103. Jereb D, Bauer K, Martin JE: Developing a correction factor to determine the set tidal volume when using helium through a Dräger Dura 2 ventilator, *Respir Care* 48:1093A, 2003.
104. Baxter TD, Coulliette H: A comparison of three ventilators and their oxygen consumption efficiency [abstract], *Respir Care*, 2009.
105. Donnelly D, Walsh BK: Heliox utilization by two ICU ventilators: a bench study [abstract], *Respir Care*, 2009.
106. Gluck EH: Helium-oxygen mixtures in intubated patients with status asthmaticus and respiratory acidosis, *Chest* 98:693–698, 1990.
107. Cheifetz I: *Heliox in intubated patients: rationale and evidence*, Las Vegas, 2003, American Association of Respiratory Care 49th International Respiratory Congress. December 4-7.
108. Polito A, Fessler H: Heliox in respiratory failure from obstructive lung disease [letter], *N Engl J Med* 332:192–193, 1995.
109. Jolliet P, Tassaux D, Thouret JM, et al.: Beneficial effects of helium:oxygen versus air:oxygen noninvasive pressure support in patients with decompensated chronic obstructive pulmonary disease, *Crit Care Med* 27:2422–2429, 1999.
110. GU Meduri, Cook TR, Turner RE, et al.: Noninvasive positive pressure ventilation in status asthmaticus, *Chest* 110:767–774, 1996.
111. Sinderby C, Beck J: Neurally adjusted ventilatory assist (NAVA): an update and summary of experiences, *Neth J Crit Care* 11:243–252, 2007.
112. Talmor DS, Fessler HE: Are esophageal pressure measurements important in clinical decision-making in mechanically ventilated patients? *Respir Care* 55:162–172, 2010.
113. Talmor D, Sarge T, Malhotra A, et al.: Mechanical ventilation guided by esophageal pressure in acute lung injury, *N Engl J Med* 359:2095–2104, 2008.
114. Lourenco RV, Cherniack NS, Malm JR, et al.: Nervous output from the respiratory center during obstructed breathing, *J Appl Physiol* 21:527–533, 1996.
115. Beck J, Sinderby C, Lindstrom L, et al.: Effects of lung volume on diaphragm EMG signal strength during voluntary contractions, *J Appl Physiol* 85:1123–1134, 1998.
116. Aldrich TK, Sinderby C, McKenzie DK, et al.: Electrophysiological techniques for the assessment of respiratory muscle function, *Am J Respir Crit Care Med* 166:548–558, 2002.
117. Sinderby C, Spahija J, Beck J, et al.: Diaphragm activation during exercise in chronic obstructive pulmonary disease, *Am J Respir Crit Care Med* 163:1637–1641, 2001.
118. Barwing J, Ambold M, Linden N, et al.: Evaluation of the catheter positioning for neurally adjusted ventilatory assist, *Intensive Care Med* 35:1809–1814, 2009.
119. Sinderby C, Beck J, Spahija J, et al.: Voluntary activation of the human diaphragm in health and disease, *J Appl Physiol* 85:2146–2158, 1998.
120. Beck J, Weinberg J, Hamnegard CH, et al.: Diaphragmatic function in advanced Duchenne muscular dystrophy, *Neuromuscul Disord* 16:161–167, 2006.
121. Bordessoule A, Emeriaud G, Delnard N, et al.: Recording diaphragm activity by an oesophageal probe: a new tool to evaluate the recovery of diaphragmatic paralysis, *Intensive Care Med* 36:1978–1979, 2010.
122. deWit M, Miller KB, Green KA, et al.: Ineffective triggering predicts increased duration of mechanical ventilation, *Crit Care Med* 37:2740–2745, 2009.
123. Thille AW, Cabello B, Galia F, et al.: Reduction of patient-ventilator asynchrony by reducing tidal volume during pressure-support ventilation, *Intensive Care Med* 34:1477–1486, 2008.
124. Thille AW, Rodriguez P, Cabello B, et al.: Patient-ventilator asynchrony during assisted mechanical ventilation, *Intensive Care Med* 32:1515–1522, 2006.
125. Beck J, Reilly M, Grasselli G, et al.: Patient-ventilator interaction during neurally adjusted assist in low birth weight infants, *Pediatr Res* 65:663–668, 2009.
126. Levin S, Nguyen T, Taylor N, et al.: Rapid disuse atrophy of diaphragm fibers in mechanically ventilated humans, *N Engl J Med* 358:1327–1335, 2008.
127. Knisely AS, Leal SM, Singer DB: Abnormalities of diaphragmatic muscle in neonates with ventilated lungs, *J Pediatr* 113:1074–1077, 1988.
128. Thille AW, Brochard L: Double triggering during assisted mechanical ventilation: is it a controlled, auto-triggered, or patient triggered cycle? [reply to Chen CW], *Intensive Care Med* 33:744–745, 2007.
129. Chiumello D, Polli F, Tallarini F, et al.: Effect of different cycling-off criteria and positive end-expiratory pressure during pressure support ventilation in patients with chronic obstructive pulmonary disease, *Crit Care Med* 35:2547–2552, 2007.
130. Girard TD, Kress JP, Fuchs BD, et al.: Efficacy and safety of a paired sedation and ventilator weaning protocol for mechanically ventilated patients in intensive care (awakening and breathing controlled trial): a randomized controlled trial, *Lancet* 371:126–134, 2008.
131. Sinderby C, Beck J: Proportional assist ventilation and neurally adjusted ventilatory assist: better approaches to patient ventilator synchrony? *Clin Chest Med* 29:329–342, 2008.
132. Spahija J, de Marchie M, Albert M, et al.: Patient-ventilator interaction during pressure support ventilation and neurally adjusted ventilatory assist, *Crit Care Med* 38:518–526, 2010.
133. Chatburn RL, Mireles-Cabodevila E: Closed-loop control of mechanical ventilation: description and classification of targeting schemes, *Respir Care* 56:85–102, 2011.
134. Sinderby C, Navalesi P, Beck J, et al.: Neural control of mechanical ventilation, *Nat Med* 5:1433–1436, 1999.
135. Servo Education, NAVA Tutorial: Maquet critical care, *Solna Sweden, Order No* 66(79):145, 2000.
136. Leiter JC, Manning HL: The Hering-Breuer reflex, feedback control, and mechanical ventilation: the promise of neurally adjusted ventilatory assist, *Crit Care Med* 38:1915–1916, 2010.
137. White C, Seger B, Lin L, et al.: The effect of NAVA on parameters of ventilation in the pediatric intensive care unit, *Respir Care* 55:1598, 2010.
138. Howard D, Stein H: Neonates ventilated with NAVA have better blood gases than those ventilated with SIMV/PC with PS [abstract], *Respir Care* 43, 2009.
139. Coisel Y, Chanques G, Jung B, et al.: Neurally adjusted ventilatory assist in critically ill postoperative patients: a crossover randomized study, *Anesthesiology* 13:925–935, 2010.
140. Demoule A, Schmidt M, Cracco C, et al.: Neurally adjusted ventilatory assist increases respiratory variability and chaos in acute respiratory failure, *Am J Respir Crit Care Med* 179:a3648, 2009.
141. Brander L, Leong-Poi H, Beck J, et al.: Titration and implementation of neurally adjusted ventilatory assist in critically ill patients, *Chest* 135:695–703, 2009.
142. Noblet T: Effect of bubble CPAP and high flow nasal cannula therapy on the electrical activity of the diaphragm in a premature infant, *Respir Care* 54(1537):a678892, 2009.
143. Stein H, Howard D: Neonates ventilated with NAVA do not have an increased incidence of IVH, pneumothorax or NEC/perforation compared to those ventilated with SIMV/PC with PS [abstract], *Respir Care* 43, 2009.
144. MacIntyre N: Talk to me? Toward better patient-ventilator communication [editorial], *Crit Care Med* 38:714–715, 2010.
145. Navalesi P, Colombo D, Della Corte F: NAVA ventilation: a review, *Minerva Anestesiol* 76:346–352, 2010.

APPENDIX A

Answer Key

PART 1: REVIEW QUESTIONS ANSWER KEY

Chapter 1

Basic Terms and Concepts of Mechanical Ventilation

1. Figs. 1.2 and 1.8 show how to draw these graphs. During spontaneous, quiet breathing, pleural pressure is always negative and ranges from approximately −5 to −10 cm H_2O. With positive pressure ventilation, pleural pressure can become positive at end inspiration but usually returns to its resting negative value (−5 cm H_2O) during exhalation.
2. 5 × 1.36 = 6.8 cm H_2O
3. Unit A would receive the greatest volume for the same pressure during a given inspiratory time. Unit A also has a shorter time constant (Time constant = R_{aw} × C). Unit B fills more slowly because it has increased resistance; that is, it has a greater (longer) time constant.
4. Unit B will fill more quickly. It is less compliant than unit A; therefore its time constant (R_{aw} × C) will have a smaller value. Stiff alveoli with poor compliance fill quickly, but they do not fill with the same volume for a given pressure. If pressure delivery to both units is the same, unit A will have a greater volume. Recall that C = ΔV/ΔP and ΔV = C × ΔP.
5. Lung units vary in filling and emptying times on the basis of their compliance and resistance. Less compliant lung units fill faster, and those with higher R_{aw} fill more slowly.
   A. Time constant = 0.1 second
   B. Time constant = 0.025 second
   C. Time constant = 1.0 second
   D. Time constant = 0.25 second
   E. Time constant = 1.5 seconds
   F. Time constant = 0.15 second
   G. Units ranked in order from slowest to fastest: e, c, d, f, a, and b
6. C
7. D
8. B
9. C
10. D
11. B
12. A
13. D
14. D
15. B
16. C
17. D

Chapter 2

How Ventilators Work

1. Bird Mark 7 ventilator. These devices typically have been used for administering intermittent positive pressure breathing treatments.
2. LTV 1000 and LTV 1125 Ventilators (Becton, Dickinson and Company, Franklin Lakes, NJ); Newport HT 50 Ventilator (Newport Medical Instruments, Costa Mesa, CA).
3. Negative pressure ventilator.
4. When the ventilator begins inspiratory gas flows through the main inspiratory tube, gas also flows through the expiratory valve line to the exhalation valve, closing the valve (see Fig. 2.8A cutaway). During exhalation, the flow from the ventilator stops and the exhalation valve opens. The patient is able to exhale passively through the expiratory port in the exhalation valve.
5. Rotary drive piston.
6. A
7. C
8. D
9. B
10. C
11. C
12. A

Chapter 3

How a Breath Is Delivered

1. $P_{mus} + P_{vent} = V/C + (R_{aw} \times flow)$
2. The volume of air in the lungs depends on the elastic recoil pressure, which is the pressure resulting from alveolar tension on the volume within alveoli. Elastic recoil pressure (P_E) is the elastic load offered by the lungs and chest wall that must be overcome to move air into the lungs during inspiration. Note that elastance is the inverse of compliance.
3. A
4. Pressure-targeted ventilation, pressure ventilation.
5. A
6. With pressure-controlled breaths, the pressure waveform is constant during inspiration, and volume and flow can vary with changes in lung characteristics, the set pressure, or the patient's inspiratory effort. With volume-controlled breaths, volume delivery is constant, and the volume and flow waveforms remain unchanged during inspiration. The pressure waveform can vary with changes in lung characteristics.
7. Pressure and flow.
8. C
9. B
10. A

11. True.
12. D
13. A
14. D
15. B

Chapter 4
Establishing the Need for Mechanical Ventilation

1. A
2. D
3. B
4. D
5. B
6. D
7. C
8. C
9. A
10. C
11. D
12. A
13. B
14. A
15. B

Chapter 5
Selecting the Ventilator and Mode

1. C
2. C
3. D
4. A
5. D
6. C
7. A pressure of 30 cm H_2O to start would provide approximately the same V_T delivery in VC-CMV and would be a safe starting point. This patient requires high pressures for delivery of V_T. P_{alv} should be kept below 30 cm H_2O.
8. Apparently, the ventilator has a fixed flow and pattern during inspiration that is not adequate for the patient's needs. The therapist should increase the inspiratory flow and see if this solves the problem. Another possible solution is to switch to another ventilator that allows additional flow on demand during VC-CMV.
9. There are several options: (1) switch the patient to PC-IMV with a lower mandatory rate; (2) switch the patient to PSV; (3) switch to VS. Although any of these options would allow for more spontaneous ventilation, it is important to monitor the S_pO_2, V_T, and $\dot{V}_E$ closely.
10. A dual control mode that limits pressure (e.g., PRVC) may be appropriate for this situation. This mode would limit pressure delivery while simultaneously allowing for a targeted volume in an effort to maintain the patient's minute ventilation.

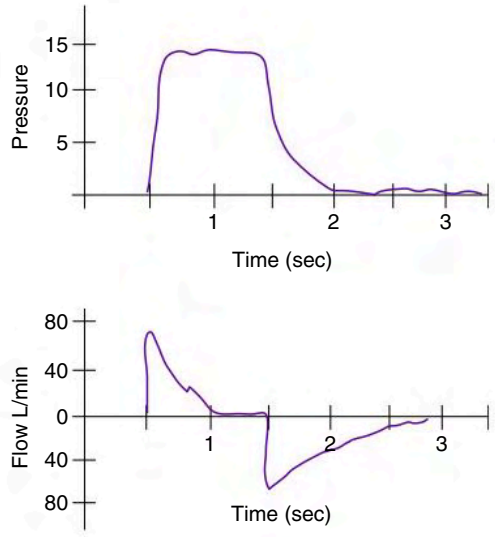
Chapter 6
Initial Ventilator Settings

1. $C_T = 100$ mL/33 cm $H_2O = 3$ mL/cm H_2O
   Volume lost to circuit = 3 mL/cm $H_2O \times 15$ cm $H_2O = 45$ mL
2. The $\dot{V}_E = 700$ mL $\times$ 15 = 10.5 L/min
   Set V_T (250 mL) − Volume lost (45 mL) = About 205 mL
3. $\dot{V}_E = 700$ mL $\times$ 15 = 10.5 L/min
   A. If the flow is set at 30 L/min using a constant flow pattern, the flow in L/s is 30 L/60 s = 0.5 L/s.
   B. The TCT based on the set machine *f* of 12 breaths/min is 5 seconds.
   C. The TCT based on the actual machine *f* of 15 breaths/min is 4 seconds.
   D. The T_I based on the set *f*, flow, and V_T:T_I = 0.7 L/(0.5 L/s) = 1.4 seconds.
   E. The T_E when *f* is 12 is 3.6 seconds.
   F. The T_E when *f* is 15 is 2.6 seconds.
4. C
5. The patient is actively breathing during the plateau period.
6.
   A. The patient's BSA is approximately 2.12 m^2.
   B. His ideal body weight is 160 lb, or about 73 kg.
   C. Before correction for body temperature, the $\dot{V}_E$ is 8.48 L/min. This is reduced by 18% (10% per degree Celsius). Eighteen percent of 8.48 is about 1.53 L. 8.48 − 1.53 is a final V_E of 6.95 L/min. In postoperative open heart surgery patients, the body temperature is sometimes cooler than normal. Patients warm up quickly and usually recover from the anesthesia quickly. As the patient warms up, the V_E would need to be increased. Either CMV or IMV modes would be appropriate with a safe rate and V_T setting.
   D. Because the surgeon is concerned about pressure, you may want to choose PC-CMV.
   E. V_T would be targeted between 6 and 8 mL/kg IBW (i.e., 438 and 584 mL/kg). The patient weighs 210 lb; however, V_T is based on IBW, not actual weight. This is a common mistake among new practitioners. (Note that the V_T would be targeted between 570 mL and 760 mL [range of 6–8 mL/kg IBW] if the actual body weight of 210 lb was used.) However, V_T is based on IBW, *not* actual weight; this is a common mistake among new practitioners.
   F. An appropriate rate based on a V_T of about 438 mL (0.438 = 16 breaths/min; based on a V_T of 0.584 L) would be (6.95 L/min)/0.584 L = 11 breaths/min.
   G. Because the physician is concerned about pressure, a low compensating PEEP of about 3 cm H_2O would be a good starting point.
7. The patient's IBW is 105 + 5(64 − 60) = 125 lb, or 56.8 (57) kg. However, she weighs 195 lb (about 87 kg); therefore she has a large BSA (about 1.94 m^2). Her estimated $\dot{V}_E$ (assuming normal body temperature, etc.) is 3.5 $\times$ 1.94, or about 6.8 L/min. If the $\dot{V}_E$ had been based on IBW, it would have underestimated her metabolic rate. However, her V_T must be based on IBW.

Remember, when a person gains weight, his or her lungs do not get bigger.

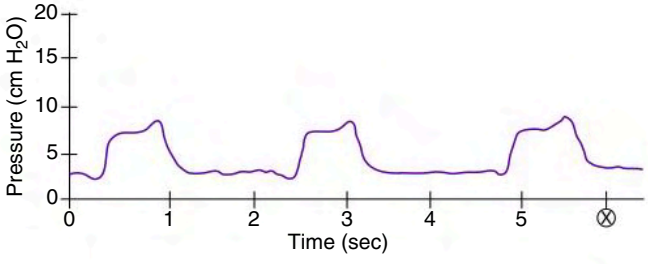
Using the calculated IBW, an appropriate V_T for a patient with ARDS would be in the range of 4 to 8 mL/kg. For the patient this range would be about 230 to 450 mL. A respiratory rate of 15 (using 8 mL/kg) to 30 (using 4 mL/kg) breaths/min would be in the target range. A higher rate may accompany a lower V_T. It might be appropriate to start in the middle of the ranges (e.g., 6 mL/kg). For example, setting a V_T of 350 mL and a rate of 20 breaths/min PV would probably provide patient comfort and would be suitable for this patient, particularly based on her diagnosis. The pressure could be set to target the desired V_T.

8. Try increasing the IPAP to 11 cm H_2O and reevaluate the patient. The V_T should be 420 to 560 mL (range, 6–8 mL/kg IBW), and the spontaneous rate should be below 25 breaths/min.
9. The $\dot{V}_E$ should be increased by 10%, or 5% for every degree >99° F.
10. Change to a descending waveform with volume ventilation would change the pattern of pressure delivery, resulting in a decrease in PIP. This was an actual case. After making the adjustment, the resulting PIP was 41 cm H_2O and the P_{plat} was 32 cm H_2O without any other changes.
11. The lung characteristics are deteriorating; that is, either R_{aw} is increasing or the lung compliance is decreasing, or both.
12. Volume support ventilation.



(NOTE: This figure should read pressure–time and flow–time curves during PC-CMV.)

13. About 14 cm H_2O.



(NOTE: This figure should read pressure–time curve during PSV.)

