Chapter 1

Clinical perspectives

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Introduction

Unless you are on cardiopulmonary bypass or in deep hypothermia, you must breathe, that is, you must ventilate your lungs to pick up oxygen and deliver carbon dioxide (CO_2) from the lungs to the outside. The detection – breath after breath – of appropriate volumes of gas and concentrations of CO_2 in the exhaled gas (it is no longer air!) proves, in one stroke, several important facts:

- CO_2 is being generated by metabolic processes during which the body utilizes oxygen.
- Venous blood brings the CO_2 from the periphery to the heart.
- The heart pumps blood through the lungs.
- Ventilation of the lungs – spontaneous, manual, or mechanical – conveys the CO_2 and other gases to the outside. As long as no contrivance, such as a ventilator, is attached to the patient, the journey of CO_2 ends here as far as we are concerned.

Subsequent chapters in this book will deal in detail with CO_2 production, transport, and analysis. In this chapter, we will examine different time- and volume-based capnograms, and invite the reader to analyze them with a clinical eye, with a special focus on problems related to ventilation – by far the most common clinical application of capnography.

First a word of caution: a capnogram, whether time- or volume-based, presents only a snapshot. Even a trend plot running over several minutes represents but a brief episode in a phase of a patient's disease. More often than not, capnography is recruited to help with the diagnosis and interpretation of an acute process (intubation, embolism, bronchospasm, adjustment of ventilation, bicarbonate infusion, etc.). The body has uncounted mechanisms to compensate for disturbances. These corrective efforts overlap, and are accomplished at different speeds, some taking a few breaths and others days to reach a new equilibrium. They can affect cardiac output, pulmonary blood flow, ventilation, acid–base balance, and renal physiology. When capnographic data during such unsteady states are observed, we must be aware of the fact that capnography can tell only a small part of the story and that the data in front of us are likely to change until a new steady state has been reached.

The normal time-based capnogram

For many years, the only widely available capnographic display plotted PCO_2 along a time axis. The phases were labeled in different ways, as shown in Figures AP1 and AP2 (page 462).

Time-based capnography can use either an on-airway (or "mainstream") method, which uses a cuvette containing a cell in which the concentration of CO_2 is assessed, or a sidestream system, which relies on aspirating gas close to the patient's face and transfering it via a long capillary tube to the gas analyzer.

Difficulties arise when we try to determine when in the respiratory cycle the phases were recorded. Figure 1.1 shows tracings obtained during mechanical ventilation of an anesthetized patient. The time plots represent top to bottom: flow, mainstream capnogram, sidestream capnogram, and airway pressure.

Observe that the mainstream capnogram precedes the sidestream capnogram by the transport time of gas in the capillary connecting the sampling port (usually on the "Y" of the breathing circuit close to the patient's mouth) to the gas analyzer. At the end of inspiration, the deadspace of the patient will be filled with air. Thus, the first exhaled gas (about 150 mL for the average adult) of anatomic deadspace without CO_2 will not be recognized by the capnograph. Phase I (without CO_2) of the capnogram, therefore, contains a little exhaled

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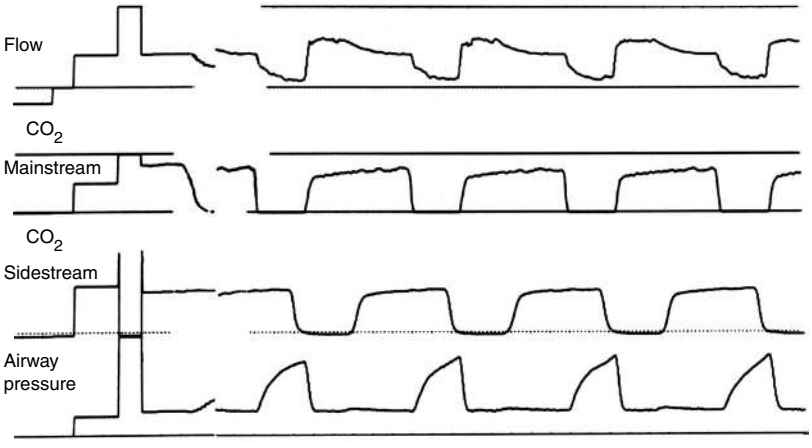


Figure 1.1 Tracing from a patient during controlled ventilation using a circle breathing system. Tracings (from top to bottom) are flow, mainstream capnogram, sidestream capnogram, and airway pressure. Observe that flow and pressure show relatively short fluctuations with inspiration and expiration, and that the sidestream capnogram is out of phase. With sidestream analyzers, the gas has to be carried from the patient to the analyzer through a capillary. Inspiration and expiration on the pressure and flow recording are not simultaneous with inspiration and expiration on the capnogram. The plateaus of the capnograms extend into the respiratory pause and last until the next inspiration arrives.

