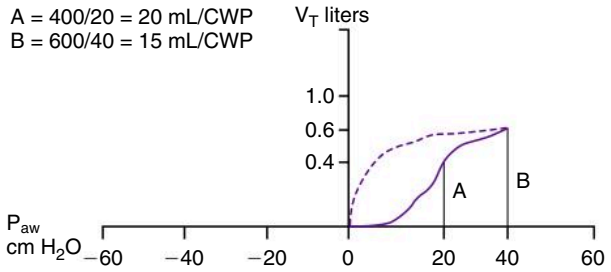


Fig. 9.37 Pressure-volume loop showing the effects of overdistention of the lung during pressure-controlled intermittent mandatory ventilation (PC-IMV). Point *A* indicates the pressure at which tidal volume delivery is optimized in terms of pressure. Point *B* represents the peak inspiratory pressure. *CWP,* Capillary wedge pressure.

Inspiratory Rise Time Control: Sloping or Ramping

A pressure breath produces a high flow at the beginning of inspiration. With small-diameter endotracheal tubes (i.e., increased R_{aw}), the high flow through the narrow opening creates turbulence. As a result, a pressure overshoot can occur at the beginning of the pressure curve before the pressure adjusts to the set value.

If the flow and pressure delivery are tapered at the start of inspiration, the waveform can be adjusted to reduce this overshoot. Most current acute care ventilators have a function that can taper the flow during pressure ventilation. When tapering is used, the pressure curve may no longer be constant but may be tapered at the beginning of the breath.

Inspiratory flow delivery during PC-CMV can therefore be adjusted with an inspiratory rise time control, also called a *slope control* (Fig. 9.38).

Flow Cycling During Pressure Support Ventilation

The normal flow-cycling mechanism of pressure support ventilation (PSV) is discussed in Chapters 3 and 5. Flow cycling occurs when the ventilator detects a decreasing flow, which represents the end of inspiration. The ventilator's software determines the point at which flow cycling occurs; in most ventilators this point is a percentage of the peak flow measured during inspiration.

Unfortunately, no single flow cycle percentage is ideal for all patients. Patients with COPD or increased R_{aw} have a slower flow rate drop-off during inspiration with pressure ventilation than do patients with normal R_{aw}. Because flow does not drop normally, patients with COPD are more comfortable with a higher flow cycle percentage (e.g., 40%). The clinician can determine the appropriate cycling criterion by evaluating the P-T curve during PSV. If an active rise in pressure occurs at end inspiration, the flow cycle percentage may be increased to reduce the amount of expiratory work the patient must perform.

The graphics in Fig. 9.39 show two flow cycle percentages. What is the peak flow in *A?* What is the flow value where inspiratory flow ends? What is the approximate flow cycle percentage?* What is the peak flow in *B?* What is the flow value at which inspiratory flow ends? What is the approximate flow cycle percentage in *B?*†

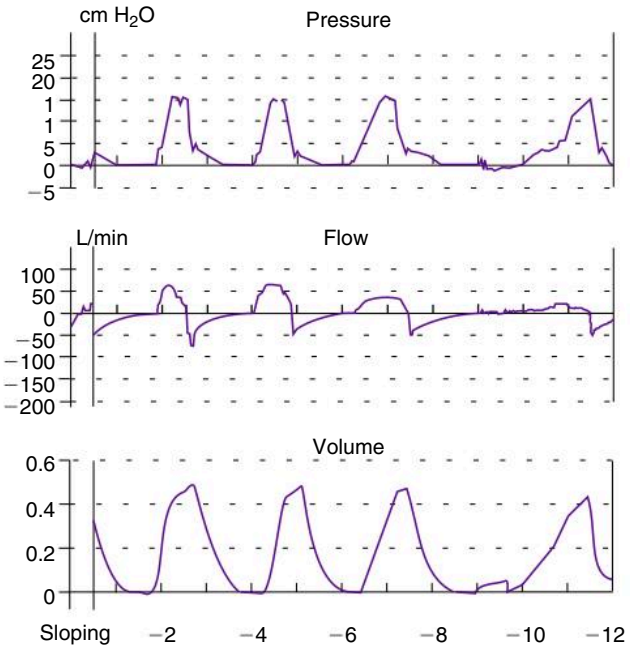


Fig. 9.38 Changes in the gas delivery system produced by adjusting the pressure slope, or rise time function, during pressure-targeted ventilation. (See text for additional information.) (Redrawn from Nilsestuen JO, Hargett K: Managing the patient-ventilator system using graphic analysis: an overview and introduction to Graphics Corner, *Respir Care* 41:1105—1122, 1996.)

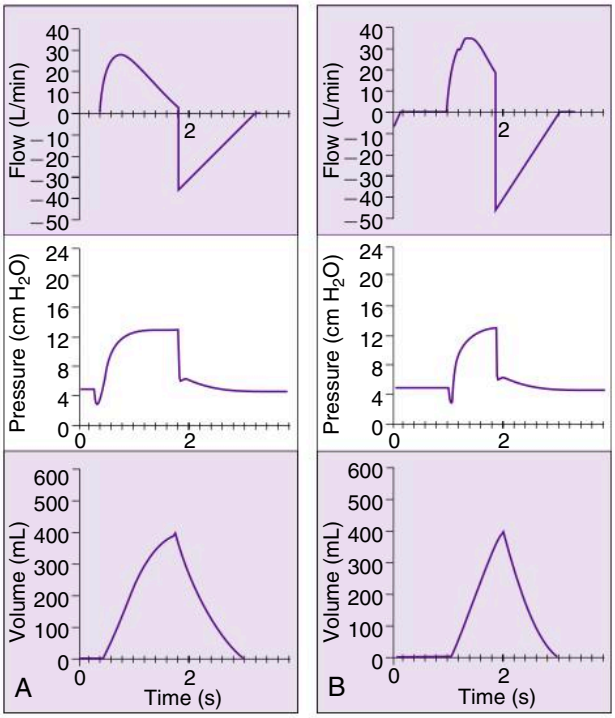


Fig. 9.39 Effect of changes in termination flow during PSV. (A) A low-percentage flow cycle is set so that inspiratory time (T_I) is longer. (B) A higher-percentage flow cycle is set so that T_I is shorter. (See text for additional explanation.) (From Hess DR, MacIntyre NR, Mishoe SC, et al.: *Respiratory care principles and practice,* Philadelphia, PA, 2002, WB Saunders.)

Case Study 9.2

A patient receiving PC-CMV demonstrates auto-PEEP on several breaths on the flow scalar (see *arrow* in Fig. 9.40, A). The therapist recommends a change in the inspiratory time setting to increase expiratory time. After 5 minutes on the new settings the following flow scalar was seen (see Fig. 9.40, B). Does the flow scalar show any evidence that the change reduced the level of auto-PEEP? Note that the expiratory flow now reaches the zero *(see arrow)* before the onset of the next breath, indicating that the change reduced the level of auto-PEEP.

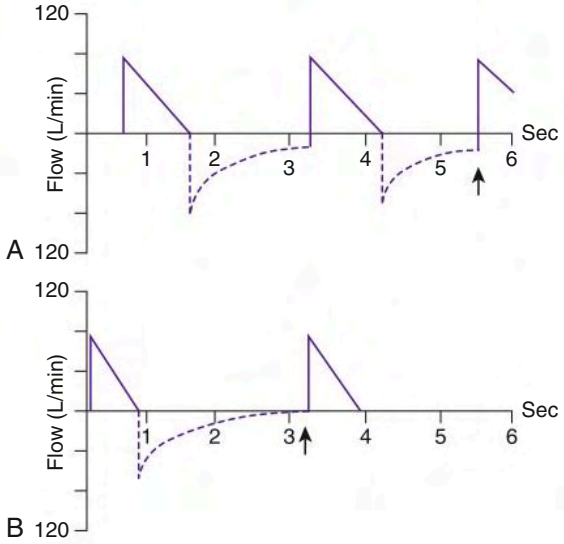


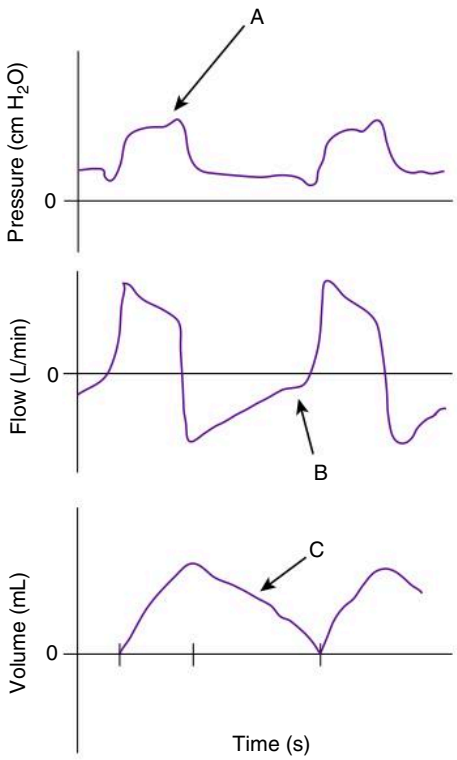
Fig. 9.40 Flow scalars for a patient showing the presence of auto-PEEP. See Case Study 9.2 for additional details.

SUMMARY

- Modern microprocessor ventilators provide graphic waveforms, including flow, volume, and pressure scalars and pressure-volume and flow-volume loops.
- Ventilator graphics can be used to monitor ventilator function, evaluate a patient's response to the ventilator, and help the clinician adjust ventilator settings.
- It is important to comprehend how the ventilator measures, computes, and displays various parameters. Clinicians must also study actual ventilator graphics to fully understand the usefulness of this application.
- The scalars most often displayed on the ventilator screen are P_{awo}, flow, and V.
- Pressure and flow scalars can provide an effective tool for identifying the PIP, PEFR, and presence of leaks in the patient circuit and auto-PEEP during VC-CMV.
- The flow waveform during volume-targeted ventilation may be set as rectangular or descending, although it is by default a descending waveform in pressure-targeted ventilation.
- The pressure waveform varies with changes in static lung compliance (C_S) and R_{aw} during volume-targeted ventilation. During pressure-targeted ventilation, changes in C_S and R_{aw} will affect the flow and volume waveforms.
- As lung characteristics deteriorate, the pressure delivered to a patient during PVS remains constant but the delivered volume may decrease.
- Pressure-volume loops can alert the clinician to changes in a patient's lung compliance, airway resistance, and work of breathing.
- Pressure and flow scalars are useful to detect patient-ventilator asynchrony.
- Flow-volume loops allow the clinician to evaluate a patient's response to bronchodilator therapy during mechanical ventilation. These loops can also be used to detect leaks and auto-PEEP.

REVIEW QUESTIONS *(See Appendix A for answers.)*

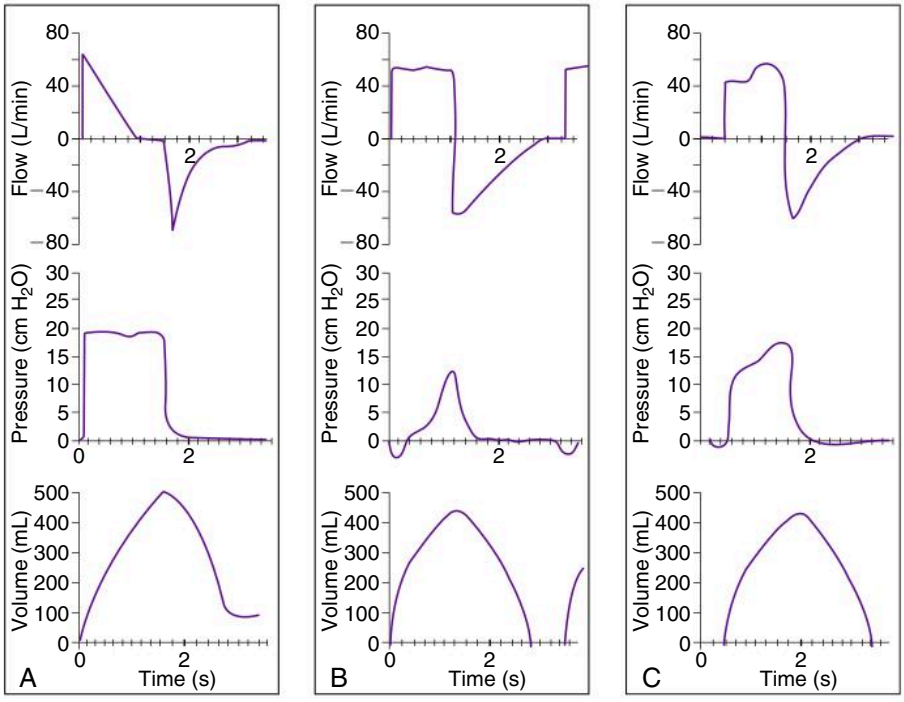
1. Refer to the scalars for pressure, flow, and volume in pressure support ventilation (PSV) in the figure below to answer the following questions.
   A. What caused the pressure spike indicated by arrow A on the pressure-time waveform?
   B. What ventilator parameter might be adjusted to eliminate this problem?
   C. What caused the flow waveform during exhalation indicated by arrow B?
   D. What parameters might be adjusted on the ventilator to eliminate this problem?
   E. What pulmonary change is suggested by the exhalation volume waveform indicated by arrow C?
   F. Is the flow cycle percentage set at a high or low percentage of peak flow?
   G. Is there any indication of inadequate inspiratory flow?



Pressure, flow, and volume scalars for PSV (see Review Question 1)

2. Use the scalars for a specific mode of ventilation in the figure below, A, to answer the following questions.

A. What target variable is illustrated?
B. What is the set pressure?
C. What is the delivered V_T?
D. What is the P_{plat}?
E. What problem is indicated by the volume-time curve?

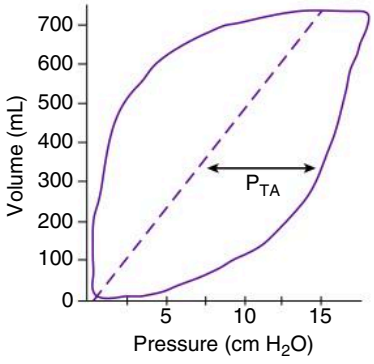


Flow, pressure, and volume scalars for three different ventilation situations (see Review Questions 2 through 4).

3. The scalars in the figure in Question 1, B are for a different mode of ventilation from that in the figure in Question 2, A. Use the scalars in part B to answer the following questions.
   A. What is the target variable illustrated and how do you determine the mode?
   B. What is the total cycle time?
   C. Is the breath patient triggered or time triggered?
   D. What problem is indicated by the pressure scalar?

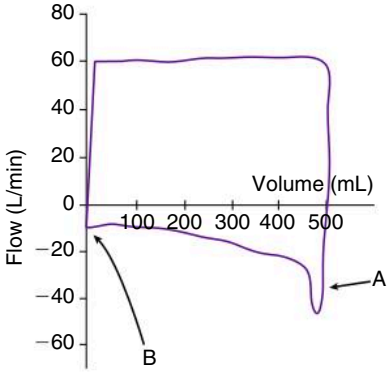
4. Answer the following questions with regard to the scalars for VC-CMV shown in the figure in Question 2, *C*.
   A. What is the set flow?
   B. Why is the flow delivery variable during inspiration?
   C. What causes the change in flow delivery and how does this affect volume delivery?

5. Answer the following questions using the figure below.
   A. What is the PIP?
   B. What is the approximate delivered V_T?
   C. Has a PEEP been set?
   D. What is the compliance?
   E. What is the approximate P_{TA} during inspiration as indicated by the double-headed arrow? Is this normal?
   F. From the appearance of this P-V loop, what do you think is the patient's primary problem?



P-V loop (see Review Question 5).

6. Answer the following questions using the figure below.
   A. What is the target variable in this figure?
   B. What are the flow setting and flow waveform?
   C. What is the V_T delivery?
   D. What causes the artifact indicated by arrow *A*?
   E. What does arrow *B* indicate?
   F. What might be the cause of this patient's pulmonary problem?



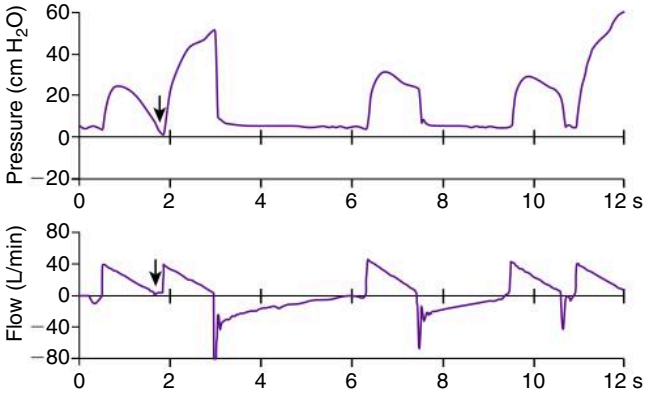
F-V loop (see Review Question 6).

7. A ventilator is set for volume-targeted ventilation, constant flow, and control mode. What will happen to the PIP, P_{plat}, T_I, and V_T if lung compliance (C_L) decreases? (Assume that the pressure limit is not reached.)

8. A ventilator is set for pressure-targeted ventilation, patient triggering, and time cycling. What will happen to the set pressure, T_I, and V_T if C_L increases? (Assume that the pressure limit is not reached.)

9. A patient receiving pressure ventilation has a C_L of 15 mL/cm H_2O (0.015 L/cm H_2O). The pressure is set at 35 cm H_2O. The ventilator is time cycled at 2 seconds. Flow drops to zero before the end of inspiration.
   A. What will the P_{alv} be?
   B. What is an estimated volume delivery?
   C. C_L changes to 30 mL/cm H_2O with improvement in the patient's lung condition. What will happen to the flow and volume delivery?
   D. How would you change volume delivery to return it to its previous value?

10. What type of asynchrony is shown in the figures below?
    A. Flow asynchrony
    B. Trigger asynchrony
    C. Termination asynchrony
    D. Cycle asynchrony



11. What would be your suggestion to resolve the problem shown in Question 10?
    A. Increase PEEP.
    B. Reduce inspiratory time.
    C. Adjust trigger sensitivity.
    D. Use a variable flow breath type.