14. A

Chapter 7
Final Considerations in Ventilator Setup

1. Initial $\dot{V}_E = 4 \times 1.5 = 6$ L/min
   V_T is based on IBW
   IBW = 106 + 6(8) = 154 lb, or 70 kg
   V_T range of 6 to 8 mL/kg = 280 to 560 mL
   Example: At 6 mL/kg, initial V_T = 420 mL (0.42 L)
   Rate using selected V_T and $\dot{V}_E = \dot{V}_E/V_T = 6/0.42 = 14$; f = 14 breaths/min
2. Over a 2-hour period, the $\dot{V}_E$ increased from 6.5 to 13 L/min. The patient is using accessory muscles to breathe, and the pressure–time curve indicates an inadequate flow. PIP has also increased. These findings suggest the development of air trapping; therefore evaluate for the presence of intrinsic PEEP. Increase the inspiratory gas flow and reassess the pressure–time curve. Assess the patient's ventilatory pattern to determine whether the problem persists. Another possibility is to switch to VC-IMV + PSV; however, inspiratory flow would still need to be increased and PSV would have to be set at an appropriate level.
   Other questions might also be asked. Can you think of any? For example, what is the S_pO_2? Have the ABG values changed? Have breath sounds changed? What does the flow–time curve look like?
3. (NOTE: A number of solutions are possible for this problem. The following answer is provided as one option.) Initial V_T is selected at 6 mL/kg (420 mL). Because this patient has a history of COPD, a lower V_T setting was selected to reduce the risk for lung injury. $\dot{V}_E$ is set at 8 L/min: 4 × BSA = 4 × 1.78 = 7.12 L/min. Increase $\dot{V}_E$ by 5% per degree Fahrenheit: T = 102°F, or 15%. Increase by 1.05 L/min to a total $\dot{V}_E$ of 8.2 L/min. Rate is set at 19 breaths/min ($\dot{V}_E/V_T = f$). Flow is set at 80 L/min using the descending flow pattern. F_IO_2 is set at 0.5 initially. (NOTE: Because the patient is breathing spontaneously, a lower f and $\dot{V}_E$ could be used, depending on the clinician's preference. The F_IO_2 could be lower as well. The settings selected here would provide a minimum safe initial form of ventilation and could be adjusted on the basis of patient assessment.)
   Resistance is estimated at P_{TA}/Flow, or 8 cm H_2O/80 L/min: 8 cm H_2O/(1.33 L/s) = 6 cm H_2O/(L/s). This answer is only an estimate because the flow is not constant. Pressure support is set at 6 cm H_2O. This is an example of initiating ventilatory support in a patient with COPD. Note the use of a lower V_T setting, the $\dot{V}_E$ increase because of the patient's temperature, and the use of pressure support for spontaneous breaths set high enough to overcome the resistance imposed by the airway. The patient's breathing must be evaluated to ensure comfort, patient–ventilator synchrony, and reduced work of breathing (WOB).
4. Postoperative patients and victims of drug overdose are examples of patients who are most likely to have normal lungs yet

still require ventilation. Appropriate settings for normal lungs are as follows:

VC-CMV or PC-CMV

V_T = 6 to 8 mL/kg

f = 10 to 15 breaths/min

Descending or constant flow waveform

T_I = About 1 second initially

PEEP ≤5 cm H_2O; kept P_{plat} <30 cm H_2O

F_IO_2 ≤0.5 (titrate to patient's normal; maintain P_aO_2 >70 mm Hg and S_pO_2 ≥92%)

For this patient a V_T range of 480 to 640 mL would be appropriate. $\dot{V}_E$ would equal 4 × 1.8 = 7.2 L/min. Rate would range from 11 to 18 breaths/min. An F_IO_2 of 0.5, a PEEP of 3 cm H_2O, a descending flow ramp, and a VC-CMV would all be appropriate settings.

Chapter 8

Initial Patient Assessment

1. C. *a, b,* and *d* refer to times when a ventilator check is performed.
2. A
3. D
4. B. V_{Danat} is equal to about 180 mL (80 kg IBW × 2.2 lb = 176 lb). With an ET in place, the V_{Danat} is reduced by about half to 90 mL. The HME adds about 50 mL of mechanical dead space. V_D is about 140 mL. $V_T - V_D = V_A$; $V_A = 400 - 140 = 260$ mL/breath.
5. B. When the flow is zero (before exhalation begins), the pressure on the pressure–time curve for the same time interval will be P_{plat}.
6. A
7. C
8. D (the difference between PIP and P_{plat} is increasing).
9. An increase in R_{aw}, as might occur with bronchoconstriction or mucous plugging.
10. V_T = 0.55 mL, V_{Danat} = 150 mL, $\dot{V}_A = (0.55 - 0.15) \times 8 =$ 3.2 L/min
11. This scenario actually describes the use of MLT. The best choice is *B*; check the cuff pressure and delivered volume. It is the safest choice without knowing whether MLT or MOV is used.
12. The cuff pressure is much too high and will result in reduced blood flow to the adjacent tracheal walls.

The tube is too small for this patient. The best choice is *C*; change the ET to a size 8 Fr.

13. C. The change in V_T may be the result of an improvement in lung characteristics (e.g., a decrease in R_{aw} or an improvement in C_L). Listen to the breath sounds, evaluate the flow–time and volume–time curves, and check the current acid–base status. Changes in V_T may lead to respiratory alkalosis. The preset pressure may need to be reduced.
14. C. Because no air leak is heard, MOV probably has been used to inflate the ET cuff. The measurement technique is accurate if done appropriately: that is, if the system was pressurized before the pilot balloon connection was attached to the manometer. This is a low pressure and would not interfere with tracheal blood flow.
15. Answer *A* is the most immediate fix. If the patient is to be ventilated for an extended period, it might be appropriate to change the ET (answer *B*).

Chapter 9

Ventilator Graphics

1.
   A. The *arrow* indicates an active exhalation by the patient.
   B. The flow cycle criterion might be increased to a higher percentage so that T_I is shorter.
   C. The expiratory flow pattern at *arrow B* indicates air trapping.
   D. Shortening the T_I might allow a longer time for exhalation and eliminate this problem. Suctioning of the patient or administration of a bronchodilator might be indicated to reduce R_{aw}. Because this is PSV, reducing the set pressure might reduce both V_T and $\dot{V}_E$, thus reducing air trapping. However, this type of adjustment would have to be monitored to ensure that the patient is still adequately ventilated.
   E. The prolonged exhalation of volume suggests airway resistance; checking a flow–volume loop might confirm this finding.
   F. It is set at a high percentage of the peak flow.
   G. The pressure–time curve does not have a concave appearance; however, the curve would not take this shape with pressure-targeted ventilation. The patient can get as much flow as needed, and the pressure will be maintained. The ventilator would increase flow to achieve the set pressure if the patient were actively inhaling. As long as a rise time or slope does not reduce inspiratory flow, inadequate flow should not be a problem.
2. Answers to questions regarding the figure featured below Question 2: flow, pressure, and volume scalars for three different ventilation situations, *A*:
   A. The target variable is pressure. The mode could be PC-CMV, but there are not enough breaths to determine whether it is PC-CMV or PC-IMV. The breath shown is time triggered, pressure targeted, and time cycled (flow drops to zero before end inspiration).
   B. The set pressure is 20 cm H_2O.
   C. V_T is 500 mL.
   D. P_{plat} is 20 cm H_2O. Because the flow drops to zero during inspiration, there is a period of no flow before exhalation begins. The pressure at that time is the same at the ventilator and in the lungs.
   E. A leak is present; the inspired and expired volumes are not the same.
3. Answers to questions regarding the figure featured below Question 2: flow, pressure, and volume scalars for three different ventilation situations, *B*:
   A. The target variable is volume. The mode of ventilation is VC-CMV. You can tell it is volume targeted because it has a constant flow curve. You can tell it is CMV because you can see the beginning of a second breath, and it is also a mandatory breath.
   B. The total cycle time in this example is 3.5 seconds.
   C. The breath is patient triggered.
   D. On the basis of the pressure–time curve, the problem is inadequate flow.

4. Answers to questions regarding the figure featured below Question 2: flow, pressure, and volume scalars for three different ventilation situations, *C*:
   A. The set flow is about 40 L/min.
   B. The patient is actively inhaling during inspiration, and the ventilator is providing additional flow. This is probably a newer-generation ICU ventilator with a responsive inspiratory valve.
   C. Volume delivery will increase because the patient can demand more flow than the set amount.
5. Answers to question 5 regarding the Figure of P-V loop:
   A. APIP is about 17.5 cm H_2O.
   B. The delivered V_T is about 700 mL.
   C. The pressure—volume loop begins at a pressure of zero; therefore no PEEP has been set.
   D. Compliance is about 40 mL/cm H_2O.
   E. P_{TA} is about 7 cm H_2O, which is slightly higher than normal.
   F. The appearance of the pressure—volume loop indicates that the patient has increased R_{aw}.
6. Answers to question 6 regarding the Figure of F-V loop:
   A. The target variable is volume because the flow is constant.
   B. The flow setting is 60 L/min, and the flow waveform is constant (rectangular).
   C. V_T is 500 mL.
   D. The artifact indicated by *arrow A* is the compressible volume from the patient circuit leaving the circuit at the beginning of exhalation.
   E. Arrow *B* indicates air trapping (auto-PEEP).
   F. Increased R_{aw}.
7. PIP and P_{plat} will increase, and T_I and V_T will stay the same.
8. P_{set} and T_I will remain constant, but V_T will increase.
9.
   A. P_{alv} is 35 cm H_2O. Compliance has nothing to do with the answer. We know the pressure because flow drops to zero during inspiration (the alveolar and preset pressures equilibrate).
   B. V_T will be 0.525 L: Volume = Pressure × Compliance; Volume = 35 cm H_2O × 15 mL/cm H_2O = 525 mL (0.525 L).
   C. After compliance improves to 30 mL/cm H_2O, volume delivery will increase: Volume = 35 cm H_2O × 0.03 mL/cm H_2O = 1.05 L (1050 mL). P_{TA} will be approximately the same because the generating pressure is the same and R_{aw} is not increased. (Actually, P_{TA} will decrease slightly as the flow drops off when P_{alv} approaches the preset pressure.)
   D. Reduce the preset pressure until the desired V_T is achieved. (This would be approximately half the previous setting because compliance doubled [about 17—18 cm H_2O].)
10. B
11. C

Chapter 10

Assessment of Respiratory Function

1. As Fig. 10.8 illustrates, the capnographic waveform is divided into four phases. In phase 1, the initial gas exhaled is from the conducting airways, which contain low levels of CO_2. During phase 2, alveolar gas containing CO_2 mixes with gas in the anatomical airways and the CO_2 concentration increases. In phase 3, the curve plateaus as alveolar gas is exhaled (this phase is often referred to as the *alveolar plateau*). Note that the concentration of CO_2 at the end of the alveolar phase (just before inspiration begins) is called the *end-tidal* PCO_2 or $P_{et}CO_2$. On inspiration (phase 4), the concentration falls to zero.
2. The pressure—time product, which is an assessment of transdiaphragmatic pressure during the inspiratory portion of the breathing cycle, is a way of estimating the contributions of the diaphragm during inspiration.
3. C
4. C
5. A
6. C
7. C
8. D
9. A
10. C
11. A
12. C
13. C
14. D
15. B
16. D

Chapter 11

Hemodynamic Monitoring

1. A
2. C
3. C
4. B
5. B
6. B
7. B
8. C
9. C
10. B
11. A
12. D
13. D
14. A
15. C
16. D
17. B
18. A
19. A
20. A

Chapter 12

Methods to Improve Ventilation in Patient—Ventilator Management

1.
   A. Mandatory $\dot{V}_A = 8 \times (400 - 140) = 2.08$ L/min; spontaneous $\dot{V}_A = 25 \times (225 - 140) = 2.13$ L/min; total $\dot{V}_A = 2.08 + 2.13 = 4.21$ L/min
   B. $C_S = 400/20 = 20$ mL/cm H_2O
   C. The patient has a respiratory acidosis. In addition, his spontaneous breathing rate is high, and his spontaneous V_T is low, indicating a high WOB. His P_{set} is within safe limits. The CO_2 can be reduced by increasing the

pressure to increase the V_T or by increasing the set rate. Increasing the P_{set} to 29 cm H_2O theoretically would increase the V_T to 870 mL (about 14 mL/kg) and reduce the P_aCO_2 to 40 mm Hg. This is a high V_T. On the other hand, increasing the set rate to 12 breaths/min would also reduce the CO_2 to 40 mm Hg. In addition, using PSV to overcome the work required to move air through the resistance of the ET would reduce the patient's spontaneous WOB. PSV also could be used to reduce the P_aCO_2 instead of changing the mandatory rate or volume.

2. Increase the mandatory *f* to 10 breaths/min to return the patient to a P_aCO_2 closer to his normal value. Add PSV of about 8 cm H_2O for spontaneous breaths to reduce spontaneous WOB.
3. IBW for this patient is 106 + 6(4) = 130 lb, or 59 kg. The set V_T is too high and has been based on actual weight, not IBW. PIP and P_{plat} are high because of the large V_T. Reduce the volume to 500 mL. The current $\dot{V}_E$ of 9 L/min results in a normal acid–base status. With P_{plat} at 35 cm H_2O for a volume of 1.00 L, C_S is about 29 mL/cm H_2O. A V_T set at 0.5 L should produce a safer P_{plat}. The rate could be adjusted to 16 to 18 breaths/min, which would provide an adequate $\dot{V}_E$.
4. No change should be made at this time. If the ICP can be managed with medical intervention, the patient does not need to be hyperventilated.
5. 14 Fr (9 × 3 = 27/2 = 13.5); B. About 22 cm; C. −100 to −120 mm Hg.
6. B
7. C
8. C
9. A
10. C. Although a pMDI is more efficient, an SVN delivers more medication.
11. D
12. D
13. C
14. B
15. D
16. D
17. C
18. D

Chapter 13
Improving Oxygenation and Management of Acute Respiratory Distress Syndrome

1. B
2. B
3. C
4. C
5. A
6. D
7. C
8. B
9. D
10. O_2 transport is an acceptable answer; another is C_S. More than one answer is possible. You might want to discuss your response with an instructor or mentor.
11. B
12. C
13. D
14. B. The current recommendation is to use the deflation portion of a static P-V loop to establish PEEP. Setting PEEP 2 to 4 cm H_2O above the LIP on an SPV curve is also acceptable.
15. B
16. B
17. A
18. C
19. A
20. D
21. C

Chapter 14
Ventilator-Associated Pneumonia

1. B
2. C
3. C
4. A
5. B
6. C
7. A
8. B
9. C
10. B

Chapter 15
Sedatives, Analgesics, and Paralytics

1. B
2. C
3. D
4. C
5. A
6. C
7. D
8. C
9. D
10. The TOF technique allows the clinician to assess the extent of neuromuscular blockade during pharmacologically induced paralysis. With this technique, two electrodes are placed on the skin along a nerve path, often near a hand, foot, or facial nerve. An electrical current consisting of four impulses is applied to the peripheral nerve over 2 seconds; the muscle contractions (twitches) produced provide information about the level of paralysis.

Chapter 16
Extrapulmonary Effects of Mechanical Ventilation

1. D
2. C
3. B
4. A
5. Decreasing P_aO_2 values in patients with respiratory failure has been shown to cause a reduction in renal function and urine flow. A P_aO_2 below 40 mm Hg (severe hypoxemia) can dramatically interfere with normal renal function.
6. A
7. B
8. The amount of blood flowing to the brain is determined by the cerebral perfusion pressure (CPP), which is calculated by subtracting the intracranial pressure (ICP) from the mean systemic arterial blood pressure (MABP). Because

PPV (with or without PEEP) can reduce cardiac output and MABP, it is reasonable to assume that CPP would also decrease during PPV in these patients.

9. C
10. A

Chapter 17

Effects of Positive Pressure Ventilation on the Pulmonary System

1. D
2. C
3. The RT might consider performing a recruitment maneuver that would include recruitment to open the lung, derecruitment to establish the deflation point (UIP during derecruitment), and then rerecruiting the lungs. It might be argued that a static pressure—volume curve could establish an LIP, but current research supports the use of the deflation point (see Chapter 13).
4. In patients with high chest wall and abdominal pressures, the restriction of lung movement caused by a rigid thoracic compartment may provide some protection from elevating transpulmonary pressures. The same would be true if a patient were in the prone position. In this situation the increase in P_{plat} probably is permissible and will not cause VILI. Another option might be to allow the P_aCO_2 to increase to maintain a lower P_{plat}.
5. D
6. C
7. The temptation might be to sedate this patient for anxiety. Another concern might be the patient's heart, considering the ECG changes. Before any medications are administered, the patient must be assessed further. ABG analysis may reveal respiratory acidosis because all these findings are consistent with that condition.
8. A
9. D
10. D
11. D
12. C
13. B
14. A
15. Auto-PEEP (air trapping).

Chapter 18

Troubleshooting and Problem Solving

1. The breath sounds and the ET marking at the teeth suggest that the tube may have migrated into the right mainstem bronchus when the patient was moved. The RT should check the original tube position from the chart. The tube must be withdrawn about 3 to 4 cm, and the breath sounds must be reevaluated. A chest radiograph may also be required.
2. D
3. C
4. D
5. D
6. Tachypnea; nasal flaring; diaphoresis; use of accessory muscles; retraction of the suprasternal, supraclavicular, and intercostal spaces; paradoxical or abnormal movement of the thorax and abdomen; abnormal findings on auscultation; tachycardia; and possibly arrhythmias and hypotension. When severe distress is confirmed, the initial step is to disconnect the patient from the ventilator and carefully begin manual ventilation. If the patient's distress immediately resolves, the problem originates with the ventilator. If the distress does not resolve, the problem originates with the patient. Continue assessment until the problem is solved.
7. Intubation apparently resulted in insertion of the tube into the right mainstem bronchus. After 4 hours of ventilation at a high O_2 percentage, the left lung has collapsed, resulting in the dull percussion of this area, absence of breath sounds, and tracheal shifting. The tube must be repositioned, breath sounds must be reevaluated, and a chest radiograph is necessary to confirm tube placement.
8. The most likely cause is an air leak around the tube cuff. Inflate the cuff using MLT or minimum occluding volume and measure the cuff pressure. Listen again over the tracheal area to make sure the cuff is not leaking. Another possible cause is migration of the ET upward, above the vocal cords. Check the centimeter markings at the teeth.
9. D. The drop in volume on a pressure-limited machine could be caused by leaks, a decrease in CL, an increase in R_{aw}, or gastric distention from air swallowing from high pressures, resulting in decreased diaphragmatic excursion or a change in the patient's inspiratory effort. Perform a general visual assessment of the patient. Check around the face mask for leaks and listen to breath sounds.
10. Breath sounds suggest the presence of a pneumothorax, possibly a tension pneumothorax, in the right hemithorax. Contact a physician immediately for placement of a chest tube and continue to ventilate the patient manually with 100% O_2.
11. The graphics demonstrate that auto-PEEP is present (flow does not return to zero before a mandatory breath begins, similar to the findings in Fig. 18.8), that the machine is not responding to patient effort, and that inspiratory flow is set too low for patient demand (concave appearance of pressure—time curve, similar to Fig. 18.3). Increase inspiratory flow to shorten T_I and lengthen T_E to try to eliminate auto-PEEP and provide the needed flow demand. If this is unsuccessful, consider using external PEEP to make triggering easier for the patient. Perhaps VC-CMV is not the best mode for this patient. Consider other alternatives, such as changing the flow waveform.
12. B. A leak exists in the system.
13. The patient will need further assessment, including vital signs, pulse oximetry, ABG values, and a chest examination. More information about the ventilator settings is needed. An immediate concern is the high cuff pressure requirements. The tube size is too small for this patient; change to a larger tube.
14. It may be appropriate to do a recruitment maneuver on this patient because this condition is secondary, nonpulmonary ARDS. Resetting PEEP after recruitment may be indicated. Another option is to use permissive hypercapnia. Eliminate any contraindications to this treatment and assess the patient's tolerance of this ventilation technique. The patient may need sedation but may already be sedated because the I/E ratio is 1:1.
15. D. All the choices are possibly correct in this situation, depending on the circumstances.
    A. If the patient is on a face mask rather than connected with a TT or ET, heated humidity may not be necessary and, in

fact, may be uncomfortable if used. The heated humidifier may not have been turned, may not be set to a warm enough temperature, or may have been turned off the last time the equipment was changed.