gas. Finally, a respiratory pause at the end of expiration will leave stagnant gas in the cuvette of the mainstream analyzer or under the sampling port of the sidestream analyzer. Hence, time-based capnograms show the end of exhalation only when end-tidal values are abruptly interrupted by an incoming fresh breath that washes away the CO_2. If phase III of a time-based capnogram is horizontal, we cannot separate the end-expiratory portion that may represent a respiratory pause from an ongoing exhalation delivering a steady level of CO_2. Indeed, should the patient be in respiratory arrest, for example, the plateau would eventually slowly decay as the sidestream (gas aspirating) analyzer continues to aspirate air (or gas from the breathing circuit).

Artifacts

Before interpreting the capnogram, we must ascertain that artifacts have not distorted the tracing. Two sources of distortions can be recognized as detailed below.

Mechanical artifacts

Improper calibration of the gas analyzer can be a cause of a distorted tracing, as discussed in the chapter dealing with technical specifications and standards (Chapter 36: Technical specifications and standards). A leak in the sampling tube of a sidestream gas analyzer can allow air to be aspirated and, thus, dilutes the sampled CO_2. Obstruction of the sampling catheter will cause the capnogram to be dampened, slurring the up- and down-slopes of the capnogram and causing falsely high inspired and falsely low end-expired CO_2 values.

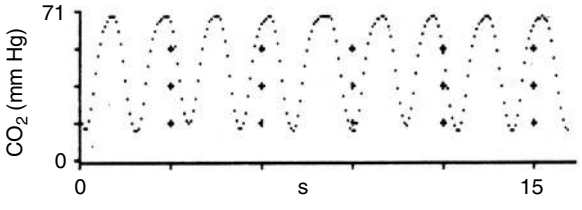


Figure 1.2 A capnogram without a well-defined plateau does not enable end-tidal partial pressure of CO_2 ($P_{ET}CO_2$) to be deduced. End-tidal values are reported to be 70.5 mm Hg; however, they are likely to be much higher in this tachypneic child. Observe that the inspired values show a PCO_2 of 14.9 even though no rebreathing occurred. The respiratory rate exceeded the capnograph's power of resolution. A capnogram without a plateau in phase III may not give meaningful end-tidal values for any other gas exhaled by the patient. Inspect the capnogram before accepting the data presented by the instrument as valid.

Observe that the sidestream capnograms are a little more rounded than the on-airway capnograms (Figure 1.1); this indicates that the sidestream capnographic signals have undergone some damping brought about when the front of the gas column traveling in the long capillary tube undergoes some mixing with adjoining gas. This damping problem becomes more troublesome with rapid respiration, as shown in Figure 1.2. With rapid ventilation, as encountered in pediatric anesthesia, the system might not have sufficient time to reach 100% of the required response, thus displaying higher than actual inspired and lower than actual expired CO_2 values. The response time of capnographs are discussed in the chapter dealing with technical specifications and standards (Chapter 36: Technical specifications and standards).

A water trap with a large internal volume (Figure 1.3) can also introduce artifacts when high

