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Who should be monitored?

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 (CO₂) from the lungs to the outside. Detecting – breath after breath – appropriate volumes of gas and concentrations of CO₂ in the exhaled gas (it is no longer air!) proves in one stroke several important facts:

1. CO₂ is being generated by metabolic processes during which the body utilizes oxygen.
2. Venous blood brings the CO₂ from the periphery to the heart.
3. The heart pumps blood through the lungs.
4. Ventilation of the lungs – spontaneous or manual or mechanical – conveys the CO₂ and other gases to the outside. As long as no contrivance, such as a ventilator, is attached to the patient, the journey of CO₂ ends here, as far as we are concerned. This adds up to an enormous amount of important information should we suspect a problem with CO₂ production, its transport to the heart and to the lungs and its elimination in the exhaled gas. Subsequent chapters in this book will deal in considerable detail with specific issues of CO₂ production, transport, and analysis. Here 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.

Before presenting different capnographic patterns a word of caution. A capnogram, whether time or volume based, presents 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.). We must remember that the body has uncounted mechanisms to compensate for disturbances. These mechanisms can affect cardiac output, pulmonary blood flow, ventilation, acid–base balance, and renal physiology. In short, every acute change in ventilation and circulation with effects on CO₂ homeostasis will induce changes. When we observe capnographic data during such unsteady states, 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. Clinicians too! help the body to make this new steady state as physiologic as possible.

The normal time-based capnogram

For many years the only widely available capnographic display plotted CO₂ along a time axis. The phases were labeled in different ways, for example as shown diagrammatically in Figure 1.1. Time-based capnography can use either an on-airway (or “mainstream”) method, which uses a cuvette containing a cell in which the concentration of CO₂ is assessed or a side-stream system, which relies on aspirating gas close to the patient’s face and bringing 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 1.2 shows tracings obtained during mechanical ventilation of an anesthetized patient. The time plots represent from top to bottom flow, mainstream capnogram, side-stream capnogram, and airway pressure.

Observe that the mainstream capnogram precedes the side-stream 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 dead-space of the patient will be filled with air. Thus, the first exhaled gas (about 150 ml for the average adult) of anatomic dead-space without CO₂ will not be recognized by the capnograph. Phase I (without CO₂) of the capnogram, therefore, contains a little exhaled gas. Finally, a respiratory pause at the zero.
end of expiration will leave stagnant gas in the cuvette of the mainstream analyzer or under the sampling port of the side-stream analyzer. Thus, time-based capnograms show the end of exhalation only when end-tidal values are abruptly interrupted by a breath that washes away the CO₂. 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 CO₂. Indeed, should the patient be in respiratory arrest, the plateau would eventually slowly decay as the gas-aspirating side-stream analyzer begins to aspirate air (or gas from the breathing circuit). Despite these limitations, time-based capnography has been widely adopted and has greatly enhanced the safety of patients at risk of inadequate ventilation.

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**

Here we include improper calibration of the gas analyzer as discussed in Chapter 39. A leak in the sampling tube of a side-stream gas analyzer can allow

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**Figure 1.1** The capnogram has four phases: I: inspiration; II: the appearance of CO₂ during early expiration; III: the plateau phase, with or without a respiratory pause; and IV: the descending slope of the inspiratory phase. With a respiratory rate of 10 breaths/min, one respiratory cycle would last 6 s. The ratio of inspiration to expiration is assumed to be 1:2. Gas assumes to be dry; barometric pressure (P_b) is 760 mmHg.

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**Figure 1.2** Tracing from a patient during controlled ventilation using a circle breathing system. Tracings from top to bottom are flow, mainstream capnogram, side-stream capnogram, and airway pressure. Observe that flow and pressure show relatively short fluctuations with inspiration and expiration and the side-stream capnogram is out of phase. With side-stream 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.
air to be aspirated and thus dilute the sampled CO₂. One special circumstance can lead to the “steepled” capnogram discussed in Chapter 6 (Capnography during anesthesia). Obstruction of the sampling catheter would cause the capnogram to be dampened, shunting up- and down-slopes of the capnogram and causing falsely high inspired and falsely low end-expired CO₂ values.

Observe that the side-stream capnograms are a little more rounded than the on-airway capnograms (Figure 1.2). Thus, the side-stream 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 1.3. 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 CO₂ values. The response time of capnographs are discussed in Chapter 39.

