BOX 17.13 Endotracheal (ET) and Tracheostomy Tube Complications

ET Complications

Damage to the Nasal Passages, Lips, or Eyes

- *During insertion*: Facial trauma, damage to the nasal structures, lips, or eyes
- *While in place:* Lip ulceration, pressure necrosis to the soft tissues, erosion of nasal septum, increased airway resistance from a small lumen tube
- *During and after extubation:* Nasal stricture

Damage to the Oropharynx

- *During insertion:* Traumatic damage to the oropharyngeal soft tissues, dental accidents, retropharyngeal or hypopharyngeal perforation
- *While in place:* Grooving of the hard palate from chronic pressure, dental deformities from constant pressure

Damage to the Larynx and Trachea

- *During insertion:* Soft tissue damage (bleeding and swelling), laryngeal trauma, laryngospasm
- *While in place:* Laryngeal injury (ulceration, edema, bleeding); laryngeal muscle dysfunction; subglottic edema; necrosis over the arytenoid cartilages and the vocal cords; trauma to mucosa covering the cricoid cartilage in infants; necrosis of tissue leading to the innominate artery and uncontrolled bleeding; tracheal injury (ulceration, edema, bleeding, tracheomalacia, cartilage and mucosal necrosis); laryngotracheal web formation; laryngotracheal granuloma; tracheal dilation; irritation of the carina; tracheoesophageal fistula; spontaneous dislocation of the tube (into the right mainstem, too high in trachea, extubation); squamous metaplasia of respiratory epithelium
- *During and after extubation:* Laryngospasm, laryngeal edema, glottic injury, laryngotracheal granuloma, laryngeal stenosis (glottic, subglottic), laryngeal motor dysfunction (vocal cord paralysis), cricoarytenoid ankylosis, tracheomalacia, tracheal dilation, tracheal stenosis, perichondritis, laryngeal chondritis, laryngotracheal web

Complications With Artificial Airways

- *During intubation:* Intubation of the right mainstem bronchus, bronchospasm, pulmonary aspiration, barotrauma, cardiopulmonary arrest, hypoxemia, cardiac arrhythmias, cervical and spinal cord injuries, patient discomfort
- *While in place:* Patient discomfort, difficulty in communicating, pain, retching, salivation, malnutrition, sinusitis, otitis media, atelectasis, pneumonia, pulmonary aspiration, decreased mucociliary transport, ineffective cough, contamination of the airway from silent aspiration during suctioning and invasion of the normal lung defenses, bronchial mucosal damage from suctioning
- *During and after extubation:* Hoarseness, sore throat, dysphagia, bronchospasm, aspiration, cardiac arrest

Mechanical Problems With Tubes

- Disconnection, kinking, obstruction from secretions, patient biting on the tube, displacement of the tube tip into the tracheal endothelial layer or against the side of the trachea or carina

Mechanical Problems Associated With the Cuff

- Compression of the tube by the cuff, excessive pressure (>25 mm Hg) from overinflation leading to tracheal necrosis, leaking or rupturing of the cuff causing inadequate ventilation, laceration of the cuff during insertion, leaking around the cuff preventing adequate ventilation, damage to the pilot balloon or connection preventing cuff inflation

Complications Associated With Tracheostomy*

- *During the surgical procedure:* Bleeding, thyroid injury, inappropriate incision position (too high or too low), injury to the recurrent laryngeal nerve, pneumothorax, tracheoesophageal fistula, subcutaneous emphysema, mediastinal emphysema, placement of the tube into the pretracheal space, cuff laceration during insertion, cardiac arrest, hypoxia
- *While in place:* Patient discomfort; infection of the wound or trachea, bleeding (skin vessel, tracheoarterial fistula); tracheal injury (inflammation, bleeding, ulceration, necrosis); tracheal dilation; web formation; perforation of trachea; granuloma formation; pseudomembrane formation; irritation of the carina; tracheoesophageal fistula; sepsis; mediastinitis; atelectasis; pneumonia; aspiration; subcutaneous emphysema; mediastinal emphysema; pneumothorax; decannulation; reduced mucociliary transport; ineffective cough; mechanical problems with the tube or cuff (see ET); squamous metaplasia of respiratory epithelium
- *During and after decannulation:* Tight stoma making decannulation difficult, patient discomfort, scarring, keloid formation, persistent open stoma, dysphagia, tracheal stenosis, tracheomalacia, tracheal granuloma, tracheal web formation, or tracheal dilation

*See Chapter 20 for additional information on tracheostomy tubes.

Care must be used during the insertion, maintenance, and removal of artificial airways. High-volume/low-pressure cuffs of good quality used for as short a time as possible will reduce the incidence of complications.

SUMMARY

- As with other forms of medical treatment, there are risks and complications associated with mechanical ventilation.
- *Barotrauma* implies trauma that results from using high pressures. *Volutrauma* implies damage from high distending volumes.
- *Ventilator-associated lung injury (VALI)* is the term generally used when referring to lung injury occurring in humans that has been identified as a consequence of mechanical ventilation (e.g., ventilator-associated pneumonia, air trapping, patient-ventilator asynchrony, extraalveolar gas [barotrauma] such as pneumothorax and pneumomediastinum).
- *Ventilator-induced lung injury (VILI)* describes lung injury that occurs at the level of the acinus. It is the microscopic level of

injury that includes biotrauma, shear stress, and surfactant depletion.
- Mechanical stress disrupts normal cell function, strains normal cell configuration, and can also lead to an inflammatory response and the release of injurious chemical mediators in the lungs.
- During controlled ventilation in which spontaneous ventilation is absent, alveolar collapse is most likely to occur in dependent areas of the lung. Because these are the areas that receive the most blood flow, an increased mismatching of ventilation and perfusion and increased dead space ventilation occur.
- Acute hypoventilation can occur in patients receiving ventilatory assistance if adequate alveolar ventilation is not provided. Hypoventilation will result in an increased P_aCO_2 and an acidotic pH. Rapidly rising P_aCO_2 levels and falling pH values can lead to serious problems.
- Hyperventilation results in a lower than normal P_aCO_2 and a rise in pH. Patient-induced hyperventilation is often associated with hypoxemia, pain and anxiety syndromes, circulatory failure, and airway inflammation.
- Ventilator-induced hyperventilation is generally caused by inappropriate ventilator settings. Weaning becomes more difficult when a patient has experienced prolonged hyperventilation.
- A number of factors can increase the risk for auto-PEEP, including the presence of COPD, high minute ventilation, increased airway resistance, increased lung compliance, high respiratory frequency, inverse-ratio ventilation, and low inspiratory flow.
- The presence of auto-PEEP will also make it more difficult for spontaneously breathing patients to trigger a ventilator breath even when sensitivity settings are appropriate.
- It is generally agreed that breathing enriched-O_2 mixtures for an extended period can increase the risk for pulmonary complications.
- Patient-ventilator asynchrony occurs when the patient's inspiratory efforts and flow demands are not met by the ventilator. Asynchrony is generally identified as trigger asynchrony, flow asynchrony, cycle asynchrony, mode asynchrony, PEEP asynchrony, and closed-loop ventilation asynchrony.
- Various problems can arise with the use of artificial airways. These include complications associated with the artificial airway itself, infection of the patient's airway, excessive heat to the airway from humidification systems, and inadequate or excessive humidification.

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

1. The peak pressure alarm is activated on a patient receiving mechanical ventilatory support. Peak pressures have increased from 25 to 50 cm H_2O in the past 30 minutes. While listening to a patient's breath sounds, the respiratory therapist notices absence of breath sounds over the entire right hemothorax. The patient is unconscious and nonresponsive. Which of the following actions would assist the respiratory therapist in determining the cause of the problem?
1. Percuss over the right thorax
2. Increase the pressure limit setting to 60 cm H_2O
3. Recommend a STAT chest radiograph
4. Deflate the ET cuff

A 1 only
B 2 only
C 4 only
D 1 and 3 only

2. Further evaluation of the patient reveals the following: A chest radiograph shows increased radiolucency on the right and absence of vascular markings on the right. The trachea is deviated to the left. Neck veins are distended. The patient is cyanotic. What immediate action(s) should the practitioner take at this time?
1. Call a physician STAT
2. Disconnect the patient from the ventilator and manually support ventilations
3. Increase the pressure limit
4. Increase the ventilator volume

A 1 only
B 3 only
C 1 and 2 only
D 3 and 4 only

3. A patient with ARDS is difficult to oxygenate: F_IO_2 is 0.8, PEEP is 12 cm H_2O, and P_aO_2 is 63 mm Hg. The physician requests that the respiratory therapist perform what maneuver that might help establish an optimum PEEP for the patient?

4. A patient with ARDS requires high PEEP levels. Plateau pressures are approximately 35 cm H_2O and PEEP is 16 cm H_2O. The patient's abdomen is turgidly overdistended. The respiratory therapist is concerned about the high plateau pressure. Should the respiratory therapist reduce the ventilating pressures?

5. VILI is associated with which of the following?
1. Washout or alteration of surfactant
2. Shear stress
3. Damage to pulmonary microvasculature
4. Possible release of inflammatory mediators from pulmonary cells

A 1 only
B 4 only
C 1 and 3 only
D 1, 2, 3, and 4

6. Overdistention injury of the lungs is associated with release of what substance(s) from the lungs into the bloodstream?

A Bacterial endotoxins
B Surfactant
C Cytokines
D Mucus

7. During mechanical ventilation, a patient appears to be "fighting the ventilator." He is anxious, agitated, and hypertensive. The patient's skin is hot and moist. The electrocardiogram shows peaked T waves and ST-segment depression.

Potassium level is elevated. What further assessment and therapy might be needed for this patient?

8. A patient on PC-CMV has initial ABG findings as follows: P_aO_2 of 101 mm Hg, P_aCO_2 of 60 mm Hg, and pH of 7.30. The respiratory therapist should:
A Increase minute ventilation to this patient
B Decrease pressure setting
C Change the ventilation mode
D Gradually, over several days, increase the minute ventilation to this patient

9. During mechanical ventilation, hyperventilation, particularly in patients with COPD, can cause which of the following?
1. Muscle twitching and tetany
2. High pH values
3. Air trapping
4. Cardiac arrhythmias
A 2 only
B 1 and 3 only
C 2, 3, and 4 only
D 1, 2, 3, and 4

10. During mechanical ventilation with VC-IMV, a respiratory rate of 4 breaths/min and a V_T of 600 mL, a patient has a spontaneous rate of 24 breaths/min between machine breaths. Pressure support is set at 5 cm H_2O. S_pO_2 is 95%. The spontaneous V_T ranges from 175 to 275 mL. The patient is using accessory muscles to breathe. ABG results are within normal range. The patient's ideal body weight is 70 kg. Which of the following might be appropriate?
A Increase the V_T setting on the ventilator
B Increase the F_IO_2
C Decrease the IMV rate
D Increase pressure support to 10 cm H_2O

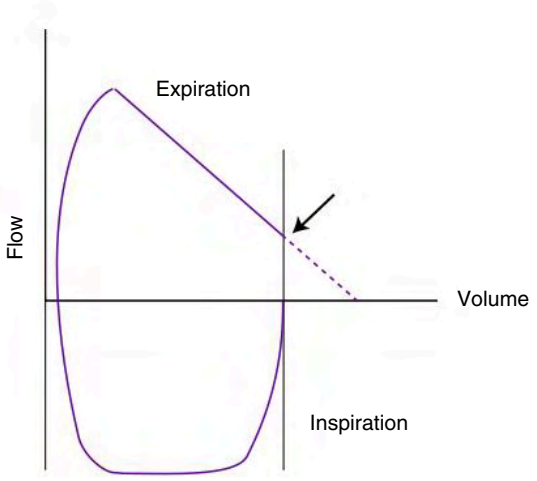
11. Reducing the WOB can be accomplished by using which of the following?
1. Increased inspiratory flow rates
2. Increased ventilatory sensitivity
3. Putting the patient in an upright position
4. Ensuring the patency of the ET
A 1 only
B 2 and 3 only
C 1, 3, and 4 only
D 1, 2, 3, and 4

12. A patient with ARDS is on 15 cm H_2O of PEEP after a recruitment maneuver. F_IO_2 is 0.85. Ventilation is good, but P_aO_2 is only 94 mm Hg. What might the respiratory therapist recommend for improving the patient's oxygenation?
A Set the F_IO_2 at 0.5 to avoid oxygen toxicity
B Reduce the PEEP to 10 cm H_2O
C Change the patient to the prone position
D Recommend an increase in minute ventilation

13. Mean airway pressure for a patient is 21 cm H_2O. The respiratory therapist increases mean airway pressure to 25 cm H_2O by increasing PEEP. P_{plat} is now 35 cm H_2O. This might result in which of the following?
A An increase in cardiac output
B VILI
C A decrease in FRC
D An increase in dead space

14. A patient will require 10 to 14 days of mechanical ventilation and is orally intubated with a standard ET. Ventilation and plateau pressures are adequate on VC-CMV with a volume of 450 mL and a rate of 14 breaths/min. ABGs are P_aO_2 of 101 mm Hg, a P_aCO_2 of 41 mm Hg, and a pH of 7.40. What changes might the respiratory therapist recommend at this time?
A Make no changes
B Increase the V_T
C Change the ET to provide CASS
D Decrease the F_IO_2

15. What type of problem is indicated in the following figure?



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CHAPTER 18

Troubleshooting and Problem Solving

OUTLINE

KEY TERMS

- Ascites
- Asynchrony
- Problem
- Pulmonary angiogram
- Thrombolytic therapy

LEARNING OBJECTIVES

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

1. Identify various types of technical problems encountered during mechanical ventilation of critically ill patients and describe the steps that can be used to protect a patient when problems occur.
2. Name at least two possible causes for each of the following alarm situations: low-pressure alarm, high-pressure alarm, low positive end-expiratory pressure (PEEP)/continuous positive airway pressure alarms, apnea alarm, low or high tidal volume alarm, low or high minute volume alarm, low or high respiratory rate alarm, low or high F_IO_2 alarm, low-source gas pressure or power input alarm, ventilator inoperative alarm, and technical error message.
3. Determine the cause of a problem using ventilator graphics from a patient-ventilator system.
4. Assess a patient on mechanical ventilation who is experiencing sudden dyspnea and identify the cause of the problem.
5. Describe the signs and symptoms associated with patient-ventilator asynchrony.
6. Explain the correct procedure for determining whether a problem originates with the patient or the ventilator during patient-ventilator asynchrony.
7. List four ways the addition of a nebulizer powered by an external source gas can affect ventilator function.
8. Recognize abnormalities in ventilator graphics and patient response in the event of inadequate gas flow delivery to a patient.
9. Identify the causes and potential problems related to electrolyte imbalances and their causes.
10. Recognize the signs and symptoms of a respiratory infection.
11. Identify a problem associated with an artificial airway or a mask used for noninvasive positive pressure ventilation.
12. Recognize the presence of auto-PEEP using ventilator graphics.
13. Suggest appropriate interventions for a patient who has experienced a right mainstem intubation and for a patient with a pneumothorax using physical assessment data.