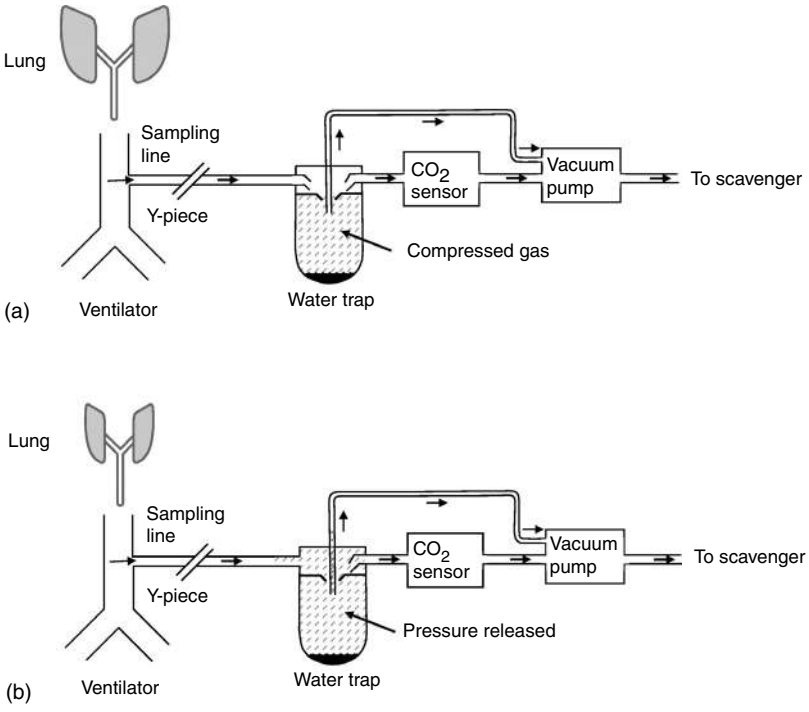


Figure 1.3 Capnogram artifact and water traps. Large water traps (10 mL) produce artifact, which has its origin in the phase of respiration and whose appearance depends on respiratory rate. (a) At the end of inspiration, the system is pressurized at peak airway pressure (P_{aw}) and filled with fresh gas, except for the lower part of the water trap, which holds a gas mixture containing CO_2 (shaded). (b) At the beginning of expiration, P_{aw} decreases to baseline. The pressurized gas mixture in the lower part of the water trap expands and some flows into the sampling tube, the CO_2 content of which is eventually detected by the capnograph. Its appearance on the capnograph depends on what part of an earlier breath is moving through the water trap when the P_{aw} drops to baseline. With constant sampling, flow, and tube length, it depends on respiratory rate. [Modified from: van Genderingen HR, Gravenstein N. Capnogram artifact during high airway pressures caused by a water trap. *Anesth Analg* 1987; **66**: 185–7.]

airway pressures during inspiration compress gas in the trap [1]. This gas expands during expiration and enters the gas stream to be analyzed, thereby introducing an artifact [2]. Modern sidestream capnographs therefore use small water traps and/or filters.

Clinical artifacts

The smooth outline of the capnogram might be dented by the patient taking a breath while undergoing mechanical ventilation (see examples – Figures 9 and 10 in the Appendix). Pattern #10 has been baptized a "curare cleft," an unfortunate appellation. Calling it a curare cleft implies that not enough muscle-relaxant drugs were given so that the patient was capable of initiating a breath. Instead of focusing on incomplete relaxation, the clinician should ask why the patient attempts to breathe while being mechanically ventilated. The answer may be that the patient's partial pressure of CO_2 in arterial blood ($PaCO_2$) exceeds the physiological limits and that in the face of partial paralysis, a troubled respiratory center attempts to correct hypercarbia. Increasing the minute ventilation would be a better measure than deepening the muscle relaxation. An alternative explanation might be that the patient, unable to signal pain because of almost complete paralysis, gasps in desperation. Rather than blocking the response with deeper muscle paralysis, the patient should be better anesthetized. Finally, a "curare cleft," can be generated by pushing on the patient's chest, as might well happen when the surgeon leans on the chest during an operation. Only if the clinician is persuaded that none of these explanations apply and that a hiccup, for example, must be held responsible for the "curare cleft", and that the brief inspiratory efforts interfere with the surgical procedure, should the degree of muscle relaxation be increased.

Finally, cardiogenic oscillations may ripple the down-slope of the capnogram (Figure 13 in the Appendix). These interesting, heart-rate-synchronous, small inspirations and expirations provide evidence that cardiac contractions and relaxations in the chest cause fluctuations of the lung volume with tidal volumes of about 10 mL, the recording of which generates a pneumocardiogram [2]. Evidence of these cardiogenic tidal volumes can also be seen in the movement of the inspiratory and expiratory valves of an anesthesia breathing system. During the respiratory pause in mechanical ventilation, the valves can be seen to flutter synchronously with the heartbeat.

In summary, a capnogram should have four well-defined phases. Figure 1.4 lists points to be considered

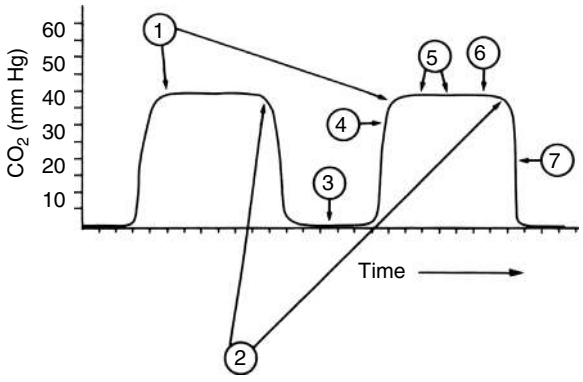


Figure 1.4 (1) *Plateau/onset* – Is there a pattern demonstrating that the patient is being ventilated? (2) *Plateau/end* – Are peak values appropriate? Are the ventilator settings and the patient's respiratory pattern consistent with the capnogram and capnographic findings? (3) *Baseline* – Is the inspired CO_2 tension zero (normal baseline), or is there evidence for rebreathing (elevated baseline)? (4) *Upstroke* – Is there evidence for slow exhalation (slanted upstroke)? (5) *Plateau/horizontal* – Is there evidence of uneven emptying of lungs? (6) *Plateau/smooth* – Is expiration interrupted by inspiratory efforts? (7) *Downstroke* – Is the downstroke steep, or is there evidence of slow inspiration or partial rebreathing?

when deciding whether or not to accept a capnogram of a quality sufficient for clinical interpretation.