Bibliography

1. Branson RD: Enhanced capabilities of current ICU ventilators: do they really benefit patients? *Respir Care* 36:362–376, 1991.
2. Branson R: Understanding and implementing advances in ventilator capabilities, *Curr Opin Crit Care* 10:23–32, 2004.
3. Brochard L, LeNouche F: Pressure support ventilation. In Tobin MJ, editor: *Principles and practices of mechanical ventilation*, ed 2, New York, NY, 2006, McGraw-Hill.
4. Cairo JM: *Mosby's respiratory care equipment*, ed 10, St. Louis, MO, 2018, Elsevier.
5. Chatburn RL: Classification of mechanical ventilators, *Respir Care* 37:1009–1025, 1992.
6. Chatburn RL: A new system for understanding mechanical ventilators, *Respir Care* 36:1123–1155, 1991.
7. Croci M, Pelosi P, Chiumello D, et al.: Regulation of pressurization rate reduces inspiratory effort during pressure support ventilation: a bench study, *Respir Care* 41:880–884, 1996.
8. Dhand R: Ventilator graphics and respiratory mechanics in the patient with obstructive lung disease, *Respir Care* 50:246–261, 2005.
9. Du HL, Ohtsuji M, Shigeta M, et al.: Expiratory asynchrony in proportional assist, *Am J Respir Crit Care Med* 165:972–977, 2002.
10. Durbin CG: Applied respiratory physiology: use of ventilator waveforms and mechanics in the management of critically ill patients, *Respir Care* 50:287–293, 2005.
11. Harris RS, Hess DR, Venegas JG: An objective analysis of the pressure-volume curve in the acute respiratory distress syndrome, *Am J Respir Crit Care Med* 161:432–439, 2000.
12. Hess DR: Mechanical ventilation strategies: what's new and what's worth keeping? *Respir Care* 47:1007–1017, 2002.
13. Hess DR, Branson RD: Mechanical ventilation. In Hess DR, MacIntyre NR, Mishoe SC, et al., editors: *Respiratory care principles and practice*, ed 2, Sudbury, MA, 2002, Jones and Bartlett.
14. Kacmarek RM, Hess D, Stoller JK: *Monitoring in respiratory care*, St. Louis, MO, 1993, Mosby.
15. Kondili E, Prinianakis G, Georgopoulos D: New concepts in respiratory function, *Br J Anaesthesiol* 91:106–119, 2003.
16. Lucangelo U, Bernabé F, Blanch L: Respiratory mechanics derived from signals in the ventilator circuit, *Respir Care* 50:55–65, 2005.
17. Nilsestuen JO, Hargett K: Managing the patient-ventilator system using graphic analysis: an overview and introduction to Graphics Corner, *Respir Care* 41:1105–1122, 1996.
18. Nilsestuen JO, Hargett KD: Using ventilator graphics to identify patient-ventilator asynchrony, *Respir Care* 50:202–234, 2005.
19. Rodriguez PO, Bonelli I, Setten M, et al.: Transpulmonary pressure and gas exchange during decremental PEEP titration in pulmonary ARDS patients, *Respir Care* 58(5):754–763, 2013.
20. Sanborn WG: Monitoring respiratory mechanics during mechanical ventilation: where do the signals come from? *Respir Care* 50:28–52, 2005.
21. Sassoon CSH, Mahutte CK: What you need to know about the ventilator in weaning, *Respir Care* 40:249–256, 1995.
22. Waugh JB, Deshpande VM, Brown MK, et al.: *Rapid interpretation of ventilator waveforms*, ed 2, Upper Saddle River, NJ, 2007, Prentice Hall.
23. Yamada Y, Du HL: Effects of different pressure support termination on patient-ventilator synchrony, *Respir Care* 43:1048–1057, 1998.
24. Henning RJ, Shubin H, Weil MH: The measurement of the work of breathing for the clinical assessment of ventilator dependence, *Crit Care Med* 5:264, 1977.
25. Benditt JO: Esophageal and gastric pressure measurements, *Respir Care* 50:68–75, 2005.

CHAPTER 10

Assessment of Respiratory Function

OUTLINE

KEY TERMS

- Capnography
- Fractional hemoglobin saturation
- Functional hemoglobin saturation
- Indirect calorimetry
- Metabolic monitor
- Pressure broadening
- Pulse oximetry
- Qualitative
- Quantitative
- Transcutaneous monitoring

LEARNING OBJECTIVES

On completion of this chapter, the reader will be able to do the following:

1. Describe the principle of operation of the pulse oximeter.
2. Identify physiological and technical factors that can influence the accuracy of pulse oximetry readings.
3. Describe how various clinical conditions can affect CO-oximetry, oxygen saturation (S_aO_2) and pulse oximetry, and oxyhemoglobin saturation (S_pO_2).
4. Discuss the normal components of a capnogram.
5. Give examples of pathophysiological conditions that can alter the contour of the capnogram.
6. Discuss how arterial-to-end-tidal partial pressure of carbon dioxide ($P_{[a\text{-}et]}CO_2$) is affected by changes in ventilation-perfusion relationships.
7. Discuss how volumetric CO_2 tracings can be used to assess gas exchange during mechanical ventilation.
8. Describe how exhaled nitric oxide measurements can be used in the management of patients with asthma.
9. Explain the theory of operation of transcutaneous PO_2 and PCO_2 monitors and list the clinical data that should be recorded when making transcutaneous measurements.
10. Provide the respiratory quotient (RQ) value associated with substrate utilization patterns in normal, healthy subjects.
11. Discuss some clinical applications of metabolic monitoring in critically ill patients.
12. Briefly describe devices that are used to measure airway pressures, volumes, and flows during mechanical ventilation.
13. Calculate mean airway pressure, dynamic compliance, static compliance, and airway resistance.
14. Identify pathological conditions that alter lung compliance and airway resistance and measurements of the work of breathing.
15. Define the pressure-time product and discuss its application in the management of mechanically ventilated patients.

Procedures and devices such as pulse oximetry, capnography (capnometry), transcutaneous monitoring of blood gases, exhaled nitric oxide (NO), indirect calorimetry, and bedside lung function testing have made it possible for respiratory therapists to monitor respiratory function noninvasively in mechanically ventilated patients. When it is used appropriately, noninvasive monitoring can provide valuable information for clinicians managing patients receiving ventilatory support. However, if it is used indiscriminately, it can be distracting and confusing for the clinician and economically costly for the patient.

Fig. 10.1 Pulse oximeter. (Courtesy Nonin Medical, Plymouth, Minn.)

NONINVASIVE MEASUREMENTS OF BLOOD GASES

Pulse Oximetry

Hypoxemic events in mechanically ventilated patients are most often associated with apnea, airway obstruction, equipment failure or disconnection, and incorrect gas flow settings. Visual recognition of hypoxemia by physical examination is often unreliable because of intraobserver variability, differences in patients' skin pigmentation, and interference by ambient lighting.[1,2] Laboratory measurement of arterial blood gases (ABGs) remains the gold standard for measuring the level of hypoxemia (see Evolve website for a review of ABGs); however, this procedure is performed intermittently and may fail to detect transient hypoxic episodes.

Pulse oximetry provides continuous, noninvasive measurements of arterial oxygen (O_2) saturation.[3] A sensor is placed over a digit, an earlobe, the forehead, or the bridge of the nose; this sensor measures the absorption of selected wavelengths of light beamed through the tissue (Fig. 10.1). For example, oxyhemoglobin can be differentiated from deoxygenated hemoglobin by shining two wavelengths of light (660 and 940 nm) through the sampling site. As Fig. 10.2 illustrates, at a wavelength of 660 nm (red light), deoxygenated hemoglobin absorbs more light than oxyhemoglobin. Conversely, oxyhemoglobin absorbs more light at 940 nm (infrared [IR] light) than does deoxygenated hemoglobin.

Pulse rate is determined by relating cyclical changes in light transmission through the sampling site with blood volume changes that occur during ventricular systole and diastole. That is, as local (e.g., finger, toe, or earlobe) blood volume increases during ventricular systole, light absorbency increases and transmitted light decreases. In contrast, as blood volume decreases during diastole, absorbency decreases and transmitted light increases. Fig. 10.3 illustrates the pulsatile or alternating current (AC) and nonpulsatile or direct current (DC) components of a typical pulse oximetry signal.

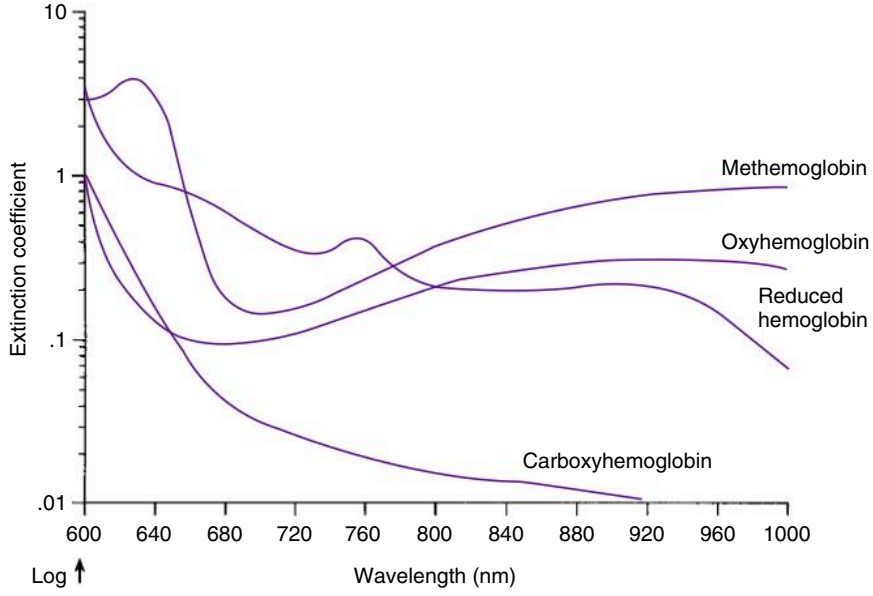


Fig. 10.2 Absorption characteristics of four types of hemoglobin: reduced hemoglobin, oxyhemoglobin, carboxyhemoglobin, and methemoglobin.

The percentage of oxyhemoglobin present can be determined by first calculating the ratio of absorbencies for pulsatile and nonpulsatile flow, at the two specified wavelengths, or

$$\frac{\text{Red}}{\text{Infrared}} = \frac{\dfrac{\text{Pulsatile}_{660\text{nm}}}{\text{NonPulsatile}_{660\text{nm}}} \div \text{Pulsatile}_{940\text{nm}}}{\text{NonPulsatile}_{940\text{nm}}}$$

This ratio is then applied to an algorithm that relates ratios of these two absorbencies to oxyhemoglobin saturation.[4]

Physiological and Technical Concerns

Pulse oximeters are generally accurate for O_2 saturations greater than 80%.[5,6] Pulse oximeter saturations less than 80% should be confirmed with laboratory analysis of ABGs, including CO-oximetry.[7,8] A number of physiological and technical factors can influence the accuracy of pulse oximetry measurements, including low perfusion states, the presence of dysfunctional hemoglobins and dyes, variations in patients' skin pigmentation, and ambient light interference. The following is a summary of various factors that can influence pulse oximetry readings. A more detailed discussion of each of these factors can be found in the references listed at the end of this chapter.

Low Perfusion States. It should be apparent that the accuracy of a pulse oximeter reading depends on the identification of an arterial pulse. In cases in which perfusion is low, such as hypovolemia, the pulse oximeter may not be able to accurately identify a pulsatile signal, resulting in either an intermittent or absent S_pO_2 reading. Other situations that may contribute to this problem include administering peripheral vasoconstrictors, hypothermia, and heart-lung bypass (i.e., extracorporeal membrane oxygenation).[9] Although some oximeters are better than others in dealing with low perfusion states, compensation for the weak signal associated with low perfusion states is limited. The reason for this limitation is that a low perfusion state produces a low signal-to-noise ratio and thus a signal that can potentially be altered by motion artifacts.

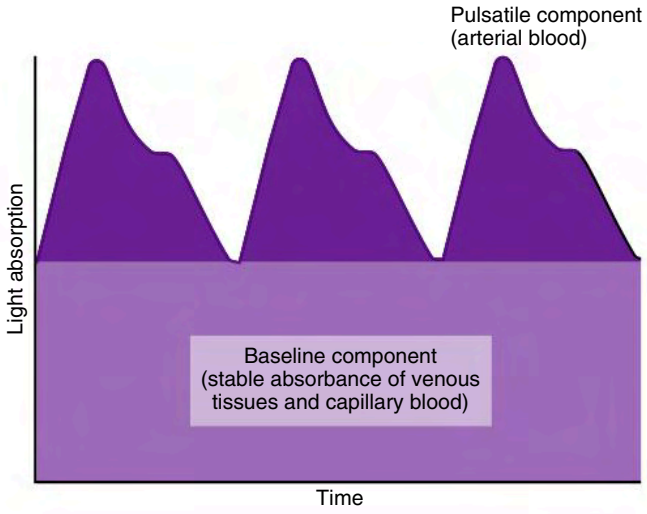


Fig. 10.3 Output signal generated by pulse oximeter illustrating pulsatile and nonpulsatile components. Saturation is based on the ratio of light absorption during pulsatile and baseline phases. (From Kacmarek RM, Stoller JK, Stoller, Heuer AJ: *Egan's fundamentals of respiratory care*, ed 12, St. Louis, MO, 2021, Elsevier.)

The Masimo Signal Extraction Technology (SET) (Masimo Corp, Irvine, CA) pulse oximeter is a relatively new processing system that uses special algorithms to minimize interference from motion artifacts. Conventional pulse oximetry assumes that arterial blood accounts for the pulsations, or AC component, of the pulse oximetry signal and venous blood produces a nonpulsatile, or DC component, of the signal. During patient motion, venous blood may contribute to the pulsatile signal and cause the pulse oximeter to underestimate the S_pO_2 because it cannot distinguish between arterial and venous blood. Masimo SET signal processing identifies the venous blood signal, isolates it, and, using adaptive filters, cancels the noise and extracts the arterial signal. When tested on healthy individuals simulating various motion artifacts, the Masimo SET oximeter exhibited a much lower error rate compared with conventional pulse oximeters. Studies with critically ill patients also demonstrated fewer false alarms of hypoxemia compared with conventional pulse oximeters.[10]

Dysfunctional Hemoglobins and Dyes. Adult blood typically contains four types of hemoglobin: reduced or deoxygenated hemoglobin (HHb), oxyhemoglobin (O_2Hb), carboxyhemoglobin (COHb), and methemoglobin (MetHb). Two terms that are often used when describing O_2Hb saturation are fractional and functional saturations. **Fractional hemoglobin saturation** is calculated by dividing the amount of O_2Hb measured by the sum of concentrations of all four types of hemoglobin present, or

$$\text{Fractional } O_2\text{Hb} = O_2\text{Hb} \,/\, [\text{HHb} + O_2\text{Hb} + \text{COHb} + \text{MetHb}]$$

Functional hemoglobin saturation is calculated by dividing the O_2Hb concentration by the concentration of hemoglobin capable of carrying O_2, or

$$\text{Fractional}O_2\text{Hb} = \frac{O_2\text{Hb}}{[\text{HHb} + O_2\text{Hb}]}$$

Laboratory CO-oximeters measure all four types of hemoglobin by using separate wavelengths of light to identify each species, whereas pulse oximeters use only two wavelengths to quantify the amount of O_2Hb and HHb present. Laboratory CO-oximeters are therefore capable of reporting fractional O_2Hb saturations, and pulse oximeters are typically described as evaluating functional hemoglobin saturations.

This description of a pulse oximeter capability may be somewhat misleading because, as Fig. 10.2 shows, O_2Hb and COHb have similar absorption coefficients for red light (660 nm), whereas COHb is relatively transparent to IR light (940 nm). Additionally, MetHb and HHb have the same absorption coefficients for red light; however, MetHb demonstrates a greater absorbency for IR light (940 nm) than does O_2Hb. Accordingly, the presence of significant levels of COHb, as occurs in carbon monoxide poisoning, will lead to an overestimation of S_pO_2 (Key Point 10.1).[8] Methemoglobinemia, a potential complication of administering nitric oxide, benzocaine (topical anesthetic), and dapsone (an antibiotic used to treat malaria and *Pneumocystis*

***Key Point* 10.1** Abnormal hemoglobin, such as carboxyhemoglobin (smoke exposure), produces an erroneously high S_pO_2. If abnormal hemoglobin levels are suspected, a CO-oximeter should be used to evaluate the oxygen saturation.

jiroveci), can cause erroneous S_pO_2 values because MetHb absorbs both red and IR light.[6,11] If enough MetHb is present to dominate all pulsatile absorption, the pulse oximeter will measure a ratio of red to IR of 1:1, corresponding to an S_pO_2 of about 85%. Consequently, the pulse oximeter reading will overestimate or underestimate the *true oxyhemoglobin* saturation, depending on whether the actual S_aO_2 is less than or greater than 85%.[9,11]

It is well documented that intravascular dyes can adversely affect S_pO_2 values by absorbing a portion of the incident light emitted by the pulse oximeter diodes. Scheller and colleagues[12] demonstrated that injection of methylene blue and indigo carmine into human volunteers caused a false drop in S_pO_2, whereas indocyanine green had little effect on pulse oximeter values.

Nail Polish. Nail polish (particularly blue and black nail polish) can affect S_pO_2 readings. It has been suggested that nail polish causes light to be shunted around the finger periphery (called optical shunting).[13,14] The transmitted light never comes in contact with the vascular bed; consequently, SpO_2 values can be erroneously high or low, depending on whether this light takes on a pulsatile character. Placing the device over the lateral aspects of the digit rather than over the nail can largely alleviate this problem.

Skin Pigmentation. Theoretically, skin pigmentation should have no effect on pulse oximeter readings. In practice, however, S_pO_2 readings are typically higher for patients with dark skin pigmentation. For example, an S_pO_2 of 95% in an African American patient may actually represent an S_aO_2 of only 92%.[15-17] Many clinicians therefore use higher threshold values (i.e., S_pO_2 >92%) for initiating O_2 therapy in African American patients. Although using higher target S_pO_2 values does not lead to untreated hypoxemia in most of these patients, some will have arterial O_2 pressure (P_aO_2) values as high as 200 mm Hg when therapy is based on measured S_pO_2.

Bilirubin, a breakdown product of heme metabolism, is the pigment responsible for the yellow discoloration seen in jaundiced patients. Although an elevated bilirubin level (>20 mg/dL) has been shown to affect O_2Hb values recorded with CO-oximetry, pulse oximetry measurements do not appear to be affected by hyperbilirubinemia.[18,19]

Ambient Light. Direct sunlight and external light sources (e.g., fluorescent lights, heat lamps, fiberoptic light sources, surgical lamps) have been shown to affect S_pO_2 readings adversely.[20-22] Most commercially available pulse oximeters attempt to compensate for this interference by continually cycling the transmitted red and IR light on and off at a rate of about 480 cycles per second.

Clinical Applications

The usefulness of pulse oximetry as an early warning system for detecting hypoxemia in patients with unstable oxygenation status is well recognized. Pulse oximetry is an excellent trending device in critically ill patients, providing a continuous display of O_2 saturation. However, changes in S_pO_2 may not represent an equivalent change in actual S_aO_2.[23,24] This discrepancy is particularly evident when pulse oximetry is used in the neonatal intensive care unit (ICU). Although it is generally used to trend O_2 saturations in neonates, pulse oximetry is not used as a basis for prescribing O_2 therapy in neonates; most neonatologists prefer to base O_2 therapy decisions on P_aO_2 rather than O_2 saturation.[25,26]

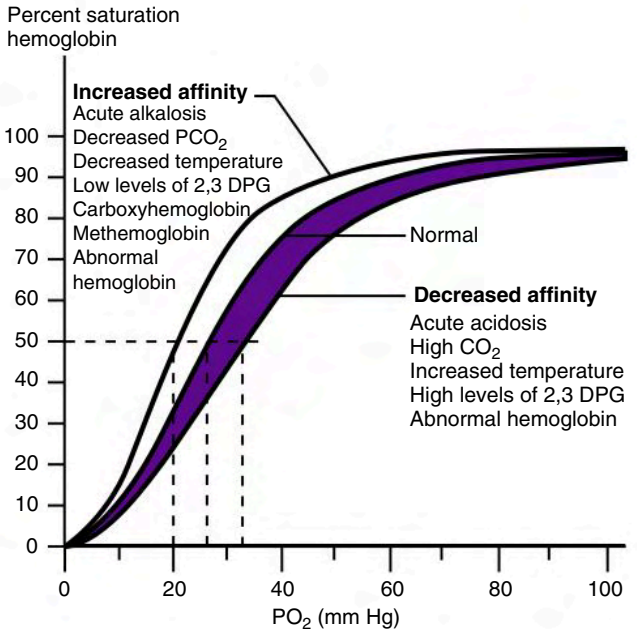


Fig. 10.4 Oxyhemoglobin dissociation curve for arterial blood. *2,3 DPG,* Diphosphoglycerate.