14. Describe the potential problems associated with using a heated humidification system during mechanical ventilation.
15. Use a ventilator flow-volume loop to assess a patient's response to bronchodilator therapy.
16. Make recommendations about ventilator parameters for a patient with acute respiratory distress syndrome.
17. Recommend adjustment of flow-cycle criteria during pressure support ventilation based on ventilator graphics.

Troubleshooting, in the context of mechanical ventilation, involves the identification and resolution of technical malfunctions in the patient-ventilator system. Troubleshooting can be thought of as purposeful resolution of inappropriate and potentially dangerous situations.

Previous chapters have reviewed key concepts used in the management of patients receiving mechanical ventilation. This chapter discusses common technical problems encountered during mechanical ventilation and presents basic problem-solving strategies to ensure patient safety.

DEFINITION OF THE TERM *PROBLEM*

A **problem** can be defined as a situation in which a person finds discord or is uncomfortable with a matter that cannot be immediately resolved. Interestingly, a situation that might be uncomfortable for one person may not appear to be a problem for another person. For example, a respiratory therapist in the intensive care unit (ICU) may note that audible and visual alarms have activated for the intravenous pump. Unless it is part of this person's job function, the respiratory therapist might not perceive this as a problem that must be resolved and may simply contact the nurse. In contrast, if a ventilator alarm were activated, the same respiratory therapist would accept the responsibility of going to the patient's bedside to assess the situation and finding a solution to the problem. Therefore individuals must perceive an event as a problem and want to find a meaningful solution; that is, a situation must create discomfort that forces someone perceiving it to take action toward a resolution.

The ability to define a problem is particularly important to clinicians caring for critically ill patients. The lives of patients receiving ventilatory support may ultimately depend on the mechanical ventilator. Indeed, a matter of minutes can mean life or death. Because potential mishaps can occur with the patient-ventilator system, it is essential that clinicians develop the ability to identify and correct associated problems.

Solving Ventilation Problems

The first step in solving any problem is to assess the situation at hand carefully. The next step is to gather and analyze pertinent data, which should point to a number of viable solutions.

When a solution is attempted, the clinician's observations of the patient's response are critical. If the response is positive and leads to the correct remedy, the problem is resolved. If not, the clinician must undo what was attempted and try to determine the reason the particular solution failed before attempting another approach. If the problem cannot be resolved, the clinician should seek help.

Determining the cause of the problem can help prevent its recurrence. Note the steps taken to solve the problem shown in Box 18.1 and Fig. 18.1. Which individual or individuals first perceived it as a problem? What steps were taken to determine the cause? What was the final resolution?

Notice that the volume delivered by the ventilator (the inspiratory portion of the volume curve) is smaller than the volume exhaled, possibly because the patient is actively exhaling. A patient evaluation reveals that this is not the case.

The respiratory therapist, unable to explain the cause immediately, uses a respirometer to measure the volume coming from the volume delivery port, patient, and exhalation valve. The three volumes are equal. Still unable to determine the cause, the respiratory therapist contacts the individual who performs the maintenance checks on the ventilator. The respiratory therapist learns that the expiratory transducer was calibrated for a heated humidifier; however, a heat-moisture exchanger (HME) is being used with this patient. Although recalibrating the transducer would resolve the issue, it is not a life-threatening situation and therefore no immediate action is required.

PROTECTING THE PATIENT

It is important to understand that ensuring patient safety is the foremost obligation of the clinician. Whenever an alarm activates on a ventilator or monitoring device, the clinician should first make sure that the patient is receiving adequate ventilation and oxygenation. Initially, this can be accomplished by visually assessing the patient's level of consciousness, use of accessory muscles, and chest

BOX 18.1 Troubleshooting a Problem Using Ventilator Graphics

While doing rounds in the intensive care unit, a pulmonologist notices a ventilator graphic display showing the expiratory portion of the volume curve dropping below baseline (see Fig. 18.1). He contacts the respiratory therapist and inquires about possible causes.

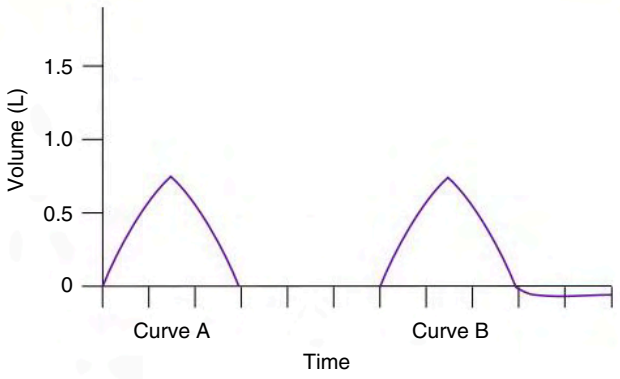

Fig. 18.1 Curve *A* shows a normal volume-time curve. Curve *B* shows the expiratory portion of the volume below the zero baseline.

wall movements. During this initial assessment, the alarm should be checked and silenced. Auscultation of the chest can establish the presence of adequate breath sounds, and checking the S_pO_2 can provide information about the patient's heart rate and oxygen (O_2) saturation. If the patient is in acute distress, demonstrating labored breathing, pallor, diaphoresis, and apparent anxiety, along with deterioration of breath sounds and a decreasing S_pO_2, immediate action is required. When a serious problem is detected, the patient may need to be disconnected from the ventilator and receive manual ventilation with a resuscitation bag. (After the patient is stabilized, the clinician can review the cause of the alarm activation and obtain help from other personnel if necessary.)

A self-inflating resuscitation bag can be used temporarily for ventilation of a distressed patient. When it is used properly, the resuscitation bag allows for the assessment of lung characteristics, because the clinician can check ("feel") a patient's lung and chest wall compliance and airway resistance manually. Manual ventilation must be performed cautiously to avoid inappropriate patterns of ventilation, excessive pressures (i.e., above 40 cm H_2O), and barotrauma.[1,2] Additionally, ventilator disconnection of a patient with acute respiratory distress syndrome (ARDS) who is ventilated with a high level of positive end-expiratory pressure (PEEP) (15–25 cm H_2O) can cause derecruitment of the lung, resulting in O_2 desaturation. Manual ventilation must also be used judiciously because disconnecting the patient from the ventilator can also result in contamination of the patient's airway, which in turn can increase the patient's risk for developing ventilator-associated pneumonia (see Chapter 14).

IDENTIFYING THE PATIENT IN SUDDEN DISTRESS

The term *patient-ventilator asynchrony* is typically used to describe the phenomenon that occurs when patients are unable to breathe comfortably with the mechanical ventilator. The phrase "fighting the ventilator" is sometimes used to describe an individual who is apparently doing well while receiving mechanical ventilation but suddenly develops acute respiratory distress.[3,4] This situation is particularly challenging for most clinicians because the patient is unable to verbalize his or her discomfort (Key Point 18.1). (Sometimes clinicians can gain valuable information from patients simply by asking direct ["yes" or "no"] questions.[5])

The sudden onset of dyspnea can be identified by observing the physical signs of distress (Fig. 18.2), including tachypnea; nasal flaring; diaphoresis; accessory muscle use; retraction of the suprasternal, supraclavicular, and intercostal spaces; paradoxical or abnormal movement of the thorax and abdomen; abnormal findings on auscultation; tachycardia; arrhythmia; and hypotension.[3,4] Pulse oximetry, capnograph readings, ventilator graphics, peak inspiratory pressure (PIP), plateau pressure (P_{plat}), and exhaled volumes may have changed and may provide information to help identify the cause of the problem.

***Key Point* 18.1** "The problems we created cannot be solved with the same level of thinking that we had when we created them."—*Albert Einstein.*

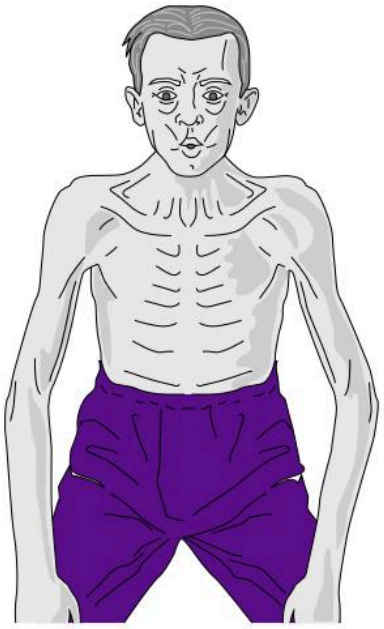

Fig. 18.2 Physical signs of severe respiratory distress. (From Copstead LC, Banasik, JL: *Pathophysiology,* ed 4, Philadelphia, PA, 2009, Saunders.)

BOX 18.2 Causes of Sudden Respiratory Distress in Patients Receiving Mechanical Ventilation

Patient-Related Causes
- Artificial airway problems
- Bronchospasm
- Secretions
- Pulmonary edema
- Pulmonary embolus
- Dynamic hyperinflation
- Abnormal respiratory drive
- Alteration in body posture
- Drug-induced problems
- Abdominal distention
- Pneumothorax
- Anxiety

Ventilator-Related Causes
- System leak
- Circuit malfunction or disconnection
- Inadequate F_IO_2
- Patient-ventilator asynchrony
  - Inappropriate ventilator support mode
  - Inappropriate trigger sensitivity
  - Inappropriate inspiratory flow setting
  - Inappropriate cycle variable
  - Inappropriate PEEP setting
  - Problems with closed-loop ventilation

From Tobin MJ: What should a clinician do when a patient "fights the ventilator"? *Respir Care.* 1991;36:395–406.

Patient-ventilator **asynchrony** can be caused by a number of factors. Box 18.2 lists the most common causes of sudden respiratory distress in patients receiving mechanical ventilation. (See

BOX 18.3 Management of Sudden Severe Respiratory Distress in a Patient on Mechanical Ventilation

1. Disconnect the patient from the ventilator.
2. Begin manual ventilation using a self-inflating resuscitation bag containing 80% to 100% O_2; maintain normal ventilating pressures, and use a PEEP attachment if the patient has been on high PEEP ($\geq$10 cm H_2O).
3. Manually evaluate compliance and resistance through bag ventilation.
4. Perform a rapid physical examination and assess monitored indexes and alarms.
5. Check the patency of the airway by passing a suction catheter.
6. If death appears imminent, treat the most likely problems: pneumothorax and airway obstruction.
7. Once the patient's condition has stabilized, perform a more detailed assessment and provide any additional treatment required.

Modified from Tobin MJ: What should a clinician do when a patient "fights the ventilator"? *Respir Care* 36:395–406, 1991.

the section in Chapter 17 on steps to reduce the work of breathing [WOB] during mechanical ventilation.)

Evaluation of the ventilator graphics and settings can be used to resolve most of these problems. For example, autotriggering of breaths can occur if the sensitivity setting (inspiratory trigger) is too sensitive, resulting in an excessive number of triggered breaths. Patient-ventilator asynchrony is often associated with a patient's feelings of panic, which can be relieved by encouraging the patient to not "fight the ventilator" and relax while breathing with the ventilator. Selection of the appropriate mode and correct adjustment of the ventilator can eliminate most problems and reduce the need for sedation. (See Chapter 17 for more detailed information on patient-ventilator asynchrony.)

Identifying the cause of patient-ventilator asynchrony can be accomplished using a relatively simple approach (Box 18.3). If the patient is in *severe* distress, the first step is to disconnect the patient from the ventilator and carefully ventilate the patient using a manual resuscitation bag (i.e., avoiding excessive airway pressure). If the patient's distress resolves immediately, the problem is with the ventilator; if the distress does not resolve, the problem is typically because of the patient experiencing anxiety or pain (Key Point 18.2).

***Key Point* 18.2** In cases in which patient-ventilator asynchrony is identified, the clinician should disconnect the patient from the ventilator and carefully provide ventilation to the patient using a manual resuscitation bag.

PATIENT-RELATED PROBLEMS

The most common patient-related problems encountered during mechanical ventilation involve the placement and patency of the artificial airway or the presence of a pneumothorax, bronchospasm, and excessive secretions (see Box 18.2).

BOX 18.4 Causes of Airway Problems That Can Lead to Sudden Respiratory Distress

- Tube migration (flexion and extension of the head and neck can move the endotracheal tube [ET] in the airway an average of 2 cm down and up, respectively)
- Migration of the ET above the vocal cords
- Migration of the ET into the right mainstem bronchus
- Rupture or leakage of the ET cuff
- Kinking of the ET
- Patient biting the ET
- Airway secretions and mucous plugging of airways
- Impingement of the ET on the carina or airway
- Cuff herniation over the end of the ET (less of a problem with the current endotracheal cuffs)
- Development of a tracheoesophageal fistula
- Rupture of the innominate artery

BOX 18.5 Emergency Treatment for Rupture of the Innominate Artery

Rupture of the innominate artery is a potentially serious airway complication, and the mortality rate for this condition is high. It is usually seen in the first 3 weeks after a tracheostomy. The immediate indication is blood spurting from the tracheotomy site.

To stop the hemorrhage, the cuff should be overinflated or pressure should be applied internally with a finger inserted through the stoma. The finger is inserted as far as possible toward the carina and then pulled forward in an attempt to compress the artery against the posterior aspect of the sternum.

Airway Problems

Examples of acute airway problems include kinking of the endotracheal tube (ET), impingement of the tube on the carina, and displacement of the tube upward, above the vocal cords or into the right mainstem bronchus (Box 18.4). Rupture of the innominate artery can also occur (this is usually seen with tracheostomy tubes) (Box 18.5).[4] An unusual case study reported a fatal tracheal-innominate artery fistula caused by fixed positioning of the Hi-lo Evac tube to the left upper molar.[6] (This type of ET is used for continuous aspiration of the subglottic secretions.) Apparently, the continuous suction pressure against the soft tissue of the trachea for an extended period resulted in erosion of the tissue and, eventually, exposure of the innominate artery. This catheter has been redesigned to reduce the risk for this complication.

A quick check of the patient's oral cavity can sometimes reveal whether the ET is kinked or the patient is biting it. The clinician can easily determine whether the tube has been inserted too far or if it is too shallow in the airway by looking at the tube's centimeter markings at the airway opening. A properly positioned oral ET typically shows a centimeter marking at the teeth at approximately 23 cm for men (range, 22–24 cm) and 21 cm for women (range, 20–22 cm). If the centimeter markings are less than the ranges cited, the tube is too high in the airway. If the marking is greater than the ranges cited, the tube has migrated deeper into the airway.