Interpreting an artifact-free, time-based capnogram

Cardiovascular issues

The presence of a capnogram signifies that the patient's lungs are perfused. In cardiac arrest, the lungs will not be perfused, but with successful resuscitation, CO_2 will appear in the exhaled gas (as discussed in greater detail in Chapter 20: Cardiopulmonary resuscitation). In general, the capnogram will give evidence of acutely reduced pulmonary perfusion coincident with a drop in cardiac output. Figure 1.5 shows an example of momentarily induced ventricular fibrillation as practiced during implantation of a pacemaker/defibrillator. This will produce a typical pattern of decreasing capnographic tracings. During the first seconds of arrest without pulmonary perfusion, the lung yields quickly decreasing amounts of CO_2 from the stagnant blood or from lung tissue. With successful defibrillation and re-establishment of pulmonary perfusion, CO_2 once again appears in the exhaled breath. Of course, with continued cessation of pulmonary blood flow and continued ventilation, the capnogram will eventually show zero CO_2. If ventilation is stopped during cardiac arrest, a time-based sidestream capnogram will gradually reach zero values as the system continues to aspirate gas (with many devices about 200 mL/min), thus eventually aspirating breathing circuit gas. An on-airway (mainstream) system might show steady values (high or low) if the gas in the cuvette of the system remained stationary.

Some changes in end-tidal values develop slowly, and are thus more readily recognized in trend plots. For example, showers of air emboli can produce areas of alveolar deadspace (ventilated but not perfused alveoli), perhaps associated with a decrease in cardiac output. Shortly thereafter, the air bubbles either pass through the lungs or make it into the alveoli to be exhaled. This process causes the tell-tale transient dip in end-tidal CO_2 values as shown in Figure 1.6. This capnogram is from a patient undergoing a posterior fossa operation in the sitting position and suffering from a typical shower of air emboli. Such ventilation/perfusion $\dot{V}/\dot{Q}$ abnormalities are discussed in greater detail in Chapter 31 (Ventilation/perfusion abnormalities and capnography).

Pulmonary issues

The most important use of capnography in the field, in the intensive care unit, and in the operating room comes with the establishment of an artificial airway. Intubation of the esophagus instead of the trachea still kills people who depend on a tracheal tube for ventilation. Capnography indicates whether or not the tube is in the esophagus. Details of this essential application of capnography in different settings are discussed in considerable detail in several subsequent chapters.

In an artifact-free capnogram, normal end-tidal CO_2 values (between 35 and 45 mm Hg) suggest normal ventilation. However, because a $\dot{V}/\dot{Q}$ mismatch (see Chapter 31: Ventilation/perfusion abnormalities and capnography) can cause the end-tidal values to appear normal while arterial values are high, the clinician will consider other evidence to confirm adequate ventilation. First, the clinician will need to assess the minute volume in the light of the patient's age and weight. We are reassured if the patient's end-tidal CO_2 values are within the normal range and tidal volume and minute ventilation fall within the ranges given in Table 1.1. However, observe that the adult range of minute ventilation covers a wide span. In general, recumbent patients under anesthesia requiring mechanical ventilation