A review of the relationship between S_aO_2 and P_aO_2 (i.e., the oxyhemoglobin dissociation curve) is helpful before the use of pulse oximetry to detect hypoxemia is discussed. S_aO_2 varies with the P_aO_2 in a sigmoidal or S-shaped manner. (See Fig. 10.4 for the oxyhemoglobin dissociation curve for arterial blood.) For O_2 saturations greater than 90% saturation, P_aO_2 may rise considerably without much change in S_aO_2. When saturation is less than 80%, the PO_2 values fall rapidly. Although an S_aO_2 of 97% is considered normal, maintaining an O_2Hb saturation of at least 90% is considered acceptable for adult patients. As a result, many algorithms for O_2 therapy typically use 90% or slightly higher as an indication for increasing the fractional inspired O_2 concentration (F_IO_2). (As mentioned, because dark skin pigmentation can lead to erroneously high S_pO_2, many clinicians use a threshold of 94% —95% for these patients as an indication for adjusting the F_IO_2.) In the case of hyperoxygenation, pulse oximeters can provide limited information about P_aO_2 values because of the flat portion of the oxyhemoglobin dissociation curve above 90%.

Several criteria should be met to ensure that pulse oximetry values are meaningful. Box 10.1 contains a summary of the American Association for Respiratory Care (AARC) clinical practice guidelines for pulse oximetry. These guidelines provide valuable information that practitioners should use to determine whether S_pO_2 values are valid.

Advances in light-emitting diode technology have led to simplification of pulse oximeter transmitters and sensors, making them easy to use and available at a relatively low cost. Pulse oximetry probes are available in neonatal, pediatric, and adult sizes. The response time of the pulse oximeter (i.e., the time required for the pulse oximeter to detect a change in central oxygenation level [left-heart PO_2]) depends on the location of the probe. The lag time is shortest for probes placed on the earlobe; on the finger, the lag time is longer by 12 seconds or more than for the earlobe; and probes placed on the toe have the longest lag time.

BOX 10.1 Summary of the American Association for Respiratory Care Clinical Practice Guideline for Pulse Oximetry

Indications

On the basis of current evidence, pulse oximetry is useful for the following:

1. Monitoring arterial oxyhemoglobin saturation
2. Quantifying the arterial oxyhemoglobin saturation response to therapeutic intervention
3. Monitoring arterial oxyhemoglobin saturation during bronchoscopy

Contraindications

Pulse oximetry may not be appropriate in situations in which measurements of pH, P_aCO_2, and total hemoglobin are required. The presence of abnormal hemoglobin may be a relative contraindication.

Limitations

A number of factors, agents, and situations can affect readings and limit precision and performance of pulse oximetry. These include the following:

1. Motion artifacts
2. Abnormal hemoglobin (particularly carboxyhemoglobin and methemoglobin)
3. Intravascular dyes
4. Exposure of the measuring sensor to ambient light sources
5. Low perfusion states
6. Skin pigmentation
7. Nail polish
8. Low oxyhemoglobin saturations (i.e., below 83%)

Monitoring

The following information should be recorded during pulse oximetry:

1. Probe type and site of measurement, date and time of measurement, patient position, activity level
2. F_IO_2 and mode of supplemental O_2 delivery
3. Arterial blood gas measurements and CO-oximetry results that may have been made simultaneously
4. Clinical appearance of the patient (presence of cyanosis, skin temperature)
5. Agreement between pulse oximeter heart rate and heart rate determined by palpation or electrocardiographic recordings

From AARC Clinical Practice Guideline: Pulse oximetry, *Respir Care* 37:891–897, 1992.

As mentioned earlier, pulse oximetry is used routinely to monitor continuously the oxygenation status of critically ill patients with unstable oxygenation status and to monitor O_2 saturation during surgery and bronchoscopy. Pulse oximetry can be particularly useful in the ICU for titrating F_IO_2 and positive end-expiratory pressure (PEEP) in mechanically ventilated patients and for monitoring the oxygenation status of patients undergoing chest physical therapy and suctioning.[22]

Although pulse oximetry can be effective when prescribing O_2 therapy in hospitalized patients, it may not be as useful in prescribing O_2 therapy for homecare patients. Carlin and colleagues[25] demonstrated that the use of pulse oximetry only can disqualify a significant number of patients applying for reimbursement for O_2 therapy. Under the guidelines of the Centers for Medicare and Medicaid Services, a patient must demonstrate a P_aO_2 of 55 Torr or lower or a saturation of 88% or lower to qualify for O_2 therapy.[24] It is important to recognize that any of the previously mentioned physiological or technical problems can significantly affect S_pO_2 measurements. To resolve this problem, invasive ABG analyses should be done in chronically ill patients to establish the need for O_2 therapy. Case Study 10.1 involves pulse oximetry.

Case Study 10.1

Causes of Cyanosis

You are preparing a patient for bronchoscopy. As you are administering an aerosol treatment with benzocaine, you note that the patient appears to be cyanotic but does not demonstrate any signs of distress. Pulse oximetry readings indicate that the S_pO_2 is 85%. You immediately obtain arterial blood gases that demonstrate a pH of 7.36, PCO_2 of 42 Torr, and PO_2 of 80 Torr. Explain the cause of the cyanosis. What diagnostic test would confirm this explanation?

CAPNOGRAPHY (CAPNOMETRY)

Capnography is the measurement of carbon dioxide (CO_2) concentrations in respired gases. Although the terms *capnography* and *capnometry* are often thought to be synonymous, capnography describes the continuous display of CO_2 concentrations as a graphic waveform called a *capnogram;* capnometry involves the display of exhaled CO_2 numerically without a waveform.[27,28]

Technical Considerations

Both chemical and spectroscopic methods can be used to perform capnometry. Chemical devices that rely on a disposable colorimetric detector provide **qualitative** estimates of exhaled CO_2. Spectroscopic devices (e.g., IR, Raman, acoustic, mass spectroscopy) provide **quantitative** data on the concentration of expired CO_2. Because chemical and IR analyzers are most often used for mechanically ventilated patients in the critical care setting, the following discussion will focus on these devices.

Chemical Methods

Chemical (colorimetric) capnometers are handheld devices composed of specially treated filter paper in a plastic casing that can be attached to the patient's endotracheal tube (ET) (Fig. 10.5). The amount of CO_2 present in the patient's inspired and exhaled gas can be estimated by noting the color changes in the filter paper. For example, the Nellcor Adult/Pediatric Colorimetric CO_2 Detectors (Medtronics Minimally Invasive Therapies, Minneapolis, MN) respond to exhaled CO_2 with a simple color change from purple to yellow. The color changes correspond to CO_2 concentration ranges, which are displayed on the device: "A" corresponds to a range of approximately 0.03% to less than 0.5% CO_2 (<4 mm

Fig. 10.5 Nellcor Adult/Pediatric Colorimetric CO_2 Detector. (Medtronics Minimally Invasive Therapies, Minneapolis, Minn.)

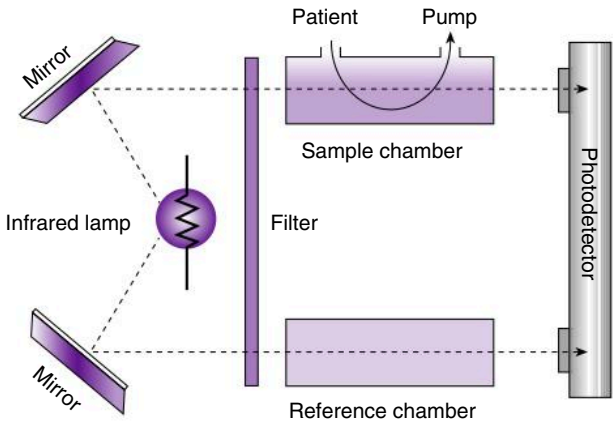


Fig. 10.6 Schematic diagram of a double-beam positive infrared capnograph. (From Kacmarek RL, Stoller JK, Heurer AJ, editors: *Egan's fundamentals of respiratory care*, ed 9, St. Louis, MO, 2009, Mosby.)

Hg); "B" indicates a range of 0.5% to <2.0% (4 to <15 mm Hg); and "C" indicates a range of 2.0% to 5.0% (15–38 mm Hg).

Chemical capnometers are particularly useful in emergency situations in assessing airway placement.[29] It is important to understand that changes in the color of the filter paper are the result of a chemical reaction that affects the pH of the filter paper. (Note that if acidic liquids such as regurgitated gastric contents contact the filter paper, an irreversible color change will occur and render the device unusable.) Proper placement of an ET can be determined because the color of the paper will change continually as inhaled and exhaled CO_2 levels vary while the patient breathes into and out of the device. In some cases, ET placement in the trachea rather than the stomach may be difficult to determine because the patient's gastric PCO_2 may be elevated after receiving mouth-to-mouth breathing or if the patient has recently ingested a carbonated beverage.

Infrared Spectroscopy

IR spectroscopy is based on the principle that molecules containing more than one element absorb IR light in a characteristic manner.[28] CO_2 absorbs IR radiation maximally at 4.26 μm. The concentration of CO_2 in a gas sample can be estimated because its concentration is directly related to the amount of IR light absorbed. The presence of other gases (e.g., water [H_2O] and nitrous oxide [N_2O]) can adversely affect the accuracy of CO_2 measurements by causing a phenomenon called **pressure broadening**. This phenomenon occurs because the peak absorbance of IR radiation by CO_2 lies between the peak absorbencies of H_2O and N_2O. The presence of these gases increases the absorption of IR radiation and results in erroneously high CO_2 readings. Pressure broadening can be minimized by removing water vapor from the gas sample before it is analyzed and by using electronic filters to subtract the IR absorption by gases other than CO_2.[21]

Fig. 10.6 is a schematic of a double-beam, positive-filter capnograph. The gas is drawn into a cuvette inside the sample chamber. IR radiation is beamed through the cuvette and through a reference chamber containing CO_2-free gas. The CO_2 in the sample chamber absorbs some of the radiation, reducing the amount of radiation that reaches the detector. The difference between the radiation transmitted through the sample cell and the radiation transmitted through the reference is converted into an electrical signal, which is amplified and displayed. The displayed value can be displayed in millimeters of mercury (mm Hg) representing the partial pressure or as percent CO_2 (%CO_2).

In clinical practice, IR analyzers are typically classified according to the method of sampling of respired gases. Fig. 10.7 illustrates two methods: sidestream sampling devices and mainstream sampling devices. In sidestream sampling devices, gas from the airway is extracted through a narrow plastic tube to the measuring chamber, which is located in a separate console. In mainstream devices, the sampling chamber attaches directly to the ET and analysis is performed at the airway.

Sidestream sampling devices show a slight delay between sampling and reporting times because of the time required to transport the sample from the airway to the measuring chamber. The plastic tube that transports the sample of gas to the analyzer is prone to plugging with water and secretions, which interferes with the delivery of gas to the analyzer. Contamination with ambient air, caused by leaks in the sample line, is also a concern.

In mainstream sampling devices the analyzer is attached directly to the ET; therefore, no delay occurs between sampling and reporting times. However, this type of device adds a small amount of dead space to the airway. The analyzer must be properly supported because the added weight it places on the artificial airway increases the possibility of dislodgement or complete extubation. Also, because the analyzer is attached directly to the airway, it is handled often and subject to damage from mishandling (e.g., dropping).

Physiological Considerations

Inspired air contains essentially no CO_2 ($\approx$0.3% CO_2). Expired air normally contains about 4.5% to 5.5% CO_2, which is primarily the product of cellular metabolism. Fig. 10.8 illustrates a capnogram for a healthy, resting adult breathing room air. The waveform, which displays the fractional concentration of expired CO_2 (F_ECO_2) versus time, is divided into four phases. In phase 1, the initial gas exhaled is from the conducting airways, which contain low levels of CO_2 from inspired air. During phase 2, alveolar gas containing CO_2 mixes with gas exhaled from the anatomical airways and the CO_2 concentration rises. In phase 3, the curve plateaus as alveolar gas is exhaled (this phase is often referred to as the *alveolar plateau*). The concentration of CO_2 at the end of the alveolar phase (just before inspiration begins) is referred to as

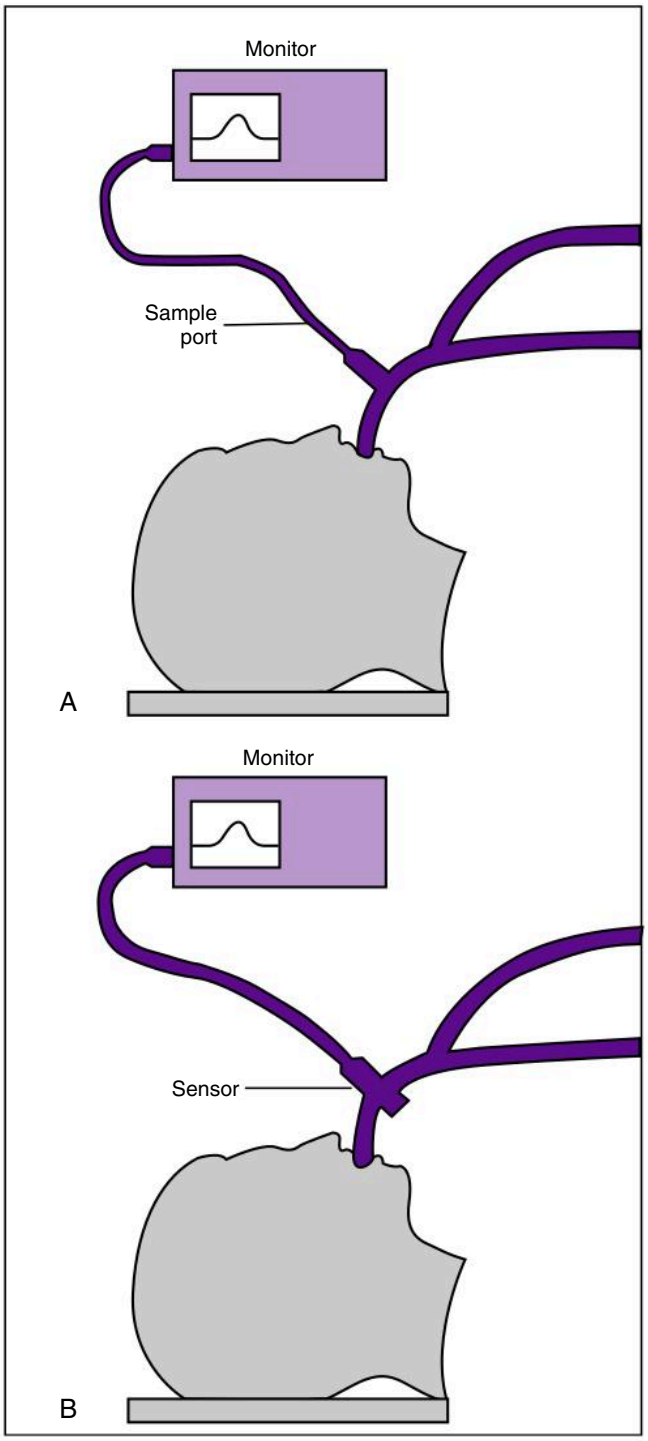


Fig. 10.7 Schematic illustrating (A) sidestream and (B) mainstream capnographs. (From Cairo JM: *Mosby's respiratory care equipment*, ed 11, St. Louis MO, 2022, Elsevier.)

the *end-tidal* PCO_2, or $P_{et}CO_2$. In phase 4 (inspiration) the concentration falls to zero.

The $P_{et}CO_2$ is dependent on the alveolar PCO_2 (P_ACO_2), which is ultimately influenced by CO_2 production ($\dot{V}CO_2$) and the effectiveness of ventilation (i.e., matching of ventilation to perfusion). The production of CO_2 is primarily determined by the

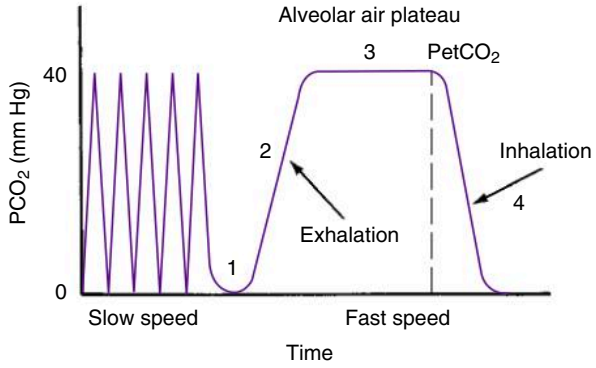


Fig. 10.8 Capnogram from a normal, healthy, resting subject breathing room air.

metabolic rate. Fever, sepsis, hyperthyroidism, and seizures increase metabolic rate and $\dot{V}CO_2$. Hypothermia, starvation, and sedation reduce metabolic rate and $\dot{V}CO_2$.

The relationship between ventilation and perfusion of the lung and gas exchange (i.e., P_ACO_2) can be expressed using $\dot{V}/\dot{Q}$ relationships.[30] Fig. 10.9 shows three $\dot{V}/\dot{Q}$ relationships that can potentially affect the level of alveolar PCO_2. In Fig. 10.9A, ventilation and perfusion are equally matched. The partial pressure of arterial CO_2 (P_aCO_2) and P_ACO_2 are nearly equal. The $P_{et}CO_2$ is normally about 4 to 6 mm Hg lower than the P_aCO_2. In Fig. 10.9B, ventilation decreases relative to perfusion (low $\dot{V}/\dot{Q}$). The P_ACO_2 eventually equilibrates with the mixed venous PCO_2 $\dot{V}/\dot{Q}$. Clinical situations in which this type of $\dot{V}/\dot{Q}$ relationship can exist throughout the lung (leading to higher than normal $P_{et}CO_2$ values) include respiratory center depression, muscular paralysis, and chronic obstructive pulmonary disease (COPD). In Fig. 10.9C, ventilation is higher than perfusion (high $\dot{V}/\dot{Q}$). Physiological dead space ventilation increases, and the P_ACO_2 approaches inspired air (0 Torr). Decreased $P_{et}CO_2$ values are found with this type of $\dot{V}/\dot{Q}$ relationship in patients with pulmonary embolism, excessive PEEP (extrinsic or intrinsic), and any disorder marked by pulmonary hypoperfusion.

Clinical Applications

Capnography has been shown to be a useful measurement in both spontaneously breathing and mechanically ventilated patients. Box 10.2 summarizes the key points of the AARC's clinical practice guideline for using capnography/capnometry in ventilated patients.[27] The following section discusses some of the most common uses of capnography.

Capnograph Contours

Changes in the contour of the capnogram can be used to detect increases in dead space ventilation, hyperventilation and hypoventilation, apnea or periodic breathing, inadequate neuromuscular blockade in pharmacologically paralyzed patients, and CO_2 rebreathing. They can also be used to monitor the effectiveness of gas exchange during cardiopulmonary resuscitation (CPR) and to detect accidental esophageal intubation.