B. The heater may not be functioning.

C. If the system had recently been changed, condensate would not have had a chance to build up in the circuit.

D. Depletion of the water supply to the humidifier reservoir could also result in lack of rain-out and inadequate humidification.

16. The patient's condition improved after treatment.
17. If a chest tube is in place, a leak may exist from the lung into the chest drainage system.
18. From the pressure graphic and the patient's description, the patient is apparently not getting sufficient inspiratory gas flow. The pressure setting seems inadequate considering the C_S and R_{aw}. The flow can be determined by rearranging the equation for calculating R_{aw}. (NOTE: During PSV and PCV, P_{set} may be used in place of P_{TA} to calculate the answer: $R_{aw} = P_{TA}/Flow$ [or $P_{set}/Flow$].) The maximum available flow is approximately equal to P_{set} divided by R_{aw} ($Flow = P_{set}/R_{aw}$). Flow = 12 cm H_2O/12 cm H_2O/L/s = approximately 1 L/s, or 60 L/min. This flow is apparently inadequate for the patient. The pressure needs to be increased to increase the available flow. (NOTE: If you got this answer correct, you're really beginning to use analytical thinking. This required the use of some advanced problem-solving skills and a modified equation that wasn't previously used. [Sometimes questions are used for teaching!])
19. The graphics show that T_I is short and flow is not returning to zero during inspiration. Increasing the T_I provides more time for P_{set} to reach the alveolar level and increase V_T delivery.
20. The patient's lung characteristics may have changed, and the current inspiratory pressure and/or flow are now too low, resulting in a longer T_I.
21. The machine is not set sensitive enough to the patient's inspiratory effort.
22. The patient has developed auto-PEEP since the setting change. A possible solution is to increase inspiratory gas flow to shorten T_I and increase T_E. Another possible solution is to return the ventilator to the previous settings and use a lower V_E to eliminate auto-PEEP.
23. Further information about the patient is needed before a solution can be found. Does the patient have ALI or ARDS? Is a recruitment maneuver indicated? Currently the PEEP is at zero. At the very least, PEEP apparently needs to be set above the P_{flex} point. It would be better, depending on the patient's problem, to use the deflection point on the pressure—volume curve after a recruitment maneuver.

Chapter 19

Basic Concepts of Noninvasive Positive Pressure Ventilation

1. B
2. C
3. D
4. B
5. A
6. C
7. B
8. A
9. B
10. A
11. C
12. D
13. C
14. D
15. C

Chapter 20

Weaning and Discontinuation From Mechanical Ventilation

1. D
2. D
3. B
4. C
5. B
6. None of these techniques has proved to be unquestionably better than the others. IMV may increase weaning time compared with PSV and T-piece weaning.
7. A cuff leak test should be done on mechanically ventilated adults who meet extubating criteria and are deemed high risk for postextubation stridor. For adults who have failed a cuff leak test but are otherwise ready for extubating, it is suggested administrating systemic steroid for at least 4 hours before extubating.
8. A
9. D
10. D
11. C
12. A
13. A
14. B
15. D
16. A
17. B
18. C
19. A
20. A

Chapter 21

Long-Term Ventilation

1. C
2. B
3. D
4. C
5. D
6. A
7. C
8. C
9. C
10. A
11. D
12. B
13. B
14. C
15. C
16. D
17. A
18. A
19. A
20. B

21. D
22. B
23. C
24. D
25. D
26. Severity of illness; longevity of illness; multiple medications (sedatives, analgesics, psychotropics, steroids); sleep disruption; delirium; anxiety; depression.
27. Setting a long T_I and setting a PEEP level. Possibly increasing V_T if a significant leak is noted with cuff deflation.
28. Obstruct a port with the finger to direct the flow of gas through the vocal cords.
29. Deflate the cuff.
30. Comatose/unconscious patient, foam cuff in place or cuff must remain inflated, increased and/or thick secretions, severe upper airway obstruction, increased R_{aw} and increased compliance that may cause air trapping (e.g., COPD), ET in place (not TT), reduced lung compliance, and laryngeal and pharyngeal dysfunction.

Chapter 22
Neonatal and Pediatric Mechanical Ventilation

1. C
2. D
3. C
4. B
5. B
6. A
7. D
8. B
9. A
10. D
11. C
12. C
13. B
14. B
15. A
16. A
17. C
18. B

Chapter 23
Special Techniques Used in Ventilatory Support

1. D
2. A
3. D
4. D
5. A
6. C
7. B
8. C
9. B
10. C
11. A
12. D
13. A
14. C
15. B
16. C
17. B
18. D
19. D
20. A

PART 2: CASE STUDY ANSWER KEY

Chapter 1: Basic Terms and Concepts of Mechanical Ventilation
Case Study 1.1

Determine Static Compliance (C_S) and Airway Resistance (R_{aw}). Compliance is 0.5/19, or 0.0263 L/cm H_2O (26.3 mL/cm H_2O), and R_{aw} is estimated to be 5 cm H_2O/L/s. The patient's compliance is very low, suggesting that some condition is making the lungs stiffer than normal and increasing her work of breathing. R_{aw} is low considering that the patient has an artificial airway in place.

Chapter 2: How Ventilators Work
Case Study 2.1

Ventilator Selection. Because the patient's room has only piped-in O_2, you would have to select an electrically powered ventilator with a built-in compressor. You could use an external 50 psig compressor, although some compressors are noisy. The availability of the O_2 source would allow you to provide O_2 as needed for the patient.

Chapter 3: How a Breath Is Delivered
Case Study 3.1

Patient Triggering

Problem 1: The machine is not sensitive enough to the patient's effort. The clinician should increase the ventilator sensitivity control.

Problem 2: The machine is too sensitive and is auto-triggering into inspiration. The clinician should reduce the sensitivity.

Case Study 3.2

Premature Breath Cycling. The ventilator pressure-cycled when the patient coughed and increased the peak airway pressure sensed by the ventilator during the breath. With newer ICU ventilators, excess pressures are less likely to occur when a patient coughs because these devices incorporate "floating" exhalation valves.

Chapter 4: Establishing the Need for Mechanical Ventilation
Case Study 4.1

Stroke Victim. The patient should be intubated to protect the airway and immediately evaluated for cerebral reperfusion therapy. The patient is typically admitted to the ICU after reperfusion therapy is completed for further evaluation (i.e., vital signs, S_pO_2 monitoring, ECG, breath sounds, ABG values, electrolytes, and neurological status). Mechanical ventilation may be necessary if the patient remains unconscious and unresponsive. In cases in which the patient is not receiving mechanical ventilation, aerosol therapy delivered through a Briggs T-adapter is required to prevent drying of secretions. Additional information regarding the treatment of stroke can be found at http://stroke.ahajournals.org/.

Case Study 4.2

Unexplained Acute Respiratory Failure. There are at least two possible solutions:

1. The problem may be drug related (e.g., narcotic overdose); try naloxone (Narcan).
2. Intubate and begin ventilation; assess further with vital signs, S_pO_2 monitoring, ECG, breath sounds, ABG values, electrolytes, blood alcohol levels, toxicology screening, and neurological status evaluation.

Case Study 4.3

Ventilation in Neuromuscular Disorders

Case 1: In spite of this patient's inability to maintain a tight seal around the mouthpiece, the measured parameters are acceptable. It is prudent, however, to use a mouth seal for this patient for subsequent measurements. Continue to monitor MIP and VC for at least every 8 hours. Request an evaluation of an anticholinesterase challenge. Keep the patient NPO and provide suctioning at the bedside until swallowing ability can be evaluated. Monitor S_pO_2 and/or ABG values if symptoms become worse.

Case 2: The history and symptoms suggest Guillain-Barré syndrome. Assessment of this patient should include measurement of MIP and VC along with arterial blood gas analysis. A reduced MIP (<15 cm H_2O) and VC (<10–15 mL/kg) plus the presence of acute ventilatory failure would be consistent with respiratory muscle weakness associated with Guillain-Barré syndrome. Mechanical ventilation is indicated. Consider the advantages and disadvantages of using noninvasive ventilation and oral versus nasal endotracheal intubation, or consider a tracheostomy. The RT may also suggest an anticholinesterase challenge to confirm the diagnosis.

Case Study 4.4

Asthma Case. The patient's condition appears to be improving. Continue drug therapy, adjusting the medication dosages and frequency as needed according to Expert Panel Review—3 recommendations. Continue to monitor the patient.

Chapter 5: Selecting the Ventilator and Mode

Case Study 5.1

What type of breath is it?

1. This is a patient-controlled machine breath. The V_T is delivered by the ventilator and the ventilator cycles the breath.
2. This is a spontaneous breath, and these variables describe CPAP. The patient is controlling the start time (trigger) and the V_T (cycle).

Case Study 5.2

Pressure Control (PC-CMV) or Volume Control Ventilation (VC-CMV)

1. When it is desirable to target P_aCO_2, volume control ventilation may be used because it can guarantee volume delivery and minute ventilation. With pressure control, ventilation changes in lung compliance or R_{aw} will result in changes in V_T, which can ultimately affect P_aCO_2.
2. Pressure control ventilation should be used when the goal is to avoid high pressures.

Chapter 6: Initial Ventilator Settings

Case Study 6.1

Minute Ventilation ($\dot{V}_E$) Needs. The ordered $\dot{V}_E$ is 500 mL × 12, or 6000 mL/min, or 6 L/min. The estimated $\dot{V}_E$ based on BSA (and metabolism) is 7 L/min. You might use VC-CMV with a set V_T of 500 mL and a minimum rate of 12 breaths/min. Assuming that the patient is not apneic and can also initiate spontaneous breaths, she could trigger additional breaths if needed to achieve a minute ventilation for the estimated metabolic needs.

Case Study 6.2

Minute Ventilation ($\dot{V}_E$), Tidal Volume (V_T), and Respiratory Rate. He has a BSA of 2.15 m^2. His IBW at 6 lb, or 72 inches, is 106 + 6(12) = 178 lb (81 kg). If a V_T of 7 mL/kg is used, the V_T is about 570 mL. V_E is 4 × 2.15 = 8.6 L/min, and the rate is (8.6 L/min)/(0.570 mL), or about 15 breaths/min.

Case Study 6.3

Inspiratory/Expiratory Ratio (I/E) and Flow. Her IBW is 145 lb (66 kg). (Use Fig. 6.1 and determine her BSA. Using actual weight and height BSA = 1.98 m^2.) Her $\dot{V}_E$ = BSA × 3.5 for a female. (Initial $\mathbf{V_E}$ = 1.98 × 3.5 = 6.93 L/min.) An initial V_T setting of 6 mL/kg IBW would be a reasonable starting point (V_T = 6 mL/kg × 66 kg = 396 mL or approximately 400 mL). An initial rate would be: $f = \dot{V}_E/V_T$, f = (6.93 L/min)/400 mL = (6930 mL/min)/400 mL = 17.3 breaths/min. Setting the rate at 17 breaths/min would be appropriate. A constant flow of 30 L/min or 0.5 L/s is set. Her $T_I = V_T$/flow (L/s), T_I = 400 mL/(0.5 L/s), T_I = 400 mL/(500 mL/s), T_I = 0.8 second), her T_E = TCT − T_I; TCT = 60 s/(17 breaths/min) = 3.5 seconds; (T_E = 3.5 s − 0.8 s; T_E = 2.7 second), and her I/E ratio would be 1:3.4 (T_I = 0.8 second; T_E = 2.7 second; $I/E = [T_I/T_I]/[T_E/T_I]$ = [0.8 s/0.8 s]/[2.7 s/0.8 s]). This ratio would allow ample time for exhalation. In fact, the flow rate might be reduced if high peak pressures were present. (See the following section on inspiratory flow and flow patterns for more information on flow rates and peak pressures.)

Case Study 6.4

Tidal Volume (V_T) During Pressure Control Continuous Mandatory Ventilation (PC-CMV). Assuming all other factors remain constant, the initial patient compliance (C) is 350 mL/12 cm H_2O = 29.1 mL/cm H_2O. To achieve a V_T of 550 mL, remember that C = ΔV/ΔP, and ΔP = ΔV/C; 550 mL/(29.1 mL/cm H_2O) equals a required pressure of 18.9 cm H_2O.

Case Study 6.5

Inspiratory Flow Termination in Pressure Support Ventilation (PSV)

Problem 1: The breath will end when flow drops to 12.5 L/min (i.e., 25% of peak flow).

Problem 2: The ventilator will continue to deliver flow until the maximum T_I limit is reached (usually 2–3 seconds, depending on the ventilator model).

Problem 3: The pressure–time graphic will show a sudden rise in the pressure at the end of the breath that is 2 to 3 cm H_2O higher than the preset pressure level. This rise in pressure will be detected by the ventilator and will pressure cycle the breath.

Case Study 6.6

Pressure-Regulated Volume Control (PRVC). Because less pressure is required to deliver the same volume, the patient's lung characteristics may be improving. This would mean a drop in R_{aw} or an increase in C_S. The therapist may want to assess the patient to confirm this finding.

Chapter 7: Final Considerations in Ventilator Setup

Case Study 7.1

Auto-PEEP and Triggering. The patient must generate −8 cm H_2O to bring P_{alv} to 0, plus −1 cm H_2O to trigger the ventilator. Total effort is −9 cm H_2O.

Case Study 7.2

Key Questions for ARDS Patient

1. The initial indications for ventilation in this patient are increased WOB, impending respiratory failure, refractory hypoxemia, and inability to tolerate mask CPAP.
2. In terms of precipitating factors, he sustained a fracture of the left femur, which may cause fat emboli, and chest injuries that resulted in a pneumothorax. Bilateral fluffy infiltrates are present on a chest radiograph, and he has refractory hypoxemia (low PaO_2 on a high F_IO_2).
3. He is 6 feet 2 inches tall and weighs 258 lb. BSA = 2.42 m^2; initial $\dot{V}_E$ = (4 × BSA) = 9.68 L/min. Because this patient has experienced trauma, his metabolic rate may be elevated, which must be kept in mind when he is ventilated. IBW = 106 + 6(14) = 190 lb (86.4 kg); using values of 4 to 8 mL/kg, a V_T range of 345 to 690 mL is appropriate. The initial V_T of 690 mL (8 mL/kg) can be used to establish an initial *f* and will allow for further evaluation of the V_T setting when P_{plat} and auto-PEEP can be measured. $f = \dot{V}_E/V_T$ = 9.68/0.690 = 14 breaths/min. Flow should be set at 100 L/min to keep T_I short, and a descending ramp waveform should be used. VC-IMV + PSV or VC-CMV modes are equally acceptable. Pressure-controlled ventilation can be used instead of volume-controlled ventilation. It would be helpful to start with VC-CMV to establish the pressure needed to achieve the desired volume in pressure ventilation. PC-IMV + PSV and PC-CMV are equally acceptable. An initial PEEP of ≥5 cm H_2O and an F_IO_2 of 1 should be set.

Case Study 7.3

Troubleshooting: The Pulse Oximeter. Patients with CHF often demonstrate peripheral vasoconstriction, thus limiting blood flow to their extremities. The pulse oximeter cannot register a value unless it detects a pulse (see Chapter 10). The respiratory therapist could try warming the patient's hand or using another site, such as the other hand or a toe. Testing the function of the pulse oximeter to see whether it is working properly is also a good idea.

Chapter 8: Initial Patient Assessment

Case Study 8.1

The Importance of Documentation. The legal inference is that if treatment is not documented, it is not done. The therapist must prove that care was provided despite a lack of documentation. He argued that he did not want to repeat the same information over and over and that he had "charted by exception." Fearing that a jury might not believe the therapist, the hospital settled the case out of court.

Case Study 8.2

Circuit Disconnect. The low-pressure alarm did not activate for several reasons. First, it may have been set too low and was not sensitive enough to the drop in pressure. Second, the bedding may have been occluding the Y-connector, thus keeping the pressure in the circuit above the low-pressure alarm level. The low-pressure alarm and the low V_T alarm must be set appropriately to avoid these types of situations. With a low volume returning to the exhalation side of the ventilator, the drop in V_T would have been detected and the low V_T alarm would have been activated. This would have alerted the respiratory therapist to a problem before the patient's monitor indicated tachycardia.

Case Study 8.3

Cuff Inflation Techniques. The MLT was used to set the cuff pressure so that a small leak existed at 13:00. This can be noted by the difference of 20 mL between the set volume (520 mL) and the measure exhaled volume (500 mL). Several hours later (the set volume is the same), the patient's condition improves such that less pressure is required to deliver the same volume. With a lower ventilating pressure, a leak no longer exists around the cuff. Note that the cuff pressure (20 mm Hg) is equal to about 27 cm H_2O. This is enough cuff pressure to prevent a leak when the ventilating pressure is only 23 cm H_2O. The MLT could be repeated if maintaining a leak is desirable for this patient.

Case Study 8.4

Patient Assessment Cases

Problem 1: Findings suggest that the patient has a respiratory infection. A chest radiograph and laboratory studies, including a white blood cell count, might confirm this diagnosis. The low-pitched rattles heard on auscultation (rhonchi) indicate secretions in the large airways. The patient may need to be suctioned. Also, a culture and sensitivity test of a sputum sample may be in order. Antibiotics may be indicated.

Problem 2: Pneumothorax on the left side.

Problem 3: Right mainstem intubation.

Case Study 8.5

Evaluating C_S and R_{aw} During Mechanical Ventilation

Problem 1: The increase in PIP and P_{TA} while P_{plat} remains constant indicates a change in R_{aw}. Listen to the breath sounds and try to determine the cause of the change (e.g., secretions, kink in the ET tube, bronchospasm).

Problem 2: The decrease in V_T indicates a change in lung characteristics. Listen to the breath sounds and try to determine whether R_{aw} may have increased (i.e., presence of low-pitched rattling breath sounds [rhonchi] or wheezing) or whether the tube has become progressively occluded or is obstructed in some way. Crackles or changes in the percussion note may indicate a change in lung parenchyma that might reduce compliance (e.g., the development of pneumonia) or a completely obstructed airway that is causing the distal portion of the lung to collapse. Try to determine the cause of the change and correct it. Increase the set pressure to maintain ventilation if necessary.

Chapter 9: Ventilator Graphics

Case Study 9.1

The two different breath types can be easily distinguishable by analyzing the pressure and flow scalars (see Fig. 9.11).

Case Study 9.2

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.

Chapter 10: Assessment of Respiratory Function

Case Study 10.1

Causes of Cyanosis. The patient had an adverse reaction to the benzocaine and developed methemoglobinemia, which could be verified by performing CO-oximetry. CO-oximetry would allow you to directly measure methemoglobin levels in the patient's blood. The treatment of acute methemoglobinemia is intravenous administration of methylene blue.

Case Study 10.2

Capnography During Intubation. Capnography is often used to assess the ET placement. In this case the ET was placed in the esophagus, thus preventing the detection of any exhaled CO_2. Listening to breath sounds and reviewing the patient's chest radiographs can confirm this finding.

Case Study 10.3

Dead Space Ventilation. The application of +10 cm H_2O of PEEP may have caused a reduction in pulmonary perfusion resulting in an increase in dead space ventilation, which is evidenced by the shifting of the $SBCO_2$ curve to the right and the reduction in PCO_2. The reduction in S_pO_2 is consistent with an altered $\dot{V}/\dot{Q}$ ratio, which would be associated with compression of the pulmonary capillaries by an excessive amount of $PEEP_E$ being administered.