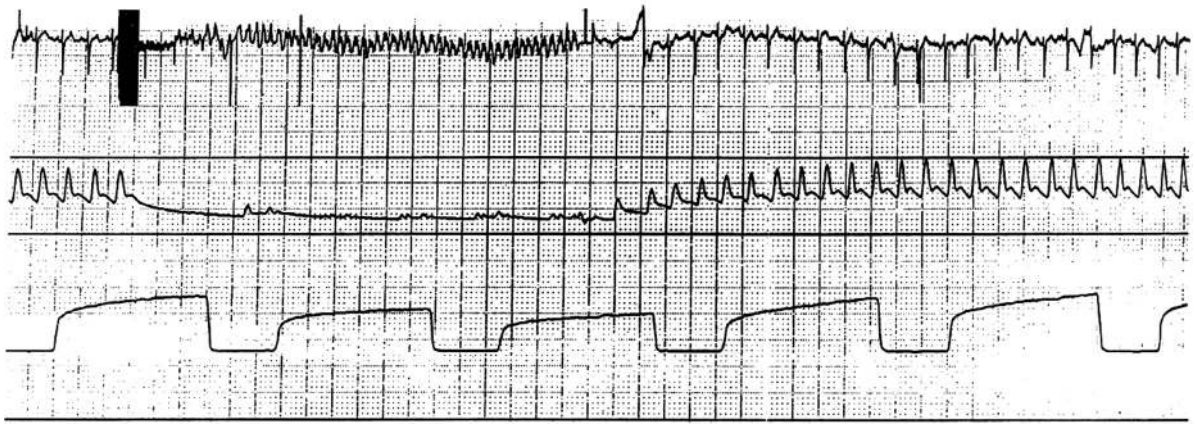


Figure 1.5 A patient undergoing the implantation of an automatic internal cardiac defibrillator was monitored with electrocardiogram (ECG) (top), radial arterial pressure (middle), and mainstream capnography (bottom). Induced ventricular fibrillation (black areas in ECG) and defibrillation are apparent in the ECG tracing. Observe decay of arterial pressure. During absent pulmonary blood flow, the patient's lungs were ventilated, and, with two breaths, the $P_{ET}CO_2$ decreased from 35 mm Hg before fibrillation to 22 mm Hg before defibrillation.

Figure 1.6 The capnogram shows a trend of slow decrease in peak expiratory CO_2 from about 34 to a low of 22 mm Hg, and then an increase to 35 mm Hg. Inspiratory values remained normal. This trend is compatible with a brief shower of air emboli in a patient undergoing a posterior fossa craniectomy in the sitting position.

Table 1.1 Average respiratory values for resting, healthy patients

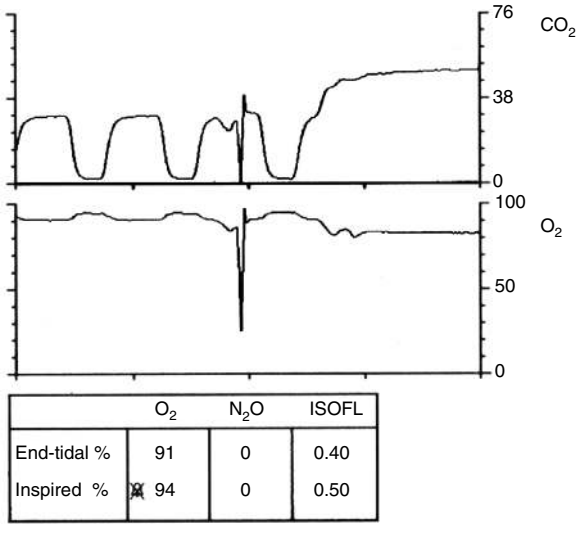
Parameter	Adult range	Neonatal range
Respiratory rate	10–15 breaths/min	30–40 breaths/min
Tidal volume	6–10 mL/kg	5–7 mL/kg
Minute ventilation	4–10 L/min	200–300 mL/kg/min

need larger tidal volumes to maintain normal blood gas values than spontaneously breathing patients sitting upright. The selection of the optimal minute ventilation must also take into account the deadspace ventilation. Every tidal volume ventilates deadspace as well as the alveoli. If we wish to double the minute ventilation, we *might* double the respiratory rate. However, if we increase the respiratory rate without changing the tidal volume, deadspace ventilation is increased in parallel with alveolar ventilation. If the beginning tidal volume is small enough to tolerate, then increasing the tidal volume instead of changing the respiratory rate would greatly improve alveolar ventilation without increasing deadspace ventilation.

Figure 12 in the Appendix shows a capnogram from an asthmatic patient. The reported end-tidal CO_2 pressure of 42 mm Hg is likely to be distinctly lower than the $PaCO_2$ of this patient, as the patient does not show a plateau of phase III, and the still-rising values were interrupted by the next inspiration.

If the plateau of the capnogram (phase III) does not become almost horizontal before the next breath brings the transition to phase IV, we must wonder how long the CO_2 levels would have continued to rise had an inspiration not interrupted exhalation. Patients with obstructive lung disease, such as asthma, will often show such a sloping phase III. The end-tidal partial pressure of CO_2 ($P_{ET}CO_2$) will then faithfully fail to represent $PaCO_2$. Asthmatic patients exhibiting such a sloping phase III of the capnogram often respond to the inhalation of bronchodilators with improvement of their capnogram and rising $P_{ET}CO_2$ until the improved gas exchange has corrected the problem.

Small tidal volumes will represent relatively low effective alveolar ventilation; that is, with shallow breathing, deadspace will make up more than the usual 30% of tidal volume. In such circumstances, the end-tidal CO_2 values might appear normal, and the



	O_2	N_2O	ISOFL
End-tidal %	91	0	0.40
Inspired %	94	0	0.50

Figure 1.7 A patient undergoing thoracotomy was intubated with an endotracheal tube that enables the blocking of one mainstem bronchus while collecting gas from the blocked lung as well as the ventilated lung. The left part of the capnogram is produced by the ventilated lung, showing a $P_{ET}CO_2$ of 29 mm Hg. The $PaCO_2$ was 46 mm Hg. The right part of the capnogram represents gas sampled distal to the blocker in the right lung showing a PCO_2 of 48 mm Hg. The PCO_2 of the mixed venous blood sampling through a pulmonary arterial catheter was 49 mm Hg.