Phase 3 (i.e., alveolar plateau) becomes indistinguishable from airway obstruction (i.e., increased physiological dead space) as occurs in COPD (Fig. 10.10A). Hyperventilation is characterized by a reduction in P_aCO_2 and therefore $P_{et}CO_2$ (Fig. 10.10B). Conversely, hypoventilation is associated with elevated P_aCO_2 and

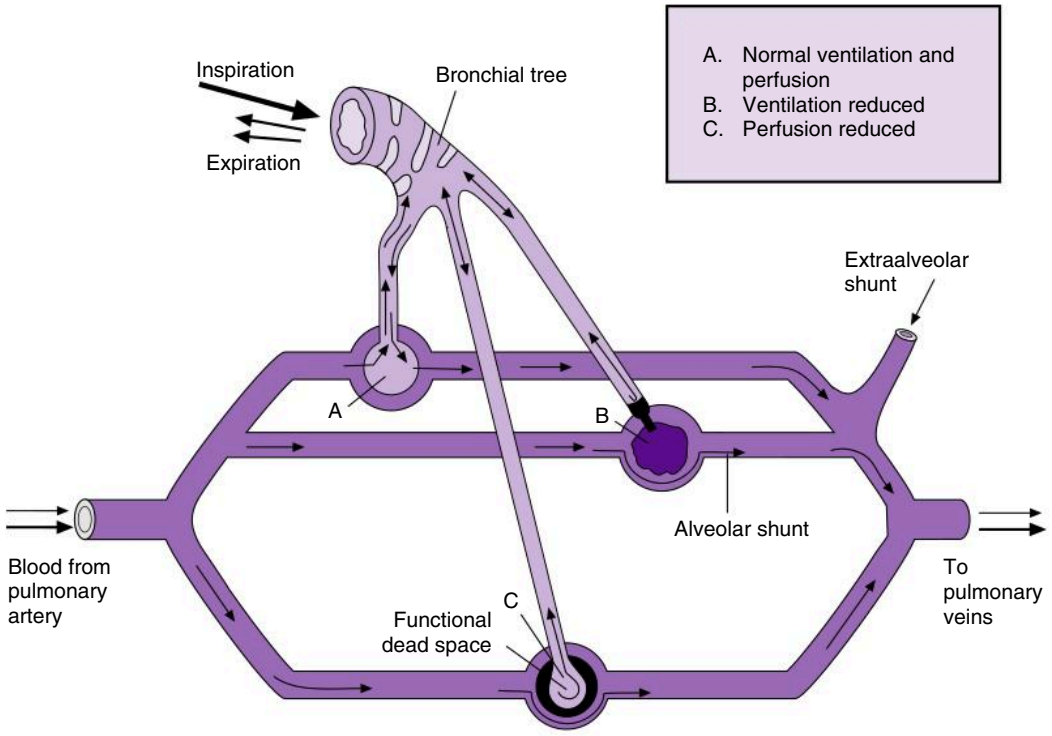


Fig. 10.9 Ventilation-perfusion relationships: (A) Normal. (B) Low $\dot{V}/\dot{Q}$. (C) High $\dot{V}/\dot{Q}$. (From Despopoulos A, Silbernagl S: *Color atlas of physiology,* ed 4, New York, 1991, Thieme Medical Publishers.)

BOX 10.2 Summary of American Association for Respiratory Care Clinical Practice Guideline for Capnography/Capnometry During Mechanical Ventilation

Indications

On the basis of current evidence, capnography is useful for the following:

1. Monitoring the severity of pulmonary disease and evaluating the response to therapy, especially therapy intended to improve V_D/V_T and ventilation-perfusion ($\dot{V}/\dot{Q}$) relationships. It may also provide valuable information about therapy directed at improving coronary blood flow.
2. Used as an adjunct to verify that tracheal rather than esophageal intubation has taken place.
3. Graphic evaluation of the integrity of the patient-ventilatory interface.
4. Monitoring the adequacy of pulmonary and coronary blood flow.
5. Screening patients for pulmonary embolism.
6. Detection of CO_2 rebreathing and the waning effects of neuromuscular blockade.
7. Monitoring CO_2 elimination.
8. Optimization of mechanical ventilation.

Contraindications and Complications

There are no absolute contraindications to capnography in mechanically ventilated adult patients. Mainstream devices increase the amount of dead space added to the ventilator circuit. The sampling rate of respired gases when using sidestream analyzers may be high enough to cause autotriggering when flow triggering of mechanical breaths is used. The effect is inversely proportional to the size of the patient. The gas-sampling rate can diminish delivered tidal volume in neonates and small patients while using volume-targeted or volume-controlled ventilation.

Monitoring

During capnography, the following should be recorded:

1. Ventilatory variables, including tidal volume, respiratory rate, PEEP, inspiratory/expiratory ratios, peak airway pressures, and concentrations of respiratory gases
2. Hemodynamic variables, including systemic and pulmonary pressures, cardiac output, shunt, and $\dot{V}/\dot{Q}$ imbalances

Limitations

Although capnography can provide valuable information about the efficiency of ventilation, in addition to systemic, pulmonary, and coronary perfusion, P_aCO_2 should be routinely determined by standard arterial blood gas analysis. Leaks in the ventilator circuit or leaks around the tracheal tube can lead to inaccurate measurements of expired CO_2. The reliability of the contour of the capnogram can also be affected by the stability of the minute volume, tidal volume, cardiac output, and CO_2 body stores. High breathing frequencies may exceed the response capabilities of the capnograph and therefore affect the integrity of the capnogram recorded. Low cardiac output may cause a false-negative result when attempting to verify the endotracheal tube (ET) position in the trachea. Positioning the ET in the pharynx and the presence of antacids and carbonated beverage in the stomach can lead to false-positive results when assessing ET placement.

From AARC Clinical Practice Guideline: Capnography/capnometry during mechanical ventilation, 2011, *Respir Care* 56(4):503–509, 2011.

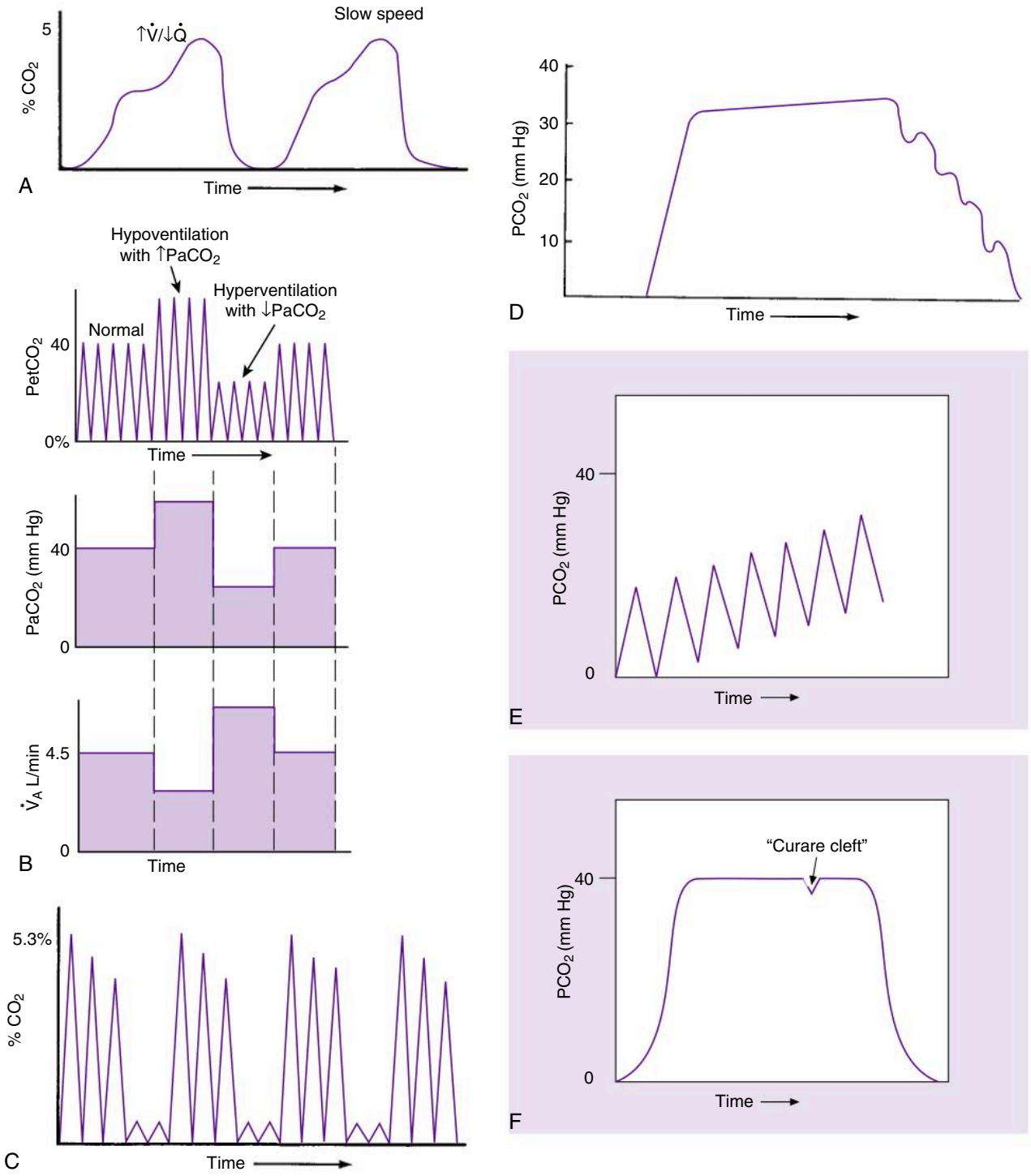


Fig. 10.10 Representative capnograms demonstrating (A) chronic obstructive pulmonary disease, (B) hypoventilation and hyperventilation, (C) Cheyne-Stokes breathing, (D) cardiac oscillations, (E) rebreathing exhaled air, and (F) "curare cleft." P_aCO_2, Arterial carbon dioxide pressure; PCO_2, partial pressure of carbon dioxide.

$P_{et}CO_2$ (see Fig. 10.10B). Fig. 10.10C is a capnogram of a patient with Cheyne-Stokes breathing. During bradypnea, phase 4 will occasionally show cardiac oscillations resulting from the motion of the beating heart transferred to the conducting airways (Fig. 10.10D). Failure of the capnogram to return to baseline is an indicator of rebreathing of exhaled gas (Fig. 10.10E). Fig. 10.10F shows a capnogram for a paralyzed patient, demonstrating the characteristic "curare cleft" during phase 3. This is a positive sign that the paralyzed patient is receiving insufficient neuromuscular blockade; however, other factors can contribute to this capnographic finding, such as patient-ventilator asynchrony.

Case Study 10.2

Capnography During Intubation

After considerable difficulty, an endotracheal tube is inserted without visualization of the trachea into a patient's airway during CPR. Capnography results show a $P_{et}CO_2$ of 3 Torr; a standard ABG measurement demonstrates a P_aCO_2 of 75 Torr. Explain the cause of this discrepancy in the capnography and ABG results.

***Key Point* 10.2** Capnography can be a valuable adjunct to verify tracheal rather than esophageal intubation during CPR.

Capnography can be used to detect pulmonary blood flow cessation, such as occurs with pulmonary embolism or during cardiac arrest. The American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care recommend the use of continuous quantitative waveform capnography for confirmation and monitoring of ET placement during CPR.[29] Laboratory studies suggest that the capnogram can be used as an indication of the progress and success of CPR. These studies demonstrated that $P_{et}CO_2$ increases as $\dot{V}/\dot{Q}$ is restored to normal.

As mentioned, capnography can be used to detect accidental esophageal intubation. The gastric PCO_2 is generally equal to room air; therefore failure to detect the characteristic changes in CO_2 concentration during ventilation possibly indicates esophageal intubation. However, low perfusion of the lungs is associated with low $P_{et}CO_2$ and should not be confused with esophageal intubation. Also, the gastric PCO_2 may be elevated after mouth-to-mouth breathing or if the patient recently ingested a carbonated beverage. Case Study 10.2 gives an example situation of the use of capnographic data (Key Point 10.2).

Arterial to Maximum End-Expiratory PCO_2 Difference

The $P_{(a\text{-}et)}CO_2$ for tidal breathing should be approximately 4 to 6 mm Hg. It is elevated in patients with COPD, left-sided heart failure, and pulmonary embolism caused by an increase in physiological dead space.[30,31] Another technique that can be used to further evaluate the severity of the disease is to compare arterial PCO_2 measurements with maximum expired PCO_2 measurements (the arterial to maximum expiratory PCO_2 gradient). With this technique, the expired PCO_2 recorded at the end of a maximum exhalation is compared with the P_aCO_2. The difference is normally minimal. Patients with COPD and left-sided heart failure do not show an arterial to maximum expired PCO_2 difference, whereas patients with pulmonary embolism do show an increased gradient. Fig. 10.11 shows the capnographic appearance of these differences.

Volumetric Capnometry

In addition to end-tidal CO_2 ($EtCO_2$) monitoring, another available application is volumetric capnometry. $EtCO_2$ monitoring focuses on exhaled CO_2 plotted over time, whereas volumetric capnometry focuses on exhaled CO_2 plotted relative to exhaled volume.[4] The Philips Respironics NM3 monitor is an example of a capnometer that can provide this type of monitoring (Fig. 10.12) (Philips Respironics, Inc., Murrysville, PA). (NOTE: The Philips Respironics NM3 monitor can also be used to estimate cardiac output noninvasively; see Chapter 11.)

Description of the Single-Breath CO_2 Curve

The single-breath CO_2 graph ($SBCO_2$) is produced by the integration of airway flow and CO_2 concentration; it is presented on a breath-to-breath basis. As shown in Fig. 10.13, the graph can provide information on anatomical dead space, alveolar dead space (when P_aCO_2 is known), and CO_2 elimination (VCO_2) for each breath. If a horizontal line is drawn at the top of the curve, representing $\%CO_2$ in arterial blood, three distinct regions of the curve are established.

Area X represents the actual amount of CO_2 exhaled in the breath, assuming that no exhaled air is rebreathed. In other words, the area under the $SBCO_2$ curve is the volume of CO_2 in a single breath. Adding all the single breaths in a minute gives the $\dot{V}CO_2$, the same results that would occur if exhaled gas were collected using a Douglas bag. Thus the CO_2 monitor can provide information about the volume of CO_2 in one breath ($\dot{V}CO_2$ VCO_2) and the volume of CO_2 produced in 1 minute ($\dot{V}CO_2$).

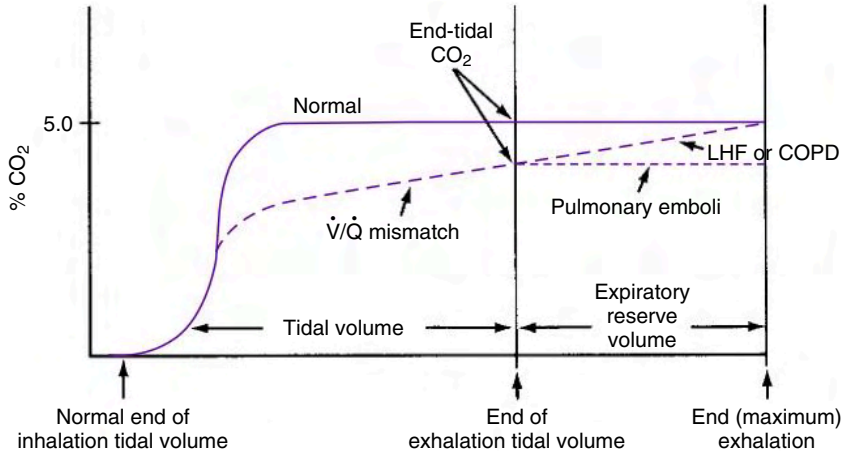


Fig. 10.11 $P_{(a\text{-}et)}CO_2$ for normal and forced expiratory capnogram. (From Darin J. Capnography, *Curr Rev Respir Ther* 3:146—150, 1981; Erickson L, Wollmer P, Olsson CG, et al.: Diagnosis of pulmonary embolism based upon alveolar dead space analysis, *Chest* 96:357—362, 1989; and Hatle CJ, Rokseth R: The arterial to end expiratory carbon dioxide tension gradient in pulmonary embolism and other cardiopulmonary diseases, *Chest* 66:352—357, 1974.)

Area Y represents the amount of CO_2 that is not eliminated because of alveolar dead space (i.e., ventilated alveoli that are poorly perfused or receive no perfusion at all). *Area Z* represents the amount of CO_2 that was not eliminated because of the anatomical dead space.

The relationship of the areas (with the added arterial blood line) provides some important parameters for analysis. The ratios of the areas created in the $SBCO_2$ curve are the same as the relationship seen in the Enghoff modified Bohr equation:

$$\frac{[P_aCO_2 - P_ECO_2]}{P_aCO_2} = \frac{(Y + Z)}{(X + Y + Z)}$$

where P_aCO_2 is arterial partial pressure for CO_2, and P_ECO_2 is mixed expired partial pressure for CO_2. (XYZ were defined previously.) Four major factors influence the amount of CO_2 exhaled: CO_2 production, perfusion of the lungs, diffusion, and ventilation.

Altering the production of CO_2 or any one of the factors involved in transport of CO_2 without compensation will result in changes in P_aCO_2 and the volume of CO_2 eliminated through the lungs.[32,33]

As previously mentioned, conditions that can alter the volume of CO_2 produced are related to changes in metabolic rate. For example, $\dot{V}CO_2$ increases in patients with sepsis, fever, severe burns to the body, or trauma and with increases in work of breathing (WOB; i.e., the respiratory muscles produce additional CO_2). If metabolic rate increases and ventilation does not increase, P_aCO_2 rises, and therefore the amount of CO_2 exhaled during the $SBCO_2$ maneuver increases.

Reductions in perfusion to the lungs, as occurs with pulmonary emboli and reductions in cardiac output, can lead to changes in the contour of the $SBCO_2$ curve. As perfusion to the lung decreases, the phase 2 portion of the curve shifts to the right, showing increased dead space in the system (i.e., less CO_2 exhaled). In this situation, physiological dead space increases. In addition, the area under the curve contains less CO_2; therefore less $\dot{V}CO_2$ VCO_2 per breath is eliminated because of the fact that less CO_2 is being delivered to the alveoli. (It is important to recognize that overall CO_2 production is not reduced.)

Application of PEEP can also alter the contour of the volumetric $SBCO_2$ curve. As PEEP is increased from 0 to 15 cm H_2O, the phase 2 portion of the curve shifts to the right because of expanding airways (increasing PEEP keeps the airways open) and reduced perfusion. The addition of PEEP can cause compression of the pulmonary capillaries and a decrease in perfusion to the lungs, reducing effective perfusion to the ventilated alveoli. This change represents an increase in alveolar dead space. The slope of phase 2 decreases as well; this is a result of lower CO_2 concentration occurring at an identical volume point on the *x*-axis, causing a rise in P_aCO_2.

Case Study 10.3 provides an example of how volumetric capnography can be used clinically.

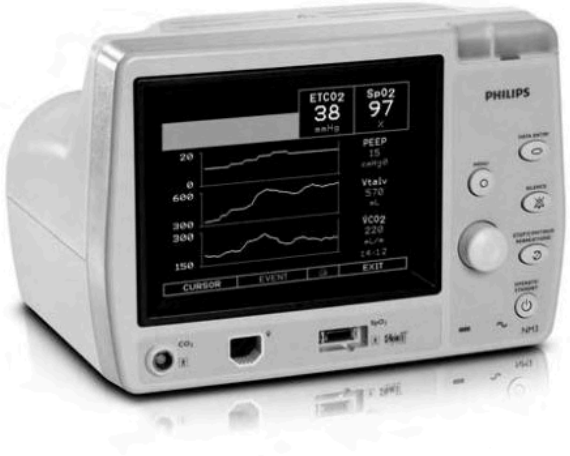


Fig. 10.12 An NM3 monitor. (Courtesy Philips North America Corporation, Andover, MA.)

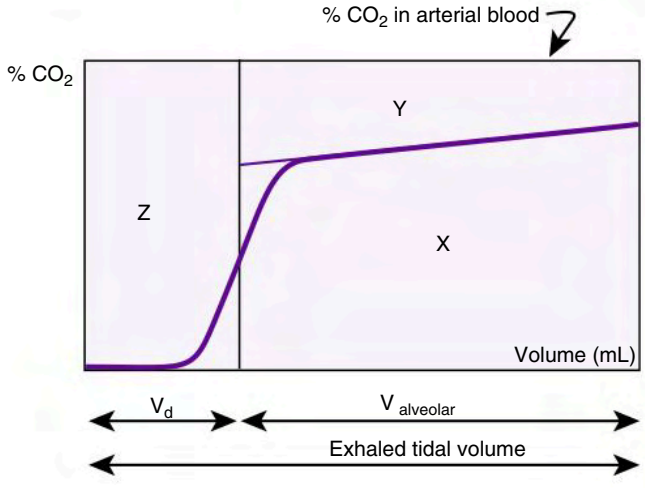


Fig. 10.13 Graph of exhaled volume (*x*-axis) versus $\%CO_2$ (*y*-axis). A horizontal line drawn at the top of the curve represents the $\%CO_2$ in arterial blood. Three distinct regions are illustrated: Area X represents actual CO_2 exhaled in one breath; area Y is the amount of CO_2 not eliminated because of alveolar dead space; and area Z is the amount of CO_2 not eliminated because of anatomical dead space. $V_{alveolar}$, Alveolar volume; V_d, dead space volume. (Redrawn from material from Philips North America Corporation, Andover, MA.)