Chapter 11: Hemodynamic Monitoring

Case Study 11.1

Evaluation of Pressure Tracing

1. The catheter is located in a pulmonary artery.
2. Systolic and diastolic pressures are approximately 40/25 mm Hg. These values are measured during exhalation.

Case Study 11.2

Cardiac Index and Stroke Index

C.I. $= \dot{Q}/BSA = (3\ L/min)/(1.7\ m^2) = 1.76\ L/min/m^2$

S.I. $= SV/BSA$; $SV = \dot{Q}/HR = 3\ L/min/110\ beats/min = 0.027$ L/beat or 27 mL/beat. S.I. $= SV/BSA = 27\ mL/beat/1.76\ m^2 = 15.34\ mL/beat/m^2$. Cardiac index and stroke index are both lower than normal.

Case Study 11.3

Application of the Fick Principle. At 13:00 $\dot{Q} = (350/[20 - 14] \times 10) = 4.7$ L/min. At 15:00 $\dot{Q}$ is $(350/[20 - 12] \times 10) = 4.4$ L/min. The decrease in CVO_2 is associated with a decrease in cardiac output.

Case Study 11.4

Stroke Work

Left ventricular stroke work (LSW) = MAP × SV × 0.00136. And LSWI = LSW/BSA.

Before the medication LSW $= 80 \times 60 \times 0.00136 = 6.53$ kg-m.

LSWI $= 6.53\ kg\text{-}m/1.5\ m^2 = 4.35\ kg\text{-}m/m^2$.

After the medication LSW $= 100 \times 70 \times 0.00136 = 9.52$ kg-m.

LSWI $= 9.52\ kg\text{-}m/1.5\ m^2 = 6.35\ kg\text{-}m/m^2$.

Case Study 11.5

Hemodynamic Monitoring: After Open-Heart Surgery. Calculate $C(a-V)O_2$ for before and after treatment: (ignore the dissolved portion).

Before: $C_aO_2 = 0.90 \times 13 \times 1.34 = 15.7$ vol%; $CVO_2 = 0.75 \times 13 \times 1.34 = 13.1$ vol%; $C(a-V)O_2 = 2.6$ vol%. Calculate cardiac output before: $\dot{Q} = (V \times 100 = (250/2.6) \times 100 = 9620$ mL/min or 9.62 L/min.

After: $C_aO_2 = 0.98 \times 13 \times 1.34 = 17.1$ vol%; $Cv_2 = 0.65 \times 13 \times 1.34 = 11.3$ vol%; $C(a-V)O_2 = 5.8$ vol%.

Calculate cardiac output after: $\dot{Q} = (V \times 100 = (230/5.8) \times 100 = 3956$ mL/min or 3.96 L/min.

Cardiac output has dropped, and the $C(a-V)O_2$ has increased as a result.

Case Study 11.6

Hemodynamic Monitoring: Chest Injury. It was determined that the pulmonary artery was torn during surgery and the patient's liver had been injured by a retractor. The low hemoglobin is one indication of internal bleeding. The patient's hemodynamic status appears to be good. The only value that was significantly out of the normal range was the pulmonary artery pressure (PAP), which was probably elevated because of the medications the patient was receiving.

Case Study 11.7

ICU and Hemodynamic Assessment. This case describes a patient with a pulmonary embolism, which is suggested by the refractory hypoxemia present. The fact that the PAP pressures are elevated with a normal PAOP is representative of a pulmonary condition as opposed to left heart failure. The CVP is within normal limits, indicating that the condition is acute because the right side of the heart has not yet been affected by the elevated pulmonary artery pressures.

Chapter 12: Methods to Improve Ventilation in Patient–Ventilator Management

Case Study 12.1

Hyperventilation. The flow–time scalar shows that flow does not return to zero during exhalation before another mandatory breath occurs. Auto-PEEP (air trapping) is present. It is important to check ventilating pressures and keep P_{plat} below 30 cm H_2O to

prevent lung injury. The physician should be notified that, in an effort to normalize the pH, the high $\dot{V}_E$ is causing auto-PEEP.

Case Study 12.2

Assessment During Suctioning. A sudden tachycardia is a possible complication of suctioning. The respiratory therapist should immediately stop the procedure, provide O_2 (100%), and ensure that the patient is receiving adequate ventilation, preferably using the ventilator to do so.

Case Study 12.3

Evaluation of Bronchodilator Therapy. Yes, the PIP decreased, the P_{TA} decreased by nearly 50%, and the PEFR increased by more than 50%.

Case Study 12.4

Evaluating Fluid Status. The most likely problem is low fluid volume. Fluid replacement is recommended.

Chapter 13: Improving Oxygenation and Management of Acute Respiratory Distress Syndrome

Case Study 13.1

Myasthenia Gravis. The patient has respiratory acidosis. The P_aO_2 indicates moderate hypoxemia. A common reaction by clinicians in this situation is to increase the F_IO_2. However, the cause of the hypoxemia is the elevated CO_2. An increase in P_aCO_2 of 1 mm Hg will reduce the P_aO_2 by 1.25 mm Hg. The P_aCO_2 is about 40 mm Hg above normal; therefore the P_aO_2 will be about 50 mm Hg below its actual value. The most appropriate way to increase the P_aO_2 is to increase ventilation.

Case Study 13.2

Changing F_IO_2

$$\text{Desired } F_IO_2 = (60 \times 0.75)/40 = 1.13$$

No, this is not possible. You cannot give more than 100% O_2. Along with increasing the F_IO_2 to 1, another method of improving oxygenation is to use PEEP.

Case Study 13.3

Problem Solving: Infant CPAP. This is a common problem encountered when using CPAP in the treatment of infants. The flow from the CPAP device is leaking out of the infant's mouth because the infant is crying. The CPAP levels cannot be maintained. Once the infant stops crying, the problem will correct itself.

Case Study 13.4

Selecting Optimum PEEP. P_aO_2 progressively increases as expected with an increase in PEEP and FRC. PVO_2 is low initially (about 50% saturation, assuming that pH is close to normal), progressively improves to 38 mm Hg, and then declines. At the same setting (20 cm H_2O PEEP), BP falls. PVO_2 declines because of a drop in cardiac output (assuming that $\dot{V}O_2$ is constant). The optimum PEEP level for the patient at this time is 15 cm H_2O. The next step is to reduce the F_IO_2.

Case Study 13.5

Changing Patient Position. The most likely cause is a $\dot{V}/\dot{Q}$ mismatch caused by rotation of the affected lung in the dependent position. With unilateral lung disease, it is best to position the good lung down, in the dependent position. Another possible problem is thromboembolism; repositioning of patients sometimes causes clots to move. A third possible problem is a compromised airway; the airway would need to be checked for proper function.

Chapter 14: Ventilator-Associated Pneumonia

Case Study 14.1

Patient Case—VAP. His CPIS is 7 and his condition does warrant initiation of a broad-range antibiotic.

Case Study 14.2

Patient Case—Methicillin-Resistant *S. aureus*. An appropriate antibiotic regimen would include linezolid (600 mg every 12 hours) or vancomycin.

Chapter 15: Sedatives, Analgesics, and Paralytics

Case Study 15.1

Patient Case—Discontinuing Lorazepam. Long-term use (longer than just a few months) of lorazepam can lead to physical dependence. The most common side effects that can occur when this patient suddenly stops taking his medication include nausea, vomiting, agitation, and insomnia. Other possible side effects include tremors, muscle cramping and spasms, and seizures.

Case Study 15.2

Patient Case—Agitated Patient. Fentanyl, which is a synthetic opioid, would be an effective choice for treating this patient. Fentanyl possesses analgesic and sedative properties. Morphine would not normally be used with this type of patient because of her unstable hemodynamic status.

Case Study 15.3

Patient Case—Asynchrony. Asynchrony cannot be corrected through ventilator adjustment; therefore it is appropriate to use a sedative and, if necessary, a paralyzing agent to reduce patient-ventilator asynchrony.

Case Study 15.4

Patient Case—Neuromuscular Blocking Agent. The NMBA that would be appropriate for this patient would be a depolarizing agent such as succinylcholine. It is indicated in patients who are hemodynamically stable. It can cause increases in intracranial pressure and should not be used in patients with cerebral edema and head trauma. Succinylcholine has been widely used for cases of emergency intubation because of its relatively low cost, rapid onset of action, and short duration of action.

Chapter 16: Extrapulmonary Effects of Mechanical Ventilation

Case Study 16.1

The Effects of Ventilator Changes on Blood Pressure. The increases in V_T and respiratory rate have resulted in a substantial increase in the mean airway pressure, which in turn has caused the patient's BP to drop. With this large a V_T and this high an RR, auto-PEEP may also be contributing to the rising peak and mean airway pressures. The RT should determine whether the V_T setting is appropriate for this patient (maximum approximately 8 mL/kg IBW). The RT also might recommend the use of VC-IMV or PC-

IMV, rather than VC-CMV, and reduce the mandatory rate. PSV might be added to support spontaneous breaths.

Chapter 17: Effects of Positive Pressure Ventilation on the Pulmonary System

Case Study 17.1

Peak Pressure Alarm Activating. Physical findings indicate the presence of a right-sided pneumothorax. A physician should be contacted immediately for an order for a chest radiograph and to begin treatment. The respiratory therapist should stay with the patient and make sure the pneumothorax does not become a tension pneumothorax. Appropriate emergency equipment (e.g., an emergency resuscitation cart) should be kept close at hand. Depending on the circumstances, the respiratory therapist may need to provide manual ventilation for the patient until treatment can be administered.

Case Study 17.2

Patient Case—Acute Pancreatitis. The crackles in the basilar and posterior areas may indicate atelectasis and the opening and closing of alveoli in dependent areas. An increase in PEEP is indicated, and a recruitment maneuver might also be considered (see Chapter 13).

Case Study 17.3

Appropriate Ventilator Changes. The ABG values after 7 days of PPV are normal. However, this patient's baseline ABG values suggest chronic CO_2 retention. The patient has been hyperventilated with the ventilator, and the kidneys have reduced the bicarbonate level to normal. When the mandatory rate is reduced for weaning, the patient's P_aCO_2 rises, stimulating spontaneous ventilation. Unfortunately, the patient cannot maintain a normal P_aCO_2 and pH, as suggested by the high spontaneous rate. To correct the problem, the patient's mandatory rate must be reduced gradually until normal baseline ABG values are restored (i.e., pH = 7.38, P_aCO_2 = 51 mm Hg, P_aO_2 = 58 mm Hg, HCO_3- = 29 mEq/L). Appropriate PSV for spontaneous breaths should also be provided.

Case Study 17.4

Difficulty Triggering in a Patient With COPD. The new $\dot{V}_E$ is 11.7 L/min. The increase in $\dot{V}_E$ resulted in auto-PEEP, which caused the rise in PIP and the transient drop in exhaled V_T that occurred after the change. Also, the patient is unable to trigger the ventilator, another possible indication of air trapping.

Chapter 18: Troubleshooting and Problem Solving

Case Study 18.1

Evaluating Severe Respiratory Distress in a Ventilated Patient. The patency of the airway can be used to rule out upper airway obstruction; auscultation of the patient's breath sounds can be used to rule out any sudden change in the patient's lung condition, such as the presence of secretions or the occurrence of a pneumothorax. The sudden O_2 desaturation that occurs simultaneously with a drop in end-tidal CO_2 suggests the possibility of a pulmonary embolus. This cannot be confirmed easily and requires further radiographic evaluation.

In this case a pulmonary embolism was later confirmed. Aside from attempts to increase oxygenation, little can be done for this problem through ventilator management and immediate medical intervention is required.

Case Study 18.2

Evaluating Peak Inspiratory Pressure (PIP) and Plateau Pressure (P_{plat}) in Volume-Controlled (VC) Ventilation. On the basis of pressure findings, C_L is progressively decreasing. PIP and P_{plat} are increasing while the difference between them (P_{TA}) remains relatively constant, which suggests that R_{aw} has not changed. ARDS is a possible diagnosis based on the physical findings, chest radiograph, and patient's history.

Case Study 18.3

Evaluating PIP and Volume in Pressure Control Ventilation. This patient, with a history of asthma, may be experiencing increased bronchial constriction and increased secretions. The drop in volume may be associated with increased R_{aw}. The flow—volume loop indicates increased R_{aw}, and the wheezing supports this finding. Activation of the alarms resulted from two different causes. The high-pressure alarm was activated by the patient's coughing and forceful exhalations; the low-volume alarm was activated by the reduction in volume delivery associated with increased R_{aw} during pressure ventilation. The patient most likely needs bronchodilator therapy and possibly intravenous corticosteroids and suctioning.

Case Study 18.4

Problem Solving Using Ventilator Graphics. The flow—time and volume—time curves in Fig. 18.7 show a greater inspired volume than exhaled volume, which indicates a leak in the system.

Case Study 18.5

Evaluating a Ventilator Problem. In this situation the exhalation valve was malfunctioning. A gas leak apparently occurred through the valve, causing the baseline pressure to begin dropping. The ventilator increased gas flow delivery to try to compensate for the drop in PEEP. This moved air through the exhalation valve, which was measured by the flow transducer, resulting in an upward swing of the volume curve at the end of exhalation. The problem was corrected when the expiratory valve was changed. Perhaps you can think of another possible cause of this situation.

Chapter 19: Basic Concepts of Noninvasive Positive Pressure Ventilation

Case Study 19.1

Patient Selection for NIV. Oxygenation and respiratory status appear to be acceptable, but close assessment reveals several risk factors that may compromise the patient's safety. The patient's ability to cough and swallow has deteriorated, reflecting her inability to protect the airway adequately. This places her at a very high risk for aspiration. The patient also has become more agitated and confused in the past hour, which could indicate worsening hypercarbia. The correct action at this time would be to intubate the patient and initiate invasive ventilation. Delaying this process would cause further clinical deterioration and increase morbidity and mortality.

Case Study 19.2

Monitoring and Adjusting NIV. The symptoms of dyspnea, agitation, and increased respiratory rate reveal inadequate clinical improvement from NIV. Two things need to be considered at this time. Currently, the patient's average exhaled V_T is only 3 to 4 mL/kg of the patient's body weight; this contributes to his rapid respiratory rate and may promote auto-PEEP. The practitioner should attempt to increase the patient's exhaled V_T to 6 to 8 mL/kg by increasing IPAP. The use of a full-face mask may increase the potential for CO_2 rebreathing, especially if EPAP levels are not set adequately. Increasing EPAP levels will increase the flow of gas to the mask during exhalation and reduce the potential for rebreathing of CO_2. Increasing EPAP also may reduce WOB. However, if EPAP is increased without increasing IPAP, the gradient between IPAP and EPAP (or the pressure support level) will decrease, resulting in a lower delivered V_T. Therefore if EPAP is increased, IPAP also must be increased to ensure adequate pressure support for greater V_T delivery to the patient. (NOTE: If auto-PEEP is present, elevating EPAP may also make it easier for the patient to trigger a breath [see Chapter 6].)

Case Study 19.3

Common Complications of NIV. Improvement in gas exchange and other symptoms related to chronic hypoventilation may take several weeks to occur for those who use NIV only intermittently. Patients who can tolerate NIV for at least 4 to 6 hours in each 24-hour period are most likely to show improvement in symptoms. This patient's lack of compliance and intolerance of NIV is most likely responsible for his poor physiological improvement. Nasal dryness and congestion are common complications of NIV, and every effort should be made to minimize its occurrence. A room-temperature humidifier attached to the CPAP machine adds moisture and often is helpful for patients with nasal drying or congestion. Cold, dry air coming directly from the CPAP mask may increase nasal resistance by means of increased nasal congestion. Heated humidification is more expensive but may be attempted in particularly difficult cases. Nasal irritation and congestion may be treated with nasal sprays containing steroids, ipratropium, or antihistamines. Patients with persistent difficulties may benefit from referral to an ear, nose, and throat specialist.

Treatment with decongestants, inhaled (nasal) steroids, or cromolyn sodium; humidification; and in some cases nasal surgery can control nasal symptoms in most patients. However, a few will continue to find nasal CPAP uncomfortable because of high nasal resistance.

Chapter 20: Weaning and Discontinuation From Mechanical Ventilation

Case Study 20.1

Evaluation of Weaning Attempt. The patient's spontaneous rate has risen progressively as the spontaneous V_T has decreased. Without any further information, these two findings strongly suggest that the patient's WOB has dramatically increased as the mandatory IMV rate has decreased. To assist the patient, return the IMV rate to a higher level (e.g., 4 breaths/min or more) and add PSV to support the patient's spontaneous breathing efforts. The use of low levels of PEEP/CPAP (3–5 cm H_2O) is also appropriate. Furthermore, evening is approaching. The patient probably needs to rest for the night, which means a return to full ventilatory support.

Case Study 20.2

Calculation of Rapid Shallow Breathing Index (RSBI)

Patient 1: The index is 10/0.4, or 25, which indicates readiness to wean from mechanical ventilation.

Patient 2: The index is 30/0.25, or 120; therefore weaning is not recommended.

Case Study 20.3

Failed Weaning Attempt. One possible cause relates to cardiac function. On the basis of the patient's history of a myocardial infarction, he may be experiencing increased left ventricular preload and a shift in blood volume to the central veins, which may lead to cardiogenic pulmonary edema, as demonstrated by the rise in wedge pressure, despite the lack of dysrhythmias. One solution to this problem might be the administration of diuretics in an effort to reduce the fluid volume and treat the cardiac problem.

Chapter 21: Long-Term Ventilation

Case Study 21.1

Patient Case—Difficulty Weaning. It would be appropriate to assess the patient further to determine why he is having difficulty weaning from the ventilator and whether he meets the criteria for discharge. It may be related to his chronic condition (ALS) or his previous MIs left him unable to support ventilation. In either case, he may require slower weaning attempts. If his condition is medically stable and meets the criteria for discharge, transfer to an intermediate or long-term care facility probably would be the best course.

Case Study 21.2

Patient Case—Communication Difficulty. The respiratory therapist might consider a speaking valve. This might help the patient phonate without requiring a change of the tracheostomy tube. Careful evaluation of the patient's ability to use the valve would be required to ensure that the apparatus is beneficial considering the level of ventilatory support.

Chapter 22: Neonatal and Pediatric Mechanical Ventilation

Case Study 22.1

Assessment and Treatment of a Newborn. Clinical assessment of this patient should include prompt measurement of vital signs and S_pO_2. Clinical signs call for nasal CPAP or surfactant administration. The patient should be attached to an electrocardiograph–respiratory rate monitor, and a chest radiograph should be taken immediately. The typical approach for a patient this size is to first start with nasal CPAP, and if the infant shows ongoing signs of respiratory distress and increasing oxygenation, then a blood gas should be obtained. Additionally, he may benefit from a dose of surfactant with immediate extubation to CPAP. ABG analysis should be considered. On the basis of the history and clinical presentation, respiratory distress syndrome is the most likely diagnosis and should be ruled out first.

Case Study 22.2

Adjustments to Home Therapy. The RT primarily faces the challenge of helping clear and manage this patient's secretions. The patient may have developed pneumonia. However, the chest radiograph showed extensive atelectasis in the right middle and lower lobes. Atelectasis was also seen on the left at the lingular area. Many patients with neuromuscular disorders can be

ventilated quite effectively with noninvasive devices. However, secretions, which can obstruct and cause plugging of the airways, persistently affect some patients.