If a serious airway problem cannot be resolved quickly, the clinician should provide ventilation of the patient manually to assess airway patency. If ventilation of the patient cannot be provided in this manner, an appropriately sized suction catheter should be passed through the artificial airway. If the catheter does not pass freely (and the patient is not biting on the ET), the tube is most likely obstructed. The cuff should then be deflated, which may allow some air to move around the tube in spontaneously breathing patients. If the obstruction cannot be cleared, the tube must be removed immediately and the patient ventilated with a resuscitation bag until reintubation is possible.[7,8]

Pneumothorax

Pneumothorax is another possible problem that must be detected during positive pressure ventilation and treated quickly. Pneumothorax can be recognized as increased WOB if a patient is conscious. For example, the patient may demonstrate nasal flaring, use of accessory muscles, uneven chest wall movement, and absence of breath sounds on the affected side. Auscultation and percussion of the chest, cardiovascular assessment, and ventilating pressure evaluations usually can distinguish a pneumothorax from other problems. Table 8.1 provides physical and radiological findings commonly seen in patients with pulmonary disorders such as pneumothorax, asthma, emphysema, pneumonia, and pleural effusion.

If a tension pneumothorax is strongly suspected and cardiopulmonary arrest is imminent, a 14- or 16-gauge needle is inserted into the second intercostal space at the midclavicular line, over the top of the rib on the affected side. If the patient's condition is stable, a confirming chest radiograph can be obtained with chest tube placement and pleural drainage. The following case study describes the case of a patient with sudden respiratory distress.

Clinical Scenario: A Case of Sudden Respiratory Distress

A 45-year-old woman has been receiving ventilation for 3 weeks for respiratory failure arising from severe asthma. Treatment has included administration of sedatives, antidepressants, and corticosteroids and bronchodilator therapy. Several attempts at weaning her from the ventilator, including a course of noninvasive positive pressure ventilation, have been unsuccessful. At 36 hours after successful placement of a tracheostomy tube, the patient's ventilator high-pressure alarm activates. The respiratory therapist performs an assessment and suctions the patient's airway, which is determined to be patent. Breath sounds are diminished but present bilaterally. Within 10 minutes the high-pressure alarm activates again. The respiratory therapist returns to the room and notes that the patient appears to be coughing forcefully. The patient suddenly develops ventricular tachycardia and loses consciousness. The respiratory therapist finds it extremely difficult to apply ventilation through the tracheostomy tube. The tube is pulled, and the patient is immediately and successfully reintubated with an oral ET. Unfortunately, the resuscitation effort fails, and a postmortem chest radiograph shows bilateral pneumothoraxes.

The clinicians conclude that thickened secretions had blocked the airways, causing air trapping distally, and that increased pressures in the thorax from the patient's forceful coughing caused the bilateral pneumothoraxes.

Bronchospasm

Bronchospasm may be manifested as dyspnea, wheezing, evidence of increased WOB (i.e., such as heightened use of accessory muscles); lack of coordination of chest or abdominal wall movement; retraction of the suprasternal, supraclavicular, and intercostal spaces; and increased airway resistance (R_{aw}) (as evidenced by increased PIP and transairway pressure [PIP − P_{plat}]). Wheezing associated with increased R_{aw} from airway hyperreactivity, such as occurs with asthma, can be treated with bronchodilators and parenteral corticosteroids. Wheezing can also be associated with cardiogenic problems and pulmonary emboli.

Secretions

Evaluation of the patient's secretions can be useful in differentiating a variety of problems. (See Table 12.2 for a list of sputum findings and possible associated problems.) Drying of secretions is most often associated with inadequate humidification (Key Point 18.3). Copious amounts of secretions can occur with pulmonary edema and certain pulmonary disorders (e.g., cystic fibrosis). Depending on their characteristics, secretions may also suggest the presence of a respiratory infection; however, this is not usually a problem with a sudden onset. It is essential that the patient is provided appropriately warmed and humidified air.[8] Suctioning should be performed only when indicated rather than according to a fixed schedule. Bronchial hygiene may include postural drainage and percussion and therapeutic bronchoscopy. (See Chapter 12 for additional information about airway clearance techniques.)

***Key Point* 18.3** Drying of secretions is most often associated with inadequate humidification.

Pulmonary Edema

Pulmonary edema can be either cardiogenic or noncardiogenic in origin and should be managed accordingly. Cardiogenic pulmonary edema can occur suddenly and often manifests with thin, frothy, white to pink secretions. In cases in which cardiogenic pulmonary edema is suspected, the clinician should check for additional evidence of a cardiac problem, such as electrocardiographic findings, elevated blood pressure, evidence of neck vein distention, a history of heart disease, and data from a pulmonary artery catheter, if available (see Chapter 11). Cardiogenic pulmonary edema and heart failure can often be managed successfully with medications that reduce preload, increase contractility, and reduce afterload, such as furosemide (Lasix), digoxin (Lanoxin), enalapril maleate (Vasotec), and morphine.

Noncardiogenic pulmonary edema or pulmonary edema that is caused by an increase in pulmonary capillary permeability (e.g., ARDS usually develops over a day or two and is not a sudden-onset problem. The management of ARDS is discussed in Chapter 13.

Dynamic Hyperinflation

Dynamic hyperinflation of the lungs that occurs with auto-PEEP can lead to difficulty with ventilator triggering.[9] Auto-PEEP can also cause cardiovascular problems, such as hypotension and reduced cardiac output. One of the best ways to detect the presence of auto-PEEP is through the evaluation of the ventilator waveforms.

Auto-PEEP should be suspected whenever flow does not return to baseline in either a flow-time scalar or a flow-volume loop. Efforts to reduce auto-PEEP can be aided by reducing the inspiratory time (T_I), minute ventilation ($\dot{V}_E$), and R_{aw}. As previously mentioned, auto-PEEP hinders a patient's ability to trigger the ventilator (see Fig. 7.1). (See Chapter 7 for a discussion of how raising the applied PEEP [extrinsic PEEP] can be used to ease breath triggering in patients with chronic obstructive pulmonary disease [COPD] when auto-PEEP cannot be completely eliminated.)

Abnormalities in Respiratory Drive

Inadequate output from the respiratory centers of the brain can occur as a result of heavy sedation, acute neurological disorders, or neuromuscular blockage.[10] However, these conditions are more likely to reduce respiratory function than to produce sudden respiratory distress. Increased output from the respiratory centers is associated with pain, anxiety, increased peripheral sensory receptor stimulation, medications, increased ventilatory needs, and inappropriate ventilator settings.

Change in Body Position

Changes in the patient's position can be associated with accidental extubation, bending and twisting of the patient circuit, and in some cases alterations in the patient's level of oxygenation. Reductions in oxygenation can occur with repositioning of the patient so that the diseased lung is placed in a dependent position. It can also occur with sudden airway obstruction by a mucous plug, secretions, or clot migration that leads to a pulmonary embolus. (Changing the patient's body position can cause a thrombus to dislodge and migrate, causing a pulmonary embolus.)

Drug-Induced Distress

It is important to recognize that when acute respiratory distress develops in a patient on ventilation in the ICU and the cause cannot be readily identified, possible causes may be related to medications the patient takes or possible chemical dependency. For example, intravenous morphine, which is commonly used to relieve pain, can cause nausea, hypotension, disorientation, hallucinations, fever, constipation, and respiratory arrest. This may be especially true in older patients, whose ability to metabolize and clear medications may be diminished.

Patients with chemical dependency (alcohol, drug, or tobacco) present additional clinical management issues, particularly if the ICU staff is unaware of a patient's chemical dependency. Sudden interruption of use of the chemical by the patient may cause symptoms of withdrawal syndrome, such as anxiety, restlessness, irritability, insomnia, and inability to focus attention.

Abdominal Distention

Abdominal distention can be associated with air being introduced into the stomach (e.g., via a nasogastric tube) and with a number of disorders, including **ascites**, abdominal bleeding or obstruction, and liver or kidney disorders. Some of these conditions have a slower onset than others, but all cause an upward pressure on the diaphragm, restricting its downward movement. This restriction of diaphragm movement can lead to atelectasis in the basilar areas of the lungs, ventilation-perfusion abnormalities, and hypoxemia.

Pulmonary Embolism

Pulmonary embolism (PE) is another acute-onset problem that can lead to patient-ventilator asynchrony. The rapid onset of hypoxemia from a large embolus leads to all the signs of distress previously described. The patient typically demonstrates the presence of bilateral breath sounds, indicating that both lungs are receiving ventilation (i.e., PE interferes with perfusion, not ventilation). With PE the heart rate, blood pressure, and respiratory rate are elevated. Even with high ventilator rates and flows, the patient may use accessory muscles to breathe and may become pale. Checking airway patency and ventilating pressures and increasing the F_IO_2 may not reverse the arterial O_2 desaturation (S_aO_2). Disconnection from the ventilator and manual ventilation also will not help relieve the distress.

PE is an emergency that often leaves the clinician feeling helpless to determine the cause and treatment (Case Study 18.1). Capnography findings can, however, provide a clue to the presence of a PE. A decrease in the end-tidal carbon dioxide ($P_{et}CO_2$) value compared with previous readings and a widening of the arterial-to–end-tidal partial pressure CO_2 gradient ($P_{(a-et)}CO_2$) may suggest the presence of an embolus. Demonstration of the presence of a PE usually requires a **pulmonary angiogram** and computed tomography. **Thrombolytic therapy**, such as the use of alteplase (recombinant tissue plasminogen activator [tPA]; Actilyse) or reteplase (Retavase), may be appropriate.

VENTILATOR-RELATED PROBLEMS

Clinicians typically rely on algorithms to identify problems with the patient-ventilator circuit. A relatively quick way to identify whether the problem is a ventilator-related problem is to determine whether the patient's respiratory distress is relieved by manual ventilation with 100% O_2 via a self-inflating resuscitation bag. If the intervention relieves the respiratory distress, the problem is probably associated with the ventilator or applied ventilator-management strategy.

Case Study 18.1

Evaluating Severe Respiratory Distress in a Ventilated Patient

While performing a patient-ventilator check, the respiratory therapist notes that the patient suddenly develops signs of severe respiratory distress. The low O_2 saturation alarm on the pulse oximeter activates. Breath sounds are equal bilaterally with no change from previous findings. The respiratory therapist disconnects the patient and performs manual ventilation using 100% O_2. A suction catheter passes through the patient's ET without difficulty; however, the patient's distress continues and O_2 saturation remains low. The therapist notes that the capnometer reading for $P_{et}CO_2$ has changed from its previous value of 35 mm Hg to 27 mm Hg. Arterial blood gas analysis indicates that the P_aCO_2 has not changed but the P_aO_2 decreased to 20 mm Hg. The $P_{(a-et)}CO_2$ has increased from 6 to 14 mm Hg. What is the cause of the patient's respiratory distress?

Leaks

Activation of low-pressure, low-volume, or low-$\dot{V}_E$ alarms typically indicates a leak in the patient-ventilator circuit is present. (As discussed later in this chapter, the presence of a leak can be verified by analyzing the various ventilator graphics available on most ICU ventilators.) Leaks are commonly caused by disconnection of the patient from the ventilator; if this is the problem, the ventilator circuit simply needs to be reconnected to the patient's artificial airway.

Leaks can also occur around the cuff of the ET. To determine whether this is the problem, the clinician should auscultate over the tracheal area for abnormal breath sounds during inspiration. To correct a cuff leak, the cuff is reinflated and the cuff pressure is rechecked. If a minimum leak technique is used, the cuff leak and tracheal air sounds should be present at peak inspiration under normal conditions. (See Chapter 8 for more detailed information on checking the circuit and evaluating for leaks.) Small ETs, such as those used with neonates, do not have cuffs and therefore allow for a minimum amount of air to leak around the tube. Migration of the ET into the upper airway, above the vocal cords, is another possible cause of a leak associated with the ET.

Circuit leaks can also occur at junctions in the patient circuit where connections exist. These may include connections at water traps, humidifiers, and HMEs; inline closed-suction catheters; temperature probes; inline metered-dose inhaler chambers; proximal airway pressure lines; capnograph (CO_2) sensors; and unseated or leaking exhalation valves. Another, less common source of leaks is a pleural drainage system. In this situation, compensation for pleural leaks can sometimes be accomplished by increasing volume delivery to the patient. The amount of air leaking through the pleural drainage system can be determined by comparing the inspiratory and expiratory tidal volume (V_T).

Leak checks are typically performed when equipment is prepared for patient use, but these checks can also be performed while the equipment is in use (e.g., if a patient circuit is changed). Newer ICU ventilators can automatically perform the patient-ventilator circuit test. A leak check can also be performed manually if a leak develops during patient ventilation. While the patient is ventilated manually, the clinician changes the ventilator mode to volume-controlled ventilation (VC-CMV) and sets the V_T to 100 mL, the flow to 20 L/min, the inspiratory pause to 2 seconds, and the pressure limit to maximum. The patient Y-connector is occluded with sterile gauze, and the ventilator is cycled manually. The circuit pressure that develops during inspiration should plateau and hold at that level, falling no more than 10 cm H_2O during a 2-second pause. If the pressure falls more than this, a significant leak is present and must be corrected. If the leak cannot be corrected quickly and easily, a change of equipment may be necessary.

Inadequate Oxygenation

A condition of inadequate oxygenation is usually signaled by activation of a low S_pO_2 alarm. Patients typically demonstrate tachycardia (although bradycardia may also occur) along with other signs of hypoxemia. (See Table 4.1 for the signs and symptoms of hypoxia.) Note that arterial blood gas analysis is required to confirm the presence of hypoxemia. It is important to recognize that worsening hypoxemia can be an ominous sign of deteriorating lung function.

Inadequate Ventilatory Support

Inappropriate $\dot{V}_E$ and ventilator settings can cause increased WOB, which can ultimately lead to patient-ventilator asynchrony. Respiratory acidosis and hypoxemia may also be seen in cases in which the patient experiences an increased WOB. (Chapter 17 contains more information on the effects of increased WOB.)

Trigger Sensitivity

As previously mentioned in Chapter 17, setting an appropriate trigger sensitivity is important to ensuring effective ventilatory support that minimizes the WOB for the patient. Autotriggering is a sign that the trigger sensitivity is set too low. Lack of ventilator response to a patient's inspiratory efforts may be the result of incorrect sensitivity settings, low-flow settings, or a poorly responsive internal demand valve. Trigger sensitivity may also be altered when a nebulizer is being used that is powered by an external gas source, which can blunt the machine's ability to sense a patient breath. Other causes are water in the inspiratory line and the presence of auto-PEEP.

Inappropriate sensitivity settings can often be corrected by simply increasing or decreasing the sensitivity setting. If these changes do not solve the problem, each of the other causes mentioned must be addressed.

Inadequate Flow Setting

A low inspiratory gas flow can be corrected by increasing the flow setting or changing the flow pattern, such as using a descending ramp rather than a rectangular flow pattern. A concave inspiratory pressure scalar during VC-CMV indicates active inspiration with inadequate flow (Fig. 18.3).[11] Changing the mode of ventilation may also be an effective means of providing adequate flow to the patient. For example, switching from VC-CMV to pressure ventilation with a volume target (e.g., pressure-regulated volume control) can change the flow pattern and sometimes relieve distress.

