $CO_2$ production ($V_{CO_2}$). The rela-
tionship between $PaCO_2$ and alveolar ventilation ($V_A$) is defined as follows:\textsuperscript{1}: $PaCO_2 = \frac{V_{CO_2}}{V_A}$

Let us consider the effect of each of these variables on $P_{ET}CO_2$. First, we have discussed the fact that, though a gradient usually exists, $P_{ET}CO_2$ will cor-
respond to $PaCO_2$ in a linear fashion.\textsuperscript{8}

Pulmonary $V_{CO_2}$ is dependent upon metabolic (tissue-level) $V_{CO_2}$, determined by the rate of aero-
bic metabolism. As the metabolic rate increases, as in hyperthermia or exercise,\textsuperscript{12} this increased meta-
bolic $V_{CO_2}$ will result in elevated $CO_2$ transport in
the mixed venous blood and excretion at the lung, reflected in rising $P_{ET}CO_2$ values. The converse is true with reduced metabolic rates, as in hypo-
thermia.

Alveolar ventilation is inversely related to $PaCO_2$\textsuperscript{1}. Thus, in hypoventilation, $PaCO_2$ will be elevated, with a corresponding increase in $P_{ET}CO_2$
(see the equation just presented), until ventilation falls so low that effective exchange of $CO_2$ is pre-
cluded, at which point $P_{ET}CO_2$ values will decrease

Figure 3
Capnogram in child anesthetized with halothane

\[ \text{Figure 3} \]
Capnogram in child anesthetized with halothane

\[ \begin{align*}
\text{A.} & \quad \begin{array}{c}
\text{9:15 AM} \\
0.75\% \text{ Halothane}
\end{array} \\
\text{B.} & \quad \begin{array}{c}
\text{9:30 AM} \\
2\% \text{ Halothane}
\end{array}
\end{align*} \]

A. shows adequate spontaneous ventilation at 0.75\% halothane. B. shows obviously depressed respirations at 2\% halothane, with attendant hypercarbia.

As seen in Figure 3, capnography allows for immediate observation of elevated $P_{ET}CO_2$ values in anesthetized, spontaneously ventilating patients and can assist the clinician in recognizing hypercarbia so that it can be treated by assisting ventilation to avoid potential arrythmias.

**Physiological changes and the capnogram**

Based upon the physiology described above, it is evident that end-tidal $CO_2$ monitoring is ex-
tremely useful as a diagnostic tool in several situations unique to anesthesia. First, and most im-
portant, is the role of $P_{ET}CO_2$ monitoring in ver-
ification of intratracheal placement of breathing tubes.\textsuperscript{14} Situations arise in clinical anesthesia prac-
tice in which endotracheal tube placement may be
difficult, and once the tube is inserted, location can be questionable. For example, in very small infants, in patients with craniofacial or anatomical airway abnormalities, and in the grossly obese patient, problems can arise with endotracheal tube placement. Once the tube has been inserted, if CO₂ is present in expired gases in the appropriate concentration of 4 to 6 volume % (28-42 torr), then intratracheal tube placement and pulmonary ventilation are assured. Ventilation of the esophagus would result in essentially no measurable end-tidal CO₂ concentration. Likewise, occlusion of the lumen of an endotracheal tube, inadvertent extubation, or disconnection of the breathing circuit during continuous capnography results in gradual or abrupt loss of the capnogram tracing. These events are illustrated in Figures 4 and 5.

Air embolus is a second important physiological event with an impact on the capnogram which may occur during anesthesia in patients in the sitting position. Veins located above the heart collapse with negative pressure; surgical openings may allow entrainment of air which then moves through the venous system. Blockage of the pulmonary circulation by air bubbles results in an increase in dead space proportional to the size of the embolus, thus reducing the alveolar and end-tidal CO₂ concentration. PₚₑₜCO₂ monitoring reliably detects this reduction and is a recommended mode of monitoring in neurosurgical cases requiring the sitting position. Air embolus is typically heralded by the onset of a mill-wheel murmur (best detected by a precordial Doppler flowmeter), and is associated with a sharp decrease in the capnogram and end-tidal CO₂ values (Figure 6). Adequate blockage of the open venous vents and embolus resolution can be verified by murmur disappearance and rise in the capnogram values to pre-embolic levels. Brechner et al. were the first investigators to identify air embolization in an early stage by utilizing end-tidal CO₂ monitoring. Early identification of the problem can prevent the disastrous entrainment of massive amounts of air.

Malignant hyperthermia represents a third situation, unique to anesthesia and potentially fatal, which end-tidal CO₂ monitoring can detect. Though the exact pathophysiology of malignant hyperthermia has yet to be elucidated, it is believed that triggering agents such as succinylcholine and potent inhalation anesthetics interfere with the excitation-contraction cycle of skeletal muscle. Thus, after initial calcium release from the T-tubules, which inhibits troponin, the actin and myosin filaments interact and contraction occurs. Malignant hyperthermia triggering agents interfere with the normal sequence of events such that calcium concentration does not fall normally following contraction, and sustained muscle contraction leading to rigidity and hypermetabolism results.

This hypermetabolic state results in a rise in temperature and increased CO₂ production as the end-product of aerobic metabolism. In a number of documented cases, the initial presenting sign of malignant hyperthermia was an unexplained increase in end-tidal CO₂ concentration in the face of unchanged ventilation. Thus, end-tidal CO₂ monitoring can be an extremely valuable diagnostic tool for detecting this dangerous pharmacogenic disease. An unexplained rise in the end-tidal CO₂ concentration can alert the practitioner to a potential malignant hyperthermia episode, which may then be definitively diagnosed in the face of other developing symptoms and treated before progression to a critical situation.