The RT immediately recruited the parents to help develop a home care plan that would provide cough assist and chest physical therapy. On the RT's recommendation, the patient was admitted to the hospital's intermediate care unit. The clinical staff, along with the parents, introduced the patient to the cough-assist device, which had never been used with this patient. The parents were taught to perform chest physical therapy. Although some nasotracheal suctioning was necessary on the first day of admission, this was soon replaced with frequent use of the cough-assist device, followed by suctioning of the hypopharynx with a Yankauer suction tube. The RT also evaluated the patient on a BiPAP ventilator and determined that both IPAP and EPAP should be increased.

Case Study 22.3

Patient Case—Acute Status Asthmaticus. Despite the helium/oxygen mixture, R_{aw} for this patient is probably quite high. The RT should evaluate pressure and flow scalar waveforms. It may be that with the T_I of 0.9 second, the set inspiratory pressure cannot be reached before the time limit. Flow, which normally tapers to zero in pressure control breaths, may be terminated prematurely. Some clinicians call this situation *flow chop*. The RT should recommend an increase in the T_I and adjustment of the inspiratory time, which would probably achieve a higher V_T. Also, the RT should monitor the patient's flow during the expiratory phase of each breath. It might be necessary to reduce the ventilator rate, providing additional expiratory time to allow expiratory flow to return to baseline.

Case Study 22.4

Recommending Changes in Ventilator Settings. Although many patients treated for pneumonia respond well to the pressure control/CMV, some develop areas of atelectasis. This may be a result of secretions and airway plugging, or it may be associated with a progressive loss of pulmonary compliance secondary to the acute pneumonitis. An early sign of progressive lung collapse is tachypnea with deteriorating V_T on this mode of ventilation. Although the patient's $\dot{V}_E$ has not dropped, oxygenation has been affected. Some might consider keeping the patient on this mode and increasing the inspiratory pressure and PEEP settings. Because the clinical condition is worsening, the patient should be switched from a pressure control mode to a volume control mode. The change to a volume control mode of ventilation will restore lung volumes, particularly FRC and V_T. It is becoming increasingly more common to use volume control or dual-control modes of ventilation in patients to prevent lung collapse that may not be able to be maintained during pressure control.

Case Study 22.5

Evaluation of PRVC Dual-Control Mode. In this example the patient is not tolerating PRVC because of a leaky ET. In some cases, dual-control modes should not be advocated with leaky ETs. In this particular case, the ventilator servo-controls or adjusts pressure based on a measured inspiratory V_T. However, because there is a large leak present, the ventilator is initially sensing a V_T that is greater than the preset volume, improved compliance, and the ventilator weans the inspiratory pressure. A larger ET leak during PRVC can result in erroneous weaning of the inspiratory pressure, placing the patient at greater risk for hypoventilation and lung collapse. Conversely, dual-control modes that regulate pressure based on exhaled V_T may overexpand lung units. Patients who have large ET leaks may be better supported using pressure control modes that will hold a constant pressure in the lungs without the risk for inappropriate weaning of the inspiratory pressure.

Case Study 22.6

Interpretation and Response to Monitored Data. The physician and RCP should continue to monitor the $\dot{V}CO_2$. With an increase in PEEP, additional lung units probably have been recruited, leading to an increase in CO_2 elimination at the airway. In this situation the $\dot{V}CO_2$ usually rises to a plateau and then drops slightly. If an inappropriate amount of PEEP had been added, the $\dot{V}CO_2$ most likely would have decreased because of an increase in dead space.

Case Study 22.7

Patient Case—Acute Respiratory Distress Syndrome Managed With HFO. The RT should suggest lowering the mean airway pressure setting. Significant recruitment of lung units may have taken place over the short time the patient has been on the oscillator. The mean airway pressure may be so high at this point that overdistention could be contributing to dead space ventilation. In this situation, reducing the mean airway pressure for a short time is often a good idea to see whether oxygenation improves.

Case Study 22.8

Determining Appropriateness of Nitric Oxide Therapy. RTs often are called upon to administer nitric oxide to patients whose condition is refractory to O_2 therapy. It is essential that the underlying cause of the hypoxemia be identified before iNO is given. In some cases, administration of iNO could worsen the patient's condition. Almost all infants and children who are candidates for iNO should undergo echocardiography (ECHO). In this infant's case, the RT asked whether ECHO had been done to confirm a diagnosis of PPHN. ECHO was ordered, and a critical coarctation of the aorta was identified. It was also determined that the aortic arch was moderately hypoplastic. Although the infant's condition was serious, he was relatively hemodynamically stable because of right-to-left shunting, presumably through the PDA and patent foramen ovale. If nitric oxide had been given, pulmonary vasodilation would have occurred, reducing the shunting of blood from the pulmonary to the systemic circulation. The result most likely would have been profound systemic hypotension.

Chapter 23: Special Techniques Used in Ventilatory Support

Case Study 23.1

Patient Assessment During HFOV. The most likely cause is a pneumothorax. Given the tracheal shift, it may be a tension pneumothorax.

Case Study 23.2

Calculating Gas Flows During Heliox Therapy. The correction factor for 70:30 is 1.6. At a flow of 10 L/min, the actual flow will be 1.6 × 10, or 16 L/min.

PART 3: CRITICAL CARE CONCEPTS ANSWER KEY

Chapter 1: Basic Terms and Concepts of Mechanical Ventilation

Critical Care Concept 1.1

Calculate Pressure. Remember that if C = ΔV/ΔP, then ΔP is ΔV/C. With a volume of 0.5 L and a compliance of 0.1 L/cm H_2O, pressure = 5 L/0.1 cm H_2O, or 5 cm H_2O. A P_{alv} change of 5 cm H_2O would be required to achieve a 0.5 L V_T in a person with normal C_L.

Chapter 2: How Ventilators Work

Critical Care Concept 2.1

Open Loop or Closed Loop. This is a closed-loop system. The ventilator is providing a specific F_IO_2 and monitors S_pO_2. The ventilator can detect changes in S_pO_2 and change the F_IO_2 setting. Whether this is a good idea is a matter of opinion. It can be argued that this would provide a safeguard for patients who suddenly become hypoxemic. It could also be argued that O_2 saturation monitors are not reliable enough and could give erroneous readings, resulting in an inappropriate ventilator response.

Chapter 5: Selecting the Ventilator and the Mode

Critical Care Concept 5.1

Volume-Controlled Breaths With Changing Lung Characteristics

1. About 1 second and it does not change among *A, B,* or *C.*
2. This is a constant flow waveform, also called a *rectangular* or *square* waveform.
3. The V_T is about 500 mL.
4. PIP at *A* is about 14 cm H_2O, at *B* it is about 25 cm H_2O, and at *C* it is about 12 cm H_2O.
5. Lung compliance decreases with pneumonia, ARDS, pulmonary fibrosis, or pulmonary scarring, such as can occur with lung cancer. Compliance also decreases with pneumothorax and pleural effusions. Changes in the abdominal wall, such as occur with ascites, can elevate the diaphragm and reduce compliance. Placing a patient in the prone position reduces thoracic compliance. Burns and surgical incisions in the chest wall reduce thoracic compliance.
6. If compliance remains the same but airway resistance increases, more pressure will be required to deliver gas flow. PIP will increase.

Critical Care Concept 5.2

Pressure-Controlled Breaths With Changing Lung Characteristics

1. The pressure curve is constant.
2. The waveform during inspiration is basically a descending (decelerating) ramp. It might also be called an *exponential descending flow waveform.*
3. The flow–time curve in *A* drops to zero just at the end of inspiration. The flow curve in *C* drops to zero before the end of inspiration.
4. The volume curve in *C* has a short plateau at the top that begins when flow drops to zero during inspiration and ends when exhalation starts. It is flat because the volume is not changing. Because the flow is zero during this time interval, no more volume is going into the lungs.
5. The volume delivery at *B* is higher than at *A* because the lungs in *B* are more compliant than the lungs in *A.*

Chapter 6: Initial Ventilator Settings

Critical Care Concept 6.1

Tidal Volume (V_T) and Ideal Body Weight (IBW). The lowest V_T would be 390 mL (6 mL/kg × 65 kg = 390 mL) and the highest V_T would be 520 mL (8 mL/kg × 65 kg = 520 mL). A woman of the same height (5 ft, 6 in) would have an IBW of 61 kg. The female's lowest V_T would be 366 mL (6 mL/kg × 61 kg = 366 mL), and the highest V_T would be 488 mL (8 mL/kg × 61 kg = 488 mL).

Critical Care Concept 6.2

Inspiratory Flow in a Time-Cycled Ventilator. TCT = 5 seconds (60 s/12 breaths = 5 seconds for each breath); I/E ratio = 1:4; T_I = 5 s/5 = 1 second; T_E = 4 seconds. Inspiration must be delivered in 1 second. Flow = Volume/Time = 0.5 L/1 s. Convert time to a minute; flow = (0.5 L)/s = (0.5 L × 60 s)/(1 s × 1 min) = 30 L/min.

Chapter 7: Final Considerations in Ventilator Setup

Critical Care Concept 7.1

The absolute humidity of a gas that is 100% saturated at normal body temperature is 44 mg/L. The humidifier provides 37 mg/L and the patient needs 44 mg/L. The humidity deficit is 7mg/L. The relative humidity (RH) deficit decreases; RH = (Absolute/Maximum capacity) × 100 = (37 mg/L)/(44 mg/L) = 85.3%.

Chapter 11: Hemodynamic Monitoring

Critical Care Concept 11.1

Fick Principle

$$\dot{Q} = \dot{V}O2/(C_aO_2 - C\bar{V}O_2)$$

$$\dot{Q} = 300 \text{ mL/min}/(180 \text{ mL/L of whole blood} - 130 \text{ mL/L of whole blood})$$

$$\dot{Q} = 6 \text{ L/min}$$

Chapter 16: Extrapulmonary Effects of Mechanical Ventilation

Critical Care Concept 16.1

Calculating Cardiac Transmural Pressure

Problem 1: First, convert to similar units: 1 mm Hg = 1.36 cm H_2O; therefore 150 mm Hg = 204 cm H_2O. $P_{TM} = P_{inside} - P_{outside}$ = (204 cm H_2) − (+ 10 cm H_2O) = 194 cm H_2O

Problem 2: P_{TM} = (204 cm H_2) − (−10 cm H_2O) = 214 cm H_2O

The higher P_{TM} value during spontaneous breathing increased the LV afterload compared with PPV.

APPENDIX B

Review of Abnormal Physiological Processes

MISMATCHING OF PULMONARY PERFUSION AND VENTILATION

Pathological pulmonary conditions can result in loss of perfusion and/or loss of ventilation to various areas of the lung. These abnormal conditions include dead space, intrapulmonary shunt, ventilation/perfusion abnormalities (also called $\dot{V}/\dot{Q}$ mismatching), and diffusion defects. Fig. B.1 illustrates an extreme case of uneven ventilation and uneven blood flow. Each of these various conditions will be reviewed.

PHYSIOLOGICAL DEAD SPACE AND ITS CLINICAL MONITORING

Physiological dead space (V_D) is defined as ventilation of lung areas that are not perfused, resulting in no gas exchange (i.e., ventilation without perfusion). Physiological dead space (V_{Dphys}) is divided into anatomical dead space (V_{Danat}) (Fig. B.2A), which comprises the conductive airways down to the level of the respiratory bronchioles where gas exchange begins; and alveolar dead space (V_{Dalv}), where alveoli are ventilated but unperfused or underperfused, respectively. Physiological dead space is therefore the sum of the anatomical and alveolar dead space: $V_{Dphys} = V_{Danat} + V_{Dalv}$.[1]

V_{Danat} is about 1 mL/lb of ideal body weight (IBW) or about 2 mL/kg of IBW. In an average-sized, 150-lb adult, the anatomical dead space is about 150 mL per breath. In normal healthy individuals, physiological dead space and anatomical dead space are approximately equal and V_{Dalv} is not significant. Anatomical dead space seldom changes in clinical situations. V_{Danat} can be lower than normal when an endotracheal tube or a tracheostomy tube is in place, bypassing the upper airway. V_D can be artificially increased by adding mechanical dead space (V_{Dmech}). (This is discussed in more detail later.)

Increased V_{Dalv} can occur with such conditions as pulmonary thromboemboli, pulmonary vascular injury, pulmonary vascular disorders, chronic obstructive pulmonary disease (COPD), and regional hypotension. Increases in V_{Dalv} cause $\dot{V}/\dot{Q}$ mismatching. V_{Dalv} can produce hypoxemia and hypercarbia. Patients with increased V_{Dalv} often have the following characteristic findings: normal lung volumes, normal lung mechanics, increased ventilation (hypoxemic response), normal distribution of ventilation in most cases, uneven distribution of capillary blood flow in the lungs, decreased diffusing capacity, hypoxemia, and normal or increased partial pressure of arterial carbon dioxide (P_aCO_2). Pulmonary embolism is an example of a classic dead space disorder. Pulmonary embolism reduces pulmonary perfusion without necessarily altering ventilation (see Fig. B.2B).

Patients receiving mechanical ventilation can have increased V_{Dalv} if high ventilating pressures cause overexpansion of the alveoli, which in turn exerts pressure on adjacent capillaries, reducing effective flow (see Fig. B.2C). Increased V_{Dalv} also can be present with increased shunting. Suppose a patient had left lower lobe atelectasis. The perfusion to that area would likely be near normal, whereas ventilation to that area would be reduced. This would represent a shunt ($\dot{Q}$ in excess of $\dot{V}$). If this patient was being mechanically ventilated, the affected lobe might not be as well ventilated as other lung areas. Pressurized gas would favor uncompromised areas, reduce their perfusion, and cause a dead

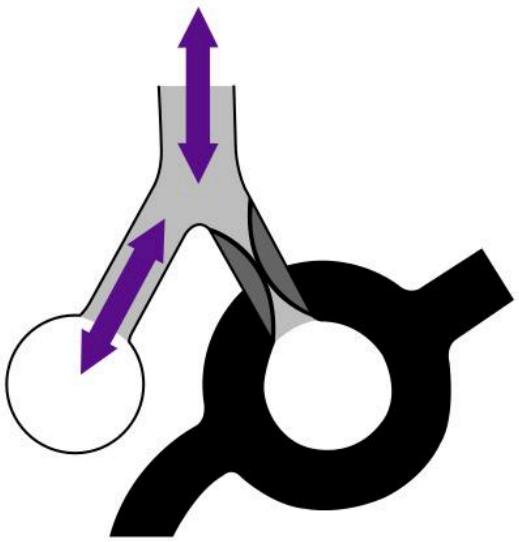


Fig. B.1 Extreme cases of uneven ventilation and uneven pulmonary perfusion. *Circles* represent groups of alveoli with their pulmonary capillary blood flow. *Gray areas* represent the conductive airways (anatomical dead space), and *arrows* represent the distribution of ventilation. The alveolus on the left receives all the ventilation but no perfusion, representing an extreme case of alveolar dead space. The alveolus on the right receives all of the blood flow but is not ventilated because of an obstructed airway. This represents an extreme case of intrapulmonary shunt. (From Comroe JH, Forster RE, DuBois AB, et al.: *The lung: clinical physiology and pulmonary function tests,* ed 2, Chicago, 1973, Year Book Med Publishers.)

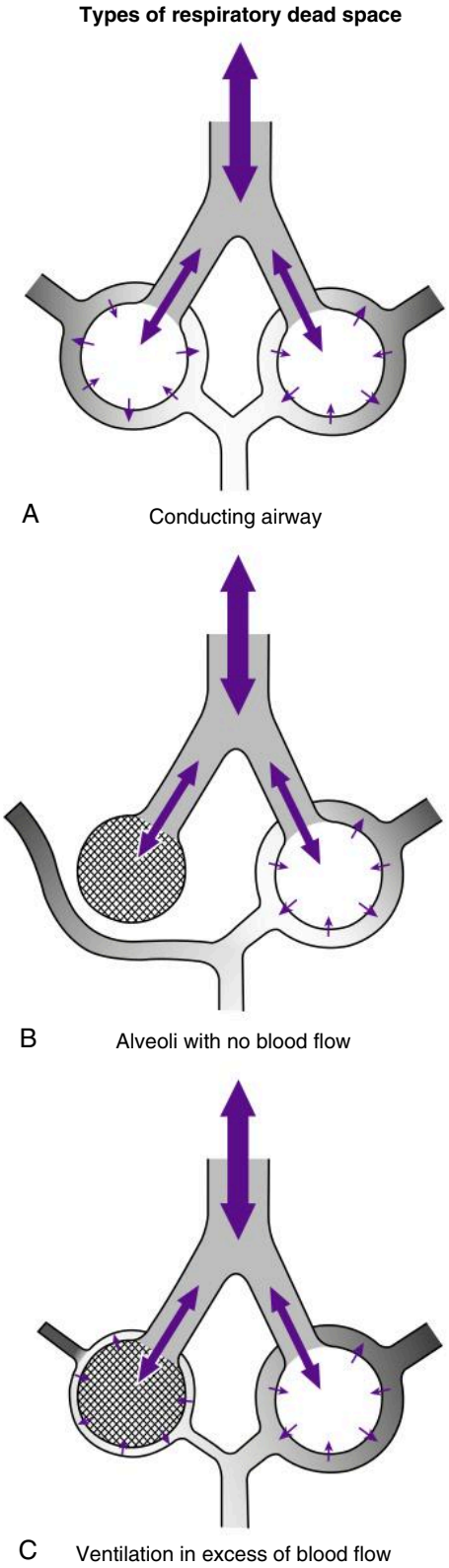


Fig. B.2 Three types of dead space. (A) Anatomical dead space represented by the conductive airways. (B) Alveolar dead space because of alveoli with no blood flow. (C) Alveolar dead space because of ventilation in excess of perfusion (alveolus on the left). See text for further explanation. (From Comroe JH, Forster RE, DuBois AB, et al.: *The lung: clinical physiology and pulmonary function tests*, ed 2, Chicago, 1973, Year Book Medical.)

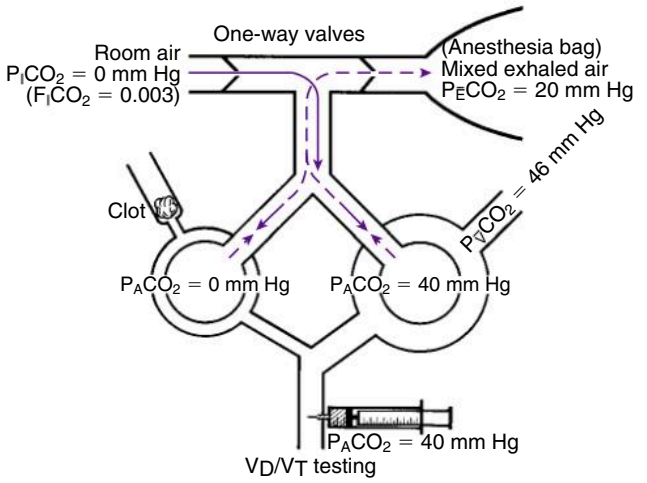


Fig. B.3 Schematic representation of the procedure for measuring inspired partial pressure of CO_2 (P_ICO_2), fraction of inspired CO_2 (F_ICO_2), partial pressure of CO_2 in the alveolus (P_ACO_2), partial pressure of mixed venous CO_2 ($PVCO_2$), mixed expired CO_2 (P_ECO_2), and arterial PCO_2 (P_aCO_2) for the calculation of V_D/V_T. See text for further explanation.

space effect to those areas. Thus a combined shunt and dead space condition would exist, as illustrated by the extreme case in Fig. B.1.