Other Examples of Patient-Ventilator Asynchrony

In addition to an inappropriate sensitivity setting and inadequate flow, other types of patient-ventilator asynchrony can occur. For example, auto-PEEP, an increased ventilatory drive, or the need for sedation may manifest as patient-ventilator asynchrony. In such cases the airway pressure (P_{aw}) usually fluctuates dramatically; the respiratory therapist must determine the cause and correct the problem (Box 18.6).[11,12] For example, with auto-PEEP the patient may have trouble triggering a breath. For patients with COPD with airflow obstruction, setting low levels of PEEP may alleviate the problem (see Box 7.1 and Fig. 7.1A and B).[9,13]

Asynchronous breathing may also be seen in patients with COPD when pressure support ventilation (PSV) is used. COPD patients often show active short inspirations and active long expirations.[14] If the patient begins to exhale actively during the inspiratory phase of PSV, the flow may not drop to the necessary cycling value to end inspiration on the pressure-supported breath, resulting in a sudden rise in the scalar at the end of the breath.[15] This problem can be avoided by having these patients use a ventilator with adjustable flow-cycling characteristics.

During PSV, another problem may occur in patients with a high-flow demand when pressures are set too low. In markedly distressed patients, the transition to exhalation may be affected by the ventilator's ability to respond quickly and open the exhalation valve.[16]

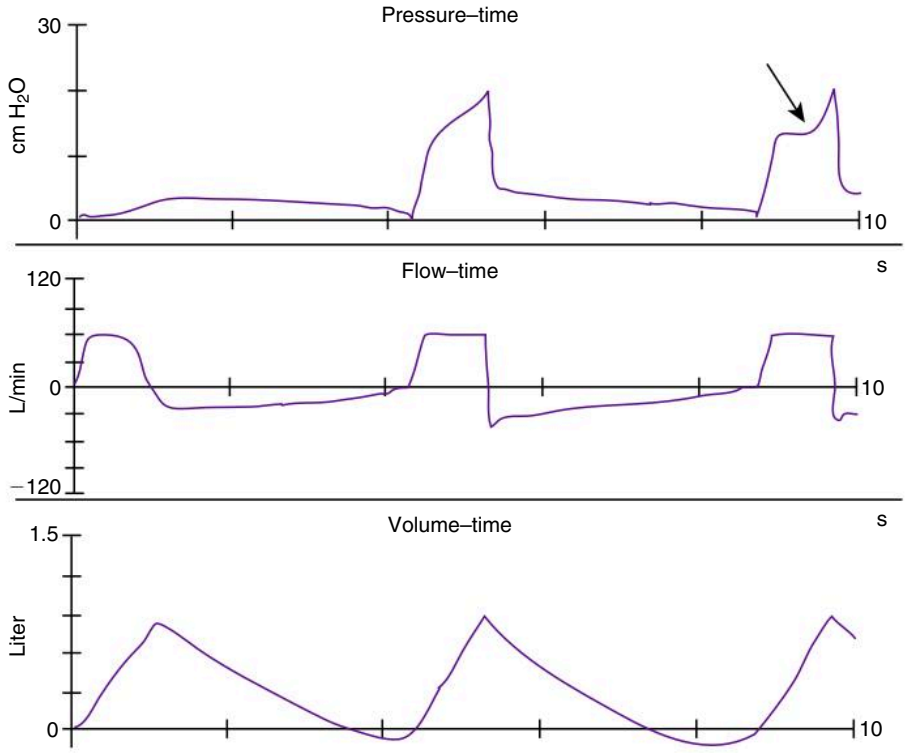

Fig. 18.3 Before the delivery of a mandatory breath (constant flow, volume-controlled continuous mandatory ventilation [VC-CMV]), patient effort reaches the trigger threshold, and a normal breath is delivered (breath on *left*). The second breath *(right)* is patient triggered, but the pressure curve *(arrow)* is concave because of the patient's active attempt to inhale. (From Nilsestuen JO, Hargett KD: Using ventilator graphics to identify patient-ventilator asynchrony, *Respir Care* 50:202—234, 2005.)

BOX 18.6 Resolution of Various Types of Ventilator Asynchrony*

- *Trigger asynchrony:* Confirm that the sensitivity level has been set appropriately, air trapping (auto-PEEP) is not present, and the patient is not agitated.
- *Flow asynchrony:* Ensure the flow is adequate and the flow delivery curve is appropriate.
- *Cycle asynchrony:* With pressure support ventilation (PSV), ensure that the patient is not exhaling actively; with pressure control ventilation (PC-CMV), make sure the duration set for the T_I is not too long.
- *Mode asynchrony:* Occurs when more than one type of breath is delivered, such as with intermittent mandatory ventilation (IMV); another mode may be more appropriate for the patient.
- *PEEP asynchrony:* Causes include overdistention from excess PEEP and atelectasis and atelectrauma from a PEEP setting that is too low; make sure PEEP is set at an appropriate level.
- *Closed-loop ventilation asynchrony:* Closed-loop mode may not be performing as needed for a particular patient. Make sure current settings are appropriate. Consider selecting another mode.

*See Chapter 17 for additional information.

If all efforts have been made to resolve a patient-ventilator asynchrony problem and a solution cannot be found, medications may be necessary. Sedatives, used either alone or with neuromuscular blocking agents, may be the most effective method to relieve severely distressed patients (see Chapter 15). However, the clinician must make a systematic attempt to find the cause of the problem and correct it as quickly as possible before recommending medications.

COMMON ALARM SITUATIONS

Ventilators and monitoring equipment have a number of alarms to notify the practitioner of changes in a patient's status. Appropriate use of these alarms is essential for patient safety (see Chapter 7).[17]

Low-Pressure Alarm

As already discussed, low-pressure alarms are most often activated by leaks (Box 18.7).[18,19] When a low-pressure alarm activates, the clinician should first check to ensure the patient is receiving ventilation. If the alarm occurs because the patient is inadvertently disconnected from the ventilator, the patient should simply be reconnected. Otherwise, the patient may need ventilation with a manual resuscitator until the source of the leak is identified. Once the problem has been identified, the clinician should reset the alarm, making sure that it is set about 5 to 10 cm H_2O below PIP.

BOX 18.7 Common Causes of Low-Pressure Alarms

- Patient disconnection
- Circuit leaks
- Disconnection of the inspiratory or expiratory tubing of the ventilator circuit
  - Humidifiers
  - Filters
  - Water traps
  - Inline metered-dose inhalers
  - Inline nebulizers
  - Proximal pressure monitors
  - Flow monitoring lines
  - Exhaled gas monitoring devices
  - Inline closed-suction catheters
- Temperature monitors
- Exhalation valve leaks
  - Cracked or leaking valves
  - Unseated valves
  - Improperly connected valves
- Airway leaks
  - Use of minimum leak technique
  - Inadequate endotracheal tube (ET) cuff inflation
  - Leak in pilot balloon or cut pilot balloon
  - Rupture of ET cuff
  - Migration of ET into upper airway above the vocal cords
- Chest tube leaks

BOX 18.8 Common Causes of High-Pressure Alarms

Conditions Related to the Airway
- Coughing
- Secretions or mucus in the airway
- Patient biting on the ET (oral intubation)
- Kinking of the ET inside the mouth or in the back of the throat
- Impingement of the ET on the trachea or carina
- Changes in the position of the ET (i.e., migration of the tube into the right mainstem bronchus)
- Herniation of the ET cuff over the end of the tube

Conditions Related to the Lungs
- Increased airway resistance (e.g., secretions, mucosal edema, bronchospasm)
- Decreased compliance (e.g., pneumothorax, pleural effusion)
- Patient-ventilator asynchrony

Changes in the Ventilator Circuit
- Accumulation of water condensate in the patient circuit
- Kinking in the inspiratory circuit
- Malfunction in the inspiratory or expiratory valves

High-Pressure Alarm

High-pressure alarms are incorporated into all current ICU ventilators (Box 18.8).[18,19] High-pressure limits are usually set about 10 cm H_2O above PIP. Conditions leading to activation of high-pressure alarms can be categorized as airway problems, changes in the patient's lung characteristics or patient-related conditions, and problems related to the patient-ventilator circuit.

Audible and visible high-pressure alarms are typically activated when a patient coughs or bites on the ET. High-pressure alarms also will become activated when secretions build up in the patient's airway. Coughing usually is self-limited and does not require treatment. Use of a bite block or oropharyngeal airway may help prevent unresponsive patients from biting on the ET. Some commercially available ET holders have built-in bite blocks. Conscious and responsive patients can be instructed not to bite on the tube; sometimes they listen. Secretions can often be removed by suctioning.

Conditions that adversely affect a patient's lung characteristics (e.g., increased airway resistance [R_{aw}] or decreased lung compliance [C_L]) or spontaneous breathing efforts can also trigger high-pressure alarms. Examples of conditions that increase R_{aw} are bronchospasm, secretions, and mucosal edema. Conditions most often seen in the ICU that are associated with decreased C_L include ARDS, pneumonia, pneumothorax, pleural effusions, abdominal distention, and ascites. Identifying R_{aw} and C_L changes can be identified on physical examination by listening to breath sounds and evaluating changes in the PIP and P_{plat} (see Table 18.1) and by interpreting ventilator graphics (Case Studies 18.2 and 18.3; Fig. 18.4).

TABLE 18.1 Patterns of Alteration in Thoracic Pressure-Volume Relationships

	CASE 1[a]		CASE 2[b]	
Parameter	1 Hour Ago	Now	1 Hour Ago	Now
Tidal volume (mL)	600	600	600	600
Plateau pressure (cm H_2O)	10	10	10	30
Peak pressure (cm H_2O)	20	40	20	40
Static compliance (mL/cm H_2O)	60	60	60	20
Dynamic characteristic (mL/cm H_2O)	30	15	30	15

[a]The plateau pressure has not changed in Case 1; therefore an airway problem should be suspected.
[b]The plateau pressure has increased in Case 2, but no increase is seen in the gradient between the peak pressure and the plateau pressure; therefore a pneumothorax, mainstem intubation, or atelectasis should be suspected.
From Tobin MJ: What should a clinician do when a patient "fights the ventilator"? *Respir Care* 36:395–406, 1991.

A high-pressure alarm may also be triggered when a patient actively breathes out of synchrony with the ventilator. PIP rises if the patient actively exhales while the ventilator is in the inspiratory phase, and this can activate the high-pressure alarm. A change in a ventilator setting or in the patient's condition that results in air trapping in the lungs (i.e., auto-PEEP) also causes the PIP to rise and may trigger an alarm.

Case Study 18.2

Evaluating Peak Inspiratory Pressure (PIP) and Plateau Pressure (P_{plat}) in Volume-Controlled (VC) Ventilation

A 28-year-old man is receiving ventilatory support with VC-CMV. A high-pressure alarm has activated on several occasions over the past 4 hours. The following parameters were noted:

Time	Volume (L)	PIP (cm H_2O)	P_{plat} (cm H_2O)
12:00	0.5	25	19
14:00	0.5	34	29
16:00	0.5	39	33

The patient had been admitted 72 hours earlier after a motor vehicle crash in which he sustained chest trauma without pneumothorax. He has been receiving ventilatory support for the past 36 hours because of severe hypoxemia and increased WOB that progressed to acute respiratory failure.

At 16:00 hours the respiratory therapist notes bilateral crackles, particularly in the lung bases, where the percussion note was dull. A chest radiograph shows bilateral infiltrates. What caused the increase in PIP and P_{plat}? Suggest a possible diagnosis for this patient based on the clinical findings.

Case Study 18.3

Evaluating PIP and Volume in Pressure Control Ventilation

A patient with a history of asthma is receiving mechanical ventilation in the PC-CMV mode after open heart surgery. Occasionally the high-pressure alarm is activated when the patient coughs or appears to try to exhale forcibly. The low V_T alarm is also activated several times. The following values were obtained while monitoring the patient:

Time	Volume (L)	PIP (cm H_2O)	*f* (breaths/min)
09:00	0.75	25	8
10:00	0.68	25	9
11:00	0.6	25	11

Fig. 18.4 shows the flow-volume loop measured on the patient at 11:00 hours. Breath sounds reveal bilateral scattered wheezes. What caused the change in volume delivery? What therapy would you recommend?

It is important to recognize that PIP may also be elevated if problems arise in the patient-ventilator circuit. Accumulated water from condensation can lead to oscillations in the gas flow through the circuit and subsequent fluctuations in P_{aw}. Water in the circuit can lead to autotriggering, increased PIP, and sometimes activation of a high-pressure alarm. Kinks in the circuit can also lead to an increased PIP and alarm activation.

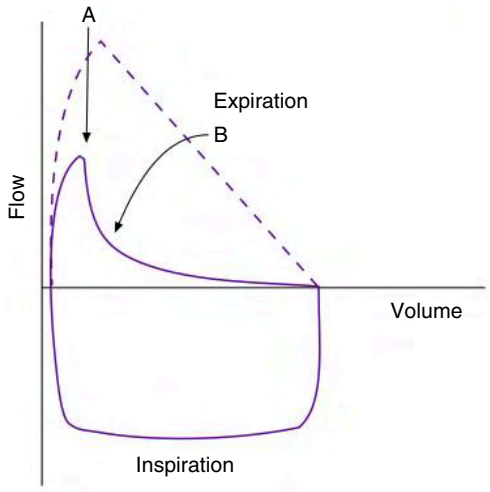

Fig. 18.4 Flow-volume loop. Inspiration occurs below the *x*-axis, and expiration occurs above it. *Arrow A* indicates the peak expiratory flow. *Arrow B* indicates increased expiratory resistance with reduced expiratory flow *(solid line)*. The *dashed line* represents the predicted expiratory flow loop for this patient.

Several possible problems should be considered when circuit pressures are rising. For example, nebulized medications can accumulate on the expiratory filter or the exhalation valve, leading to increased resistance to gas flow through the filter or valve. Consequently, the exhalation valve may be unable to open completely. If the valves are malfunctioning, they should be cleaned or replaced and the sensor should be recalibrated to correct the problem. HMEs also can accumulate excessive secretions, which can lead to increased flow resistance.[20] Notice how slowly the expiratory flow and expiratory volume return to baseline in Fig. 18.3. This is a graphic example of increased expiratory resistance. (NOTE: A partly obstructed HME can also produce this type of expiratory feature.)

Regardless of the cause of a high-pressure alarm, the patient's safety must always be the clinician's first priority (Key Point 18.4). The clinician must ensure that the patient has a patent airway and is receiving ventilation and oxygenation.

***Key Point* 18.4** Whenever a problem arises, the clinician's first priority always must be the patient's safety.

Low-PEEP/CPAP Alarms

Low-PEEP/continuous positive airway pressure (CPAP) alarms activate when the airway pressure falls below the desired baseline during PEEP or CPAP. This may occur when the ventilator cannot compensate for a leak in the circuit. Another possible cause of a low-pressure alarm is active inspiration by the patient. Active inspiration can cause the pressure to drop below the alarm setting. The machine sensitivity may not be responsive enough to the patient's effort, or the ventilator demand valve may not open quickly enough in response to flow demand. For example, if inspiratory gas flow is set too low and the demand valve does not respond to the patient's inspiratory effort, the PEEP level drops, which may activate the alarm.

