Capnographic Waveforms in the Mechanically Ventilated Patient

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Summary

A focus on patient safety has heightened the awareness of patient monitoring. The importance of clinical applications of capnography continues to grow, as reflected by the increasing number of medical societies recommending its use. Recognition of changes in the capnogram assists in clinical decision making and treatment and can increase patient safety by alerting the clinician to important situations and changes. This article describes the interpretation of capnograms and how capnogram interpretation influences airway management. Key words: capnography, capnogram, volumetric capnography, carbon dioxide rebreathing, mechanical ventilation. [Respir Care 2005;50(1):100–108. © 2005 Daedalus Enterprises]

Introduction

Carbon dioxide (CO₂) is the most abundant gas produced by the human body. The removal of CO₂ is the primary drive to breathe and a primary motivation for mechanically ventilating a patient. Monitoring the CO₂ level during respiration (capnography) is noninvasive, easy to do, relatively inexpensive, and has been studied extensively. Medical societies, representing anesthesiology,1,2 cardiology,3 critical care,4 pediatrics,5 respiratory care,6 and emergency medicine7 mandate or recommend capnography. The capnogram is a graphical representation of the level of exhaled CO₂, and it reflects both physiologic and anatomical changes. This article reviews the clinical utility of time-based and volume-based capnograms.

History of the Capnogram

Physiologists recognized early on that continuous analysis of CO₂ is important to the measurement and understanding of intrapulmonary gas mixing and ventilation/perfusion relationships. The importance of continuous analysis of CO₂ has been further “enhanced by simultaneous analysis of gas volumes.”8 However, those instruments “are rather complex and . . . not generally available.”8

Elam et al, researching the problem of CO₂ elimination from closed-circuit anesthesia systems, were among the first to report on simultaneously recorded capnography and flow profiles of human respiration.9,10 Their seminal
work on CO₂ homeostasis was published in a series of 4 papers.¹¹–¹⁴ That research included both normal and abnormal characteristics of the capnographic profile and measurements of dead space and alveolar ventilation.

In 1957, Smalhout, who is considered by many the father of clinical capnography, began using capnographs while working in the Central Military Hospital of Utrecht, The Netherlands.¹⁵ During a 7-year period leading up to the publication of his doctoral thesis in 1967,¹⁶ Smalhout collected approximately 6,000 capnograms, which documented numerous shape variations.¹⁵ Smalhout and Kalenda¹⁷ later published an atlas of strip-chart capnograms that illustrated many of the applications of capnography. However, they noted that their research represented “little more than the surface of a deep pool.”¹⁷ That atlas and Smalhout’s frequent lectures on capnography helped popularize the use of the terms “capnography,” “capnograph,” and “capnogram.” Weingarten¹⁸ summarized Smalhout and Kalenda’s contributions:

Under their direction, capnography survived a stormy gestation period as it reached maturity in The Netherlands. It was introduced in the United States at a small private meeting sponsored by a major instrument manufacturer, held in conjunction with the World Congress on Intensive Care Medicine in Washington DC, in May 1978. Five anesthesiologists attended the meeting, 2 of whom concluded that capnography would prove to be of very little value.

The earliest description in the literature of the volumetric capnogram and a method to determine “airway” dead space was from Aiken and Clark-Kennedy, in 1928.¹⁹ In 1948, Fowler,²⁰ described the single-breath test for nitrogen curve, in seeking to use uniform terminology to clarify the “meaning of dead space.” He thus divided the volumetric capnogram curve into 4 phases (I, II, III, and IV). The term “infrared CO₂ meter” was used from the 1950s and to the early 1970s, when the term “capnograph” was derived from the Godart Capnograph.

Single-breath CO₂ curves appeared in the literature as early as 1961.²¹ The concepts of dead space and CO₂ elimination were not presented in a unified framework, however, until Fletcher published his 1980 doctoral thesis²² and later publications.²³ His method became widely known as the single-breath test or single-breath CO₂ curve.

The Normal Capnogram

The normal capnogram has multiple features that allow for clinical interpretation (Fig. 1). There are no widely accepted standards for labeling of the normal capnogram.¹⁷,²⁴,²⁵ A capnogram is a time-tracing of CO₂ concentration. A capnograph has 2 recording speeds. The fast speed is approximately 12.5 mm/s, which allows interpretation of short-term changes. The slow speed is approximately 25 mm/min, which allows for identifying long-term trends. Changes in the capnogram suggest changes in the patient’s condition. Proper interpretation of the capnogram can alert a clinician to important changes and should include at least the examination of 3 key features: baseline starting at zero; sharp increase in CO₂ concentration; and steady alveolar plateau.