Observation of the shape and magnitude of the PₚₑₜCO₂ waveform can yield information about the clinical condition of the patient. To illustrate this point, the onset of bronchospasm in a suscep-

![Figure 6](image-url)

**Figure 6**

Capnography during air embolism

Vol% CO₂ 5 4 3 2 1 0

Sudden decrease in PaCO₂ indicating pulmonary air embolism. (Adapted with permission from Brechner, et al.)

![Figure 7](image-url)

**Figure 7**

Capnogram in normal lung function (A) versus obstruction due to bronchospasm (B)

A. Note sharp upstroke on expiration, indicating patient airway, even emptying of alveolar space.

B. Sloping upstroke on expiration indicates obstruction and delayed expiratory flow. Lack of definite plateau reflects uneven emptying of alveolar space.

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tible patient is heralded by gradual sloping of the initial upstroke of the capnogram due to prolonged expiration (Figure 7). This alerts the clinician to the changing condition of the patient's airway. As we see under B in Figure 7, in some patients with obstructive lung disease, the plateau phase is never reached due to delayed and incomplete emptying of partially obstructed alveoli. Gradually sloping expiratory upstrokes and loss of defined end-tidal plateau are capnographic changes which may signal the onset of bronchospasm in susceptible patients.

The cleft in the capnogram seen in Figure 8 is typically seen when breathing resumes with partial curarization of the diaphragm. Thus, this form of tracing may occur intraoperatively when patients are under light anesthesia and "buck" or at emergence during reversal of residual neuromuscular blockade, and may serve as a guide to adequacy of ventilatory effort.

As previously described, decreases in cardiac output are associated with corresponding decreases in $P_{ETCO_2}$. Thus capnography can serve as an additional monitor of cardiovascular function. In Figure 9, the capnogram wave form is seen to decrease in a child in response to elicitation of the oculocardiac reflex. This decrease in $P_{ETCO_2}$ corresponds to bradycardia and decreased cardiac output, and signals the need for therapeutic intervention.

### Anesthesia machine function and end-tidal CO₂ monitoring

End-tidal CO₂ monitoring can be useful in detecting anesthesia machine and ventilator malfunction. In some institutions, end-tidal CO₂ monitors are utilized as ventilator disconnect alarms. Thus, when the patient is placed on a mechanical ventilator, capnographic wave forms are monitored constantly. Values that are too high, associated with a steep capnogram tracing, may reflect inadequate ventilation or hypermetabolism and can be set to trigger an alarm. Values that are too low, or a flat capnogram tracing, can reflect entrainment of room air through a system leak, hyperventilation, or in the case of a flat tracing (as in Figure 4), possible ventilator disconnect. Low values also can be set to trigger an alarm. Thus, capnography can serve as an additional monitor of overall patient safety.

Inspection of the inspiratory and expiratory portions of the capnogram, as well as end-tidal and inspiratory CO₂ concentrations, can reveal anesthesia machine ventilatory malfunctions. For example, if incompetent circle system valves do not maintain unidirectional gas flow through the soda lime absorber, or if the soda lime is old or packed incorrectly with channeling of gases, the inspired concentration of CO₂ will rise. This is due to rebreathing and/or inefficient exposure of CO₂-laden expired gases to the soda lime for absorption. The end-tidal CO₂ monitor will reveal a rising concentration of CO₂ if it is set to monitor inspired gases and a continuous capnogram will show an elevated inspiratory baseline (Figure 10). This in-

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**Figure 8**

The capnographic changes seen during reversal of neuromuscular blockade (spontaneous ventilation). The cleft is thought due to lack of coordination of intercostal muscles and diaphragm.

**Figure 9**

Capnogram during the elicitation of the oculocardiac reflex in a child of 2 years

<table>
<thead>
<tr>
<th>Time (sec)</th>
<th>Vol% CO₂</th>
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<tbody>
<tr>
<td>1</td>
<td>2</td>
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<td>2</td>
<td>3</td>
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<td>9</td>
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<td>9</td>
<td>10</td>
</tr>
</tbody>
</table>

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**Figure 10**

Elevated baseline (inspiratory) values of CO₂ indicate rebreathing of CO₂ in A. A faulty valve was discovered and replaced, returning the capnogram to normal in B.
icates to the anesthetist that CO₂ is present in the inspired gases, which may cause hypercarbia, a condition that is potentially deleterious to the patient.

To summarize, end-tidal CO₂ monitoring is a valuable new non-invasive, low risk assessment tool for the anesthetist. The PETCO₂ closely "tracks" PaCO₂ values and reflects the status of ventilation, circulation and metabolism in anesthetized patients. Important physiological changes and anesthesia equipment malfunctions are more easily identifiable when the anesthetist has the ability to interpret and utilize the data from this monitor, and patient safety thus is enhanced.

REFERENCES

AUTHOR
Charles A. Griffis, CRNA, MS, is a graduate of the UCLA Program of Nurse Anesthesia and received his baccalaureate degree in nursing from the University of South Florida in Tampa, Florida. He is currently enrolled as a doctoral student in the College of Education at the University of California, Los Angeles and holds the position of director for the UCLA Program of Nurse Anesthesia. This article is based on Mr. Griffis' presentation at the 1985 AANA Annual Meeting in Anaheim, California.