Apnea Alarm

Activation of an apnea alarm may indicate patient apnea or patient disconnection, system leaks, inadequate machine sensitivity, or inappropriately set apnea parameters. An apnea alarm may also be accompanied by a low-pressure or low $\dot{V}_E$ alarm. Some ventilators have a preset apnea period of 20 seconds, whereas others allow the operator to set the apnea period. Ventilators are designed to detect spontaneous and mandatory breaths. Thus, when an apnea alarm sounds, the most obvious cause is patient apnea. After ensuring that the patient is receiving ventilation, the clinician should check the mandatory breathing rate and the machine's sensitivity. The clinician should also check for possible leaks or patient disconnection. In cases in which auto-PEEP is present, patient triggering may be more difficult, resulting in undetected efforts. A ventilator set in a spontaneous mode (e.g., PSV or CPAP) could misinterpret this as apnea.

Some ventilators provide a backup mode of ventilation when the apnea alarm is activated. Backup modes generally provide a minimum safe level of ventilation for the patient until the operator can respond to the alarm and correct the problem. Some ventilators cancel the backup mode once a patient effort is detected (e.g., PB 840, Medtronics Minimally Invasive Technologies, Minneapolis, MN).

Low-Source Gas Pressure or Power Input Alarm

A low-source gas alarm activates if the gas source fails or the high-pressure line becomes disconnected from the gas source. With ventilators equipped with an air compressor, the operator must ensure that the compressor is operating. (NOTE: Earlier-generation ventilators, such as the CareFusion Bear 1000, used an on/off switch that had to be turned on; current ICU ventilators, such as the Medtronics PB 840 and 980 ventilators, are designed with compressors that automatically turn on when there is a loss of pressure in the patient-ventilator circuit.) The operator must, however, ensure that the air and O_2 lines are connected to an active gas supply, such as a wall outlet.

Modern microprocessor-controlled ventilators require an electrical power source. If a power-loss alarm activates, the clinician should first confirm that the unit is connected to an active electrical outlet and has not been unplugged. If the electrical outlet is working and the unit fails to start, the line fuse or circuit breaker may need to be replaced or reset. Most ICU ventilators have a reset button near the "power on" switch. In the event of an overall power outage, the ventilator should be plugged into a red electrical outlet (these outlets are connected to emergency generator power supplies). Many ICU ventilators have backup battery power sufficient to operate a unit for 30 minutes to 4 hours, depending on the ventilator and the battery source.

Ventilator Inoperative Alarm and Technical Error Message

With microprocessor-controlled ventilators, an inoperative alarm or a technical error message is displayed if an internal malfunction is detected by the ventilator's self-testing systems. This most often occurs when the ventilator is first turned on. Sometimes simply turning the machine off and then back on corrects the error. If it does not, it may be necessary to replace the ventilator and contact the manufacturer's representative.

Operator Settings Incompatible With Machine Parameters

An error message or alarm (or both) is triggered if the operator tries to select a setting that is outside the range for that parameter or is incompatible with the other selected settings. For example, if the clinician tries to set a V_T of 50 mL and the V_T range for the ventilator is 200 to 2000 mL, the ventilator will indicate that that setting cannot be selected. Another example of an incompatible setting can occur when using volume control ventilation. If the operator sets an inspiratory gas flow that cannot deliver the set V_T within an acceptable time based on the set *f*, the machine produces an error message asking the operator to correct the flow or reduce the V_T.

Inspiratory-to-Expiratory Ratio Indicator and Alarm

Most current ICU ventilators do not allow the inspiratory-to-expiratory (I/E) ratio to exceed 1:1 unless the operator specifically wants to use an inverse I/E ratio. This generally requires activation of a separate control or touch pad or some similar function that alerts the operator that the ratio is being inverted.

An inverse-ratio alarm may activate if a change occurs in the patient lung's characteristic (i.e., increased R_{aw} or decreased C_L), resulting in a lower inspiratory flow. This does not happen often, however, because most ventilators have enough power to maintain the desired gas flow. Another possibility is a flow setting that is too low for the desired V_T delivery. The I/E ratio can also change when the selected waveform is changed. For example, changing from a constant flow to a descending ramp waveform may lengthen the T_I in a volume-targeted ventilation. Selection of an inspiratory pause can also lengthen T_I. In pressure-controlled ventilation, a long T_I (depending on the set *f*) may activate the I/E ratio indicator.

Other Alarms

Additional alarms may be available on many ICU ventilators, including high-PEEP/CPAP alarms, low- and high-V_T alarms, low- and high-$\dot{V}_E$ alarms, a high-*f* alarm, and low- and high-F_IO_2 alarms. High-PEEP/CPAP alarms are often activated by the same problems that lead to high-pressure alarms. Low-V_T and low-$\dot{V}_E$ alarms usually activate in situations that cause low-pressure alarms or when the patient's spontaneous ventilation has decreased for some reason. Flow-sensor disconnection, leaks, or malfunction can also cause activation of these alarms.

High-V_T, high-*f*, and high-$\dot{V}_E$ alarms can activate if the patient's $\dot{V}_E$ has increased, or when the ventilator is too sensitive to patient effort (i.e., autotriggering). High-$\dot{V}_E$ alarms also may be activated if a nebulizer powered by a separate external gas source is in line with the main circuit. These problems may be caused by inappropriate calibration, contamination, or malfunction of flow sensors.

Box 18.9 and Fig. 18.5 present several clinical situations that can be associated with alarm activation. It is important that clinicians become familiar with the types of equipment used in their facilities and the various alarm systems available.

USE OF GRAPHICS TO IDENTIFY VENTILATOR PROBLEMS

Ventilator graphic displays can provide valuable information that clinicians can use to evaluate the integrity of the patient-ventilator system (Key Point 18.5). A detailed review of ventilator graphics

BOX 18.9 Ventilator Troubleshooting: Response to Alarms and Abnormal Waveforms

If an alarm activates or an abnormal waveform appears:

1. Assess the patient's appearance to evaluate for distress.
2. Ensure the patient is receiving adequate ventilation and oxygenation.
3. If necessary, and if the patient is suffering severe distress, disconnect the patient from the ventilator and manually ventilate, adding PEEP (if needed, and increase the F_IO_2.)
4. Reassess the patient.
5. Check the activated alarm, and make sure alarm parameters have been set appropriately.
6. Once the cause of problem has been determined, resolve it.
7. If the problem cannot be resolved, change the ventilator or call for help.

Common Alarm Situations

Low-Pressure Alarm

1. Check for patient disconnection.
2. Check for leaks in the patient circuit related to the artificial airway and through chest tubes.
3. Confirm that the proximal pressure line is connected and unobstructed.
4. Low-pressure alarm may be accompanied by a low minute ventilation ($\dot{V}_E$) or low tidal volume (V_T) alarm.

High-Pressure Alarm

1. If the patient is coughing, check to determine whether secretions have built up in the airway or the patient is biting the endotracheal tube (ET).
2. Check for kinking or displacement of the ET; also check the tube's position in the airway (i.e., ensure that the ET is not inserted too far into the trachea).
3. Check whether R_{aw} has increased or C_L has decreased.
4. Ensure that the main inspiratory or expiratory lines are not kinked or obstructed.
5. Check that the patient is breathing synchronously with the ventilator.
6. Determine whether air trapping (auto-PEEP) has developed.
7. Ensure that the expiratory filter and expiratory valve are functioning properly.

Low Positive End-Expiratory or PEEP/CPAP Alarms

1. Check to determine whether the low-PEEP alarm is set below the applied PEEP level.
2. Determine whether the patient is actively inspiring below baseline.
3. Determine whether a leak is present.
4. Confirm that the patient has not become disconnected from the ventilator.
5. Ensure that the proximal airway pressure (P_{aw}) line is not occluded.

Apnea Alarm

1. Determine whether the patient is apneic.
2. Check for leaks.
3. Check the sensitivity setting to be sure the ventilator can detect patient effort.
4. Check the alarm-time interval and the volume setting, when appropriate.

Low-Source Gas Pressure or Power Input Alarm

1. Check the 50-psi gas source (e.g., wall connection, cylinder, or air compressor).
2. Check high-pressure hose connections to the ventilator.
3. Check ventilator's electrical power supply and whether it is plugged into the electrical outlet that is connected to the emergency backup system.
4. Check the line fuse or circuit breaker.
5. Try using the reset button.
6. If alarms continue, replace the ventilator.

Ventilator Inoperative Alarm or Technical Error Message

1. If an internal malfunction message is present and the ventilator is turned on, try turning the ventilator off and restarting it.
2. If alarm continues, follow message instructions or replace the ventilator.

Operator Settings Incompatible With Machine Parameters

1. Error message usually indicates that a parameter must be reset (e.g., flow is not high enough to deliver V_T within an acceptable T_I to keep the inspiratory-to-expiratory (I/E) ratio below 1:1 [based on *f*, V_T, and flow]).
2. Adjust the appropriate controls.

Inspiratory-to-Expiratory Ratio Indicator and Alarm

1. Usually indicates an I/E ratio greater than 1:1.
2. If inverse I/E ratio is a goal, disable the I/E ratio limit or ignore the audible warning.
3. If normal I/E ratios are a goal, check alarm causes:
   - If increased R_{aw} or decreased C_L has resulted in a lower flow, treat the cause.
   - If the flow setting is too low for the desired V_T delivery, increase flow or change the flow waveform.

Other Possible Alarms

1. High-PEEP/CPAP alarms
   - Causes are similar to those for high-pressure alarms.
   - In flow-cycled modes (e.g., pressure support volume [PSV]), check for system leaks.
2. Low-V_T, low-($\dot{V}_E$), and/or low-*f* alarms
   - Causes are similar to those for low-pressure alarms.
   - Determine whether spontaneous ventilation has decreased for some reason.
   - Verify that all alarms have been set appropriately.
   - Check flow sensor for disconnection or malfunction.
3. High-V_T, high-($\dot{V}_E$), or high-*f* alarms
   - Check machine sensitivity level for autotriggering.
   - Check for possible cause of increased patient ($\dot{V}_E$).
   - Ensure alarms have been set appropriately.
   - If an external nebulizer is in use, reset the alarm until the treatment is finished, then return the alarm to the appropriate setting.
   - Check the flow sensors for calibration, contamination, or malfunction.
4. Low- or high-F_IO_2 alarm
   - Check gas source.
   - Make sure built-in O_2 analyzer is functioning properly.

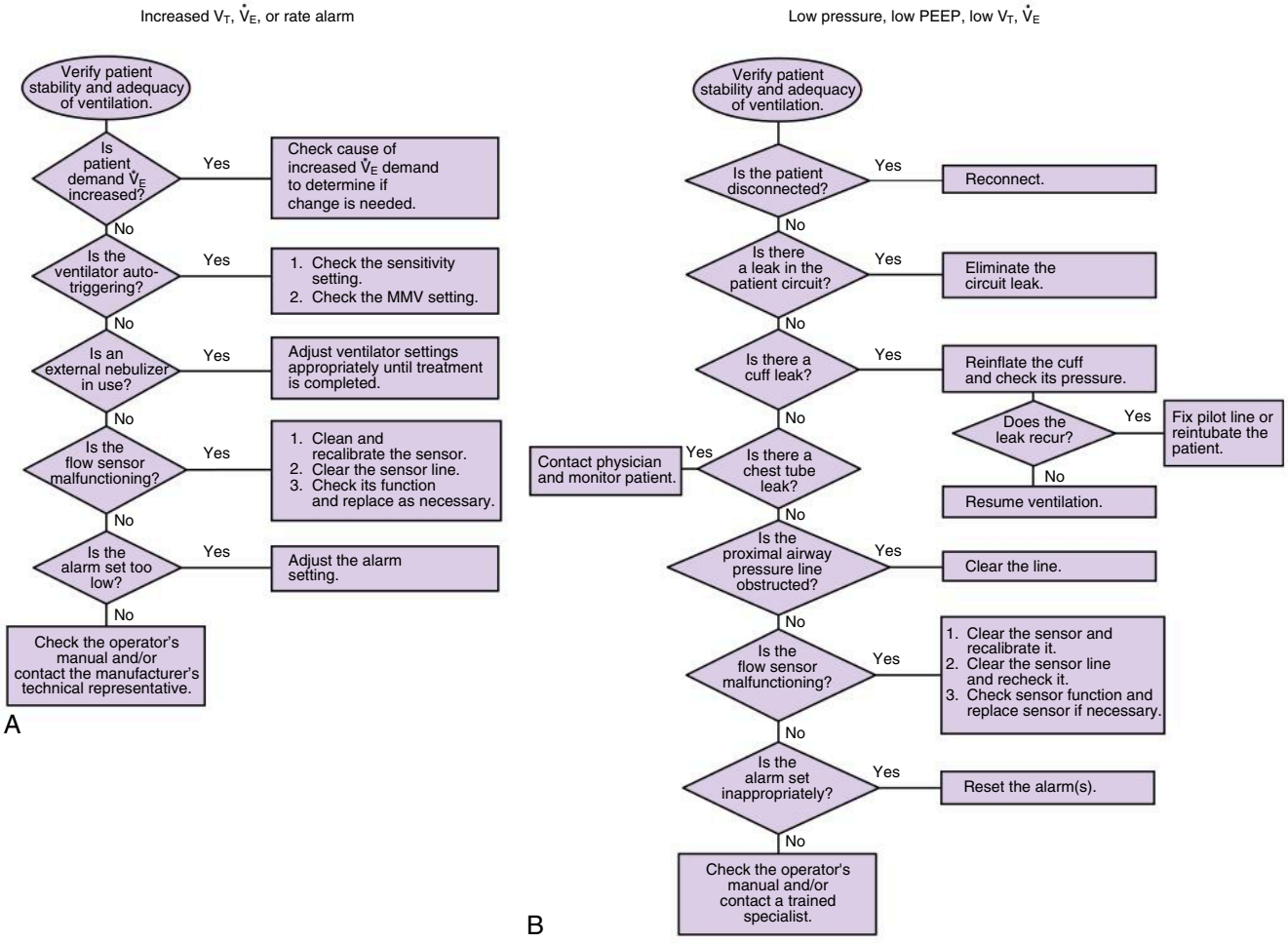

Fig. 18.5 Algorithms for troubleshooting activated alarms. (A) Increased tidal volume (V_T), minute ventilation ($\dot{V}_E$), or rate alarm. *MMV*, Mandatory minute ventilation. (B) Low-pressure, low positive end-expiratory/continuous positive airway pressure (PEEP/CPAP), low ($\dot{V}_E$), low V_T, or low rate alarm. (C) High-pressure or high PEEP/CPAP alarm. (D) Inverse I/E ratio indicator. (E) Apnea Alarm. (F) Loss of power alarm. *C*, Compliance; *ET* Endotracheal tube; R_{aw}, airway resistance; T_I, Inspiratory time; T_E, Expiratory time. (*Form Cairo JM, Pilbeam SP. Mosby;s Respiratory Care Equipment. 7th ed. St. Louis, MO, Mosby, 2004.*)

***Key Point* 18.5** Ventilator graphic displays can provide valuable information that clinicians can use to evaluate the integrity of the patient-ventilator system.

was presented in Chapter 9, and examples of ventilator-associated problems have been discussed throughout the text. The following discussion provides a summary of how ventilator graphics can be used to identify problems encountered with the patient-ventilator interface.