An elevation of the baseline (Fig. 2) indicates clinically important rebreathing of CO₂, which may be due to mechanical problems²⁶–²⁸ or therapeutic use of mechanical dead space.

Chronic disease of the lungs and airways prolongs the transitional portion of the capnogram (Fig. 3). Slow-time constant alveolar units continue to mix alveolar gas with dead-space gas. Prolonging the expiratory phase allows the end-tidal CO₂ (PₑTCO₂) to be more reflective of PₐCO₂.
That decrease in the $PETCO_2$-$P_{aCO_2}$ difference represents a decrease in dead space, and that decrease in dead space is helpful when contrasting a spontaneous breath to that of a breath delivered by a ventilator.

Inability to maintain a plateau phase may be indicative of a pneumothorax. Figure 4 illustrates the “melting away” of the alveolar plateau (ie, a dramatic change in the mean alveolar CO$_2$ concentration). After observing a falling blood-oxygen saturation, the clinician ordered a chest radiograph for this patient, who had a chest tube in place. On observation of this “melting away” of the capnogram, the chest tube was stripped. The capnogram then returned to normal and the patient’s oxygen saturation was again stable by the time the portable radiograph arrived at the bedside, so the radiograph procedure was canceled.

**Airway Management**

Capnography can help confirm the placement of an endotracheal tube (ETT) into the trachea (or misplacement into the esophagus), and can be used in various clinical environments, including in the field (by emergency medical services workers), and in the emergency department, intensive care unit, neonatal intensive care unit, and delivery room.$^{29–33}$ During ETT placement the capnogram can rapidly alert the clinician to ETT misplacement in the esophagus. A capnogram from an esophageally-placed ETT is usually a different shape and size than a normal tracheal capnogram. Additionally, capnographic waveforms from the esophagus will generally be present only for a few breaths. The shape difference between tracheal and esophageal placement is clearly visible in adults (Fig. 5)$^{31}$ and neonates (Fig. 6),$^{34}$ with rare exceptions.$^{35}$ CO$_2$ in the esophagus, which is usually present from CO$_2$ in the stomach due to exhaled gas, is removed in seconds. In neonates with low pulmonary perfusion and intracardiac shunt, the capnogram waveform may be diminished in amplitude (Fig. 7). After the initial placement and securing of the ETT, changes in ETT position can cause abrupt changes in the capnogram (Fig. 8). For example, the procedure of turning and flexing a patient to position him for a spinal tap resulted in a right main-bronchus migration of the ETT that was first indicated by a change in the capnogram. This alerted the clinician, who repositioned the ETT and com-
completed the procedure. Rapid recognition of changes in ETT placement can also avoid other problems, such as desaturation. However, though the presence of CO₂ in the ETT increases confidence that the ETT is properly placed, it does not assure that it is properly placed. Endobronchial intubation can have a normal-appearing capnogram. Proper ETT placement should be confirmed with multiple techniques.³⁶

As the lack of CO₂ is used to determine improper ETT placement, the presence of CO₂ is increasingly being used to identify improper nasogastric or oral feeding-tube placement.³⁷–⁴¹

Cardiopulmonary Resuscitation

Capnography has long been used for rapid evaluation of the effectiveness of chest compressions.⁴² CO₂ removal is more effective when a less fatigued person performs the cardiopulmonary resuscitation (Fig. 9). Falk et al⁴³ found changes in CO₂ removal during successive stages of cardiopulmonary resuscitation (Fig. 10). This included the administration of sodium bicarbonate, resulting in the by-product of CO₂ and the successful return to spontaneous circulation, shown as a dramatic increase in CO₂ removal. A mechanical problem during cardiopulmonary resuscitation can be quickly recognized with the aid of a capnogram. Figure 11 illustrates a clinical situation in which the flow to a non-self-inflating resuscitation bag was set too low, so the CO₂ was not being adequately washed out of the bag and there was excessive rebreathing of CO₂, which was identified via the capnogram. The flow was increased and CO₂ returned to baseline.

Mechanical Ventilation

A paper by Carlon et al included a wide range of capnographic waveforms from mechanically ventilated patients,⁴⁴ which supported the view that capnography assists in quickly identifying and resolving clinical and technical problems. We will discuss several of the capnograms from that paper.

Figure 12 illustrates the capnogram pattern from a fluttering expiratory valve, which can be caused by water condensation or pressure compensation by the ventilator. Note that some rebreathing is present.