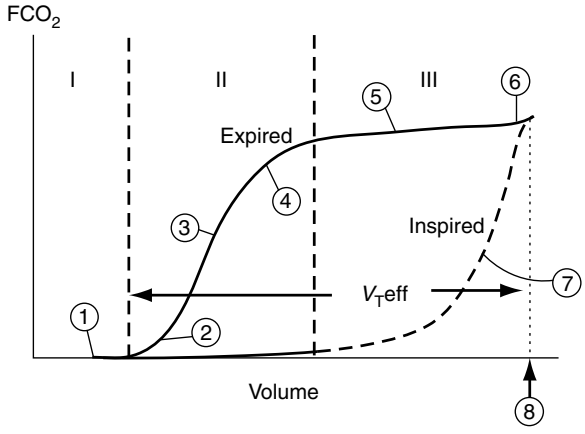


Figure 1.8 A solid line denotes the expiratory portion; the inspiratory portion, if shown, is denoted by a dashed line. The three phases are "denoted" by I, II, and III. (Numbers 1–8 represent the checklist and comments below.) (1) *Phase I* – Is the inspired CO_2 tension zero (normal baseline), or is there evidence of rebreathing (elevated baseline)? Does the volume of phase I reasonably reflect the anatomical and apparatus deadspace (in addition to possibly compressed volume if the program does not subtract this)? Please note that the vertical interrupted line for phase I does not intersect the abscissa at the deadspace volume. (2) *Angle between phases I and II* – Is the transition clearly defined? (3) *Slope of phase II* – Is there evidence for slow exhalation (slanted up-slope)? When the transition to phase III is slurred, consider obstructive pulmonary disease. (4) *Angle between phases II and III* – Is the transition clearly defined? (5) *Slope of phase III* – Is the slope almost level (children and young adults), or is there a clear gradient (i.e., evidence of uneven emptying in patients with lung disease)? (6) *End of phase III* – What is the final value? Is expiration interrupted by inspiratory efforts? Are peak values appropriate? The area under the expiratory limb represents the volume of expired CO_2. (7) *Down-slope (if inspiratory limb shown)* – Is the down-slope steep, or is there evidence of partial rebreathing? The area under the inspiratory limb represents the volume of inspired CO_2; the area between the curves represents the volume of CO_2 eliminated. (8) *Exhaled volume and exhaled CO_2 volume* – Are the values consistent with the expected value and ventilator settings?

capnogram can look quite unremarkable. Yet, an interposed large tidal volume can reveal a $P_{ET}CO_2$ much higher than expected.

Intubation of a mainstem bronchus will result in relative hyperventilation of the intubated lung, producing low $P_{ET}CO_2$ values. Once both lungs are ventilated without changing the tidal volume, the end-tidal values will normalize. In the unventilated airways, CO_2 will equilibrate with venous blood as seen in Figure 1.7.

In the discussion of time-based capnography, the question of the adequacy of ventilation – that is, the adequacy of CO_2 elimination and deadspace ventilation – pops up repeatedly. Thus, it would be nice to be able to view deadspace ventilation as it relates to tidal volume. Enter volume-based capnography.

The normal volume-based capnogram

An individual tracing of the time-based capnogram left a number of questions unanswered, which the single breath volume-based capnogram provides. In Figure 1.8, the solid line denotes the expiratory portion, and the inspiratory portion (not always shown) is denoted by a dashed line. In general, the data offered by the volume-based capnogram refine the information offered by time-based capnography. Again, we ask for an artifact-free tracing, and we consider ventilation and circulation. The phases of the capnogram can then be scanned for detailed information; the questions to be raised for each phase are numbered and enumerated in Figure 1.8. Our most important question is: is there evidence that the lungs are being ventilated? If they are not, is the endotracheal tube in the esophagus, or is the patient in cardiac arrest? Once we are reassured, we proceed to examine the details. The inspired CO_2 tension