Leaks

As previously discussed, inadvertent patient disconnection and leaks in the patient-ventilator circuit are common during mechanical ventilation. When these situations arise, low-pressure, low-volume, low-$\dot{V}_E$, or apnea alarms usually become activated. Volume-time scalars are one means of identifying leaks, as are pressure-volume and flow-volume loops (see Chapter 9 for more details). In each graphic representation, the most important indicator of a leak can be found using the expiratory volume curve. If the expiratory volume does not return to zero in any of these waveforms, a leak is present in the system (Figs. 18.6 and 18.7; Case Studies 18.4 and 18.5). In some cases, the volume tracing may drop below zero; this finding indicates that the equipment needs to be recalibrated (see Fig. 18.1). This can also occur if the patient is actively exhaling.

Case Study 18.4

Problem Solving Using Ventilator Graphics

The respiratory therapist hears a low-pressure ventilator alarm for a patient receiving VC-CMV. She evaluates the patient and finds that the individual is not in distress and is being ventilated and oxygenated. She checks the activated alarm (i.e., low $\dot{V}_E$), silences it, and saves the graphics display. Fig. 18.7 shows the saved graphs. What do these waveforms indicate?

Inadequate Flow

Patient-ventilator asynchrony can occur if a ventilator provides only a fixed flow or an inadequate flow during mechanical ventilation. In such cases the pressure-time graphic is concave and the flow curve is constant (see Fig. 18.3).

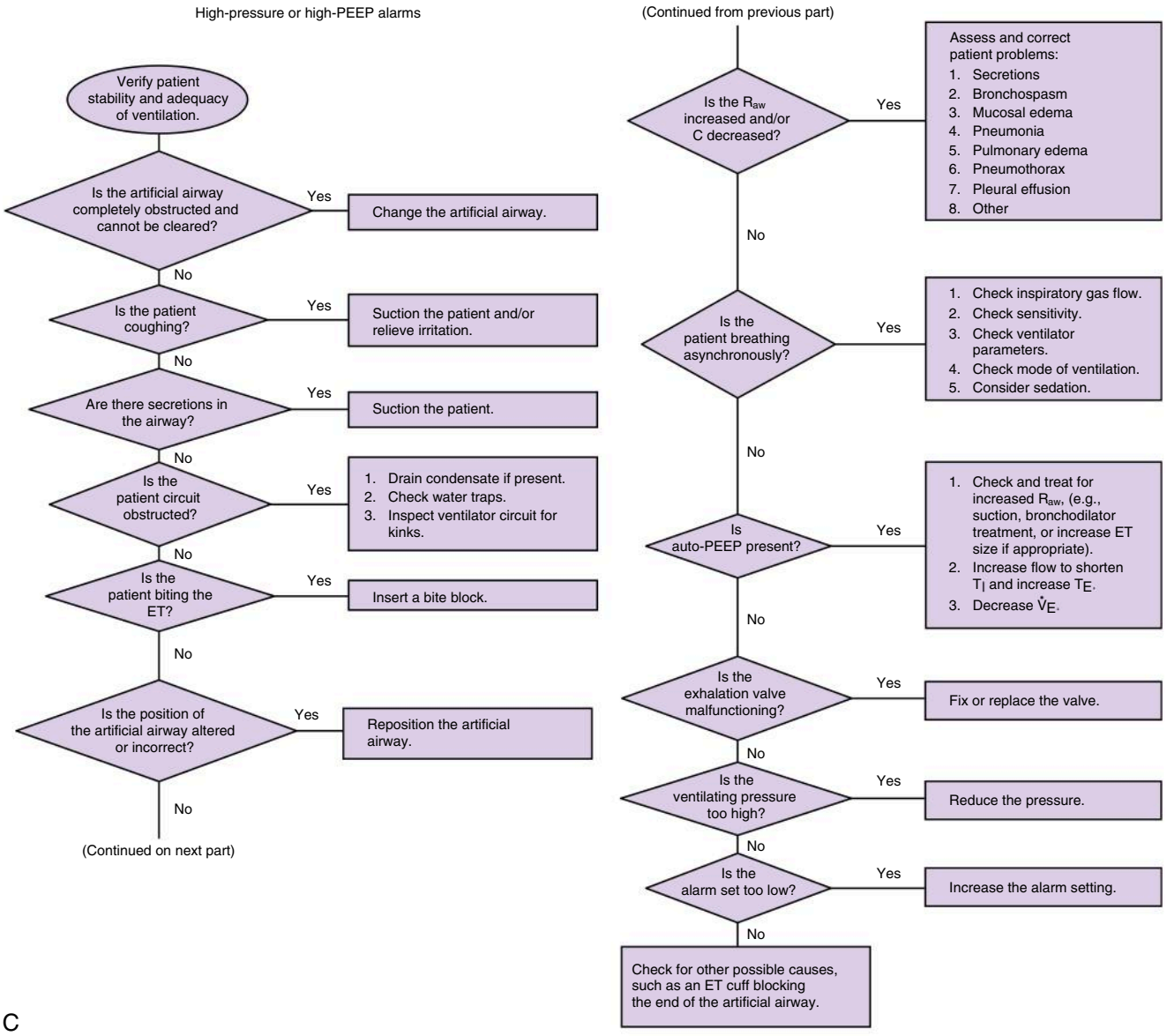

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Fig. 18.5, cont'd

Inadequate Sensitivity Setting for Patient Triggering

The ventilator's trigger sensitivity can be set too low for a patient. An inappropriately set sensitivity, such as an inadequate flow setting, increases WOB.

Overinflation

Pressure-volume loops that produce a duck-bill, or "beak," appearance can be used to identify alveolar overinflation (see Fig 13.6).

Auto-PEEP

Ventilator graphics can be used to detect auto-PEEP. If expiratory flow does not return to zero on a flow-volume loop before the next mandatory breath, auto-PEEP is present (Fig. 18.8).

Inadequate Inspiratory Time During Pressure Ventilation

One goal of pressure-controlled ventilation (PC-CMV) may be to provide a T_I long enough to achieve a slight plateau effect so that the inspiratory flow drops to zero before the end of inspiration (see Fig. 5.2C). Sometimes this plateau is not present because of changes in the patient's lung characteristics or a short T_I. The T_I may need to be adjusted, depending on the clinical situation.

Waveform Ringing

When flow and pressure delivery are high at the beginning of a breath, particularly during a pressure breath, a phenomenon known as *ringing, spiking,* or *overshoot* can result from the oscillation of air in the patient-ventilator circuit and upper airway at

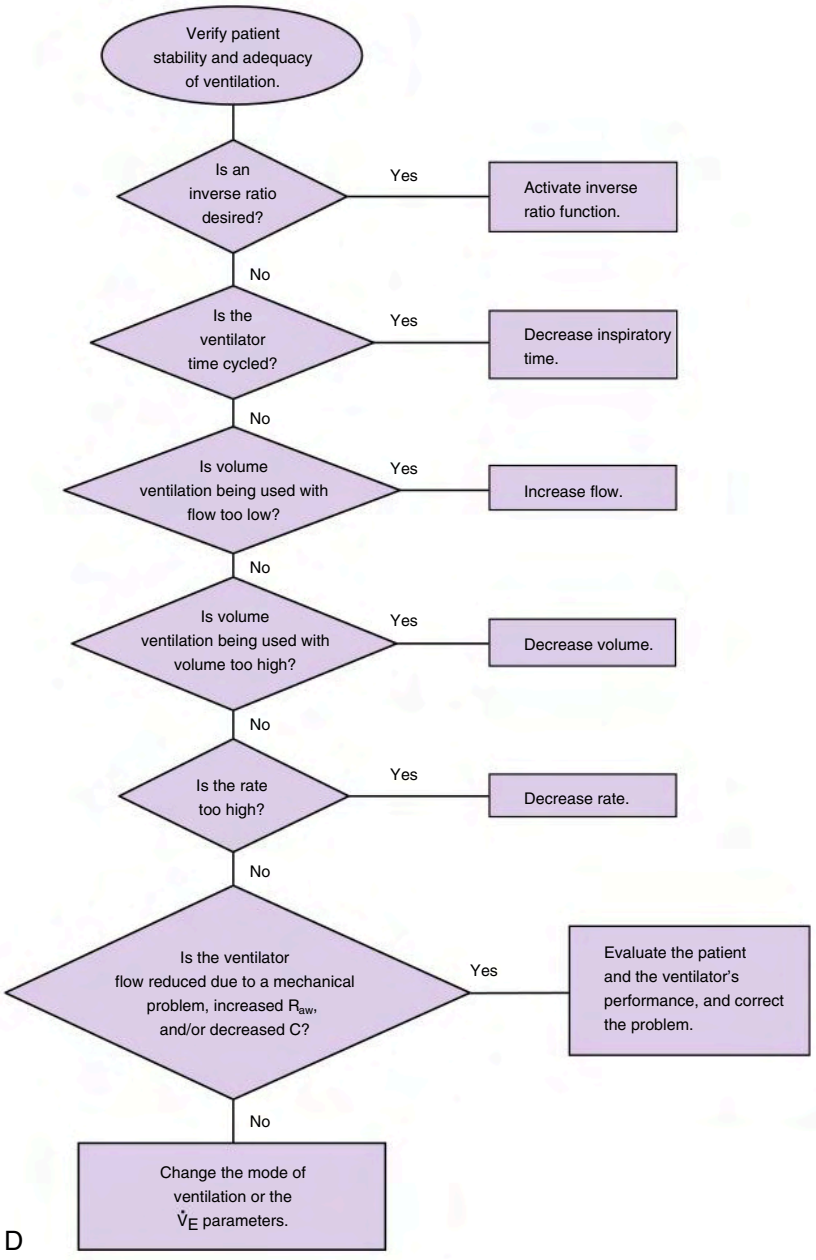


Fig. 18.5, cont'd

the beginning of inspiration (Fig. 18.9). This situation, although not life-threatening, does not represent a smooth breath delivery. Current ICU ventilators allow adjustment of gas flow and pressure delivery at the beginning of a breath to help taper flow and pressure delivery and reduce overshoot. This feature commonly is labeled the *inspiratory rise time.* Oscillation can also occur if condensation is present in the patient circuit. This latter situation can be easily remedied by draining of the circuit.

Expiratory Portion of Volume-Time Curve Below Baseline

As discussed earlier in this chapter, active exhalation may cause the expiratory portion of the volume-time curve to drop below baseline. Notice that the volume graphic may also descend below baseline if the expiratory flow sensor is out of calibration (see Fig. 18.1).

Patient-Ventilator Asynchrony

Figs. 18.3 and 18.10 show graphics for a patient breathing out of synchrony with the ventilator. As mentioned earlier, patient-ventilator asynchrony can occur when the flow and sensitivity settings are inadequate or when auto-PEEP is present. Adjustment of these parameters or switching to a servo-controlled mode (e.g., pressure-regulated volume control) or to pressure-controlled ventilation may help alleviate the problem. In some cases, the patient may also need to be sedated.

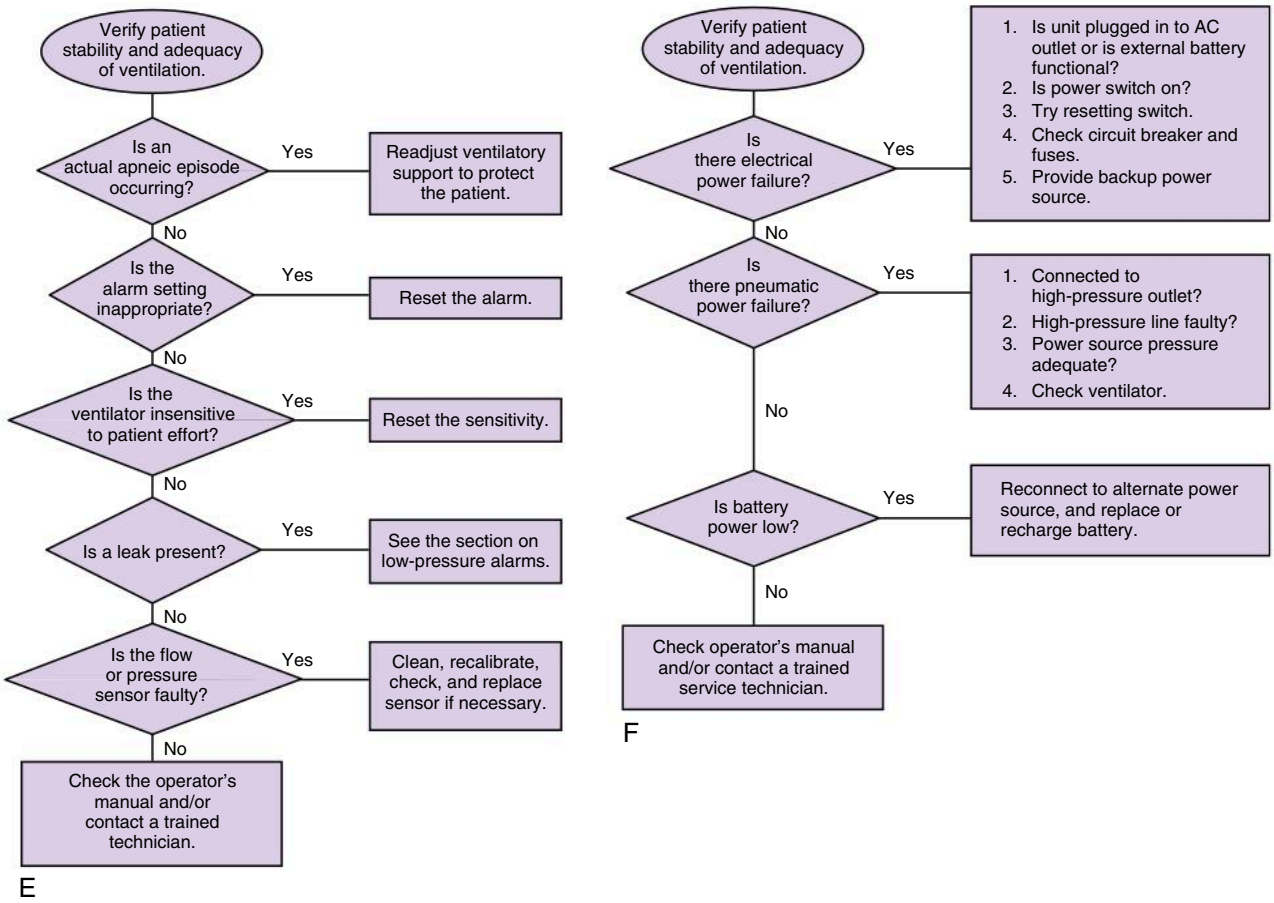
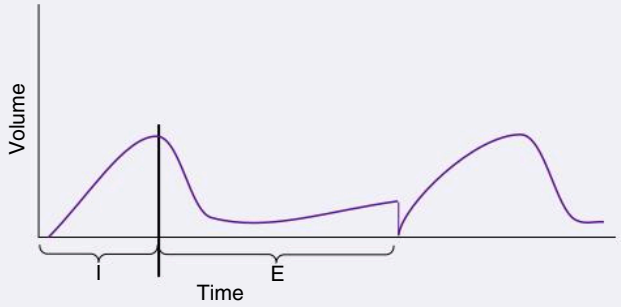


Fig. 18.5, cont'd

Case Study 18.5

Evaluating a Ventilator Problem

During ventilation of a patient with VC-CMV and 10 cm H_2O of PEEP, the respiratory therapist notices the volume-time graphic shown in the following figure. During exhalation the respiratory therapist feels an uninterrupted flow of a small amount of air from the exhalation valve, even though the patient has had no previous evidence of air trapping. What do these findings suggest?