Figure 13 illustrates conflict between mandatory breaths (ie, delivered by the ventilator) and spontaneous breaths (ie, there was patient-ventilator asynchrony during intermittent mandatory ventilation).
Figure 14 illustrates a situation in which a small respiratory effort was not detected by the ventilator during the expiratory phase. In that situation the triggering sensitivity may need to be adjusted.

Figure 15A illustrates an erratic pattern and varying $\text{PETCO}_2$. The figure shows no alveolar plateau and some rebreathing. Figure 15B shows that the breathing pattern and capnogram were normalized with pressure support of 20 cm H$_2$O. (Adapted from Reference 44, with permission.)

Figure 16 illustrates a chaotic, rapid respiratory pattern, with spontaneous breaths during mandatory (ventilator-delivered) breaths, and rebreathing, which can indicate failure of a weaning trial.

Frequent alteration of a mechanically ventilated patient’s position is common practice. Figure 17 illustrates how changing the patient’s position can affect the capnogram. While supine, the patient had a high $P_{a\text{CO}_2}$ and an abnormal capnogram. In the right-lateral position the capnogram deteriorated even more. In the left-lateral position the capnogram showed a normal waveform, with an alveolar plateau and a much lower $P_{a\text{CO}_2}$. That change in ventilation/perfusion matching allowed ventilator adjustment.

Neonatal Applications

The use of capnography during mechanical ventilation of neonates is less documented in the literature. The additional dead space, weight, mechanical problems, phase delay, and the use of uncuffed ETTs may limit the clinical value of capnography with neonates.

Arsowa et al. presented a series of capnograms that illustrate that the physiologic changes revealed by neonatal capnograms are consistent with the physiologic changes in adults. However, the shape of the normal neonatal capnogram is different (Fig. 18). Because of the smaller dead space and higher respiratory rate, the normal neonatal capnogram has a shorter time at baseline, a sharper rise in $CO_2$ concentration, and little if any alveolar plateau.

Administration of surfactant alters respiratory mechanics and changes the rate of alveolar emptying, which is reflected on the capnogram (Fig. 19). Before the administration of surfactant, the capnogram has an elevated baseline, the transitional phase has a prolonged slope, and there is no alveolar plateau. After the administration of surfactant the capnogram returns to a normal shape.
The capnogram of a neonate with pneumonia shows biphasic emptying of the lung (Fig. 20). Different time constants cause a varying rate of CO₂ removal.

A normal capnogram with a large difference between P\textsubscript{ETCO\textsubscript{2}} and P\textsubscript{aCO\textsubscript{2}} indicates substantial physiologic dead space (Fig. 21). Right-to-left cardiac shunt diverts blood away from the lung. Cardiac shunt reflects an increase in pulmonary dead space.

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The normal volumetric capnogram has the same general features as the time-based capnogram. The volumetric capnogram, which traces CO\(_2\) concentration against exhaled volume, is divided into 3 phases (Fig. 22). Using features of each phase, physiologic measurements can be calculated.

Changes in positive end-expiratory pressure (PEEP) affect the volumetric capnogram (Fig. 23). Increasing PEEP shifts the capnogram and alters the slope of phase II. Changes in pulmonary blood flow can also shift the volumetric capnogram (Fig. 24). Currently, the shift is not clinically quantifiable, but optimal PEEP, perfusion, or drug therapy may be determined by measuring that change in slope.

The volumetric capnogram also reflects changes in airway resistance (Fig. 25). A patient with severe asthma demonstrates a prolonged transitional phase, which improves over time. Drug therapy may also be monitored, as with time-based capnography.\(^48\)

Concave phase-III volumetric capnograms have been seen with obese patients and patients with increased expiratory resistance. Obese patients (Fig. 26) can have biphasic emptying and higher \(P_{ETCO_2}\) than \(P_{aCO_2}\). That difference suggests varying mechanical and ventilation/perfusion properties. The increase in expiratory resistance (Fig. 27) may reflect a slow expiratory phase with a slow accumulation of alveolar CO\(_2\). The alveoli that empty last may have more time for CO\(_2\) diffusion.\(^49\)

The volumetric capnogram has been used to diagnose pulmonary emboli. Extrapolated CO\(_2\) at 15% of total lung capacity (TLC) (late dead space). \(V_T\) = tidal volume. (Adapted from Reference 50.)