If a patient with COPD were given therapeutic oxygen (O_2), the O_2 might decrease the pulmonary artery pressure by opening up pulmonary capillaries that were previously constricted because of alveolar hypoxemia. However, these newly opened capillaries would allow blood to flow past unventilated alveoli and thus divert pulmonary blood flow away from areas of better ventilation. The result might cause an increased V_{Dalv}.

For these various reasons, it is beneficial to monitor V_D in critically ill patients and monitor for changes in V_{Dphys}. One way is to measure the ratio of dead space to tidal volume (V_D/V_T). Normal V_D/V_T is about 0.25 to 0.40. V_D/V_T can be calculated from the Enghoff modification of the Bohr equation:

$$\frac{V_D}{V_T} = (P_aCO_2 - P_ECO_2) / P_aCO_2$$

where P_ECO_2 is the mixed expired partial pressure of CO_2, V_D is the physiological dead space, V_T is the tidal volume, and P_aCO_2 is the arterial partial pressure for CO_2.

Fig. B.3 shows a classic representation of a procedure for determining V_D/V_T. In this example:

$$P_aCO_2 = 40\,\text{mm Hg}, P_ECO_2\,\text{mm Hg},$$
$$V_T = 500, \text{and } V_D/V_T = (40 - 20)/40 = 0.50$$

The amount of ventilation that is not involved in gas exchange (unperfused alveoli) is 50%. Because the V_T is 500 mL, the V_D will be 0.50 × 500 mL, or 250 mL.

Use of end-tidal CO_2 monitoring can also be useful in tracking changes in dead space. For example, when a pulmonary embolism occurs, there may be a significant decrease in the end-tidal PCO_2 and an increase in the arterial-to—end-tidal PCO_2 gradient (see Chapter 10). However, this finding by itself is not conclusive evidence of increased V_{Dalv} because end-tidal PCO_2 decreases with increased alveolar ventilation ($\dot{V}_A$) and an improvement in $\dot{V}/\dot{Q}$ matching.

BOX B.1 Calculation of Mechanical Dead Space Volume Needed to Increase P_aCO_2

$$V_{Dmech} = \frac{P_aCO_2\text{-}P_aCO_2}{P_aCO_2\text{-}(P_aCO_2)\text{-}P_ACO_2} \times (V_T\text{-}V_{Danat})$$

where V_{Dmech} = mechanical dead space to add
P_aCO_2 = actual P_aCO_2
$P_aCO_2^1$ = desired P_aCO_2
P_ACO_2 = alveolar CO_2

Assume that $\dot{V}/\dot{Q}$ is normal, then $P_aCO_2 - P_ACO_2$ is <10 mm Hg. Use $P_aCO_2 - P_ACO_2 = 5$ mm Hg
Example: $P_aCO_2 = 40$ mm Hg
$P_aCO_2 = 30$ mm Hg
$V_T = 900$ mL
$V_{Danat} = 200$ mL

$$V_{Dmech} = \frac{40 - 30}{40 - 5} \times (900 - 200)\text{mL}$$

Add 200 mL V_{Dmech} to achieve a $P_aCO_2 = 40$ mm Hg.

To determine the volume of a length of large-bore corrugated tubing:

a. Fill the tubing with water and pour it into a graduated cylinder.
b. Add the V_{Dmech} between the endotracheal tube and patient circuit Y-connector.
c. Increase the patient's O_2 percentage slightly to correct for the decreased F_IO_2 brought on by using V_{Dmech}.

Another technique for determining physiological dead space is to use volumetric capnography, as described in Chapter 10.

CALCULATION OF MECHANICAL DEAD SPACE VOLUME NEEDED TO INCREASE P_aCO_2

On rare occasions it may be beneficial to increase a patient's P_aCO_2 when hypocapnia is associated with an increase in minute ventilation that cannot be controlled for some reason (see Chapter 6). When adding V_{Dmech} is desirable, the amount needed can be calculated using the equation in Box B.1. The dead space is added between the endotracheal tube and patient Y-connector on the ventilator circuit.

SOME CAUSES OF HYPOXEMIA

Changes in the lung that compromise the ability of O_2 to transfer from the alveolus to the pulmonary capillary result in hypoxemia. This hypoxemia results in lower than normal partial pressure of arterial O_2 (P_aO_2) and a higher than normal $P_{(A-a)}O_2$ owing to one of three general causes: shunt, $\dot{V}/\dot{Q}$ V abnormalities ($\dot{V}/\dot{Q}$ mismatching), and diffusion defects. Each of these is described.

Shunt

Shunt is defined as that portion of blood from cardiac output that does not participate in gas exchange with alveolar air, that is, perfusion without ventilation. There are three different types of shunts: anatomical shunt, capillary shunt, and perfusion in excess of ventilation.[2]

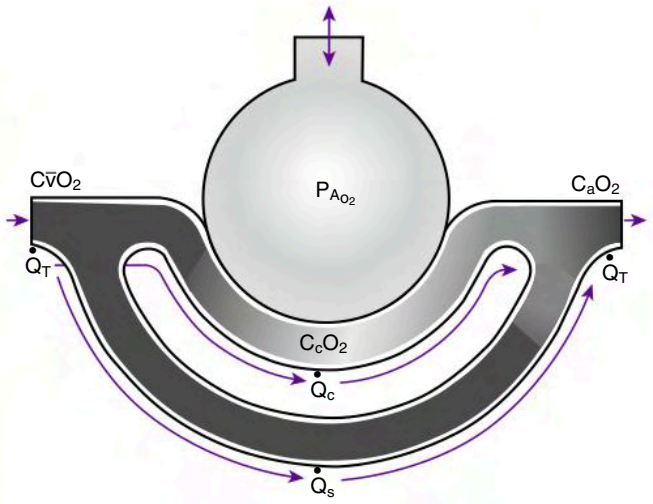


Fig. B.4 Normal alveolar gas exchange is presented by the exchange of gas between alveolar air (presented by the balloon-shaped alveolus) and the normal alveolar capillary (vessel adjacent to the alveolus). Shunted blood is represented by the blood vessel that is not in contact with the alveolus. Mixed venous O_2 content ($Cv\bar{O}_2$), alveolar capillary content (C_cO_2), arterial O_2 content (C_aO_2), cardiac output ($\dot{Q}_T$), and the shunted portion of cardiac output ($\dot{Q}_S$). See text for further explanation. (From Shapiro BA: *Clinical application of blood gases,* Chicago, 1973, Year Book Medical.)

Normal anatomical shunts are present in everyone. They are called *right-to-left shunts* because blood that would ideally return to the right side of the heart (deoxygenated blood) drains into vessels served by the left side of the heart (oxygenated blood). The normal anatomical shunt includes deoxygenated blood from bronchial veins, pleural veins, and thebesian veins, which all drain directly into the left heart (i.e., arterial blood). Normal anatomical shunt represents about 2% to 3% of normal cardiac output. Abnormal anatomical shunts can occur with vascular tumors in the lung or ventricular septal wall defects. In this latter case, blood flows from the right to left side of the heart, bypassing the pulmonary circulation. Any condition in which blood from the systemic circulation flows from the right heart to the left heart without entering the pulmonary circulation is therefore considered an anatomical shunt.

A capillary shunt is the result of blood flowing into the left heart from the pulmonary circulation without passing through ventilated regions of the lung. This intrapulmonary shunting causes hypoxemia that does not respond to O_2 therapy. In other words, there is no significant improvement in P_aO_2 with increases in fractional inspired O_2 (F_IO_2) (refractory hypoxemia). Fig. B.4 illustrates an example of a capillary shunt. Even if the partial pressure of alveolar O_2 (P_AO_2) is increased with administration of O_2, the increased P_AO_2 never comes in contact with the shunted blood. Thus shunted blood mixing with normal arterial content results in a lower than normal P_aO_2 and arterial O_2 content (C_aO_2).

Intrapulmonary shunts are associated with atelectasis, pulmonary edema, pneumonia, pneumothorax, complete airway obstruction, consolidation of the lung, acute respiratory distress syndrome, and, on rare occasions, arterial-to-venous fistulas. *True shunt* is the sum of anatomical and intrapulmonary capillary shunts.

Perfusion in excess of ventilation causes a *shunt effect,* which is also referred to as *venous admixture.* A shuntlike effect can occur in either poorly ventilated alveolar units that are well perfused or alveolar-capillary units, in which O_2 diffusion is impaired. Blood leaving these poorly ventilated areas is low in O_2. Thus a shuntlike effect occurs because of a $\dot{V}/\dot{Q}$ inequality. $\dot{V}/\dot{Q}$ abnormalities can also cause dead space effects and are complex and variable. (See section on Ventilation/Perfusion Abnormalities for further explanation.)

Calculation of Shunt

Because cardiopulmonary disorders can cause either dead space or shunt abnormalities, it is important to be able to evaluate each of these characteristic disorders. Dead space has been described previously. Calculation of a pulmonary shunt relies on the Fick equation:

$$\dot{V}O_2 = Q \times (C_aO_2 - CvO_2),$$

which states that the total O_2 consumption ($\dot{V}O_2$) expressed in milliliters per minute is equal to the cardiac output ($\dot{Q}_V$) expressed in 100 mL/min, multiplied by the arterial-to-venous O_2 content difference ($C_aO_2 - CO_2$) expressed in milliliters of O_2 divided by 100 mL of blood (vol%).

Fig. B.4 illustrates how the total cardiac output is composed of that portion of cardiac output that participates in gas exchange with alveolar air ($\dot{Q}_C$), and the shunted portion of the cardiac output that does not exchange with alveolar air ($\dot{Q}_S$).[2,3] Calculation of the pulmonary shunt can be determined by the following classic shunt equation (the derivation of the shunt equation is not included in this discussion):

$$Q / Q_T = (C_cO_2 - C_aO_2) / (C_cO_2 - CvO_2)$$

where $\dot{Q}_S$ is the shunted portion of the cardiac output, $\dot{Q}_T$ is total cardiac output, C_cO_2 is the content of O_2 of the pulmonary end-capillary after oxygenation of the blood, C_aO_2 is the arterial O_2 content, and $_2$ is the mixed venous O_2 content (i.e., pulmonary capillary blood before oxygenation). C_cO_2 is calculated on the basis of the assumption that pulmonary end-capillary PO_2 is the same as P_AO_2. Mixed venous blood can be obtained using a right heart balloon flotation catheter inserted into the pulmonary artery (see Chapter 11).

In many cases, clinicians often use a *clinical shunt equation* to estimate cardiac output and shunt. This equation, which is derived from the classic shunt equation, uses O_2 pressures rather than O_2 content values. The clinical shunt equation is

$$\dot{Q}_S / \dot{Q}_T = \frac{[(P_AO_2 - P_aO_2) \times 0.003]}{[3.5 + (P_AO_2 - P_aO_2) \times 0.003]}$$

Alveolar P_AO_2 is determined by the following equation:

$$P_AO_2 = [(F_IO_2) \times (P_B - PH_2O)] - \{P_aCO_2 \times [F_IO_2 + (1 - F_IO_2) / R]\}$$

where P_AO_2 is the alveolar partial pressure of O_2, F_IO_2 is the fraction of inspired O_2, P_B is the barometric pressure, PH_2O is water vapor pressure (at 37°C = 47 mm Hg), and R is the respiratory quotient ($R = \dot{V}CO_2/\dot{V}O_2$). An R value of 0.8 is commonly substituted in the equation and represents a normal value.

The clinical shunt equation estimates O_2 content by first assuming that the hemoglobin is the same in arterial, capillary, and mixed venous blood. It also assumes that if the P_aO_2 is greater than 150 mm Hg, both the pulmonary and arterial hemoglobin is fully saturated ($SO_2 = 100\%$). The PO_2 of capillary, arterial, and mixed venous O_2 is then multiplied by 0.003, which is the solubility coefficient of O_2. The equation also assumes that arterial and alveolar O_2 tensions are equal. Because central venous blood samples are not commonly available, the previous equation makes another assumption. It assumes that the a-O_2 content difference is normal, if the clinician establishes that the following circumstances are true[1]:

1. The patient's cardiovascular status is basically normal (i.e., blood pressure, heart rate, pulse pressure, etc.)
2. The metabolic rate is basically normal (i.e., no fever is present and the patient is resting)
3. The perfusion is basically normal (i.e., the skin is warm and dry, capillary refill is good, urine output is normal, etc.)

Patients meeting these three criteria will typically have an arterial-to—mixed venous difference of about 3.5%. Thus an average arterial-to—mixed venous O_2 difference of 3.5 is substituted into the equation.

Practitioners have often used the *clinical shunt equation,* assuming it provides useful information for patients on ventilation. However, it has been shown that the equation will be inaccurate at any F_IO_2 because of all the assumptions made in its derivation. In addition, at F_IO_2 values greater than 0.6, the equation will be as much as 10% to 12% inaccurate compared with the classic shunt equation.[4] Clinicians are advised to not make the assumptions used in the clinical shunt equation but to try to determine as much information as possible and accurately apply the classic shunt equation. Other resources are also available for more accurately estimating pulmonary shunt.[5]

Ventilation/Perfusion Abnormalities

Ideally, ventilation should be perfectly matched with pulmonary perfusion and $\dot{V}/\dot{Q}$ should be equal to 1.0. However, because of many factors, $\dot{V}/\dot{Q}$ varies throughout the lung even in normal individuals. Distribution of gas volume (ventilation) varies based on regional differences in resting lung volumes and transpulmonary pressure (i.e., the difference between alveolar and pleural pressures). Ventilation increases (in relation to overall ventilation) from the apex to the base of the lung in the upright individual. Distribution of pulmonary blood is affected primarily by gravity. Pulmonary blood flow also increases (relative to total pulmonary perfusion) from apex to base, and changes in perfusion are more dramatic than changes in ventilation as blood flow progresses toward the bases. $\dot{V}/\dot{Q}$ is much higher at the top of the lung (i.e., it is about 3.3). Ventilation and perfusion are nearly equal in the middle of the lung (i.e., $\dot{V}/\dot{Q}$ is about 1). In the bases of the lungs both ventilation and perfusion are better than at the apex of the lung. In addition, the lung bases are better perfused than ventilated, so the $\dot{V}/\dot{Q}$ is considerably lower in the bases and equals about 0.66.[6]

A $\dot{V}/\dot{Q}$ abnormality ($\dot{V}/\dot{Q}$ mismatching) is defined as an abnormal mismatching of gas exchange and pulmonary perfusion. A high $\dot{V}/\dot{Q}$ indicates that ventilation is greater than normal and perfusion is less than normal, or both. In cases in which a high

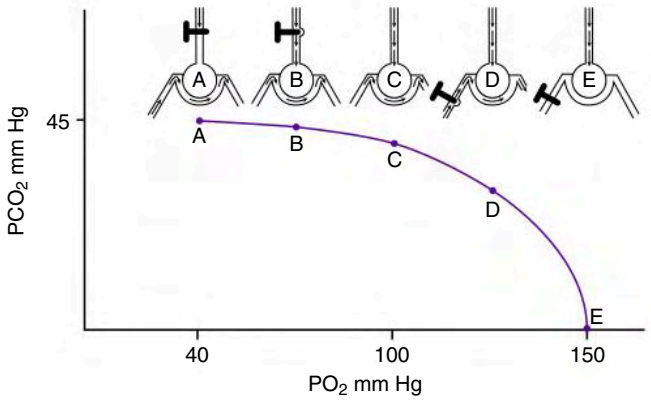


Fig. B.5 The relationship between PO_2 (mm Hg) and PCO_2 (mm Hg) with changes in the ratio of ventilation and perfusion. See text for further explanation. (Redrawn from Deshpande VM, Pilbeam SP, Dixon RJ: *A comprehensive review in respiratory care,* Norwalk, CT, 1988, Appleton & Lange.)

$\dot{V}/\dot{Q}$ ratio exists, the P_{O_2} is higher and the P_{CO_2} is lower than normal. With a low $\dot{V}/\dot{Q}$ ratio, ventilation is less than normal and perfusion is greater than normal, or both. With a low $\dot{V}/\dot{Q}$ ratio, the alveolar PO_2 is lower and the alveolar PCO_2 is higher than normal.[1,6] The hypoxemia associated with a low $\dot{V}/\dot{Q}$ ratio (shunt effect) does not respond well to O_2 therapy.

Fig. B.5 graphically illustrates the effect of regional lung changes of the $\dot{V}/\dot{Q}$ relationship on PO_2 and PCO_2 in the lung.[6] At *A* of the graph, perfusion is present without ventilation, resulting in PO_2 and PCO_2 similar to venous blood (PCO_2 is about 46 mm Hg, and PO_2 is about 40 mm Hg). This represents a pulmonary shunt, and $\dot{V}/\dot{Q}$ will be zero. At *B,* perfusion is in excess of ventilation, representing a *shunt effect.* PO_2 in this region of the lung is low. The $\dot{V}/\dot{Q}$ ratio will equal about 0.4 to 0.5. Many conditions can produce this effect, including hypoventilation, partial airway obstruction (e.g., COPD), and pulmonary interstitial disease. Pulmonary disorders with this type of $\dot{V}/\dot{Q}$ mismatching are more likely to respond to O_2 therapy. Point *C* represents a normal $\dot{V}/\dot{Q}$ relationship of 0.8, resulting in normal PO_2 and PCO_2. Point *D* illustrates ventilation in excess of perfusion. In this case the excess ventilation does not completely take part in gas exchange, producing a dead space effect. Pulmonary capillary blood from these areas is well oxygenated and may have a CO_2 lower than normal (regional hyperventilation). $\dot{V}/\dot{Q}$ values will be in excess of 1.0. Conditions that might cause this type of $\dot{V}/\dot{Q}$ mismatching are positive pressure ventilation and a decreased cardiac output. Point *E* illustrates ventilation without perfusion and is known as V_{Dalv}. A classic example of this type of $\dot{V}/\dot{Q}$ mismatch is pulmonary embolism, which was described earlier in the section on dead space. With no perfusion, alveolar PO_2 (P_AO_2) for a person breathing room air will be about 150 mm Hg and alveolar PCO_2 (P_ACO_2) will be zero in affected areas. Resulting exhaled gases will contain lower than normal PCO_2 values. $\dot{V}/\dot{Q}$ will approach infinity (i.e., 1 divided by 0).

In summary, the adequacy of gas exchange is a sum of the $\dot{V}/\dot{Q}$ distribution of gas throughout the lung. In general, inadequate ventilation relative to a normal perfusion (low $\dot{V}/\dot{Q}$ and shunt) has the greatest effect on O_2 uptake by the lung and results in hypoxemia. On the other hand, an excessive amount of ventilation relative to perfusion affects CO_2 elimination and can result in hypercapnia in severe cases.[6]

Diffusion Defects

A diffusion defect is a pathological condition resulting from impaired gas exchange across the alveolar-capillary membrane causing hypoxemia that is generally responsive to O_2 therapy. Diffusion defects occur through three possible mechanisms: a reduction of alveolar surface area, a thickened alveolar-capillary membrane, or a lower than normal O_2 pressure gradient.

A reduced surface area can occur after surgery in which, for example, a pulmonary lobe is removed (lobectomy). It can also occur in diseases that destroy alveolar tissue, such as emphysema. These structural changes to the lungs are not reversible.

Diffusion defects can occur as a result of an increase in the thickness of the alveolar-capillary membrane, which can occur with pneumonia, pulmonary or interstitial edema, pulmonary fibrosis, or any condition that thickens one or all of the components of the alveolar-capillary membrane.