A water trap with a large internal volume (Figure 1.4) can also introduce artifacts when high airway pressures during inspiration compress gas in the trap. This gas expands during expiration and enters the gas CLINICAL PERSPECTIVES 5

Figure 1.3 A capnogram without a well-defined plateau does not enable end-tidal partial pressure of CO₂ (P₄CO₂) to be deduced. End-tidal values are reported to be 70.5 mmHg; however, they are likely to be much higher in this tachypneic child. Observe that the inspired values show a PCO₂ of 14.9 even though there was no rebreathing. 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 as valid data presented by the instrument.

Figure 1.4 Capnograms 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 (Paw) and filled with fresh gas, except for the lower part of the water trap, which holds a gas mixture containing CO₂ (shaded). (b): At the beginning of expiration, Paw decreases to baseline. The pressurized gas mixture in the lower part of the water trap expands and some flows into the sampling tube, the CO₂ 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 Paw drops to baseline. With constant sampling flow and tube length, it depends on respiratory rate.
stream to be analyzed, thus introducing an artifact (van Genderingen & Gravenstein, 1987). Modern side-stream capnographs 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. Figure 1.5 shows an example. This pattern has been baptized a “curare cleft”, an unfortunate appellation. Calling it a curare cleft implies that not enough muscle relaxant drugs had been 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 might be that the patient’s partial pressure of CO₂ in arterial blood (P aCO₂) exceeds physiological limits and that in the face of partial paralysis a troubled respiratory center attempts to correct hypercarbia. Increasing 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 must be held responsible for the “curare cleft” and the brief inspiratory efforts interfere with the surgical procedure should the degree of muscle relaxation be increased.

Finally, cardiogenic oscillations might ripple the down-slope of the capnogram (Figure 1.6). These interesting, heart rate synchronous, small inspirations
and expirations give evidence that cardiac contractions and relaxations in the chest cause fluctuations of the lung volume with tidal volumes of about 10ml, the recording of which generates a pneumocardiogram (Bijaoui et al., 2001). Evidence of these cardiogenic tidal volumes can also be seen in the movement of inspiratory and expiratory valves of the 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 well-defined four phases. Figure 1.7 lists points to be considered when deciding whether or not to accept a capnogram of a quality sufficient for clinical interpretation.

**Interpreting an artifact-free time-based capnogram**

**Cardio-vascular 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 CO2 will appear in the exhaled gas, as discussed in greater detail in Chapter 20. In general, the capnogram will give evidence of acutely reduced pulmonary perfusion coincident with a drop in cardiac output. Figure 1.8 shows an example of momentary-induced ventricular fibrillation as practiced during implantation of a pacemaker/defibrillator. This will produce a typical pattern of decreasing capnographic tracings (Figure 1.8).

During the first seconds of arrest without pulmonary perfusion, the lung yields quickly decreasing amounts of CO2 either from the stagnant blood or from lung tissue. With successful defibrillation and re-establishment of pulmonary perfusion CO2 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. If ventilation is stopped during cardiac arrest, a time side-stream capnogram will gradually reach zero values as the

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**Figure 1.7**

(1) **Plateau/onset** – Is there a pattern giving evidence of ventilation? (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 CO2 tension zero (normal baseline) or is there evidence for rebreathing (elevated baseline)? (4) **Upstroke** – Is there evidence for carbon dioxide expiration (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?

**Figure 1.8** 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 PetCO2 fell from 35mmHg before fibrillation to 22mmHg before defibrillation.
system continues to aspirate gas (about 200 ml/min), thus eventually aspirating room air. 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 dead-space (ventilated but not perfused alveoli) perhaps associated with a decrease of cardiac output. Soon the air bubbles either pass through the lungs or make it into the alveoli to be exhaled. This process causes the telltale transient dip in end-tidal CO2 values as shown in Figure 1.9. This capnogram comes from a patient undergoing a posterior fossa operation in the sitting position and suffering from a typical shower of air emboli. Such ventilation/perfusion (V/Q) abnormalities are discussed in greater detail in Chapter 30.