Volume-time curve in which the volume curve descends at the beginning of exhalation and then slowly rises until the start of the next inspiratory phase.

UNEXPECTED VENTILATOR RESPONSES

Problems in some microprocessor-controlled ventilators can result from inappropriate use of or idiosyncrasies associated with the machine. The following section discusses a few noteworthy situations; the intent is not to criticize any particular ventilator but rather to make the reader aware of situations that have been reported, including unseating of the exhalation valve, excessive CPAP/PEEP levels, changes in sensitivity, inability to trigger a pressure-supported breath, and altered alarm function.

Unseated or Obstructed Expiratory Valve

The expiratory valve can be unseated if it is blocked to obtain a static compliance (C_S) reading. (This is generally a problem only if the ventilator does not have an inspiratory pause control.) The valve is unseated because a pressure buildup during this procedure causes the exhalation valve to disengage. An unseated expiratory valve may be the cause if the ventilator inoperative alarm, a low-pressure alarm, or a low CPAP/PEEP alarm activates and the patient has difficulty breathing. It is important to mention that these same alarms can be activated by other factors; therefore the actual cause must be established.

Expiratory valves may also malfunction if they become obstructed or their mobility is impaired by an accumulation of residue from medications delivered by small-volume nebulizers.

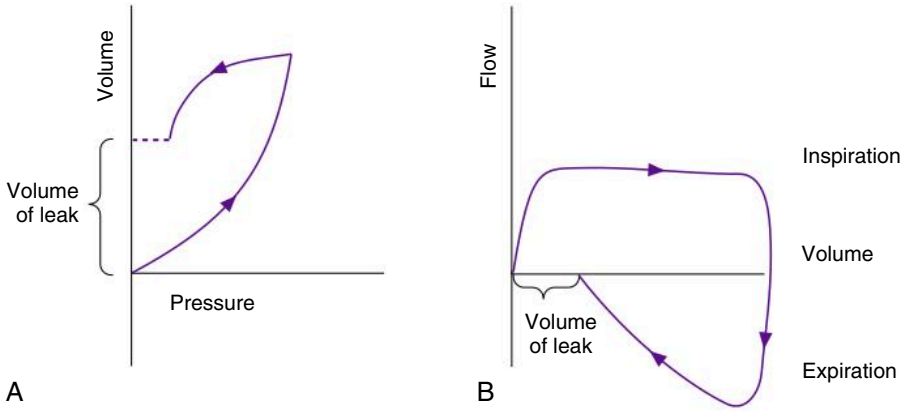


Fig. 18.6 Pressure-volume loop (A) and flow-volume loop (B) indicating an air leak.

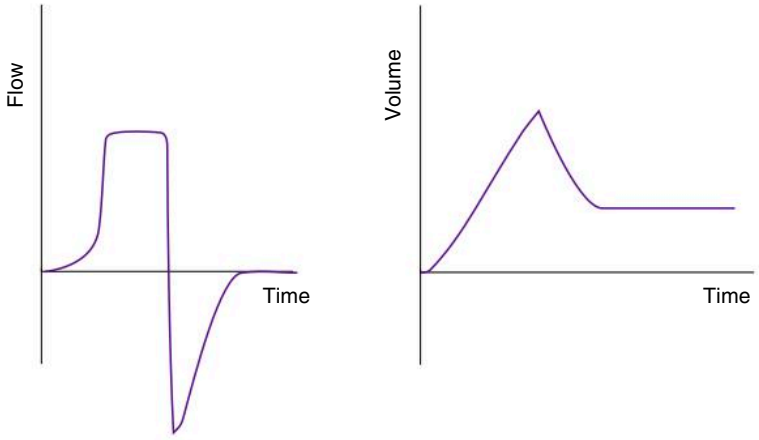


Fig. 18.7 Flow-time curve and volume-time curve demonstrating a problem.

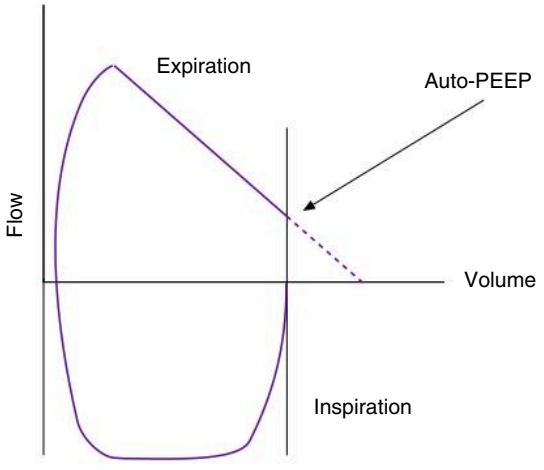


Fig. 18.8 Flow-volume loop reflecting the presence of auto—positive end-expiratory pressure. The *arrow* indicates the amount of flow at the end of exhalation.

The valves can be protected by placing bacterial filters in line, before the exhalation valve. Expiratory filters must be changed frequently to avoid increased expiratory resistance.

Excessive CPAP/PEEP

Excessive CPAP/PEEP levels (i.e., above those set by the operator) can occur in certain clinical situations. For example, a potential problem that can occur with PSV is a sudden accidental delivery of high flow and pressure because of a leak in the breathing circuit. Application of high flow to maintain CPAP/PEEP levels can cause the airway pressure to rise, and the patient may develop dyspnea, tachypnea, and tachycardia.[21,22] This problem can typically be solved by eliminating the leak.

Another problem that involves patient-circuit leaks can occur during PSV. This problem was initially reported by Fiastro and colleagues with the Puritan Bennett 7200 ventilator.[23] If a leak of more than 5 L/min develops around the cuff of the ET or somewhere in the patient circuit, the set PSV level will be maintained throughout the cycle, causing CPAP to develop in the circuit. A decrease in flow to 5 L/min is the normal mechanism that stops the PSV inspiratory phase in this ventilator. All ventilators with PSV now have a safety mechanism. If the T_I exceeds a preset time (approximately 3—5 seconds), the ventilator will cycle into expiration.

Nebulizer Impairment of Patient's Ability to Trigger a Pressure-Supported Breath

During PSV the patient must create a slightly negative pressure or drop in flow in the circuit to initiate a breath. When a

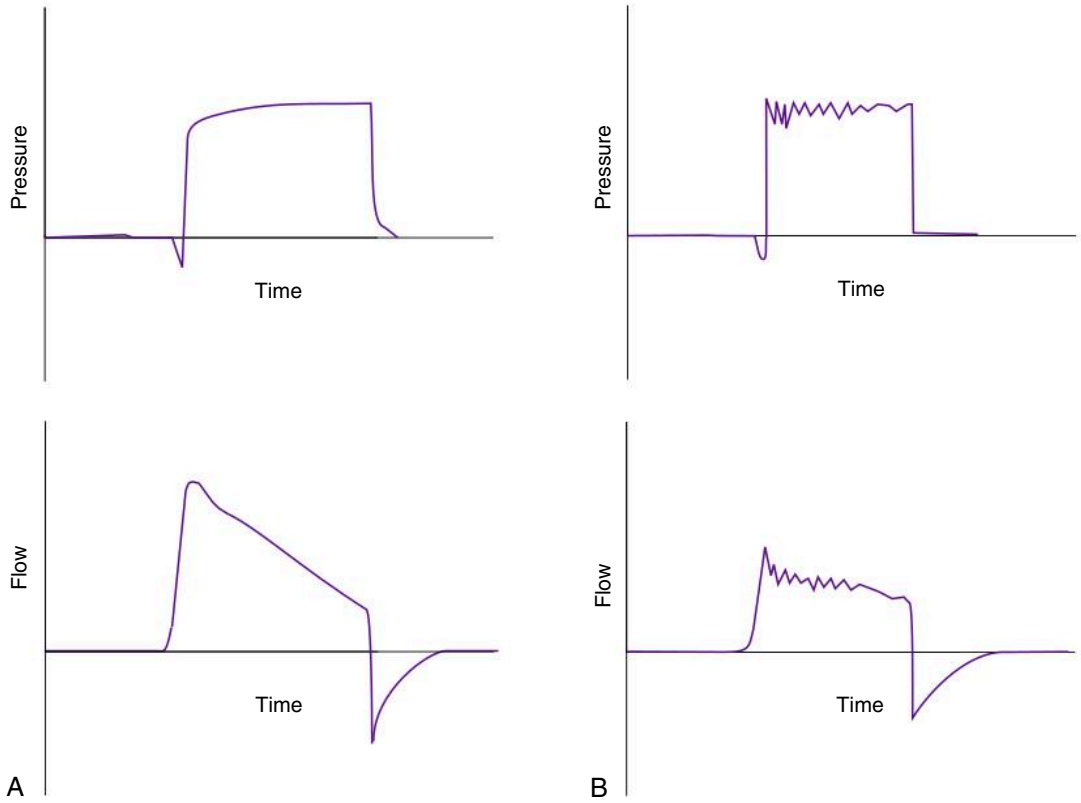


Fig. 18.9 (A) Flow-time and pressure-time curves demonstrating a normal pressure-supported breath. (B) Flow-time and pressure-time curves showing ringing (oscillations), a phenomenon that occurs with an overshoot of flow and pressure at the beginning of inspiration.

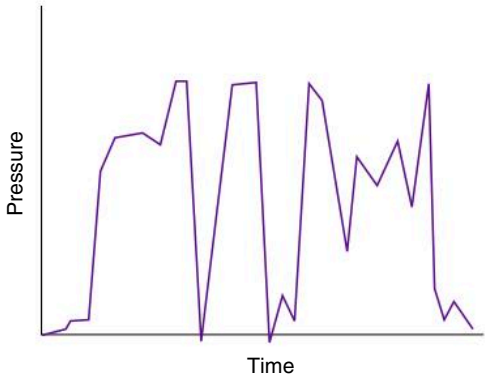


Fig. 18.10 Wide fluctuations can be seen in the pressure-time curve when the patient is actively breathing during mechanical ventilation, but the ventilator is not set up to respond to patient demand; the result is patient-ventilator asynchrony.

continuous-flow nebulizer is placed between the patient and the sensing mechanism, the patient often finds it more difficult to generate the effort to trigger the ventilator. Devices that sense ventilator triggering are usually on the inspiratory side of the ventilator. Triggering difficulty is especially apparent in older patients with COPD and weak inspiratory efforts.[24,25] (NOTE: This problem may arise with microprocessor-controlled ventilators when a nebulizer powered by an external gas source is used.)

Triggering difficulty may also occur with patients during the VC-CMV when an external nebulizer is added in line. It may not be a significant clinical problem because the set rate and volume ensure the patient will receive an adequate $\dot{V}_E$, even if the patient does not trigger the breath. Unfortunately, there is not a backup rate with PSV and volume support ventilation.[26] Therefore clinicians should use only the nebulizer provided by the ventilator's manufacturer. If one is not provided, the practitioner should use an inline metered-dose inhaler with a spacer, an ultrasonic nebulizer, or a vibrating mesh nebulizer.

High Tidal Volume Delivery

High V_T delivery can occur when externally powered small-volume nebulizers are used for aerosol delivery. Flow meters add flow to the patient circuit, which can increase the delivered V_T and result in artificially high readings of exhaled $\dot{V}_E$. Although this is not a significant problem in most situations with adults, it can be significant in neonates, and these devices should not be used for medication delivery during neonatal mechanical ventilation.

High V_T delivery can also occur in a ventilator that allows the patient to receive additional flow during inspiration on demand. Current ICU ventilators (e.g., Servo-i) provide additional flow if the airway pressure drops by 2 cm H_2O. This results in a higher V_T delivery than the set value. Set values therefore actually represent minimum V_T delivery; patients can receive the volume of air they want.

Altered Alarm Function

Some monitoring devices and ventilator controls operate on optical detectors. When exposed to intense light, such as sunlight through a window, the alarms may sound even though no change has occurred in the patient's status. This may happen with volume monitors, pulse oximeters, and other light-sensitive devices.

Electromagnetic Interference

Electrical devices that emit radio frequencies, such as cell phones, can interfere with the operation of medical equipment. Hospitals generally prohibit the use of cell phones, two-way radios, and similar equipment in locations such as the ICU, where problems are likely to occur. These devices have been known to interfere with the function of mechanical ventilators, infusion pumps, smoke detectors, and telemetry equipment. Other medical devices that can be affected include electrocardiograph monitors and machines, O_2 and apnea monitors, defibrillators, blood warmers, and dialysis units.[27]

When the purchase of a piece of equipment such as a mechanical ventilator is under consideration, the hospital should determine what built-in safeguards the manufacturer has provided to prevent electromagnetic interference.

Operator's Manuals

The operator's manuals provided by manufacturers of microprocessor-controlled ventilators typically include valuable information that can be used to address common troubleshooting problems. These manuals are a good resource of information for troubleshooting situations that are unique to the particular ventilator being used.