Single-Breath CO_2 Loop of Inspiration and Exhalation

When the $SBCO_2$ graph includes both inspiration and exhalation, a loop is produced (Fig. 10.14). The net volume of CO_2 exhaled in

Case Study 10.3

Exercise

Dead Space Ventilation

A 35-year-old, 60-kg man is admitted to the ICU after a motor vehicle accident in which he sustained multiple rib fractures. He is receiving volume-targeted mechanical ventilation and is being monitored with pulse oximetry and volumetric capnography. You are asked to increase his PEEP level from +5 cm H_2O to +10 cm H_2O. After making the change, you notice that his S_pO_2 decreases from 93% to 90%. His $SBCO_2$ curve has shifted to the right, and the $P_{et}CO_2$ decreased from 30 mm Hg to 25 mm Hg. Briefly describe why these changes may have occurred.

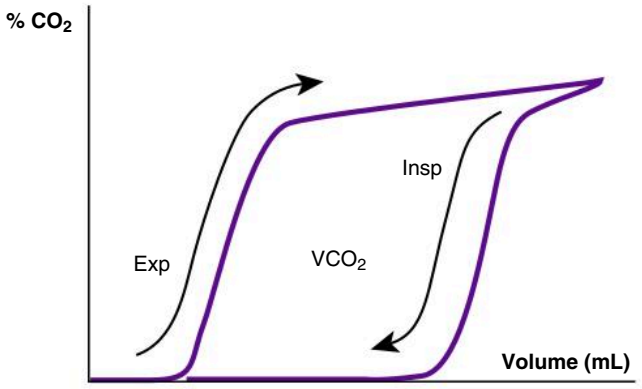


Fig. 10.14 A single-breath CO_2 curve ($SBCO_2$) recorded during inspiration and expiration. (Courtesy Ted Tabor, RRT, Paris, France.)

one breath is the area between the exhaled and inhaled CO_2 of this loop. The net CO_2 in one breath is the difference between the amount of CO_2 inhaled (which is typically negligible) and the amount of CO_2 exhaled.

Trending CO_2 Production and Alveolar Minute Ventilation Over Time

As mentioned, the Philips Respionics NM3 monitor can trend data over time. The display for this purpose reports the CO_2 produced each minute ($\dot{V}CO_2$) rather than $SBCO_2$ curves (Fig. 10.15). Trended data can be used to monitor a variety of clinical procedures. For example, during a recruitment maneuver in a patient with acute respiratory distress syndrome, trended CO_2 data will reveal a transient rise in CO_2 when previously closed alveoli are reopened. This tool can also be used in weaning patients from the ventilator. For example, if the patient's respiratory rate increases during a spontaneous breathing trial, monitoring CO_2 can help determine whether the patient's metabolic rate is increasing (and thus working the respiratory muscles) or whether a change in dead space affects the patient's ability to wean.

Trending of CO_2 can be useful for noting the time lag that can occur in CO_2 removal as a result of CO_2 stores (CO_2 bound in the cells or through bicarbonate or bound in the blood). Large stores result in extended time lags, and small stores result in short time lags. For example, if the alveolar ventilation increases, P_aCO_2 decreases and the stores of CO_2 also begin to decline; however, this second part requires time to happen and can be identified by the trending of CO_2. In this situation, if $\dot{V}_E$ increases, $SBCO_2$ increases and P_aCO_2 initially declines. After a slight delay, because the production of CO_2 remains constant, the P_aCO_2 remains low and the monitored exhaled $\dot{V}CO_2$ returns to baseline, indicating that balance has returned. Plotting $SBCO_2$ curve trends over time using a trending graph is the best way to monitor these types of changes.

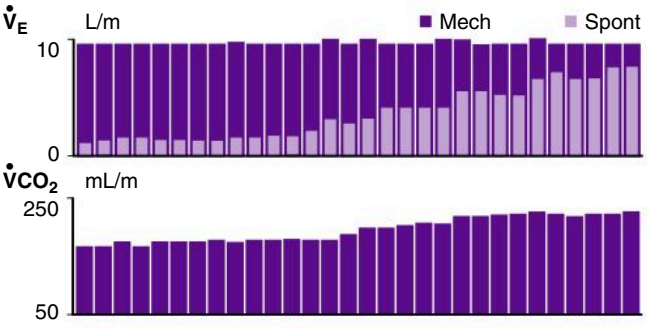


Fig. 10.15 An example of a $\dot{V}CO_2$ trended over time *(bottom bar graph)* compared with corresponding $\dot{V}_E$ *(top bar graph;* L/min). During the successful weaning trial illustrated in these graphs, mandatory breaths are reduced *(gray area, top graph)* and the patient's spontaneous breath rate increases *(dark bars in , top graph)*. At the same time, a progressive rise occurs in $\dot{V}CO_2$ as the expected work of the respiratory muscles increases. (Courtesy Philips North America Corporation, Andover, MA.)

EXHALED NITRIC OXIDE MONITORING

Nitric oxide (NO) has potent dilatory effects on the pulmonary vessels and airways. It has also been shown to facilitate coordinated beating of ciliated epithelial cells. The synthesis of NO by the body is mediated through a series of enzymes that are referred to as NO synthases (NOS), which exist in constitutive and inducible forms. The constitutive form is associated with endothelial and neural cells; the inducible NOS is particularly seen in epithelial cells. Although both forms of NOS are present in the airways, the expression of the inducible NOS appears to correlate with the level of NO found in exhaled air.

The most common method used to quantify the level of exhaled NO is chemiluminescence, which occurs when NO reacts with ozone. Exhaled NO (eNO) can be detected in exhaled gas in the range of 7.8 to 41.1 parts per billion (ppb). Note that the concentration of NO can vary with the flow of exhaled air because it is continually formed in the airways. The range of eNO is also affected by the presence of pathological conditions in addition to the patient's gender, atopic status, smoking habits, and use of medications.[33] Box 10.3 provides a list of factors that have been shown to affect the levels of eNO.

Exhaled NO is currently used as a marker for airway inflammation associated with asthma.[31] Several studies have shown a

BOX 10.3 Factors Affecting Levels of Exhaled Nitric Oxide (NO)

Conditions Associated With Reductions of Exhaled NO

- Systemic hypertension
- Pulmonary hypertension
- Cystic fibrosis
- Sickle cell anemia
- Ciliary dyskinesis

Conditions Associated With Elevated Levels of Exhaled NO

- Asthma
- Bronchiectasis
- Airway viral infections
- Alveolitis
- Allergic rhinitis
- Pulmonary sarcoidosis
- Chronic bronchitis
- Systemic sclerosis
- Pneumonia

Courtesy Aerocrine, Solna, Sweden.

positive correlation between the eNO and disease severity.[32,33] Monitoring the level of eNO can also be used to monitor the effectiveness of inhaled corticosteroid in the treatment of asthmatic patients.

TRANSCUTANEOUS MONITORING

Transcutaneous monitoring is another noninvasive method that can be used to indirectly assess a patient's oxygenation (P_aO_2) and ventilation (P_aCO_2) status. Unlike pulse oximetry and capnography, which rely on spectrophotometric analysis, transcutaneous monitoring uses modified blood gas electrodes to measure the O_2 and CO_2 tensions at the skin surface (Box 10.4).[34,35]

Transcutaneous PO_2

Devices used to monitor the transcutaneous partial pressure of O_2 ($P_{tc}O_2$) consist of a servo-controlled, heated (Clark) polarographic electrode connected to a central processing unit (Fig. 10.16A).[36-38] The electrode housing, which is covered with a Teflon membrane, attaches to the skin surface with a double-sided adhesive ring. The electrode is heated to 42° to 45° C to produce capillary vasodilation below the surface of the electrode. Note that heating improves diffusion of gases across the skin because it increases local blood

BOX 10.4 Summary of American Association for Respiratory Care Clinical Practice Guideline for Transcutaneous Blood Gas Monitoring

Setting

1. Monitoring mechanically ventilated patients (e.g., conventional modes of ventilation, high-frequency ventilation, noninvasive ventilation)
2. Bronchoscopies and procedures requiring sedation or patient-controlled analgesia
3. Sleep studies
4. Pulmonary function testing (e.g., stress testing, bronchoprovocation)
5. Trending HCO_3^- in patients with diabetic ketoacidosis
6. Apnea testing
7. Patient transport
8. Evaluation of tissue perfusion
9. Evaluation of hyperventilation during phonation of patients with vocal cord disorders
10. Titrating long-term O_2 therapy

Indications

1. Monitoring the adequacy of arterial oxygenation and/or ventilation.
2. The need to quantify the response to diagnostic and therapeutic interventions (e.g., administering enriched O_2 mixtures, application of PEEP).
3. Transcutaneous O_2 index ($P_{tc}O_2/F_IO_2$) can be used as a marker of hypoperfusion and mortality.
4. Tissue perfusion status and revascularization in wound care (e.g., during hyperbaric O_2 therapy) and peripheral vascular disease.
5. Monitoring response to therapy in patients with diabetic ketoacidosis, as $P_{tc}CO_2$ correlates with serum HCO_3^- levels.

Contraindications and Complications

Transcutaneous monitoring may be relatively contraindicated in patients with poor skin integrity and/or adhesive allergy. Complications may include thermal injury at the sensor site resulting in erythema, blisters, burns, and skin tears. Misinterpretation of data may lead to inappropriate treatment of a patient.

Monitoring

The following should be recorded when monitoring transcutaneous measurements:

1. Clinical appearance of the patient, including subjective assessment of perfusion, pallor, and skin temperature
2. Date and time of the measurement
3. Patient position
4. Respiratory rate
5. Physical activity level
6. F_IO_2 and the type of O_2 delivery device if supplemental O_2 is being administered
7. Mode of ventilator support (i.e., ventilator or CPAP settings)
8. Electrode placement site, electrode temperature, and time of placement
9. Results of simultaneous measurements of P_aO_2, P_aCO_2, and pH

Limitations

Technical and clinical factors may affect the reliability of transcutaneous readings and therefore limit the application of transcutaneous monitoring. Improper calibration, trapped air bubbles, and damaged membranes can affect the accuracy of the measurements of $P_{tc}O_2$ and $P_{tc}CO_2$. The presence of hyperoxemia (P_aO_2 >100 mm Hg) or a hypoperfused state (e.g., shock) can increase the difference between $P_{tc}O_2$ and P_aO_2.

From AARC Clinical Practice Guideline: Transcutaneous monitoring of carbon dioxide and oxygen: 2012, *Respir Care* 57(11):1955–1962, 2012.

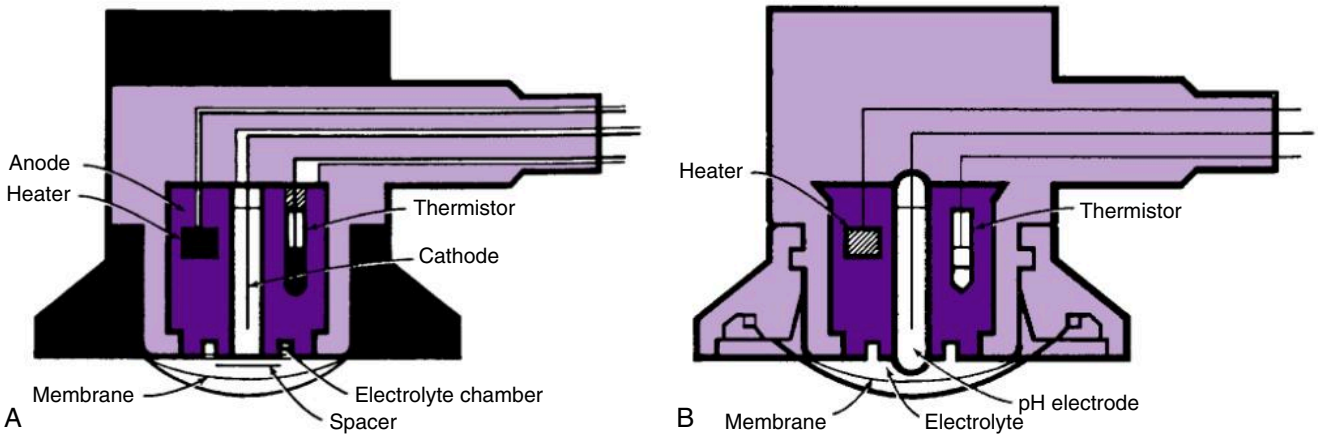


Fig. 10.16 Transcutaneous electrodes. (A) Transcutaneous partial pressure of oxygen ($P_{tc}O_2$). (B) Transcutaneous partial pressure of carbon dioxide ($P_{tc}CO_2$). (From Deshpande VM, Pilbeam SP, Dixon RJ: *A comprehensive review in respiratory care,* East Warwick, CT, 1988, Appleton & Lange.)

flow at the site of the electrode and alters the structure of the stratum corneum. The stratum corneum has been described as a mixture of fibrinous tissue within a lipid and protein matrix. It has been suggested that heating the skin to temperatures greater than 41° C melts the lipid layer, thus enhancing gas diffusion through the skin.

Although correlation of $P_{tc}O_2$ and P_aO_2 ($P_{tc}O_2/P_aO_2$ index) has been shown to be good for neonates, it is often unreliable for critically ill adult patients.[39] A decrease in peripheral perfusion caused by reductions in cardiac output or increases in peripheral (cutaneous) resistance can significantly affect the accuracy of $P_{tc}O_2$ measurements.[40,41] Data indicate that when the cardiac index is greater than 2.2 L/min/m^2, the $P_{tc}O_2/P_aO_2$ index is 0.5, whereas for a cardiac index less than 1.5 L/min/m^2 it is only 0.1.[42] Therefore hypoperfusion of the cutaneous circulation caused by pathological states (e.g., septic shock, hemorrhage, or heart failure) or increased vascular resistance (e.g., hypothermia or pharmacological intervention) can produce erroneous data. Because the $P_{tc}O_2$ is influenced by blood flow to the tissues and O_2 utilization by the tissues, changes in this value may be used as an early indicator of vascular compromise or shock.

Transcutaneous PCO_2

Measurement in the transcutaneous CO_2 partial pressure ($P_{tc}CO_2$) was introduced into clinical practice in the late 1970s after the successful use of $P_{tc}O_2$ monitors in neonatal ICU patients was demonstrated. Standard devices use a modified Stow-Severinghaus blood gas electrode, which is composed of pH-sensitive glass with an Ag/AgCl electrode (see Fig. 10.16B). As with the $P_{tc}O_2$ electrode, the $P_{tc}CO_2$ electrode is heated to 42° to 45° C. $P_{tc}CO_2$ values are slightly higher than those of P_aCO_2, primarily because of the higher metabolic rate at the site of the electrode caused by heating the skin. Most commercial instruments incorporate correction factors into their system's software to remove this discrepancy between $P_{tc}CO_2$ and P_aCO_2.

Technical Considerations

Some simple rules apply when using transcutaneous monitors. These relate to care, placement, and calibration of the electrodes. Establishing a set routine for these three tasks will help ensure accurate and useful measurements[41]:

1. The transcutaneous monitor manufacturer should have validated the transcutaneous monitor, electrodes, calibration gases, and supplies, using accepted quality control procedures and clinical reliability studies.
2. Transcutaneous electrodes are bathed by an electrolyte solution (see Fig. 10.16). This solution can easily evaporate because of the heat applied to the electrode. The electrolyte and the sensor's membrane should be changed weekly or whenever the respiratory care practitioner notices a signal drift during calibration. Because silver can become deposited on the cathode, periodic cleaning of the electrode is suggested, using the manufacturer's recommendations.
3. Before placing an electrode on the patient's skin, the site should be cleansed using an alcohol swab. In cases in which hair may be present, the site should be shaved to ensure good contact between the electrode and skin. Before applying the electrode to the skin, a drop of electrolyte gel or deionized water should be placed on the electrode's surface to enhance gas diffusion between the skin and electrode.
4. $P_{tc}O_2$ monitors are calibrated using a two-point calibration in which room air (PO_2 of about 150 mm Hg) serves as the high PO_2 of the calibration and an electronic zeroing of the system serves as the low PO_2 of the calibration.
5. $P_{tc}CO_2$ monitors are also calibrated with a two-point calibration procedure. In this calibration, a 5% CO_2 calibration gas and 10% CO_2 calibration gas are used for low and high calibration points, respectively. Electrodes should be calibrated before their initial use on a patient. Manufacturers typically suggest that the electrode should be recalibrated each time it is repositioned.
6. Reports of $P_{tc}O_2$ and $P_{tc}CO_2$ readings should include notation of the date and time of the measurement, the patient's activity level and body position, and the site of electrode placement, along with the electrode temperature. The inspired O_2 concentration and the type of equipment used to deliver supplemental O_2 should always be included. The clinical appearance of the patient, including assessment of peripheral

perfusion (i.e., pallor, skin temperature), are important data to note. In cases in which invasive ABG measurements are available, these data are recorded for comparison with $P_{tc}O_2$ and $P_{tc}CO_2$ readings.

Burns are probably the most common problem that clinicians encounter during transcutaneous monitoring. Burns can occur because the site of measurement must be heated to 42° to 45° C. Repositioning the sensor every 4 to 6 hours can help avoid this problem.[42] When transcutaneous monitoring is used with neonates, the sensor should be repositioned more often (e.g., every 2 hours).

A problem can occur with $P_{tc}O_2$ and $P_{tc}CO_2$ readings if the electrode is applied improperly. A leak-proof seal must be maintained at the skin surface for the readings to be meaningful. A leak allows room air to contact the sensor and results in higher than actual $P_{tc}O_2$ and lower than actual $P_{tc}CO_2$ readings even though the patient's clinical condition has not changed.

When combined O_2/CO_2 electrodes are used, hydroxyl (OH^-) ions produced at the PO_2 cathode may interfere with $P_{tc}CO_2$ readings. This problem has been reduced by stoichiometric consumption of OH^- by an anodized anode.[24]

BOX 10.5 Variations in Respiratory Quotient (RQ)

Substrate	RQ
Carbohydrate oxidation	1.0
Fat oxidation	0.7
Protein oxidation	0.8
Lipogenesis	>1.0

INDIRECT CALORIMETRY AND METABOLIC MEASUREMENTS

Overview of Indirect Calorimetry

Indirect calorimetry allows the clinician to estimate energy expenditure (EE) from measurements of O_2 consumption ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$).[43] This technique is based on the theory that all the energy a person uses is derived from the oxidation of carbohydrates, fats, and proteins and that the ratio of CO_2 produced to O_2 consumed (i.e., the respiratory quotient or $\dot{V}CO_2/\dot{V}O_2$) is characteristic for the fuel being burned (Box 10.5).[44] Although the use of metabolic measurements varies considerably, many clinicians are becoming comfortable with this emerging technology and recognize that metabolic measurements can provide valuable information for designing nutritional support regimens.

Technical Considerations

The most commonly used devices for indirect calorimetry are open-circuit gas exchange monitors (Fig. 10.17). They are often referred to as *metabolic monitors* or *metabolic carts*. A typical **metabolic monitor** includes analyzers for measuring the concentration of inspired and expired gases in addition to a sensor for measuring the volume and/or flow of respired gases. The O_2 analyzer is a rapid-responding polarographic or zirconium oxide O_2 analyzer. The CO_2 analyzer is a nondispersive, infrared analyzer. Volume and flow measurements can be obtained using pneumotachometers, turbine flow meters, or ultrasonic vortex flow meters. Barometric pressure and expired gas temperatures are monitored with temperature-sensitive, solid-state (integrated circuit) transducers.