The capnogram is an important tool for the clinician, in that it aids diagnosis and treatment and can increase patient safety by alerting the clinician to important situations and changes. The present review touches only on a few of the published works on capnography. Smalhout’s collection of 6,000 capnograms illustrates a wide...
range of capnographic possibilities. Smalhout states that the capnogram is the ECG of respiration. Just as the electrocardiogram is one indicator of cardiac health, the capnogram is an important adjunct to assessing respiratory function. Hopefully, additional research from clinicians will further the field and interpretation of capnograms.

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R E S P I R A T O R Y  C A R E  •  J A N U A R Y  2 0 0 5  V O L  5 0  N O  1


Discussion

Bigatello: You mentioned that you put $P_{ETCO_2}$ on-line when you place feeding tubes. I’m interested in that, because a feeding tube can go into the lungs, and a few of them also cause damage. We have a very specific protocol according to which, with intubated patients, we place every feeding tube under direct laryngoscopy. I do not have data to know whether that has decreased the incidence. But certainly measuring $P_{ETCO_2}$ will be a possible adjunct. How do you fit your particular apparatus? Does it work well for you? Is it something that you suggest on a regular basis?

Thompson: There are several publications that describe the connections for adult patients.1–4 Our protocol calls for getting aspirates and measuring the pH before we use the feeding tube.

REFERENCES


Harris: You showed a graph of the time to a steady-state CO$_2$ reading. How long does it normally take for CO$_2$ to equilibrate when you make a ventilator change? It always seems to take a very long time. And what does it depend on?

Thompson: In the majority of cases the change takes place within 20 minutes, but some patients come to full stabilization in 5–7 minutes.¹

Blanch: I have a question regarding the pediatric population. Assessing-physiologic dead space in ARDS [acute respiratory distress syndrome] is useful to predict outcome. Do you know whether measuring dead space could also be useful in pediatric patients?

Thompson: Predicting outcome is very difficult. In pediatrics we have only about a 10–15% mortality rate with pediatric ARDS. I’m not sure that we’re going to ever show any impact of these devices on pediatric ARDS. The low incidence, high survival rate, and the use of ECMO [extracorporeal membrane oxygenation] limits the number of patients available for study.

Sanborn: Is it fair to say that CO$_2$ monitoring is mainly used as a check on what you’ve done rather than as a guide to doing something? It seems that if you make a ventilator change, CO$_2$ monitoring tells you whether your adjustment was OK, rather than looking at the CO$_2$ signal in whatever way and saying, “Oh I need to do this.”

Thompson: I would say that’s true with a single-breath CO$_2$ measurement, looking at CO$_2$ elimination. I’m not sure that’s true with $P_{ETCO_2}$—with the capnograph waveform.

Durbin: About 20 years ago it was suggested that looking at the end-tidal-to-arterial CO$_2$ gradient is a way to optimize PEEP. The theory is that as you overdistend normal lung areas, you drive blood away from them, and therefore worsen V/Q [ventilation/perfusion ratio]. When you start with collapsed areas that start opening up, V/Q gets better. The balance between those 2 opposing effects allow you to optimize PEEP by simply looking at the end-tidal-to-arterial CO$_2$ gradient. That approach has not been widely used, to my knowledge, but it was reported.¹ Any thoughts on that approach?
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er MJ. Titration of PEEP by the arterial minus end-tidal CO$_2$ gradient. Chest 1984; 85(1):100–104.

Thompson: I think we would be more apt to use $V_d/V_t$ [the ratio of dead space to tidal volume] now with more modern equipment. But, yes, absolutely. I was brought up on the gradient that it’s all dead space ventilation. Shunt has very little to do with that gradient, so you are increasing alveolar dead space when you widen the gradient. I agree.

Hess: On that subject I would refer to Lluis Blanch’s 1987 paper.

REFERENCE

Blanch: Yes. We studied the effect of PEEP on the arterial minus end-tidal CO$_2$ gradient in patients with acute lung injury. At similar cardiac output, the gradient decreased with PEEP, equal to the lower inflection point of the P-V curve, and with patients who did not have a lower inflection point we applied a random PEEP level, and the behavior of the gradient was unpredictable and not correlated with changes in oxygenation. The interpretation was that PEEP induced alveolar recruitment, decreased alveolar dead space, and the gradient narrowed, whereas the contrary occurred when PEEP induced overdistension and both alveolar dead space and the gradient increased. Therefore, variations of the gradient at similar hemodynamic status might help clinicians understand the physiologic effects of PEEP.

Bigatello: I think this brings up the utility of looking at CO$_2$ elimination as an index of alveolar recruitment, which Gattinoni et al reported on. If we are recruiting the lung and not overdistending, but truly recruiting new units, we should ventilate better so the $P_{aCO_2}$ will decrease.

REFERENCE