A lower than normal alveolar-to-arterial O_2 tension gradient usually results from a lower than normal P_AO_2. It is rare that PO_2 is lower than normal with the exception of individuals residing at high altitudes (e.g., living at 10,000 feet above sea level). With a low P_AO_2, the pressure gradient between the alveolus and pulmonary capillary will be lower than normal, thus reducing the rate of gas transfer across the alveolar-capillary membrane.[1,6]

References

1. Levitzky MG: *Pulmonary physiology*, ed 8, New York, 2013, McGraw-Hill Medical, pp 58–90.
2. Shapiro BA: *Clinical application of blood gases*, Chicago, 1970, Year Book Medical.
3. Comroe JH, Forster RE, DuBois AB, et al.: *The lung: clinical physiology and pulmonary function tests*, ed 2, Chicago, 1973, Year Book Medical.
4. Warner MA, Divertie MB, Offord KP, et al.: Clinical implications of variation in total venoarterial shunt fraction calculated by different methods during severe acute respiratory failure, *Mayo Clin Proc* 58:654–659, 1983.
5. Cairo JM: *Mosby's respiratory care equipment*, ed 9, St. Louis, MO, 2014, Mosby, p 314.
6. West JB: *Pulmonary pathophysiology: the essentials*, Baltimore, MD, 1992, Williams & Wilkins.

APPENDIX C

Graphics Exercises

GRAPHING VENTILATOR WAVEFORMS

This exercise is designed to help the reader understand graphic waveforms produced by a microprocessor-controlled ventilator. By performing these graphing exercises and calculations, the reader will also gain a better understanding of the interrelationship of flow, volume, pressure, and time waveforms generated during mechanical ventilation.

Problem 1

Assume you have a patient who is receiving ventilation using volume-controlled continuous mandatory ventilation (VC-CMV) set to deliver a constant flow of gas during inspiration until it reaches the volume ordered by the physician. You are given the following information about the patient's lung characteristics and ventilator parameters:

- Compliance (C) is 0.2 L/cm H_2O
- Airway resistance (R_{aw}) is 15 cm H_2O/L/s
- Flow rate is constant at 40 L/min
- Ordered tidal volume (V_T) is 1 L (1000 mL)
- End-expiratory pressure is zero (no positive end-expiratory pressure [PEEP])

Perform the following steps using the preceding information:

- Calculate the flow rate in L/s.
- Record under variable (Fig. C.1A) the flow at each quarter-second of time that will be present during inspiration. Graph the flow at quarter-second intervals (see Fig. C.1A).
- Calculate and graph the volume delivered at each quarter-second interval during inspiration: Volume (V) = Time (T) × Flow (see Fig. C.1B).

Note that the ordered V_T of 1 L (1000 mL) was delivered in about 1.5 seconds. A volume-cycled ventilator would stop the inspiratory phase at this point. For all graphs from this point on, calculations need to be made only for times up to 1.5 seconds.

- Using the volume, calculate and graph the alveolar pressure. Recall that C = $\Delta V/\Delta P$; $\Delta P = P_{alv} - EEP$ and EEP = 0; $P_{alv} = \Delta V/C$. The volume is taken from the calculations of volume in step 3 for each quarter-second interval (see Fig. C.1C).
- Calculate and graph the transairway pressure (P_{TA}). Recall that $R_{aw} = P_{TA}/Flow$. Therefore $P_{TA} = R_{aw} \times Flow$, $P_{TA} = 15 \times 0.67 = 10$ cm H_2O. Both R_{aw} and flow have constant values. P_{TA} will have the same value for each quarter-second interval (see Fig. C.1D).
- Add the values of P_{alv} and P_{TA} to determine the airway opening pressure (P_{awo}) at each quarter-second interval. Graph these values. Note that at the zero point on the *x* and *y* axes, the P_{awo} will not go to zero. The pressure rises rapidly as the flow begins to go to the patient because the gas flow encounters the resistance of the circuit, the endotracheal tube (ET), and the patient's airways. The P_{awo} value just near zero on the *x* axis is approximately equal to the P_{TA} just to the right of the *y* axis (see Fig. C.1E).
- Compare the curves produced in the preceding exercise; the graph for flow has the same waveform as the graph for:
  - P_{alv}
  - PIP
  - P_{TA}

CHANGES IN WAVEFORMS WITH CHANGES IN LUNG CHARACTERISTICS

Ventilator Working Pressure

A ventilator that can generate pressures that greatly exceed those reached at the upper airway can deliver any waveform pattern for flow, volume, or pressure. These waveforms do not change regardless of changes in lung characteristics. Some ventilators deliver pressures of 400 to 700 cm H_2O, far greater than what is needed for ventilation of the human lung (10–35 cm H_2O).

The following sections discuss how pressure waveforms change during volume ventilation (constant flow) and how volume and flow waveforms change during pressure ventilation (constant pressure) with changes in lung characteristics.

Constant-Flow Volume Ventilation With High Working Pressure

During volume ventilation (constant flow), the volume waveform increases linearly and volume delivery is constant. Inspiration is usually time cycled or volume cycled (Fig. C.2). As a patient's lungs become less compliant or R_{aw} increases, the pressure waveforms are affected; however, the volume and flow curves remain the same. Clinically, a decrease in compliance increases peak inspiratory pressure (PIP) and P_{plat} (Fig. C.3). An increase in R_{aw} also increases PIP, whereas alveolar pressure (P_{plat}) remains fairly constant. The difference between the two (P_{TA}) increases (Fig. C.4). The ventilator provides a constant volume, even with changes in lung characteristics, as long as the ventilator is time cycled or volume cycled. If it prematurely pressure cycles as a result of reaching the set pressure limit, volume decreases.

Using other types of flow waveforms produces changes similar to those seen for P_{plat}, P_{TA}, and P_{awo}. Reduced compliance increases PIP and P_{plat}. Increased R_{aw} increases P_{TA}.

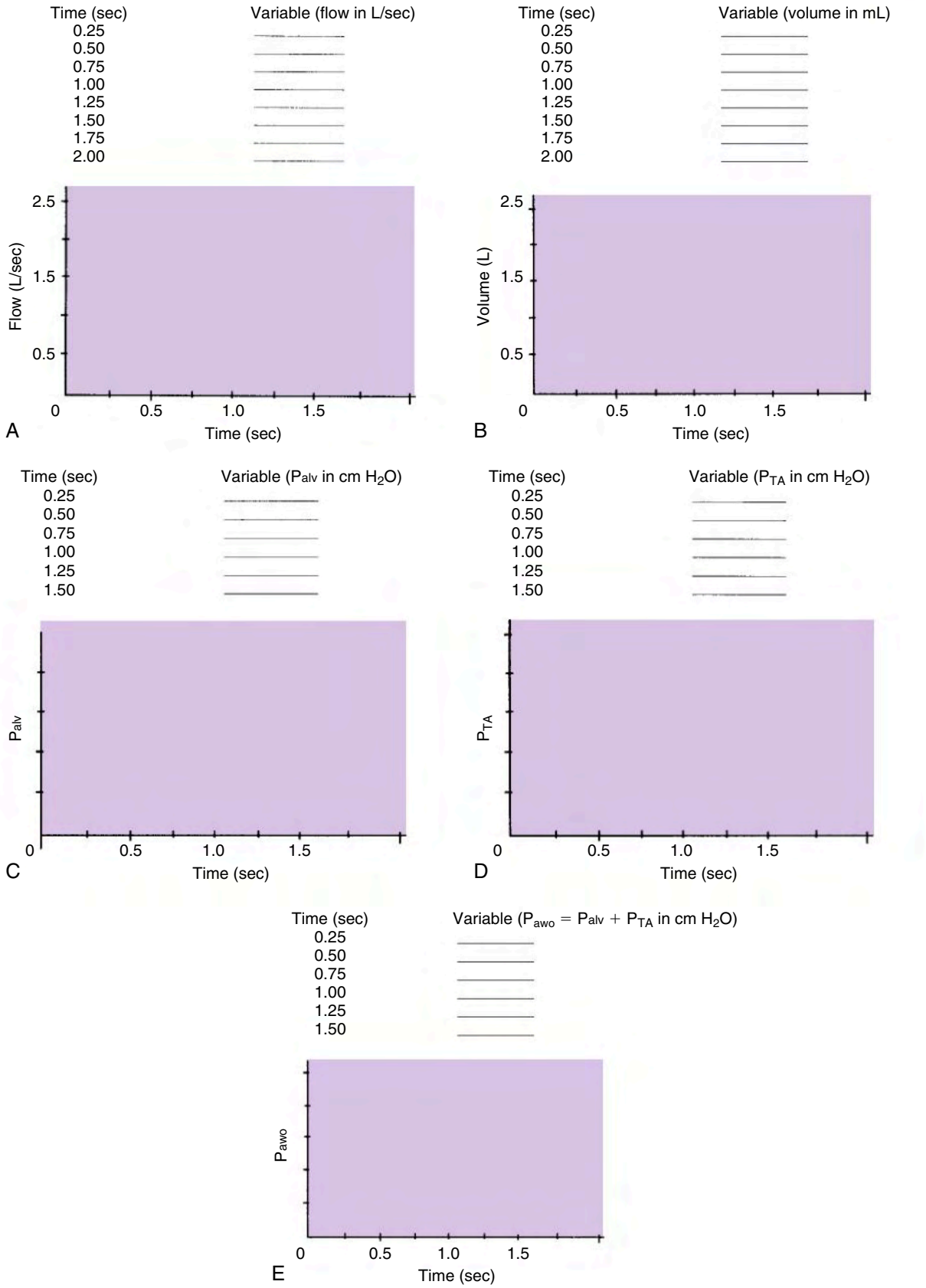
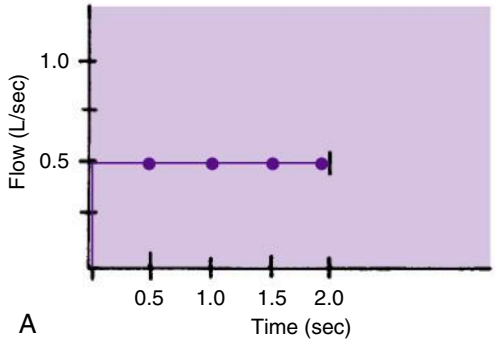


Fig. C.1 Graphing exercise. (See text for explanation. Note that the tables of Time/Variable appear directly above each graph.) (Answers to this problem can be found in Appendix A.)

Flow = Volume/Time

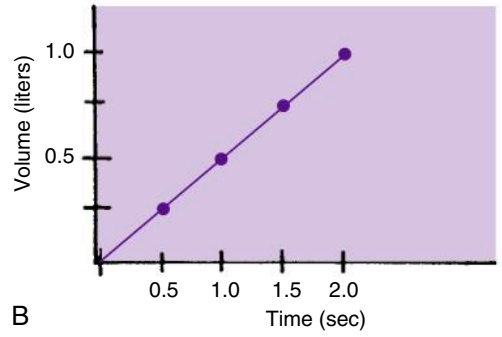
Time (sec)	Flow (L/sec)
0.5	0.5
1.0	0.5
1.5	0.5
2.0	0.5



A

Volume = Flow × Time

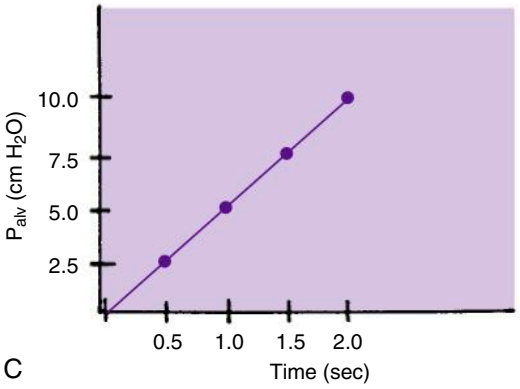
Time (sec)	Volume (L)
0.5	0.25
1.0	0.50
1.5	0.75
2.0	1.00



B

P_{alv} = Volume/Compliance

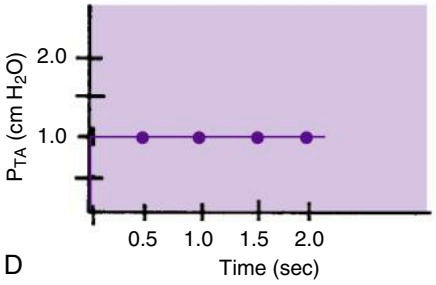
Time (sec)	P_{alv} (cm H_2O)
0.5	2.5
1.0	5.0
1.5	7.5
2.0	10.0



C

P_{TA} = R_{aw} × Flow

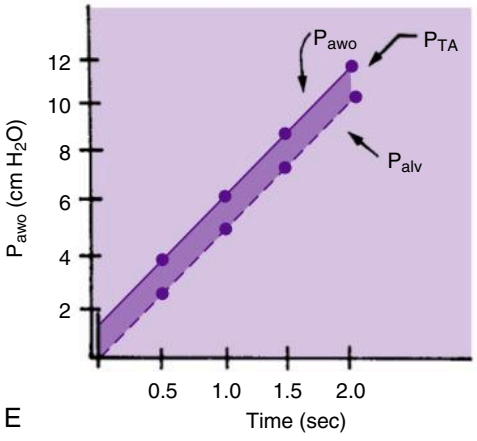
Time (sec)	P_{TA} cm H_2O
0.5	1.0
1.0	1.0
1.5	1.0
2.0	1.0



D

P_{awo} = P_{alv} + P_{TA}

Time (sec)	P_{awo} cm H_2O
0.5	3.5
1.0	6.0
1.5	8.5
2.0	11.0



E

Fig. C.2 Curves for constant (rectangular) flow under normal lung conditions (compliance [C] = 0.1 L/cm H_2O; airway resistance [R_{aw}] = 2 cm H_2O/L/s); inspiratory time [T_I] = 2 seconds. (A) Flow is constant at 0.5 L/s. (B) Volume increases at a constant rate during inspiration, achieving a tidal volume (V_T) of 1 L. (C) P_{alv} increases at a constant rate, as does volume, to a maximum of 10 cm H_2O. (D) Because flow is constant, P_{TA} is constant; this assumes that resistance and flow do not change. (E) P_{TA} is 1 cm H_2O/L/s. P_{TA} = Flow × R_{aw}. P_{alv}, Alveolar pressure; P_{awo}, upper airway pressure; P_{TA}, pressure lost to airways.

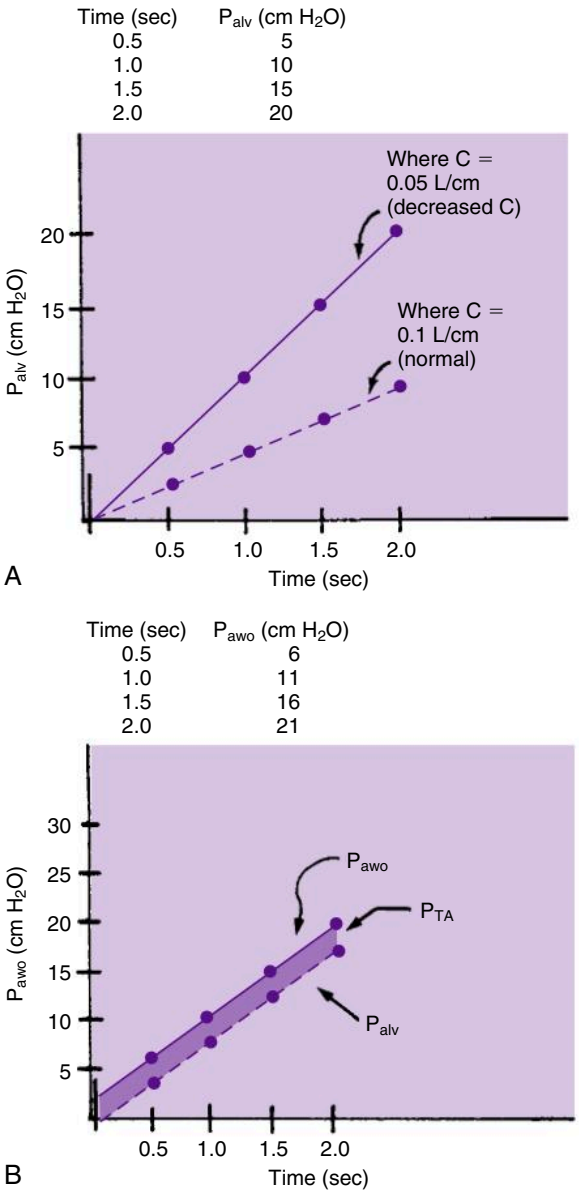


Fig. C.3 (A) Flow is constant at 0.5 L/s. T_I = 2 seconds. The volume coming from the ventilator remains the same at 1 L even though compliance (C) is reduced. P_{alv} has doubled (20 cm H_2O) in this situation because compliance is half its previous value. The *dashed line* represents the curve for normal compliance (C = 0.1 L/cm H_2O), and the *solid line* represents the curve for reduced compliance (C = 0.05 L/cm H_2O). (B) Because flow and airway resistance (R_{aw}) are constant, the pressure lost to the airways is constant (P_{TA} = 1 cm H_2O). P_{TA} is the *shaded area*. The upper airway pressure is much higher than normal because compliance is reduced. P_{alv}, Alveolar pressure; P_{awo}, upper airway pressure; P_{TA}, pressure lost to airways.

Constant-Flow Volume Ventilation With Low Working Pressure

Under normal lung conditions, a constant-flow ventilator with low to moderate working pressure (40–120 cm H_2O) creates a constant-flow waveform similar to that shown in Fig. C.5A.

When compliance is significantly reduced and resistance is increased, PIP rises and flow decreases during inspiration because of the decrease in pressure gradient between the ventilator and alveoli. When the driving mechanism no longer generates an adequate working pressure, the ventilator no longer provides a constant flow. This is not necessarily a disadvantage. The resulting modified descending ramp waveform may actually be more desirable for improving gas distribution in the lungs, but it can alter inspiratory time (T_I) (see Fig. C.5B).

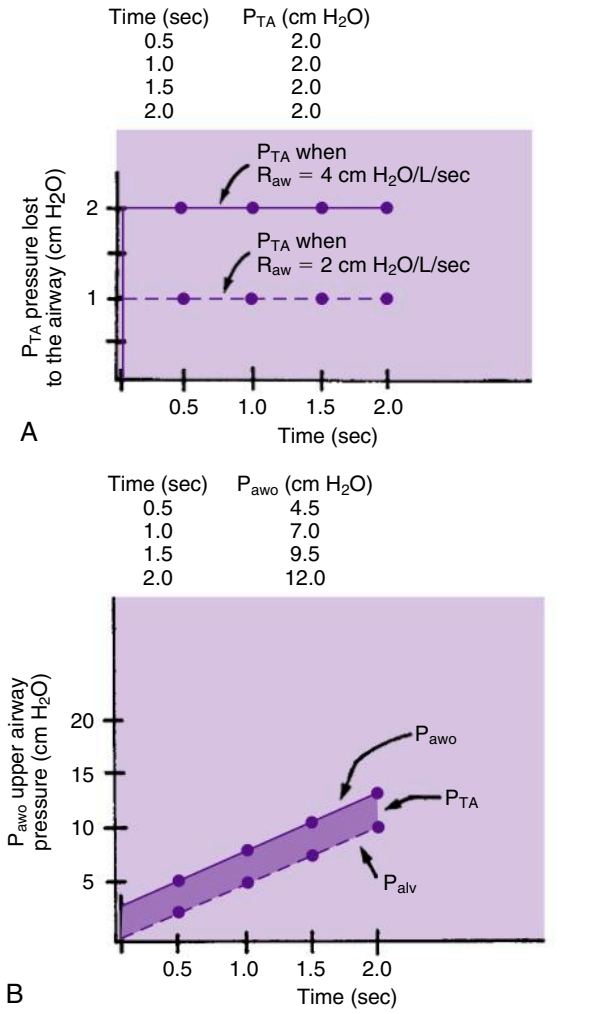


Fig. C.4 Constant-flow ventilator. The following inspiratory curves are produced when airway resistance (R_{aw}) is increased to 4 cm H_2O/L/s and compliance is normal (C = 0.1 L/cm H_2O). (A) Pressure lost to the airways (P_{TA}) is the product of flow and airway resistance (Flow × R_{aw}). With an increase in R_{aw}, P_{TA} increases to 2 cm H_2O *(solid line)* compared with normal at 1 cm H_2O *(dashed line)*. (B) Upper airway pressure (P_{awo}), the sum of alveolar pressure (P_{alv}) and P_{TA}, increases to a maximum of 12 cm H_2O because P_{TA} is increased *(shaded area)*. The difference between the peak and plateau, or P_{alv}, increases.