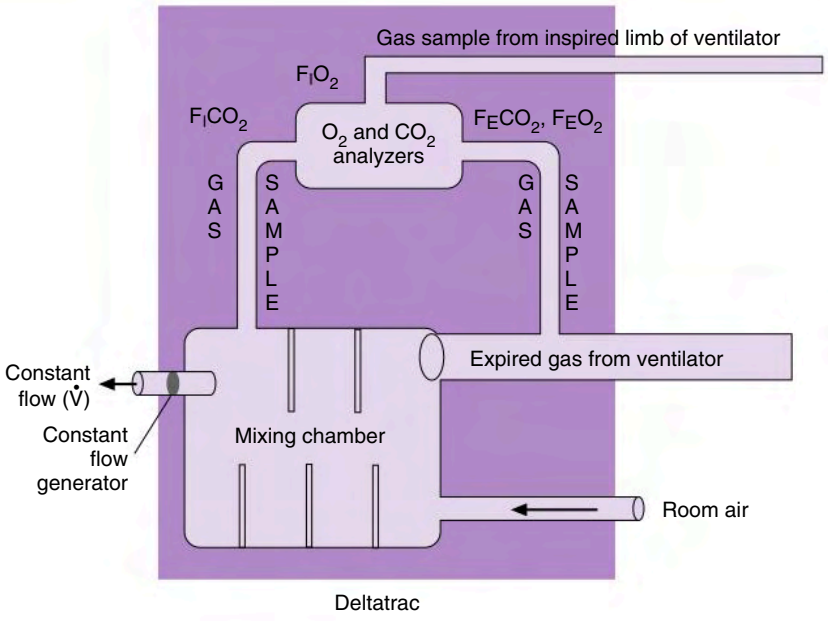


Fig. 10.17 Major components of a metabolic monitoring system. (From Weissman CM, Sadar A, Kemper MA: In vivo evaluation of a compact metabolic measurement instrument, *J Parenter Enteral Nutr* 14:216—221, 1990. Redrawn from Levine RL, Fromm RE: *Critical care monitoring,* St. Louis, MO, 1995, Mosby.)

Obtaining Indirect Calorimetry Measurements. A spontaneously breathing patient who is breathing room air can be connected to the system by having the patient breathe through a mouthpiece or mask that is attached to a nonrebreathing valve. Specially designed canopies and hoods can be used for spontaneously breathing patients who are not receiving ventilatory support.

Patients with ETs or tracheostomy tubes who are on mechanical ventilation can be connected to the system by placing the nonrebreathing valve directly onto the airway opening and directing the expired gases into the system. It is important to inflate the cuffs of ETs and tracheostomy tubes when measuring inspired and expired gases. Failure to inflate the cuff will result in loss of expired air around the tube (system leak) and erroneous measurements of $\dot{V}O_2$ and $\dot{V}CO_2$. For patients receiving a continuous flow of gas during ventilatory support, such as occurs when an external flow from a flow meter is used to power a small-volume nebulizer inline, an isolation valve must be used to ensure that only the patient's exhaled gases are delivered to the system.[45-47]

$\dot{V}O_2$ and $\dot{V}CO_2$ are calculated by comparing the fractional concentrations of O_2 and CO_2 of inspired and expired air. For patients breathing room air, the F_IO_2 can be assumed to be 0.209 and the F_ICO_2 to be 0.03. For patients receiving enriched O_2 mixtures, the F_IO_2 must be measured by the system.[46,47] Fluctuations in F_IO_2 can be caused by air leaks in the patient-ventilator-metabolic monitor system and also varying gas volumes and pressure demands, such as occur during intermittent mandatory ventilation. Unstable air-O_2 blending systems within the ventilator circuit may contribute to unstable F_IO_2 values (this problem can be prevented with the use of an external air-O_2 blender). Clinical studies have shown that some systems may not provide accurate and reproducible $\dot{V}O_2$ measurements for patient breathing F_IO_2 values greater than 0.5.[46] Box 10.6 provides a summary of the AARC Clinical Practice Guideline for using indirect calorimetry during mechanical ventilation.

Clinical Applications of Metabolic Measurements. Indirect calorimetry can provide information on EE and the pattern of substrate utilization. EE represents an individual's caloric expenditure calculated from measured $\dot{V}O_2$ and $\dot{V}CO_2$ values. EE can be calculated with the deWeir equation shown in Box 10.7. Note that urinary nitrogen (UN) is determined separately by the clinical laboratory using a 24-hour urine sample. The UN is one of the end products of protein metabolism; therefore the number of grams of nitrogen excreted in the urine is directly related to the amount of protein used by the individual. If nitrogen excretion data are not available, EE can be calculated using the modified deWeir equation. In this latter equation, it is assumed that protein represents 12% to 15% of the total EE.

EE can be expressed in kilocalories per day (kcal/day) or relative to the individual's body surface area (kcal/h/m^2). A normal, healthy adult uses about 1500 to 3000 kcal/day or about 30 to 40 kcal/h/m^2.[48]

Another method used to express the level of energy metabolism is to compare the measured EE with predicted EE, which is based on the individual's age, weight, and height. (The Harris-Benedict equations shown in Box 10.7 are examples of reference equations that have historically been used in clinical practice to estimate EE.) If the measured EE is greater than 120% of the predicted EE, a hypermetabolic state exists. Conversely, when the measured EE is less than 80% of the predicted EE, a hypometabolic state exists.

BOX 10.6 Summary of American Association for Respiratory Care Clinical Practice Guideline for Metabolic Measurement Using Indirect Calorimetry During Mechanical Ventilation

Indications

1. Metabolic measurements may be indicated in patients with known nutritional deficits and derangements. Multiple nutritional risk and stress factors that may skew predictions made using the Harris-Benedict equation (e.g., neurological trauma, chronic obstructive pulmonary disease, acute pancreatitis, multiple trauma, severe sepsis, extreme obesity, severe hypermetabolic or hypometabolic patients).
2. To measure O_2 cost of breathing in patients who fail attempts at liberation from mechanical ventilation.
3. To measure $\dot{V}O_2$ and cardiac output by the Fick equation for patients requiring hemodynamic monitoring.

Contraindications and Complications

1. Manipulation of the ventilator circuit for connection of measurement lines may cause leaks that may lower alveolar ventilation and result in hypoxemia, bradycardia, or other adverse connections.
2. Inappropriate calibration or system setup may result in erroneous results leading to incorrect patient management.
3. Isolation valves may increase circuit resistance and cause increased work of breathing and/or dynamic hyperinflation (auto-PEEP).
4. Inspiratory reserves may cause a reduction in alveolar ventilation because of increased compressible volume of the breathing circuit.

Inaccurate measurements of resting energy expenditure and RQ during open circuit measurements may be caused by:

1. Instability of F_IO_2 within a breath or breath to breath because of changes in the source gas pressure and ventilator blender characteristics
2. F_IO_2 >0.60
3. Inability to separate inspired and expired gases because of bias flow and flow-triggering systems, intermittent mandatory ventilation systems, or specific ventilator characteristics
4. Presence of anesthetic gases or gases other than O_2, CO_2, and nitrogen in the patient-ventilator circuit
5. Presence of water vapor resulting in sensor malfunction
6. Inadequate length of the measurement

Assessment of Test Quality and Outcome

1. RQ is consistent with the patient's nutritional intake.
2. RQ at rest is in the normal physiological range (0.67—1.3).
3. Variability of $\dot{V}O_2$ and $\dot{V}CO_2$ measurements should be within a physiological range (0.7—1.0).

From AARC Clinical Practice Guideline: Metabolic measurement using indirect calorimetry during mechanical ventilation—2004 revision and update, *Respir Care* 49(9):1073—1079, 2004.

Numerous factors can influence the metabolic rate, including the type and rate of nutrition that is ingested; the time of day the measurement is made; the patient's level of physical activity; and whether he or she is recovering from infection, surgery, or trauma.[49] The presence of chronic gastrointestinal, hepatic, renal,

BOX 10.7 Formulas Used During Indirect Calorimetry

Harris-Benedict Equations*

Men: Energy expenditure (EE) = 66.5 + (13.75 × weight) + (5.003 × height) − (6.775 × age)

Women: EE = 655.1 + (9.563 × weight) + (1.85 × height) − (4.676 × age)

where weight is measured in pounds, height is measured in inches, and age is determined in years.

Energy Expenditure‡†

deWeir equation: EE = [3.941 ($\dot{V}O_2$) + 1.106 ($\dot{V}CO_2$)] × 1.44 − [2.17 UN]

Modified deWeir equations: EE = [3.9 ($\dot{V}O_2$) + 1.1 ($\dot{V}CO_2$)] × 1.44

Substrate Utilization‡

Carbohydrates: dS = 4.115 $\dot{V}CO_2$ − 2.909 $\dot{V}O_2$ − 2.539 UN

Fats: dF = 1.689 ($\dot{V}O_2$ − $\dot{V}CO_2$) − 1.943 UN

Proteins: dP = 6.25 UN

dS, dF, and *dP* represent grams of carbohydrate, fat, and protein, respectively, for a fasting individual.

*Harris JA, Benedict FG, editors: *Standard basal metabolism constants for physiologists and clinicians: a biometric study of basal metabolism in man,* Philadelphia, PA, 1991, Lippincott Williams & Wilkins.

‡Burszein S, Saphar S, Singer P, et al.: A mathematical analysis of indirect calorimetry measurement in acutely ill patients, *Am J Clin Nutr* 50:227–230, 1980.

†Weir JB: A new method for calculating metabolic rate with special reference to protein metabolism, *J Physiol* 109:1–9, 1949.

endocrine, cardiovascular, and pulmonary diseases can also influence the metabolic rate.[50] Box 10.8 lists several conditions that are associated with hypermetabolic and hypometabolic states.

Prolonged starvation is associated with a decreased metabolic rate. Feeding raises metabolic rate through a mechanism referred to as *specific dynamic action*. It is thought that specific dynamic action is related to the digestion and absorption of food. EE can show diurnal variation; it is usually lowest on awakening in the morning and increases 10% to 15% by late afternoon.[50,51] This increase in EE may be related to hormonal changes that occur during the day.

Sleep is associated with a reduction in metabolic rate, whereas even the slightest exertion is associated with increases in metabolic rate. Changes in body temperature, as occur with bacterial and viral infections, can profoundly affect the metabolic rate. For example, an increase in body temperature of 1° C will cause a 10% increase in metabolic rate. Burns, long-bone fractures, and surgery can increase the metabolic rate by as much as 200%.[50]

The substrate utilization pattern is the proportion of carbohydrates, fats, and proteins that contribute to the total energy metabolism. The percentage of the total energy that a substrate contributes can be derived from measurements of the RQ (the ratio of $\dot{V}CO_2$ to $\dot{V}O_2$). The RQ levels for the various foods are known; when pure fat is burned, the RQ equals 0.7. The RQ for pure carbohydrate is 1.0, and the RQ for protein is approximately 0.8. RQ levels greater than 1.0 are associated with lipogenesis (fat synthesis) and hyperventilation. RQ levels less than 0.7 are associated with ketosis.

A healthy adult consuming a typical American diet derives 45% to 50% of his or her calories from carbohydrates, 35% to 40% from lipids, and 10% to 15% from proteins. The resultant RQ will range from 0.80 to 0.85. Note that proteins normally contribute only minor amounts to energy metabolism. The percentage of protein used represents the normal turnover rate for replenishing structural and functional proteins in the body. Proteins may contribute significantly to EE in cases of starvation. For this reason, a nonprotein RQ is usually reported to indicate the contribution to RQ made by carbohydrates and lipids.

The types of substrates ingested and the ability of the individual to use various foods determine substrate utilization. For example, feeding large amounts of glucose will raise the RQ to about 1.0, suggesting that carbohydrates are providing most of the EE. Prolonged starvation will lower the RQ to about 0.7, indicating that the individual is relying almost completely on fats for energy. Many systemic diseases will adversely affect an individual's ability to use various substrates. For example, several studies have shown that patients with severe sepsis demonstrate RQ levels of approximately 0.7 because of reliance on lipid metabolism for energy and their inability to use carbohydrates.

Monitoring of substrate utilization patterns can assist the clinician who is trying to wean patients with limited ventilatory reserve from mechanical ventilation (Key Point 10.3). It has been demonstrated that feeding these patients diets containing a high percentage of carbohydrates will raise their $\dot{V}CO_2$ to a greater extent than their $\dot{V}O_2$ (RQ approaches 1.0).[52,53] The added CO_2 load placed on these patients (remember they have limited ventilatory reserve) is greater than their own ventilatory capacity, and they fail to wean. Switching their diet to one that has a higher fat-to-carbohydrate ratio lowers their $\dot{V}CO_2/\dot{V}O_2$ ratio (RQ levels approach 0.7) and reduces the CO_2 load to the lungs. It is reasonable to suggest that this change would enhance the potential for a successful weaning outcome. See Critical Care Concept 10.1

BOX 10.8 Examples of Hypermetabolic and Hypometabolic States

Hypermetabolic States

- Pancreatitis
- Hyperthyroidism
- Pregnancy
- Drugs (e.g., stimulants)
- Hyperthermia (fever)
- Seizures
- Burns

Hypometabolic States

- Starvation
- Hypothyroidism
- Anesthesia
- Sedation
- Hypothermia
- Coma

***Key Point* 10.3** The types of substrates ingested and the ability of an individual to use various foods influence substrate utilization.

Critical care Concept 10.1

Indirect Calorimetry

Prediction equations used by clinicians to determine energy needs for hospitalized patients are derived from studies of healthy subjects. Clinicians generally agree that using standard prediction equations to estimate energy needs for critically ill patients provides values that compare poorly with measured values, such as those reported from indirect calorimetry. The energy needs of these patients tend to be quite diverse and can lead to overnutrition or undernutrition. Although stress factors can be added to the calculation of predicted caloric intake, these factors may be misleading, particularly in those patients demonstrating multisystem problems.

for a discussion of the advantages of using indirect calorimetry in the management of critically ill patients.

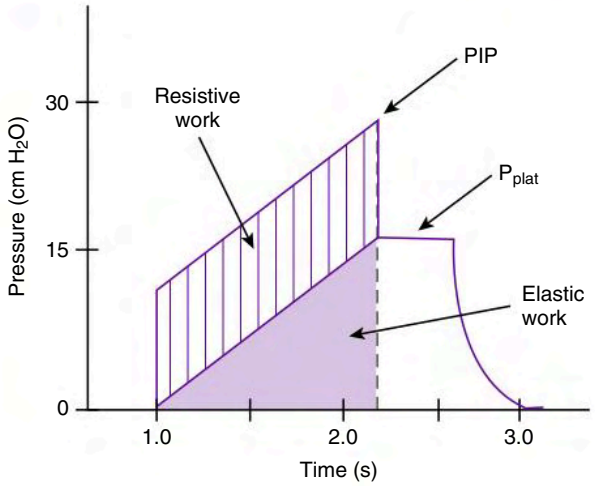


Fig. 10.18 Airway pressure tracing for a patient on mechanical ventilation. *PIP,* Peak inspiratory pressure; P_{plat}, plateau pressure.

ASSESSMENT OF RESPIRATORY SYSTEM MECHANICS

The assessment of respiratory system mechanics for patients receiving ventilatory support begins with measurements of pressure, volume, and flow events. Once these measurements have been made, the clinician can calculate derived values for respiratory system compliance, airway resistance, and the WOB.[54] Chapter 1 discusses the physiological concepts required for an understanding of respiratory mechanics in mechanically ventilated patients. The following is a brief description of the devices and techniques that are used to measure airway pressures, volumes, and flows.

MEASUREMENTS

Airway Pressure Measurements

Mechanical ventilators have traditionally allowed for measurements of airway pressures by incorporating an aneroid manometer into the ventilator circuit. With this arrangement, the manometer records pressure changes within the ventilator, which includes contributions from ventilator resistance. Measuring airway pressure near the airway opening can minimize the effects associated with ventilator resistance. Thus in the current generation of adult and neonatal ventilators, airway pressure is measured using electromechanical transducers (e.g., piezoelectric, variable capacitance, strain gauge) that connect to pressure sampling ports located near the airway opening (i.e., measurements can be made on the inspiratory limb of the ventilator circuit, the expiratory side of the circuit, or directly at the ET).

An alternative method of recording airway pressures during mechanical ventilation is to use a strain gauge pressure transducer that is normally used for measuring systemic and pulmonary arterial pressures. These transducers can be adapted for respiratory pressure measurements because respiratory pressures are similar in magnitude to those found in the systemic or pulmonary arterial vasculature.[24,55] There are several points to remember when using these types of pressure transducers. Hemodynamic pressures are recorded in millimeters of mercury (mm Hg), and respiratory pressures are recorded in centimeters of water (cm H_2O); to convert from millimeters of mercury to centimeters of H_2O, the mm Hg value is multiplied by 1.36. Second, the transducer need not be filled with fluid for making airway pressure measurements. Third, the same transducer should not be used to obtain both airway pressure and hemodynamic measurements, to avoid the possibility of introducing an air embolus into the circulation.

The most common airway pressure measurements are peak inspiratory pressure (PIP) and static, or plateau, pressure (P_{plat}) (Fig. 10.18). As discussed in Chapter 1, PIP is the maximum pressure generated during inspiration. During volume-targeted ventilation, PIP is determined by the tidal volume (V_T), peak flow, and inspiratory flow. It is also influenced by the resistance and compliance of the patient's lungs and chest wall and by ET resistance and the compliance of the ventilator circuit. During pressure-targeted ventilation, the target pressure set on the ventilator determines the PIP. Patient-triggering efforts can increase or decrease PIP, depending on the patient-ventilator synchrony.[55]

P_{plat} is the amount of pressure required to maintain the V_T within the patient's lungs during a period of no gas flow. Therefore P_{plat} reflects that alveolar pressure (P_{alv}), and it is ultimately influenced by the V_T, lung and thorax compliance, circuit elastance, and total measured PEEP [including applied and intrinsic (auto-PEEP)] (Key Point 10.4).

P_{plat} can be measured by occluding the expiratory valve at the end of a tidal inspiration. Many current ICU ventilators have an inspiratory pause control incorporated into the ventilator system that closes the inspiratory and expiratory valves at the end of inspiration so that P_{plat} measurement can be obtained. As illustrated in Fig. 10.18, P_{plat} measurements require the establishment

***Key Point* 10.4** P_{plat} reflects alveolar pressure, and it is ultimately influenced by tidal volume, lung and thorax compliance, circuit elastance, and total measured PEEP.

of a period of zero flow for 1 to 2 seconds to allow pressure equilibration to occur across the airway. This delay in pressure equilibration and establishment of the P_{plat} is the result of redistribution of the V_T and stress relaxation. True P_{plat} measurements can be obtained only during a passive inspiration, and failure to establish a stable P_{plat} can result from patient breathing activity or a leak in the ventilator circuit.[55,56]

Flow Measurements

Gas flow during mechanical ventilation can be monitored with a number of different types of flow meters, including vortex ultrasonic flow meters, variable orifice pneumotachometers, thermal flow meters, and turbine flow meters. With these devices, volume changes can be calculated by integrating the flow signal relative to time.[57,58]

Vortex ultrasonic flow meters and variable orifice pneumotachometers use resistive elements to create a pressure drop that is proportional to the flow of gas through them.[57] Vortex ultrasonic flow meters use struts to create a partial obstruction to gas flow. As gases flow past these struts, whirlpools or vortices are produced. The frequency at which these whirlpools are produced is related to the flow of gas through the struts. These devices are not affected by the viscosity, density, or temperature of the gas being measured. They are unidirectional devices and therefore cannot be used to measure inspiratory and expiratory flow simultaneously.[57] The Servo-i (Getinge, Göteborg, Sweden) uses ultrasonic transducer technology to measure expiratory gas flow. Rather than struts, two ultrasonic transducers that alternate function are used. One acts as the transmitting device and the other as the receiver (Fig. 10.19). The ultrasonic waves detect the expiratory gas flow characteristics as the patient's exhaled air moves through the expiratory cassette and provide a measure of exhaled V_Ts and flows.