should be zero; if not, this is evidence of rebreathed CO_2, as discussed in Chapter 6 (Capnography during anesthesia). A normal deadspace is assumed to occupy about 1 mL/pound (0.5 mL/kg) or, for the average adult, about 150 mL, or approximately one-third of the tidal volume. The volume-based capnogram provides a convenient opportunity to confirm this fact. A larger than normal deadspace points to either an equipment deadspace (see Chapter 6: Capnography during anesthesia), exhausted CO_2 absorber, or ventilation of unperfused lung segments (see Chapter 31: Ventilation/perfusion abnormalities and capnography). Ideally, the transition from phase I to II should be abrupt, although it usually is not because as alveolar gas passes through the deadspace, it first mixes with the deadspace gas and then rapidly displaces it. This process should result in a steep rise of the capnogram in phase II. If the alveoli empty grossly unevenly, as in severe emphysematous or obstructive lung disease, the slope will be slanted. The angle between the up-slope and the plateau indicates that the addition of CO_2 from the alveoli is now beginning to become homogeneous. A lazy up-slope and a slurred transition again indicate a troubled lung that empties its CO_2 unevenly. A horizontal (or nearly so) plateau shows a lung that fairly prodigiously adds CO_2 to every milliliter of exhaled gas. Healthy children and young adults often show nearly horizontal plateaus. Cardiogenic oscillations, as described above, can put heartbeat-synchronous ripples on the plateau. At the end of the plateau, we expect to read the true end-tidal value for CO_2, which, as already mentioned, should be between 35 and 45 mm Hg:

- If the inspiratory limb is inscribed, we would expect a steep fall of CO_2 in the inspired gas, soon reaching zero, unless the patient is rebreathing CO_2, as discussed above. The area under the inspiratory limb is the volume of inspired CO_2; and the area between the curves represents the volume of CO_2 eliminated.
- Since we have plotted the tidal volume on the abscissa, we can check the exhaled volume and compare it with the expected value for the patient. Remember that inspired and expired volumes are often not identical either because the respiratory quotient is less than 1 (more oxygen consumed than CO_2 exhaled), or because the uptake or elimination of anesthetic gases causes a discrepancy. During anesthesia, nitrous oxide is often the culprit because we may give it in relatively high concentrations (up to 70%). Its solubility coefficient of 0.47 for blood at body temperature predicts that many liters will go into solution in the body and will at the end of anesthesia again appear in the exhaled gas.

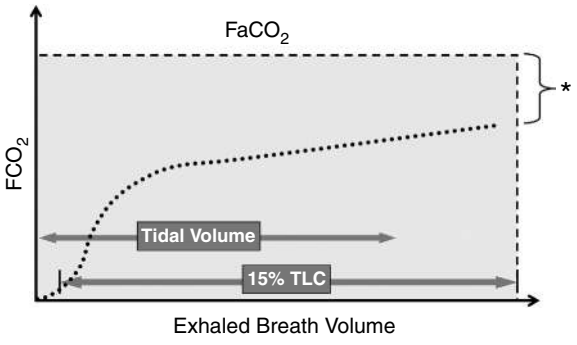


Figure 1.9 Volume-based capnogram from a patient with pulmonary embolism. Observe the large difference between end-tidal and arterial CO_2 tension. The asterisk shows the size of the alveolar deadspace at end-expiration. [Modified from: Anderson JT, Owings JT, Goodnight JE. Bedside non-invasive detection of acute pulmonary embolism in critically ill surgical patients. *Arch Surg* 1999; **134**: 869–74.]

The gas inhaled last will fill the patient's deadspace; it will be the gas exhaled first and should be free of CO_2. If it is not, the patient is rebreathing exhaled CO_2, which may be linked to the type of equipment in use or an equipment malfunction, or CO_2 is being added to the inspired gas. For example, at the end of anesthesia, some anesthetists like to add CO_2 so as to allow hyperventilation for the elimination of anesthetic gases without causing the patient to develop a respiratory alkalosis.

Figure 1.9 is from a patient who suffered a pulmonary embolism. Conditions that increase deadspace ventilation (ventilated but not perfused alveoli), such as emboli (tumor, gas, clot) or right-to-left shunts, will stand out clearly in the volume-based capnogram that shows the large deadspace. With a decrease in cardiac output, the volume of CO_2 delivered to the lungs will also decrease. As the volume-based capnogram enables the calculation of the exhaled CO_2, we can quantify the change better than with time-based capnography, which only reports the end-tidal values.

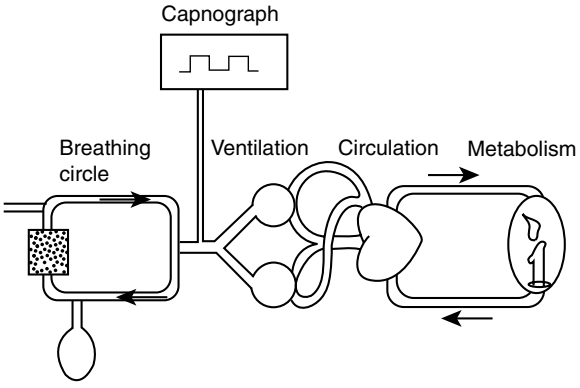


Figure 1.10 End-tidal values can be affected by a number of mechanisms, starting with the generation of CO_2 in the cell (candle), the transport of venous blood to the heart (cardiac output), the pulmonary blood flow (part of which may be shunted past ventilated alveoli), ventilation (part of which may be blocked from perfused alveoli), the breathing system (which may cause rebreathing, hyperventilation, or hypoventilation), and ending with the capnograph (which may fail because of artifacts or incorrect calibration).