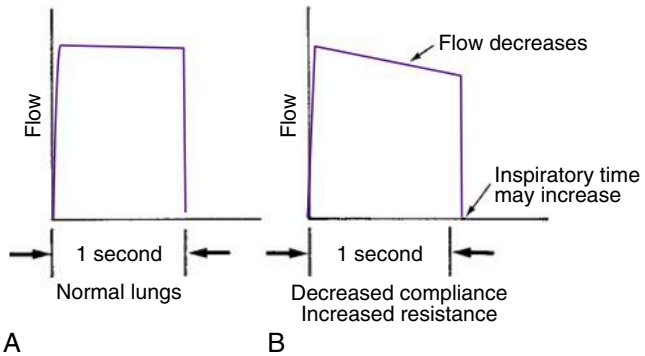


Fig. C.5 Inspiratory curves represent the changes in flow that can occur using constant flow in a ventilator with moderate to low pressure-generating capabilities. (A) Under normal conditions, flow is constant. (B) As compliance decreases and resistance increases significantly, flow decreases slightly. If the ventilator is volume cycled, volume is delivered from the ventilator but inspiratory time (T_I) may increase. This affects the inspiratory-to-expiratory ratio (I/E). These changes in lung characteristics increase P_{alv} and P_{awo} as long as the ventilator is not pressure cycled out of inspiration by reaching the preset pressure limit.

Glossary

A

absorption atelectasis Atelectasis (lung collapse) resulting from the absorption of oxygen from obstructed or partially obstructed alveoli.
acinus The smallest division of a gland; a subdivision of the lung consisting of the tissue distal to a terminal bronchiole including respiratory bronchioles, alveolar ducts, alveoli, and all other structures within.
acute cardiogenic pulmonary edema Fluid accumulation in the air spaces and parenchyma of the lungs due to increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure.
acute hypercapnic respiratory failure Inability to maintain normal PCO_2, resulting in a $PaCO_2$ >50 mmHg) over a relatively short period of time.
acute hypoxemic respiratory failure Severely decreased oxygenation (PaO_2 <50 mm Hg) that results in compromised respiratory function over a relatively short period of time.
acute-on-chronic respiratory failure An acute exacerbation of hypoxemic or hypercapnic respiratory failure in a patient with chronic respiratory failure (i.e., COPD patient who demonstrate chronic elevation of $PaCO_2$ or decreased PaO_2).
Acute Physiology and Chronic Health Evaluation (APACHE) A method of classifying the severity of illnesses in patients; a scoring system used to predict outcome.
acute respiratory failure (ARF) Any condition in which respiratory activity is completely absent or inadequate to maintain oxygen uptake and carbon dioxide clearance.
acute severe asthma Also known as *status asthmaticus.* Severe asthma episode unresponsive to repeated courses of β-2 agonist therapy. Emergency that requires immediate recognition and treatment.
adaptive support ventilation A patient-centered method of closed-loop mechanical ventilation that increases or decreases ventilatory support based on monitored patient parameters.
afterload The impedance that the ventricles must overcome to eject blood into the great vessels.
airway opening pressure (P_{awo}) Pressure at the upper airway (nose and mouth); also called *mouth pressure (P_M), airway pressure (P_{aw}), upper airway pressure, mask pressure,* and *proximal airway pressure.*
airway pressure (P_{aw}) Pressure in the upper airway; also called *mouth pressure (P_M), airway pressure (P_{aw}), upper airway pressure, mask pressure,* and *proximal airway pressure.*
alveolar dead space Alveoli that are ventilated but not perfused by pulmonary capillary blood flow (e.g., pulmonary embolism).
alveolar distending pressure The pressure difference between the inside of the lung and the outside of the lung, which is responsible for maintaining alveolar inflation; also called *transpulmonary pressure.*
amplitude The maximum value of a periodic curve or wave measured along the vertical axis.
analgesics Agents that reduce pain without causing a loss of consciousness.
anesthetic Drug or agent used to abolish the sensation of pain.
anterograde amnesia Condition in which the acquisition and encoding of new information that can potentially lead to memories of unpleasant experiences is prevented.
anterograde amnesia Inability to create new memories after the event that causes amnesia
ascites An abnormal accumulation of fluid in the peritoneal space.
assist-control (A/C) ventilation See *continuous mandatory ventilation.*
assisted breaths When all or part of the breath is generated by the ventilator and the ventilator is providing part of the work of breathing for the patient.
assisted ventilation Breath delivery that is aided by the use of a mechanical ventilatory device.
asynchrony (dyssynchrony) See *patient–ventilator asynchrony.*
automatic tube compensation A feature available in some ventilators that provides adjustable compensation for the work of breathing through an artificial airway.
auto-PEEP Inadvertent PEEP that is not set by the operator but results in a buildup of positive pressure in the lungs at the end of exhalation; commonly caused by high minute ventilation settings, particularly in the presence of airway obstruction, which result in incomplete exhalation of an inspired volume.
Autotrigger A machine-triggered breath that occurs without the patient making an inspiratory effort.

B

back pressure Pressure that builds in a tube or circuit and increases and extends backward through the tube if the normal point of exit (egress) is blocked and not restored.
balloon-tipped, flow-directed catheter The balloon-tipped, flow-directed catheter (also referred to as the *Swan-Ganz catheter* or *pulmonary artery catheter*) is a multiple-lumen catheter constructed of radiopaque polyvinylchloride.
barotrauma Injury to the lung parenchyma caused by excessive pressures in the lungs.
baseline pressure The pressure from which inspiration begins and at which expiration ends during mechanical ventilation; also known as *expiratory pressure*. Normal baseline pressure is atmospheric. Positive pressures can be applied to increase the baseline above atmospheric.
baseline variable Airway pressures measured relative to a baseline value (e.g., zero mm Hg or atmospheric pressure).
bilevel PAP A ventilatory mode that is a variation on CPAP therapy. It is characterized by the inspiratory positive airway pressure (IPAP) that is greater than the expiratory positive airway pressure (EPAP).
biofilm A thin, mucous film of bacteria that attaches to a surface.
Biot respirations Periods of apnea (10–30 seconds) followed by periods of breathing at a uniform depth; associated with central nervous system disorders (e.g., meningitis) and increased intracranial pressure.
bradycardia Heart rates in the adult less than 60 beats/min.
bronchial alveolar lavage (BAL) Medical procedure in which a bronchoscope is passed through the mouth or nose into the lungs and fluid is instilled into a small part of the lung and then recollected for examination.
bronchomalacia Weak cartilage in the walls of the bronchial tubes, occurring in children younger than 6 months, presenting with noisy breathing and/or wheezing. There is collapse of a mainstem bronchus on expiration.
bronchopleural fistulas A hole or opening between the lung and pleural space producing an air communication between the two.
bronchopulmonary dysplasia (BPD) A chronic respiratory disorder that often occurs in infants exposed to long-term ventilation. Lung tissue is scarred, and pulmonary arterial walls are thickened. Mismatching of ventilation and pulmonary perfusion exists.

C

capnography Graphic display of exhaled carbon dioxide concentration using a capnograph.
cardiac index Cardiac output divided by body surface area.
cardiac tamponade Compression of the heart caused by fluid or air in the pericardial sac; also caused by positive pressure in the lungs around the outside of the heart.
cardiac transmural pressure The pressure difference between the inside of the heart and the intrathoracic pressure.
cardiac work An estimate of the amount of work the heart must perform to eject the stroke volume.
cardiogenic pulmonary edema (CPE) Accumulation of fluid (plasma) in the pulmonary interstitial and alveolar spaces as a result of increased pulmonary capillary pressure typically associated with left heart failure.
central venous lines Catheters placed near or in the right atrium to measure central venous pressure.
chest-abdominal paradox When the chest wall and abdomen move the opposite of normal during breathing. The chest wall goes inward on inspiration while the abdomen is moving outward, and vice versa. See also *respiratory alternans*.
chest cuirass The shell-like device that fits over the thorax for the delivery of negative pressure ventilation.
Cheyne-Stokes respiration A pattern of breathing with apneas lasting 10 to 30 seconds, followed by gradual increase in the depth and rate of breaths; associated with cerebral disorders, congestive heart failure, and alterations in acid—base status such as in metabolic problems.
choanal atresia A congenital abnormality in which a bony or membranous obstruction blocks the passage between the nose and pharynx; can result in serious respiratory problems in the neonate.
cleft palate A congenital defect in which a fissure occurs in the midline of the palate; often associated with a cleft in the upper lip.
Clinical Pulmonary Infection Score (CPIS) An approach to clinical diagnosis that includes six clinical assessments with each item given a score of 0 to 2 points. The assessment criteria include fever, leukocyte count, quantity and purulence of tracheal secretions, oxygenation status, the type of radiographic abnormality, and results of a tracheal aspirate culture and Gram stain.
closed-loop system A feedback control scheme in which the actual output is measured and compared with the desired input set on the control panel (user interface). An error signal is sent to the controller if a specified difference is found between the input value and the measured output.
community-acquired pneumonia (CAP) A common infectious respiratory disease characterized by inflammation of lung tissue, acquired outside the hospital setting by inhalation or aspiration of pathogenic organisms into a lung segment or lobe. A pneumonia contracted from the environment as opposed to being acquired within a medical facility (e.g., hospital-acquired pneumonia), CAP is usually more sensitive to antibiotics.
compliance (C) The relative ease with which the structure distends; the opposite, or inverse, of elastance (e). $C = 1/e$ or $e = 1/C$.
compressible volume The volume of gas in the patient (ventilator) circuit that stays in the circuit during inspiration; also referred to as the *compressible volume* or the *volume lost as a result of tubing compliance* (C_T).
continuous mandatory ventilation (CMV) Term used most often to describe a mode that is time or patient triggered, volume or pressure targeted, and volume or time cycled. Every breath is mandatory. Also known as *controlled mechanical ventilation* and *continuous mechanical ventilation.*
continuous positive airway pressure (CPAP) Positive pressure applied to the spontaneously breathing patient during both inspiration and expiration; used for the treatment of obstructive sleep apnea and to increase mean airway pressure in critically ill patients who are able to do some spontaneous breathing.
contractility Cardiac contractility is related to the force that the ventricle generates during each cardiac cycle. The value can be estimated by using the ejection fraction.
control mode Mechanical ventilatory mode in which the ventilator controls the inspiratory pressure, volume, flow, or time.
control system The internal components of a ventilator that interpret what the operator sets on the control panel (user interface).
control variable The primary variable the ventilator control circuit adjusts to cause inspiration.
controlled ventilation Time triggering of all breaths, with breath rate established by the ventilator.
cor pulmonale Enlargement (hypertrophy) or failure of the right heart and pulmonary hypertension caused by certain pulmonary parenchymal or pulmonary vascular disorders.
critical opening pressure The pressure required to open a collapsed lung unit.
Cushing response A normal response to acute increases in intracranial pressure, which results in hypertension with bradycardia.
cycle variable The phase variable that ends inspiration.
cycling mechanism The variable by which the ventilator marks the end of inspiration.
cytokines A group of low-molecular-weight proteins secreted by specific cells; involved in cell-to-cell communication and a variety of immune responses.

D

decannulation Removal of a tracheostomy tube.
deep sulcus sign (chest radiograph) A radiographic sign indicative of the presence of a pneumothorax.
de-escalation An important method that can be used to reduce the incidence of multidrug-resistant pathogens because it reduces unnecessary use of antibiotics.
deflation point The upper inflection point on the deflation portion of the curve (UIPd), also called the *deflection point* or *deflation point.* Point at which lungs begin to collapse.
deflection point Part of the information obtained during a lung recruitment maneuver. After a full inspiration to total lung capacity, a deflation is performed and graphs are established for pressure—volume points similar to the inflation curve. A deflection point occurs where a large number of lung units collapse quickly. The deflection point is the upper inflection point during deflation.
delay-time control A comfort feature incorporated in many noninvasive pressure-targeted ventilators that allows the user to set a period of time that must elapse before inspiratory and expiratory pressures achieve prescribed levels. A very low pressure and continuous flow of gas will continue through the circuit during this interval of time. It is most often used in conjunction with the ramp feature. See *ramp*.
depolarizing agents Agents that cause skeletal muscle paralysis by causing a reversal of the resting membrane potential in excitable cell membranes.
diastole The period of cardiac relaxation and filling during the cardiac cycle.
double-circuit ventilator A ventilator in which the primary power source generates a gas flow that compresses another mechanism such as a bag or bellows.
dP/dT Change in pressure relative to time.
drive mechanism The mechanical device that causes gas flow to the patient.
dynamic characteristic Characteristic of lung function during a breath (e.g., dynamic compliance).
dyssynchrony (asynchrony) See patient—ventilator asynchrony.

E

early-onset pneumonia Ventilator-associated pneumonia (VAP) that develops between 48 and 72 hours after tracheal intubation.
Calculated as the ratio of the stroke volume to the ventricular end-diastolic volume.
elastance (e) The tendency of a structure to return to its original form after being stretched or acted on by an outside force. Elastance (e) is the inverse of compliance (C); e = 1/C.
electrical activity of the diaphragm (Edi) A minimally invasive bedside technique used in conjunction with neurally adjusted ventilator assist (NAVA) that gives information on the patient's diaphragm activity.
endogenous Growing inside the body; originating from inside the body or produced from internal causes. Compare *exogenous*.
equation of motion Mathematical equation that describes the relationships among pressure, volume, and flow during a spontaneous or mechanical breath.
erosive esophagitis Inflammation of the mucosal lining of the esophagus in which erosion occurs to the tissues. Causes include infection and irritation from a nasogastric tube or from backflow of gastric fluids from the stomach into the esophagus.
esophageal pressure Pressure measured from a balloon placed in the esophagus; used to estimate pressures and pressure changes in the pleural space.
exogenous Located or originating from outside the body (e.g., a foreign bacteria causing an infection).
expiratory positive airway pressure (EPAP) The application of positive pressure to the airway during exhalation. Compare *inspiratory positive airway pressure.*
external circuit See *patient circuit.*
external respiration The movement of oxygen and carbon dioxide across the alveolar capillary membrane.
extracorporeal membrane oxygenation A procedure in which venous blood is pumped out of the body to a heart–lung machine, where it is oxygenated and returned to the body.
extrinsic PEEP Positive end-expiratory pressure applied by the operator.
exudative Related to the oozing of fluid and other substances from cells and tissues; usually a result of infection or injury.

F

fiberoptic bronchoscopy Technique to examine breathing passages (airways) of the lungs using a thin flexible or rigid tube called a *bronchoscope* that is placed in the nose or mouth. The tip contains a small camera that projects onto a video screen.
fibrosing alveolitis A form of alveolar inflammation accompanied by dyspnea and hypoxia. Chest radiographs show diffuse infiltrates and thickening of the alveolar septa. It occurs with advanced rheumatoid arthritis and other immune disorders.
Fick principle A method for determining perfusion through an organ; based on the oxygen consumption and arterial-to-venous oxygen content difference.
flow-controlled ventilation Mechanical ventilatory mode in which the ventilator controls flow and therefore volume of gas delivered to the patient.
flow cycling When the cycle variable is flow. Inspiration ends when a specific flow is measured during inspiration by the ventilator. For example, pressure support ventilation is flow cycled.
flow limited A ventilator is considered flow limited if the flow during ventilation reaches a maximum value before the end of inspiration but does not exceed that value.
flow resistor A device with an orifice of fixed size placed in the expiratory limb of a breathing circuit; used to achieve positive expiratory pressure by creating a resistance to exhaled gas flow. As the diameter of the orifice increases in size, the provided pressure level decreases, and vice versa. Changes in the rate of gas flow also change the pressure. The higher the expired gas flow, the higher the expiratory pressure generated.
flow triggering Inspiratory flow from the ventilator begins when a set drop of flow through the patient circuit is detected during exhalation.
fractional hemoglobin saturation Calculated by dividing the amount of oxyhemoglobin measured by the amount of all four types of hemoglobin present, or fractional $O_2Hb = O_2Hb/(HHb + O_2Hb + COHb + MetHb)$.
French (Fr) size A unit of measure. The French size divided by π (3.1416) equals the external diameter of a catheter in millimeters.
frequency control Touch pad control device used to set the number of breaths delivered by a mechanical ventilator.
full ventilatory support (FVS) When the ventilator provides all the energy necessary to maintain effective alveolar ventilation.
functional hemoglobin saturation Calculated by dividing the oxyhemoglobin concentration by the concentration of hemoglobin capable of carrying oxygen, or functional $O_2Hb = O_2Hb/(HHb + O_2Hb)$.
functional residual capacity The volume of gas remaining in the lungs at the end of normal exhalation.

G

gastroprotective agents Agents such as antacids that reduce the risk for gastrointestinal bleeding.
gastrostomy or jejunostomy tube A tube used to introduce nutrients into the stomach or to remove fluids, gas, or poisons; also called a *stomach tube.*
glabella The smooth area on the frontal bone between the superciliary arches.

H

health care–associated pneumonia (HCAP) Afflicts patients who have resided in a long-term care facility or received acute care in an acute care hospital for a specified time before developing pneumonia (i.e., 2 or more days within 90 days of the infection).
heliox A mixture of helium and oxygen, usually in concentrations of 80% helium and 20% oxygen or 70% helium and 30% oxygen; used to reduce the work of breathing by lowering the density of the gas mixture.
hematogenous Originating in the blood or transported in the blood.
hertz (Hz) Unit of measure equivalent to 60 cycles (oscillations) per minute.
heterogeneous Composed of different parts or elements.
high-frequency jet ventilation Ventilation that uses rates between about 100 and 600 breaths/min; operates by using a nozzle or an injector, which creates a high-velocity jet of air directed into the lungs. Exhalation is passive.
high-frequency oscillatory ventilation (HFOV) Uses rates into the thousands, up to about 4000 cycles/min. HFOV ventilators use either a small piston or a device similar to a stereo speaker, both of which deliver gas in a "to-and-fro" motion, pushing gas in during inspiration and drawing gas out during exhalation.
high-frequency positive pressure ventilation High-frequency ventilation that employs a conventional positive pressure set at high respiratory rates (60–100 breaths/min) with lower than normal tidal volumes.
homeostasis A consistency or balance in the internal environment of the body, maintained normally by adaptive responses that promote survival and well-being.
homogeneous Comprising similar parts or elements.
hospital-acquired pneumonia (HAP) Pneumonia that occurs 48 hours or longer after admission to the hospital and results from an infection that was not incubating at the time of admission.