Variable orifice pneumotachometers are disposable, bidirectional flow measuring devices that use a variable area, flexible obstruction for measuring flow as a function of the pressure differential generated by the obstruction. They contain minimal dead space (about 10 mL) and can measure flow from 0.02 to 3.0 L/s.[59] Although the flow-pressure characteristics of these devices are nonlinear, nonlinearity is typically compensated electronically (see Fig. 10.20). Variable orifice pneumotachometers are used in the Hamilton Medical ventilators (Hamilton Medical, Bonaduz, Switzerland) (Fig. 10.20).

Thermal flow meters ("hot wire" anemometers) use sensors that are temperature-sensitive, resistive elements (e.g., thermistor beads or heated wires). These devices operate on the principle that as gas passes over the thermistor bead or heated wire, the sensor

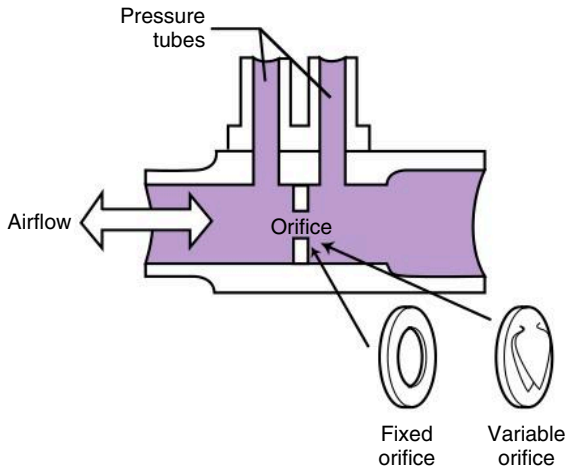


Fig. 10.20 A pneumotachograph illustrating a fixed orifice and a variable orifice (contains a moveable flap). (Redrawn from Sullivan WJ, Peters GM, Enright PL: Pneumotachographs: theory and clinical application, *Respir Care* 29:736—749, 1984.)

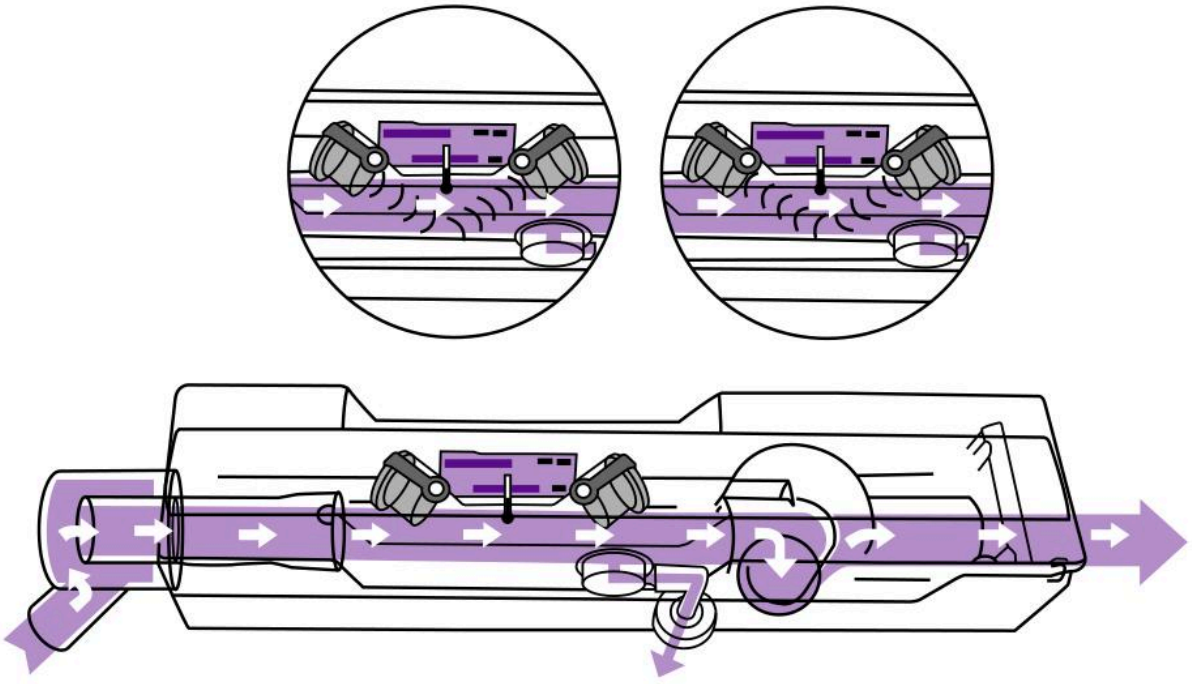


Fig. 10.19 Ultrasonic flow transducers within the Servo-i expiratory cassette. (Courtesy Getinge, Göteborg, Sweden.)

cools and its resistance changes in proportion to the gas flow. Note that the amount of cooling depends on the viscosity and thermal conductivity of the gas measured. With thermistor beads, cooling increases resistance, whereas with a heated platinum wire, cooling decreases resistance. The wire is typically heated above 37° C and protected by a low-resistance screen to prevent moisture accumulation and debris impaction on the wire. The gas flow can be calculated because the amount of power needed to maintain the temperature of the heating element above the temperature (e.g., 37° C) is related to the log of the velocity of gas flow and therefore must be linearized. Thermal flow meters are unidirectional devices and cannot be used for measuring bidirectional flows during breathing. It is important to recognize that the density and viscosity of the gas being measured can affect the accuracy and precision of the flow measurement. Correction factors for various gases can be applied through computer software.[4] Thermal anemometers are available with the Puritan Bennett 840 (Medtronics, Minneapolis, MN) and Dräger Oxylog 3000 (Dräger Medical Inc., Telford, PA) ventilators.

Turbine flow meters use a rotating vane that is placed in the path of gas flow. As gas flows through the device, the vane turns at a rate that depends on the flow rate of the gas. Gas flow therefore can be measured by counting the number of times the vane turns. This can be done mechanically by linking the vane to a needle attached to a calibrated display. The rate of gas flow can be measured electronically using a light beam that is interrupted each time the vane turns. Rotating vane devices are portable and easy to use; however, they are slow to respond to flow changes resulting from inertia and as such are inaccurate for measuring bidirectional flows. As discussed in Chapter 8 (Fig. 8.2), portable turbine flow meters can be used during patient-ventilator checks when a flow meter has not been incorporated into the ventilator's design.

Clinical Applications

Respiratory systems mechanics data can provide valuable information for the clinician caring for a patient on mechanical ventilation.[58] These data are generally divided into *measured* and *derived variables.* Measured variables include airway pressures, volumes, and airflow. Derived variables, which are calculated from measured values, include respiratory system compliance, airway resistance, and WOB. The following section is a brief description of how pathophysiological events and conditions can affect respiratory system mechanics.

Measured Variables

As mentioned previously, PIP reflects the total force that must be applied to overcome elastic and frictional forces offered by patient-ventilator systems, and P_{plat} represents that portion of the total pressure required to overcome only elastic forces. Increases in the elastance of the respiratory system (i.e., decreases in compliance of the lung and/or chest wall) will increase both peak and plateau pressures. Increases in respiratory system compliance will lower both PIP and P_{plat}. Increases in airway resistance increase PIP without concomitant increases in P_{plat}. Decreases in airway resistance (R_{aw}) will lower PIP but not affect P_{plat}. It is important to recognize that changes in inspiratory flow or V_T should not be made when monitoring R_{aw} by this method. Also, the addition of PEEP (i.e., applied and intrinsic [auto-PEEP]) will affect both PIP and P_{plat} and should be taken into account (see the discussion on R_{aw} later in this chapter) (Key Point 10.5).

Key Point 10.5 Addition of PEEP (applied and auto-PEEP) affects both PIP and P_{plat} and should be considered when assessing changes in R_{aw}.

Sudden increases in PIP should alert the clinician to a potential problem such as bronchospasm or mucus plugging. Other factors that should be checked when determining the cause of increased airway resistance include partially blocked heat-moisture exchanger (HME), incorrect ET size, water in ventilator tubing, and malfunctioning expiratory valves. Increases in PIP can be associated with barotrauma or ventilator-induced lung injury; if these conditions are suspected, P_{plat} should be monitored because alveolar overdistention and rupture are associated with high P_{alv}, which in turn results in a higher P_{plat}.

Airflow monitoring can alert the clinician to significant changes in the resistance or compliance (or both) of the patient's respiratory system. For example, high-frequency ripples on the inspiratory flow tracing can indicate turbulent flow caused by the presence of secretions in the airway or water in the ventilator circuit.[54,59] Expiratory flow limitations should be suspected if the decay in expiratory flow is linear rather than exponential (see Chapter 9). Flow measurements can be used to detect the presence of auto-PEEP, because flow will still be present at the end exhalation. This approach to detecting auto-PEEP is more of a qualitative assessment and does not provide an accurate measurement of the level of auto-PEEP (see Fig. 9.30).

Derived Variables

Mean airway pressure. The mean airway pressure (P_{aw}) represents the average pressure recorded during the respiratory cycle. It is influenced by peak inspiratory pressure, PEEP, inspiratory time (T_I), and total cycle time (TCT). It can be calculated using the following equation:

$$\bar{P}_{aw} = \frac{1}{2}\left[(PIP - PEEP) \times \left(\frac{T_I}{TCT}\right)\right] + PEEP$$

$\bar{P}_{aw}$ can also be obtained by integrating the area under the pressure-time curve. In most ICU ventilators, microprocessors incorporated into their electronic circuitry perform this calculation and provide a continuous display of P_{aw}. Oxygenation status can be significantly improved by increases in P_{aw}, and the application of PEEP has the greatest effect. However, excessive increases in P_{aw} can adversely affect cardiac performance and lead to significant reductions in cardiac output (see Chapter 11).

Dynamic and static compliances. Compliance can be simply defined as the lung volume achieved for a given amount of applied pressure.[60] Two types of compliance calculations can be used to describe this pressure-volume relationship: dynamic compliance and static compliance. Dynamic compliance considers the total impedance to volume changes (i.e., flow resistive and elastic characteristics of the patient-ventilator interface); static compliance is only influenced by the elastic characteristics of the lung-thorax unit.

Dynamic compliance is calculated by dividing the exhaled V_T by the PIP minus the PEEP, or

$$\text{Dynamic Compliance} = \text{Exhaled } V_T/(PIP - PEEP)$$

Static compliance is calculated by dividing the exhaled V_T by the P_{plat} minus the total PEEP

$$\text{Static Compliance} = \text{Exhaled VT}/(P_{plat} - \text{PEEP})$$

It is important to recognize that the effects of intrinsic PEEP can underestimate the total PEEP.

Static compliance in healthy adult subjects is approximately 100 mL/cm H_2O; it is lower in adult patients receiving positive pressure ventilation. It ranges from 40 to 50 mL/cm H_2O (men) and 35 to 45 mL/cm H_2O (women) to as high as 100 mL/cm H_2O in either sex. (Static compliance is approximately 40–50 mL/cm H_2O in pediatric patients and 10–20 mL/cm H_2O in neonates.) Pathophysiological conditions, such as pulmonary interstitial fibrosis, pleural effusion, hyperinflation, consolidation, respiratory distress syndrome, and pulmonary vascular engorgement, are associated with decreases in lung compliance. Conditions such as kyphoscoliosis and myasthenia gravis are also associated with increased chest wall elastic recoil and decreases in chest wall compliance.

Serial measurements of dynamic and static compliance provide considerably more information than single measurements can provide. For example, congestive heart failure will lead to pulmonary vascular engorgement and a reduction in both static and dynamic compliance. Diuretics therapy reduces the level of engorgement and improves static and dynamic compliance. Bronchospasm causes a decrease in dynamic compliance but does not always affect static compliance (static compliance may decrease if air trapping occurs). If bronchodilator therapy resolves the bronchospasm, dynamic compliance returns to normal.

Airway resistance

Airway resistance (R_{aw}) is the opposition to airflow from nonelastic forces of the lung. R_{aw} for the respiratory system in a ventilated patient is about 5 to 7 cm H_2O/L/s. As mentioned in Chapter 1, R_{aw} is calculated by dividing the difference between PIP and P_{plat} by the airflow (constant flow with volume ventilation), or

$$R_{aw} = \frac{(PIP - P_{plat})}{\dot{V}\left(\frac{L}{s}\right)}$$

Airway resistance is primarily determined by the diameter of the airway. A twofold decrease in airway diameter will result in a 16-fold increase in airway resistance (Poiseuille's law).† Retention of secretions, peribronchiolar edema, bronchoconstriction, or dynamic compression of the airways results in increased airway resistance. Bronchodilation results in reduced R_{aw}.

Work of breathing

The normal WOB is related to the energy required to take in a breath. WOB is normally associated with nonelastic forces from gas moving through the airway and inertial forces to move structures in the thorax. *Intrinsic* work is a result of work done to overcome these normal elastic and resistive forces and work to overcome a disorder or disease process affecting normal workloads in the lungs and thorax. For example, abnormal (increased) intrinsic work occurs in chronic bronchitis, in which the resistance to gas flow through the conductive airways increases. In fibrotic diseases of the lung, compliance is reduced and alveolar movement is restricted. This reduced movement impedes the ability of the lungs to expand, thus increasing intrinsic work.

Extrinsic work is work imposed (WOBi) by systems that are added to the patient. Common examples are the ET, trigger sensitivity, demand valve systems, the humidifying device, and the patient circuit.[61] Expiratory work is increased by the resistance offered by the exhalation valve or PEEP valve.

Work of breathing defined

In physics, work (W) is defined as the product of force (F) acting on a mass to move it through a distance (d), or $W = F \times d$. In fluid systems, such as the respiratory system, we say that work is performed when an applied force or pressure causes a volume to displace, as in inspiration and expiration. The WOB is the integral of the product of pressure and volume ($W = \int PV$). That is, work is the amount of pressure that must be generated to result in the movement of a certain volume of gas. Work is reported in kilogram-meters (kg · m) or joules (J; 0.1 kg · m = 1 J). In healthy individuals, the WOB is about 0.5 J/L, which represents only 2% to 5% of the total $\dot{V}O_2$ or 0.35 to 1.0 mL/L of ventilation. O_2 consumption by the respiratory muscles may be as high as 35% to 40% of total $\dot{V}O_2$ in patients with COPD. WOB is sometimes described by the amount of O_2 consumed by the working respiration muscles, although this is difficult to measure.[61,62] WOB can also be defined as the pressure-time product when intrapleural pressure is monitored.

Graphic representation of WOB

The WOB can now be monitored through the use of graphic displays and calculated data provided by newer microprocessor-controlled ventilators and special monitoring devices (esophageal pressure monitors). Note that the accuracy of the calculation still needs to be studied. The amount of work expended during a respiratory cycle can be estimated by multiplying the pressure changes associated with a given volume change, or $W = P \times V$. Alternatively, WOB can be calculated as $W = (PIP - 0.5 \times P_{plat})/100 \times V_T$ to estimate WOB during constant-flow passive inflation of the lungs.[63] A pressure-volume curve can be used to make this estimate.[63] Fig. 10.21 illustrates the pressure and volume changes that occur in a patient receiving controlled mechanical ventilation with a constant flow with the ventilator doing the resistive and elastic WOB.[64] Contrast this to Fig. 10.22, which shows WOB required during continuous positive airway pressure (CPAP). In this figure, WOB is the integral of airway pressure and V_T; the greater the area of the loop, the greater the WOB. Loop A is an example of a freestanding CPAP system. Spontaneous breaths occur clockwise—inspiration to expiration. Loop B is CPAP through a ventilator demand valve system and shows an increased WOBi. The area to the left of the vertical lines (baseline pressure of 5 cm H_2O) is the WOBi during inspiration. The area to the right of the line represents WOBi during expiration.

Fig. 10.23 shows the components of a spontaneous breath and a ventilator breath. It distinguishes those parts of the breath that the patient must do and those parts that the ventilator provides.[64]

Fig. 10.24 compares the components of a normal spontaneous breath with a spontaneous breath with high impedance to breathing (ET in place) and with a mandatory controlled breath.[65]

†Poiseuille's law describes the factors that affect laminar flow through flow through a smooth tube with a constant diameter. $\Delta P = V \times (8\eta 1)/\Pi r4$ where ΔP = driving pressure (dynes/cm^2), η = coefficient of viscosity of the gas, l = tube length (c 8 are constants). V = gas flow (mL/sec) radius of tube (cm) (π and 8 are constant).

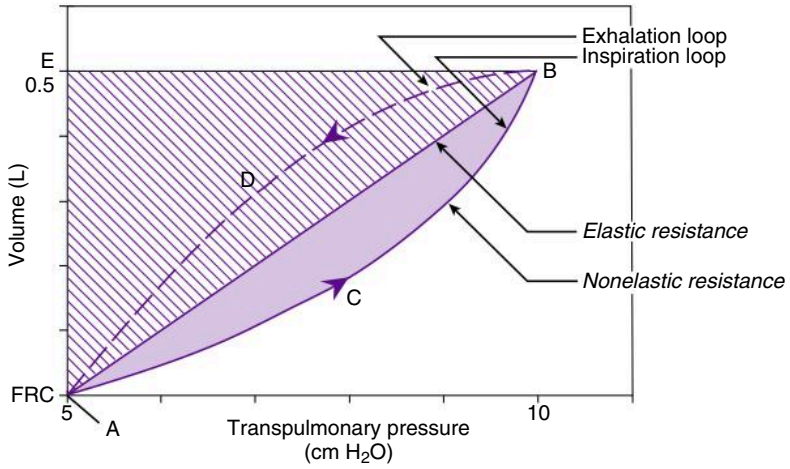


Fig. 10.21 Pressure/volume changes that occur in a patient receiving controlled ventilation with a constant-flow ventilator.

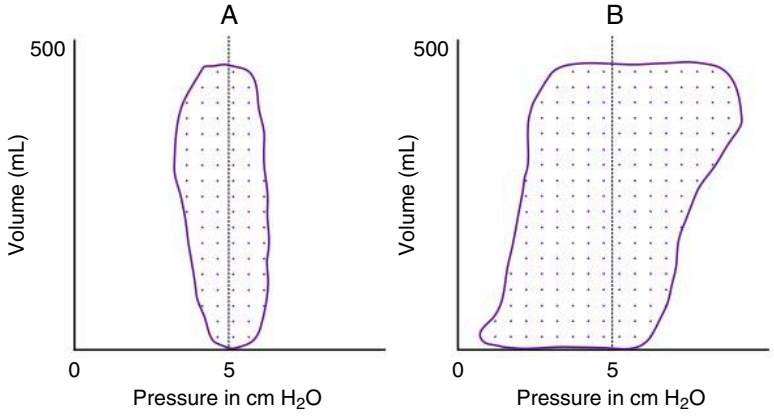


Fig. 10.22 Work of breathing (WOB) during continuous positive airway pressure (CPAP). WOB in this figure is the integral of airway pressure and tidal volume. *Loop A* is an example of a freestanding CPAP system. Spontaneous breaths occur clockwise—inspiration to expiration. *Loop B* is CPAP through a ventilator demand valve system. (From Hirsch C, Kacmarek RM, Stankek K: Work of breathing during CPAP and PSV imposed by the new generation mechanical ventilators: a lung model study, *Respir Care* 36:815—828, 1991; Kacmarek RM: The role of pressure support ventilation in reducing the work of breathing, *Respir Care* 33:99—120, 1988; and Kirby RR, Banner MJ, Downs JB: *Clinical applications of ventilatory support,* New York, 1990, Churchill-Livingstone.)