Summary

Whether using time- or volume-based capnography, many questions will confront the clinician when abnormal capnographic data call for an analysis. Figure 1.10 recapitulates the fact that many components of the system can cause trouble, starting with cellular metabolism (remember malignant hyperthermia) to mechanical problems related to the airway, ventilation, and monitors. These topics, buttressed by exhaustive references, will be discussed in detail in subsequent chapters.

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1. van Genderingen HR, Gravenstein N. Capnogram artifact during high airway pressures caused by a water trap. *Anesth Analg* 1987; **66:** 185–7.
2. Bijaoui E, Baconnier PF, Bates JHT. Mechanical output impedance of the lung determined from cardiogenic oscillations. *J Appl Physiol* 2001; **91**: 859–65.

Section 1 Ventilation

Section 1
Chapter
2

Ventilation

Capnography and respiratory assessment outside of the operating room

R. R. Kirby

Introduction

Since gas exchange is a primordial function of the lungs and the conductive airways, respiratory assessment is of paramount importance. Clinicians evaluate this function by visual observation of chest expansion, depth and rate of ventilation, use of accessory respiratory muscles, and auscultation of the quality and quantity of breath sounds. Quantitative information is obtained by determining thoracic/pulmonary compliance (change of volume related to change in pressure) and airways resistance. Other more complex techniques involve measurement of lung volumes and capacities with spirometry, which also evaluates airway patency and lung/thorax expansion. Factors that affect these measurements include pain, fatigue, and poor understanding by the patients and clinicians of how the test is to be carried out. As a result, assessment of airway obstruction or lung restriction is reliable only insofar as the patient's ability to perform the tests is optimal and unimpaired.

Perhaps the ultimate test for adequate ventilation is invasive determination of arterial CO_2 partial pressure ($PaCO_2$). In general, an elevation in $PaCO_2$ (hypercapnia) represents a decreased respiratory rate, depth, or both; inefficient alveolar ventilation (ventilation/perfusion [$\dot{V}/\dot{Q}$] inequalities); or production of CO_2 in excess of the patient's ability to excrete it. A reduction in $PaCO_2$ (hypocapnia) results from excessive alveolar ventilation in relation to CO_2 production. Measurement of $PaCO_2$, although a true reflection of ventilatory efficacy, is far from ideal since it is invasive and intermittent.

Capnography has been utilized in surgical patients for over three decades to confirm tracheal intubation and assess ventilation. Measurement of exhaled CO_2, particularly the end-tidal PCO_2 ($PETCO_2$), is an established standard of care in patient monitoring [1]. In conjunction with $PaCO_2$, capnography provides a semiquantitative assessment of $\dot{V}/\dot{Q}$ mismatch by changes in the $PaCO_2$–$PETCO_2$ gradient (normal ≤5 mm Hg). Capnograms are of three types, depending on whether the concentration of CO_2 is plotted against (1) expired volume, otherwise known as *volumetric capnogram*, (2) single breath time concentration CO_2 curve [2], or (3) time during a respiratory cycle. The latter technique is more practical for clinical use.

Capnography is increasingly employed outside the operating room as a non-invasive, continuous trend monitor of $PaCO_2$ and airway dynamics. It is of value in assessing the efficacy of cardiopulmonary resuscitation during low perfusion states or cardiac arrest, and is considered a standard of care by the American Heart Association [3]. Colorimetric capnometry is fast, convenient, and useful to verify tracheal intubation in nonoperating room settings. However, it can present problems, as was indicated by Puntervoll *et al.* [4]. They compared colorimetric methodology with mainstream capnography, and found that in emergency situations in which CO_2 containing air may be present in the esophagus, mainstream capnography should be the preferred method of verifying tracheal – and not esophageal – intubation. The colorimetric CO_2 indicator is very sensitive to low CO_2 values, and may falsely indicate correct tracheal intubation, even when the tube is in the esophagus.

As the use of capnography increases and the interpretation of abnormalities becomes more complex, categorization into useful and meaningful diagnostic and therapeutic modes is of value. The data have been classified in a simplified manner (Table 2.1) [5]. Some redundancy is noted among categories, since capnography is applicable in numerous clinical settings.

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