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 tells 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 CO2 values (between 35 and 45 mmHg) suggest normal ventilation. However, because a V/Q mismatch (see Chapter 30) can cause the end-tidal values to appear normal while arterial values are high, the clinician will consider other evidence confirming 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 CO2 values are within the normal range and his tidal volume and minute ventilation fall within the ranges given in Table 1.1. But observe that the adult range of minute ventilation covers a wide span. In general, recumbent patients under anesthesia requiring mechanical ventilation need larger tidal volumes to maintain normal blood gas values than spontaneously breathing patients sitting upright. Selection of the optimal minute ventilation must also take into account dead-space ventilation. Every tidal volume ventilates dead-space as well as the alveoli. If we wish to double minute ventilation we might double the respiratory rate. However, as we increase the respiratory rate without changing tidal volume, we increase dead-space ventilation.

Doubling tidal volume instead of changing respiratory rate would greatly improve alveolar ventilation without increasing dead-space ventilation.

During anesthesia and with mechanical ventilation typically we use relatively low respiratory rates (8–10 breaths/min) and large tidal volumes (10–12 ml/kg). Figure 1.10 shows a capnogram from a patient with chronic obstructive lung disease. The reported end-tidal CO2 pressure of 42 mmHg is likely to be distinctly lower than the P_aCO2 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

Figure 1.9 The capnogram shows a trend of slow decrease in peak expiratory CO2 from about 34 to a low of 22 mmHg, and then an increase to 35 mmHg. 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    | 7–10 ml/kg   | 5–7 ml/kg   |
| Minute ventilation | 5–10 l/min  | 200–300 ml/kg/min  |
the transition to phase IV we must wonder how long the CO₂ 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 (Figure 1.10). The end-tidal partial pressure of CO₂ (PₐCO₂) will then fail faithfully to represent PₐCO₂. V/Q mismatch is discussed in Chapter 30. Asthmatic patients exhibiting such sloping phase III of the capnogram often respond to the inhalation of bronchodilators (or general anesthesia with a halogenated agent) with an improvement of their capnogram and rising PₐCO₂ until the improved gas exchange has corrected the problem.

Small tidal volumes will represent relatively low effective alveolar ventilation, that is, with shallow breathing dead-space will make up more than the usual 30% of tidal volume. In such circumstances the end-tidal CO₂ values might appear normal and the capnogram can look quite unremarkable. Yet, an interposed large tidal volume can reveal a PₐCO₂ much higher than expected.

**Figure 1.10** Capnogram with sloping phases II and III. The patient has chronic obstructive pulmonary disease. Every mechanical breath interrupts the patient’s slow expiration so that the capnogram cannot develop end-tidal CO₂ tensions representative of alveolar arterial gas.

**Figure 1.11** A patient undergoing thoracotomy was intubated with an endotracheal tube that enables the blocking of one mainstem bronchus while collecting gas from the blocked as well as the ventilated lung. The left part of the capnogram comes from the ventilated lung showing a PₐCO₂ of 29 mmHg. The PₐCO₂ was 46 mmHg. The right part of the capnogram represents gas sampled distal to the blocker in the right lung showing a PₐCO₂ of 48 mmHg. The PₐCO₂ of the mixed venous blood sampling through a pulmonary arterial catheter was 49 mmHg.
Intubation of a mainstem bronchus will result in relative hyperventilation of the intubated lung, producing low $P_aCO_2$ 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 1.11.

In the discussion of time-based capnography the question of the adequacy of ventilation, that is the adequacy of $CO_2$ elimination and dead-space ventilation pops up repeatedly. Thus, it would be nice to be able to look at dead-space ventilation as it relates to tidal volume. Enter volume-based capnography (see also Chapters 30 and 31).

**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 1.12 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 think about the inspiratory CO2 tension 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, then the slope will be slanted. The angle between the upslope 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 shuffled transition again indicate a troubled lung that empties its anatomical and apparatus dead-space (see Chapters 30 and 31).

**Figure 1.12** 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 for rebreathing (elevated baseline)? Does the volume of phase I reasonably reflect the anatomical and apparatus dead-space? (Plus 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 dead-space 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 sudden, think of obstructive pulmonary disease.

3. 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)?

4. 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 $CO_2$.

5. Down-slope (if inspiratory limb shown): Is the down-slope steep or is there evidence of partial rebreathing? The area under inspiratory limb represents the volume of inspired $CO_2$.

6. Inspired CO2 tension: How does the ventilator compensate for CO2?

7. Exhaled volume and expired $CO_2$ volume: Are the values consistent with the expected value and ventilator setting?