Curve A represents breathing through an ET by a patient with high impedance (increased R_{aw} or decreased compliance, or both). This occurs during T-tube trials for ventilator liberation or during spontaneous breathing through a continuous flow system. Curve B represents the work done by the ventilator during a volume-controlled breath. The ventilator is doing all the WOB. A spontaneous breath under normal conditions is represented by curve C.

Some have suggested that measuring the WOB in this way may underestimate the total amount of work that a patient expends during assisted ventilation.[66] Measurements of transdiaphragmatic pressures and pressure-time products may provide accurate estimates of the WOB and the metabolic cost of breathing in mechanically ventilated patients.[67]

Pressure-time product

Measurement of the maximum inspiratory pressure (MIP) and maximum expiratory pressure (MEP) provides nonspecific information about the strength of respiratory muscles. It is possible to obtain more specific information about the contributions of diaphragmatic contractions on breathing by measuring transdiaphragmatic pressure and the pressure-time product.[68,69] *Transdiaphragmatic pressure* is a measure of the forcefulness of diaphragmatic contractions. The *pressure-time product,* which is an assessment of transdiaphragmatic pressure during the inspiratory portion of the breathing cycle, is one method of estimating the contributions of the diaphragm during inspiration. It is probably a better indication of a patient's effort to breathe than measurement of work derived from pressure-volume curves.

Fig. 10.25A shows the positioning of the two balloon-tipped catheters used to measure transdiaphragmatic pressures and thus the pressure-time product. The catheters are inserted through the nose; one is positioned in the stomach (below the diaphragm), and the other is positioned in the lower third of the esophagus (above the diaphragm). Gastric (P_{GA}) and esophageal (P_{es}) pressures are

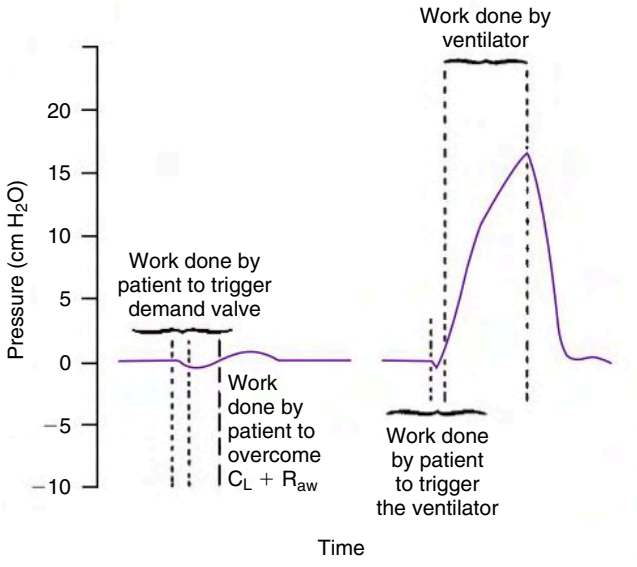


Fig. 10.23 Pressure requirements for a spontaneous *(left)* and an assisted breath *(right)*. The imposed work of breathing (WOBi) can occur during triggering of the breath. In the spontaneous breath, the patient performs work to overcome elastic and resistive forces, whereas in the assisted breath, the ventilator provides the work. C_L, Lung compliance. (From Branson RD: Enhanced capabilities of current ICU ventilators: do they really benefit patients? *Respir Care* 36:362—376, 1991.)

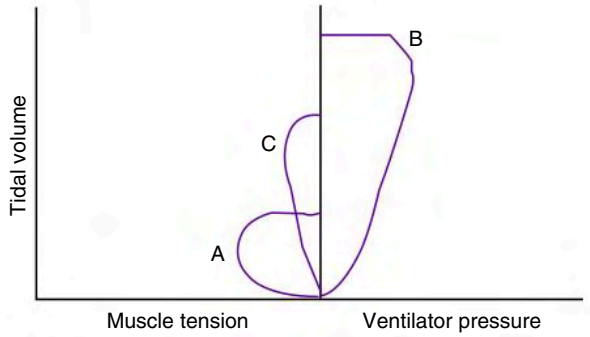


Fig. 10.24 Pressure-volume loops under various conditions. (A) Patient breathing through an endotracheal tube with high impedance (increased resistance and/or decreased compliance). (B) Patient receiving a controlled volume breath. (C) Spontaneous breath under normal circumstances. (From Kacmarek RM: The role of pressure support ventilation in reducing the work of breathing, *Respir Care* 33:99—120, 1988; MacIntyre NR: Weaning from mechanical ventilatory support: volume-assisting intermittent breaths versus pressure-assisting every breath, *Respir Care* 33:121—125, 1988.)

measured during the respiratory cycle. The electronic difference between these two pressures is referred to as the *transdiaphragmatic pressure*. When the transdiaphragmatic pressure is plotted over time, it provides a pressure-time curve that can be used to estimate the activity of the diaphragm (see Fig. 10.25B). Thus diaphragmatic activity during inspiration can be estimated by integrating the area within the curve during inspiration; the resultant value is called the *pressure-time product*. Increases in the pressure-time product indicate a greater force of contraction by the diaphragm. Conversely, decreases in the pressure-time product are associated with less muscular force.[69-71]

Although some clinicians consider the pressure-time product a useful measurement for determining the effectiveness of diaphragm function during weaning from mechanical ventilation, its use is limited in the clinical setting.[69,70]

Occlusion pressure measurements

The *occlusion pressure* ($P_{0.1s}$ or P_{100}) is the airway pressure measured after occluding the airway during the first 100 msec of a patient's spontaneous inspiration. Clinical data suggest that $P_{0.1}$ provides a useful index of ventilatory drive and may be used as a predictor of weaning success (i.e., elevated $P_{0.1}$ is associated with weaning failure). For ventilator-dependent patients, the $P_{0.1}$ has also been shown to correlate with the WOB during pressure support ventilation.[71] (See Chapter 20 for additional information on $P_{0.1}$.)

SUMMARY

- Noninvasive monitoring has become common practice in the care of mechanically ventilated patients.
- Pulse oximeters, capnographs, and transcutaneous monitors have greatly improved the respiratory care practitioner's ability to monitor changes in patients' ABG levels.
- The presence of abnormal hemoglobin, such as COHb (smoke inhalation), produces an erroneously high S_pO_2, and CO-oximetry should be performed to determine the true O_2 saturation.
- Exhaled NO measurements can be used to assess the severity of a patient's asthma exacerbation.
- Transcutaneous O_2 and CO_2 measurements provide a noninvasive method of assessing oxygenation and ventilation status.
- Indirect calorimetry provides new insights about the nutritional status of spontaneously breathing and mechanically ventilated patients.
- Several factors can influence the metabolic rate, including the type and rate of food ingested, the time of day the measurements are made, the patient's level of physical activity, and whether the person is recovering from surgery or an acute or a chronic illness.
- The substrate utilization pattern is the proportion of carbohydrates, fats, and proteins that contribute to the total EE.
- Bedside mechanics testing is an invaluable resource for optimizing mechanical ventilatory support.
- Current ICU mechanical ventilators incorporate microprocessors that can provide breath-by-breath and summative reports of pressure and flow events along with calculations of airway resistance, respiratory compliance, and WOB.
- Dynamic compliance is influenced by flow resistive and elastic characteristics of the patient-ventilator interface, whereas static compliance is influenced only by elastic characteristics of the lung-thorax unit.
- Airway resistance is determined primarily by the diameter of the airway.
- WOB is influenced by intrinsic and extrinsic factors. Intrinsic factors include the elastic and resistive forces that must be overcome during inspiration and expiration. Extrinsic factors are related to the work imposed by systems that are added to the patient, such as ETs, demand valve systems, humidifying devices, and the exhalation valve or PEEP valve.
- The pressure-time product uses transdiaphragmatic pressure measurements to estimate the contributions of the diaphragm during inspiration.

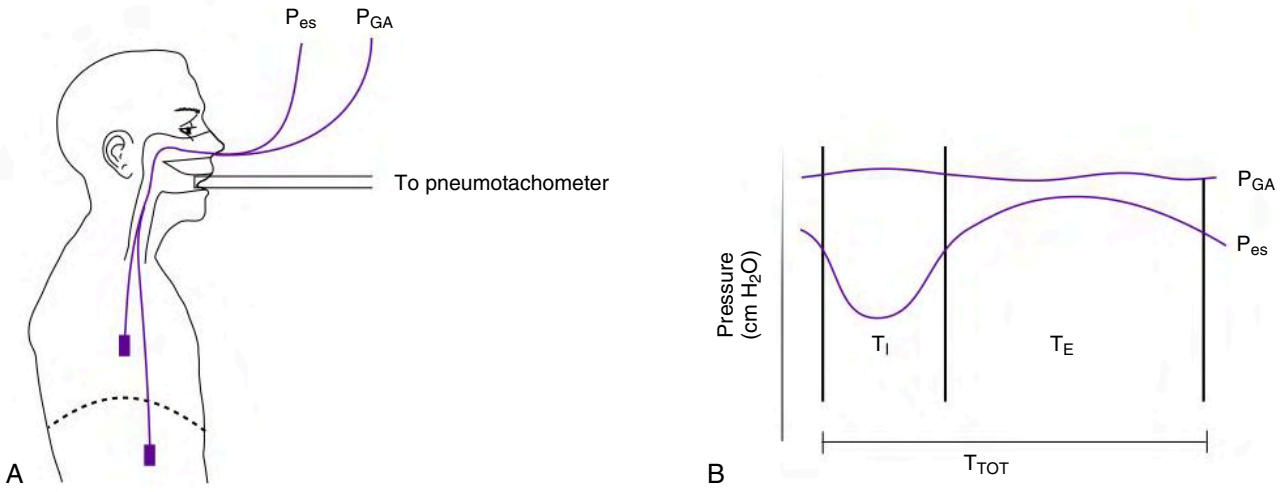


Fig. 10.25 (A) Apparatus for measuring transdiaphragmatic pressures and the pressure-time product. (B) Pressure-time curve for a spontaneous breath. The waveforms illustrate pleural pressure changes during a breath. A variety of waveforms can be displayed with the current respiratory mechanics software. P_{es}, Esophageal pressure; P_{GA}, gastric pressure; T_E, expiratory time; T_I, inspiratory time; T_{TOT}, total time.

REVIEW QUESTIONS *(See Appendix A for answers.)*

1. Draw and label the normal components of the capnogram.
2. What is the pressure-time product? How can this variable be used in the management of mechanically ventilated patients?
3. Which of the following conditions will adversely affect pulse oximeter readings?
   1. Hypovolemia
   2. Methemoglobinemia
   3. Anemia
   4. Hyperbilirubinemia
      A. 1 and 2
      B. 1 and 3
      C. 1, 2, and 3
      D. 2, 3, and 4
4. Which of the following actions is indicated when there is disparity among S_pO_2, S_aO_2, and the clinical presentation of a patient?
   A. Moving the probe to an alternative site to check for S_pO_2
   B. Replacing the pulse oximeter probe
   C. Measuring arterial O_2 saturation by CO-oximetry
   D. Disregarding the S_aO_2
5. Which of these parameters is measured to obtain a functional hemoglobin saturation?
   1. O_2Hb
   2. HHb
   3. COHb
   4. MetHb
      A. 1 and 2
      B. 1 and 3
      C. 2 and 3
      D. 3 and 4
6. An indistinct phase 3 on a patient's capnogram is most often associated with:
   A. Rebreathing exhaled gas
   B. Anemia
   C. Chronic obstructive pulmonary disease
   D. Cheyne-Stokes breathing
7. In the clinical setting, the $P_{(a\text{-}et)}CO_2$ is normally:
   A. −2 to −5 mm Hg
   B. 1 to 3 mm Hg
   C. 4 to 6 mm Hg
   D. 10 to 15 mm Hg
8. Which of the following data should be recorded when making transcutaneous measurements?
   1. Date and time of the measurement
   2. Clinical appearance of the patient
   3. Site of electrode placement on the patient
   4. Type of oxygen delivery device and the F_IO_2
      A. 1 only
      B. 2 and 4 only
      C. 1 and 3 only
      D. 1, 2, 3, and 4
9. Which of the following conditions could lead to an elevated RQ (>1.0)?
   A. Hyperventilation
   B. Starvation
   C. Diabetes mellitus
   D. Sepsis
10. Which of the following conditions is associated with hypermetabolism?
    A. Starvation
    B. Hypothyroidism
    C. Pregnancy
    D. Anesthesia
11. The following data were obtained from a mechanically ventilated patient: V_T = 600 mL, PIP = 30 cm H_2O, P_{Plat} = 20 cm H_2O. What is this patient's static compliance?
    A. 20 mL/cm H_2O
    B. 25 mL/cm H_2O
    C. 30 mL/cm H_2O
    D. 60 mL/cm H_2O

12. Which of the following conditions will cause a decrease in static and dynamic compliance?
A. Bronchospasm
B. Congestive heart failure
C. Partial occlusion of the endotracheal tube
D. Atelectasis
A. 1 and 2
B. 1 and 4
C. 2 and 3
D. 2 and 4

13. What is associated with an increase in the work of breathing in mechanically ventilated patients?
A. Bronchodilation
B. Decreased spontaneous breathing frequency
C. Switching from controlled mechanical ventilation to assisted ventilation
D. Using a larger endotracheal tube

14. Phase 3 of an $SBCO_2$ curve represents which of the following?
A. Dead space
B. Alveolar dead space
C. A mixture of airway and alveolar gas
D. Alveolar gas

15. The PEEP is increased on a patient receiving mechanical ventilation. The $SBCO_2$ curve shows a simultaneous shift to the right and an increase in the area of zone Y. This would indicate which of the following?
A. Lung recruitment and emptying of previously collapsed alveoli
B. Increased alveolar dead space from reduced pulmonary perfusion
C. Decreased P_aCO_2 from improvement in oxygenation
D. Increase in rebreathed volume

16. Which of the following will affect the level of exhaled NO in exhaled gas?
1. Presence of a pathological condition
2. Smoking habits
3. Atopic status
4. Patient's gender
1. 1 and 3 only
2. 2 and 4 only
3. 1, 2, and 4 only
4. 1, 2, 3, and 4

References

1. Nitzan M, Romem A, Koppel R: Pulse oximetry: fundamentals and technology update, *Med Devices* 7:231–239, 2014.
2. Comroe JH, Botelho SH: The unreliability of cyanosis in recognition of arterial anoxemia, *Am J Med Sci* 214:1–6, 1947.
3. Hess DR, MacIntyre NR, Galvin WF: *Respiratory care: principles and practice*. In Burlington MA, editor: ed 4, 2020, Jones and Bartlett Learning.
4. Cairo JM: *Mosby's respiratory care equipment*, ed 11, 2022, Elsevier. St. Louis MO.
5. Hannhart B, Habberer JP, Saunier C, et al.: Accuracy and precision of fourteen pulse oximeters, *Eur Respir J* 4:115–119, 1991.
6. Kacmarek RM, Stoller JK, Stoller, Heuer AJ: *Egan's fundamentals of respiratory care*, ed 12, St. Louis, MO, 2021, Elsevier.
7. Kelleher JF: Pulse oximetry, *J Clin Monit* 5:37–62, 1989.
8. Tremper KK, Barker SJ: Pulse oximetry, *Anesthesiology* 70:98–108, 1989.
9. Jubran A: Pulse oximetry, *Crit Care* l3:R11–R17, 1999.
10. Dumas C, Wahr JA, Tremper KK: Clinical evaluation of a prototype motion artifact resistant pulse oximeter in the recovery room, *Anesth Analg* 83:269–272, 1996.
11. Trillo RA, Aukburg S: Dapsone-induced methemoglobinemia and pulse oximetry, *Anesthesiology* 77:594–596, 1992.
12. Scheller MS, Unger RJ, Kelner MJ: Effects of intravenously administered dyes on pulse oximetry readings, *Anesthesiology* 65:550–552, 1986.
13. Coté CJ, Goldstein EA, Fushsman WH, et al.: The effect of nail polish on pulse oximetry, *Anesth Analg* 67:683–686, 1988.
14. Rubin AS: Nail polish color can affect pulse oximeter saturation, *Anesthesiology* 68:825, 1988.
15. Emery JR: Skin pigmentation as an influence on the accuracy of pulse oximetry, *J Perinatol* 7:329–330, 1987.
16. Ries AL, Prewitt LM, Johnson JJ: Skin color and ear oximetry, *Chest* 96:287–290, 1989.
17. Jubran A: Advances in respiratory monitoring during mechanical ventilation, *Chest* 116:1416–1425, 1999.
18. Chelluri L, Snyder JV, Bird JR: Accuracy of pulse oximetry with hyperbilirubinemia, *Respir Care* 36:1383–1386, 1991.
19. Veyckemans F, Baele P, Guillaume JE, et al.: Hyperbilirubinemia does not interfere with hemoglobin saturation measured by pulse oximetry, *Anesthesiology* l70:118–122, 1989.
20. Amar D, Neidswski J, Wald A, et al.: Fluorescent light interferes with pulse oximetry, *J Clin Monit* 5:135–136, 1989.
21. Jubran A, Tobin MJ: Monitoring during mechanical ventilation. In Tobin MJ, editor: *Principles and practice of mechanical ventilation*, ed 3, New York, NY, 2013, McGraw-Hill, pp 1139–1165.
22. Fluck RR, Schroeder C, Frani G, et al.: Does ambient light affect the accuracy of pulse oximetry? *Respir Care* 48:677–680, 2003.
23. Perkins GD, McAuley DF, Giles S, et al.: Do changes in pulse oximeter saturation predict equivalent changes in arterial oxygen saturation? *Crit Care* 7:R67–R71, 2003.
24. Kacmarek RM, Hess D, Stoller JK: *Monitoring in respiratory care*, St. Louis, MO, 2003, Mosby.
25. Carlin BW, Claussen JL, Ries AL: The use of cutaneous oximetry in the prescription of long-term oxygen therapy, *Chest* 94:239–241, 1988.
26. Baeckert P, Bucher HU, Fallenstein F, et al.: Is pulse oximetry reliable in detecting hypoxemia in the neonate? *Adv Exp Med Biol* 220:165–169, 1987.
27. Walsh BK, Crotwell DN, Restrepo RD: Capnography/Capnometry during mechanical ventilation: 2011, *Respir Care* 56(4):503–509, 2011.
28. Gravenstein JS, Jaffe MB, Paulus DA: *Capnography*, Cambridge, UK, 2011, Cambridge University Press.
29. Neumar RW, Otto CW, Link MS, et al.: Part 8: adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care, *Circulation* 122(Suppl 3):S729–767, 2010.
30. Suarez-Sipmann F, Bohm SH, Tusman G: Volumetric capnography, the time has come, *Curr Opin Crit Care* 20(3):333–339, 2014.
31. Siobal MS: Monitoring exhaled carbon dioxide, *Respir Care* 61(10):1397–1416, 2016.
32. Lúdvíksdóttir D, Janson C, Högman M, et al.: Exhaled nitric oxide and its relationship to airway responsiveness and atopy in asthma. BHR Study Group, *Respir Med* 93:552–556, 1999.
33. Franklin PJ, Stick SM, LeSouef PN, et al.: Measuring exhaled nitric oxide levels in adults: the importance of atopy and airway responsiveness, *Chest* 126:1540–1545, 2004.
34. Huch A, Huch R, Arner B, et al.: Continuous transcutaneous oxygen tension measurement with a heated electrode, *Scand J Clin Lab Med* 31:269–275, 1973.
35. Huch R, Huch A, Albani M, et al.: Transcutaneous Po_2 monitoring in routine management of infants and children with cardiorespiratory problems, *Pediatrics* 57:681–690, 1976.
36. Lubbers DW: Theory and development of transcutaneous oxygen pressure measurement, *Int Anesthesiol Clin* 25:31–65, 1987.
37. Severinghaus JS, Bradley FA: Electrodes for blood Po_2 and Pco_2 determination, *J Appl Physiol* 13:515–520, 1958.