SUMMARY

- *Problem solving* can be simply defined as determining a solution to a challenging situation. The first step in solving any problem is to carefully analyze the situation at hand. The next step is to gather and assess pertinent data, which should point to a number of viable solutions.
- The sudden onset of dyspnea can be identified by observing the physical signs of respiratory distress, including tachypnea, nasal flaring, diaphoresis, use of the accessory muscles of breathing, retraction of the suprasternal, supraclavicular, and intercostal spaces, paradoxical or abnormal movement of the thorax and abdomen, and abnormal findings on auscultation.
- Identifying the cause of patient-ventilator asynchrony can be accomplished using a relatively simple approach. If the patient is in *severe* distress, the first step is to disconnect the patient from the ventilator and carefully provide ventilation to the patient using a manual resuscitation bag. When a resuscitation bag is used properly, these devices allow for the assessment of lung characteristics.
- The most common patient-related problems encountered during mechanical ventilation involve the artificial airway or the presence of a pneumothorax, bronchospasm, and excessive secretions.
- Evaluation of the patient's secretions can be useful in differentiating a variety of patient-related problems.
- Changes in the patient's position can be associated with accidental extubation, bending and twisting of the patient circuit, and in some cases alterations in the patient's level of oxygenation.
- Activation of low-pressure, low-volume, and low-V_E alarms typically indicates that a leak in the patient-ventilator circuit is present.
- Circuit leaks typically occur at the level of the ET and at junctions in the patient circuit where tube connections exist.
- In addition to an inappropriate sensitivity setting and inadequate flow, other types of patient-ventilator asynchrony can be associated with auto-PEEP, an increased ventilatory drive, or the need for sedation.
- Activation of an apnea alarm may indicate patient apnea or patient disconnection, system leaks, inadequate machine sensitivity, or inappropriately set apnea parameters.
- A low-source gas alarm activates if the gas source fails or the high-pressure line becomes disconnected from the gas source.
- Conditions leading to activation of high-pressure alarms can be categorized as airway problems, changes in lung characteristics or patient-related conditions, and problems related to the patient-ventilator circuit.
- An error message or alarm (or both) is triggered if the operator tries to select a setting that is outside the range for that parameter or is incompatible with the other selected settings.
- Ventilator graphic displays can provide valuable information that clinicians can use to evaluate the integrity of the patient-ventilator system.
- Careful analysis is required to solve patient-ventilator system problems and troubleshoot malfunctions.
- Experience is an important part of the learning process. By using the knowledge gained through experience, practitioners can expand their ability to solve a particular problem.

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

1. A 25-year-old man receiving mechanical ventilation is rotated from the supine position onto his right side. Immediately after this move, the high-pressure alarm on the ventilator activates. On auscultation, the respiratory therapist hears breath sounds only over the right lung. The centimeter marking of the ET is 25 cm. What should the therapist do to correct this situation?

2. A constant inspiratory flow of 40 L/min is set for a patient receiving VC-CMV. The ventilator I/E ratio indicator shows that I exceeds E. How could this problem be corrected without changing V_E?
A. Shorten the expiratory time (T_E)
B. Increase the *f*
C. Lengthen the T_I
D. Increase the inspiratory gas flow setting

3. A patient with COPD is treated with prednisone, theophylline, and furosemide (Lasix). Which of the following is the most important parameter to check regularly?
A. Clotting times
B. Calcium levels
C. Potassium levels
D. Pupillary response

4. A patient on mechanical ventilatory support is suctioned for large amounts of foul-smelling green sputum. The patient has a temperature of 38° C and a normal white blood cell count. Which of the following is the most likely cause of this problem?
A. An overheated humidifier
B. Cardiogenic pulmonary edema
C. An asthma exacerbation
D. A respiratory infection

5. Which of the following are potential problems that can result when an externally powered nebulizer is added to a mechanical ventilator circuit?
1. It increases machine sensitivity.
2. Expiratory monitor readings increase from previous values.
3. It may add volume to the delivered V_T.
4. In a patient-triggered mode, the added flow must be overcome by the patient to trigger the ventilator.
A. 2
B. 3
C. 1 and 4
D. 2, 3, and 4

6. What is the first step in managing a mechanically ventilated patient in severe distress? How can you tell whether the problem originates with the ventilator or the patient?

7. A 58-year-old man is intubated orally after cardiac arrest. The patient is admitted to the ICU, and ventilatory support is provided using volume control ventilation (VC-CMV) with 100% O_2. The PIP has been increasing progressively over the past 4 hours. Auscultation of the patient's chest reveals an absence of breath sounds over the left lung and distant breath sounds over the right lung. The left hemithorax is dull to percussion, and the right chest is resonant. The trachea is deviated to the left. No chest radiograph is available. Briefly describe what is causing the problem and how it can be corrected.

8. The low-pressure and low-volume alarms activate on a ventilated patient. Auscultation over the trachea reveals a hiss during the entire mandatory breath cycle. What is the likely problem, and how would you correct it?

9. A patient is undergoing ventilation with bilevel positive airway pressure (bilevel PAP) with a full face mask. Initial pressure readings were an inspiratory positive airway pressure of 12 cm H_2O and an expiratory positive airway pressure of 3 cm H_2O, with a measured V_T of 0.55 to 0.6 L. The measured V_T with the same pressures 3 hours later is 0.3 to 0.45 L. Which of the following could be the cause of the drop in V_T?
1. Air is leaking around an underinflated ET cuff.
2. A decrease in the patient's C_L has occurred.
3. Increase in the patient's R_{aw}.
4. Ascites is restricting the patient's inspiratory efforts.
A. 1 and 2 only
B. 2 and 3 only
C. 1, 2, and 3 only
D. 1, 2, 3, and 4

10. While monitoring a patient on mechanical ventilatory support, the respiratory therapist hears the high-pressure alarm and notes that breath sounds are absent over the right lung and diminished over the left lung. The percussion note is tympanic on the right and resonant on the left. The patient's distress is not relieved when the respiratory therapist performs manual ventilation with 100% O_2. What could cause these findings, and what should be done?

11. A 70-year-old woman with COPD is on mechanical ventilation using volume-controlled continuous mandatory ventilation (VC-CMV). Although the sensitivity is at the most sensitive setting, the patient is struggling to breathe (using accessory muscles) and is unable to trigger a machine breath on her own. When breaths are delivered, the ventilator graphics show a concave pressure curve. Expiratory flow does not return to zero before the next mandatory breath is delivered. What is the likely cause of the problem?

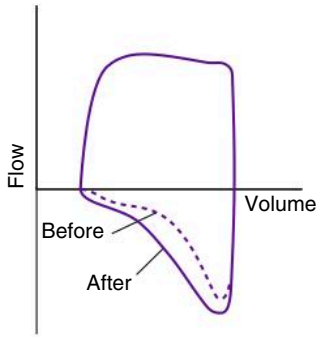
12. The low-pressure and low-volume alarms activate on a patient receiving mechanical ventilatory support. The ventilator graphics indicate that the expired volume is lower than the inspired volume. What is the likely cause of this problem? (See Fig. 18.7.)
1. Pneumothorax
2. Pulmonary edema
3. Disconnection from the ventilator
4. Increased airway resistance
A. 1 only
B. 3 only
C. 1, 2, and 3 only
D. 1, 3, and 4 only

13. A 26-year-old man who was in a motor vehicle accident is transferred from a rural hospital to the urban trauma center. He is 6-ft, 3-in tall and weighs 200 lb. He suffered trauma to the chest and left leg (fractured left femur). Currently no pneumothorax or hemothorax is present, and he has no head or neck injuries. The artificial airway is a 7-French oral endotracheal tube in the correct position. The patient requires mechanical ventilation. Cuff pressure is 38 cm H_2O to provide the minimum leak technique. Do you think any immediate changes need to be made in the current management of this patient?

14. Difficulty is encountered in the ventilatory management of a patient with acute pancreatitis and ARDS. In PC-CMV, the set pressure is 30 cm H_2O with a T_I of 2 seconds and an I/E ratio of 1:1. The patient is heavily sedated, and the rate is set at 15 breaths/min. P_aO_2 is maintained at 61 mm Hg on an F_IO_2 of 0.5 and 15 cm H_2O of PEEP. However, over the past 4 hours, pH has dropped from 7.31 to 7.22, P_aCO_2 has risen from 45 to 53 mm Hg, and P_aO_2 has dropped to 54 mm Hg as the delivered V_T has steadily decreased. What change in ventilation would you suggest?

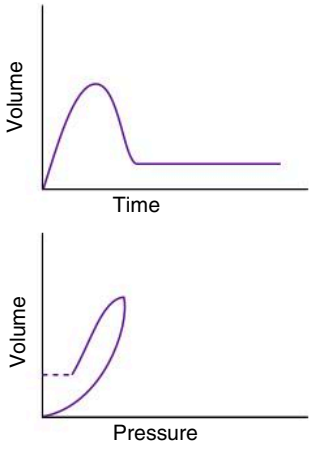
15. A respiratory therapist is monitoring a patient receiving CPAP through a freestanding system. The respiratory therapist notes that although a wick-type heated humidifier is in use, no rainout (condensate) is present in the circuit. Which of the following would be appropriate to do in this situation?
1. Nothing; this is not unusual
2. Check that the heater is working
3. Determine when the system was last changed
4. Evaluate the system to see if water has been added to the humidifier
A. 1 only
B. 2 and 3 only
C. 1, 2, and 4 only
D. 1, 2, 3, and 4

16. A patient on VC-CMV receives a bronchodilator by metered-dose inhaler. The flow—volume graphics are shown in the following figure. How would you interpret this ventilator graphic as it relates to the patient's response to therapy?



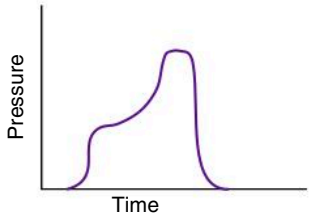
Flow-volume loop for a patient before and after bronchodilator therapy (Question 16).

17. While monitoring a patient on mechanical ventilation after open heart surgery, a respiratory therapist notes that the inspiratory volume is 550 mL and the expiratory volume is 375 mL. The ventilator volume-time and pressure-volume graphics appear in the following figure. Having established that a fairly large leak is present, the therapist checks the cuff and ventilator circuit and cannot find a leak. What could be another possible source of the leak?



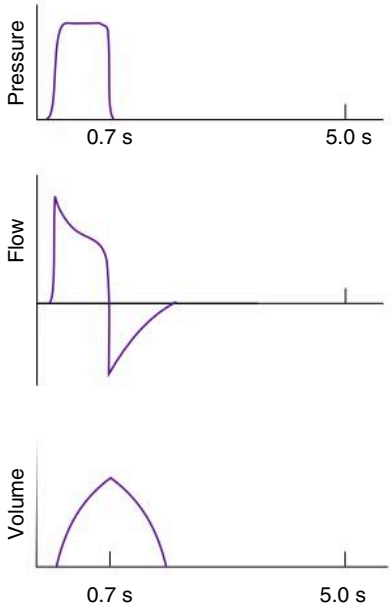
Volume-time curve and pressure-volume loop for a mechanically ventilated patient (Question 17).

18. A patient on PC-CMV has a set pressure of 12 cm H_2O. The pressure-time graphic appears in the following figure. R_{aw} is 12 cm H_2O, and static lung compliance is 30 cm H_2O. The patient is actively inspiring and appears to be "air hungry." What is the likely problem? What is the maximum gas flow available to this patient?



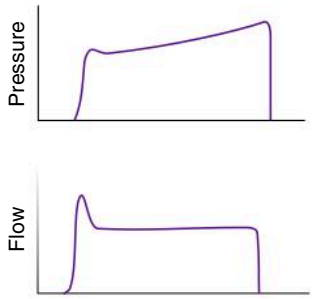
Pressure-time curve (Question 18).

19. A patient on PC-CMV has a set pressure of 30 cm H_2O, a rate of 12 breaths/min, and a T_I of 0.7 seconds. Pressure, flow, and volume scalars are shown in the following figure. V_T delivery is 0.5 L, and the patient has respiratory acidosis. The respiratory therapist wants to increase V_T. In this situation, what is the best way to increase the V_T?



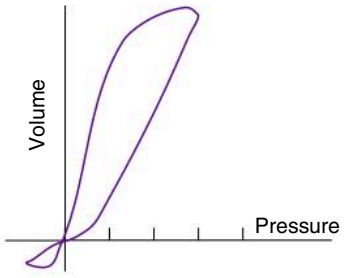
Pressure, flow, and volume scalars (Question 19).

20. During pressure augmentation (P_{aug}), the clinician notices the pressure-time and flow-time graphics (see the following figure). T_I appears to be longer compared with previous graphic displays for the same patient. How would you interpret these findings?



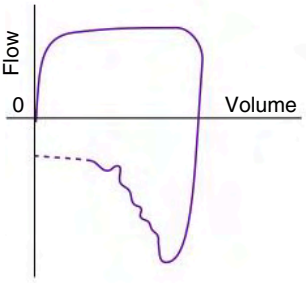
Pressure and flow scalars for a pressure augmentation (P_{aug}) breath (Question 20).

21. A patient's pressure-volume graphic is shown in the following figure. The patient is using accessory muscles to breathe during inspiration. What could be the source of this problem?



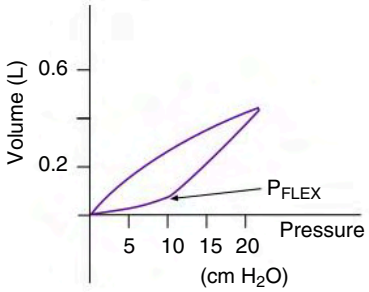
Pressure-volume curve (Question 21).

22. A respiratory therapist increases the mandatory rate to compensate for respiratory acidosis in a patient with COPD on intermittent mandatory ventilation (IMV). After the change, the PIP increases from 38 to 45 cm H_2O and P_{plat} increases from 27 to 35 cm H_2O. The flow-volume loop has also changed in appearance (see the following figure), and the patient now appears to be in distress. The patient's blood pressure has dropped from 135/95 to 125/85 mm Hg. What do you think is the problem, and what is at least one possible solution?



Flow-volume loop (Question 22).

23. PEEP therapy needs to be adjusted for a patient with severe hypoxemia. The pressure-volume loop for this patient appears in the following figure. What would be a reasonable PEEP level to set for this patient, assuming all other parameters are stable?



Pressure-volume loop (Question 23).

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CHAPTER 19

Basic Concepts of Noninvasive Positive Pressure Ventilation

OUTLINE

KEY TERMS

- Acute cardiogenic pulmonary edema
- Chest cuirass
- Community-acquired pneumonia
- Cor pulmonale
- Delay-time control
- Expiratory positive airway pressure
- Inspiratory positive airway pressure
- Inspissated secretions
- Intermittent positive pressure breathing
- Intermittent positive pressure ventilation
- Iron lung
- Nocturnal hypoventilation
- Noninvasive positive pressure ventilation
- Obstructive sleep apnea
- Pressure-targeted ventilators
- Ramp
- Simethicone agents

LEARNING OBJECTIVES

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

1. Define *noninvasive ventilation* and discuss the three basic noninvasive techniques.
2. Discuss the clinical and physiological benefits of noninvasive positive pressure ventilation (NIV).
3. Identify the selection and exclusion criteria for NIV application in the acute and chronic care settings.
4. Compare the types of ventilators used for noninvasive ventilation.
5. Explain the importance of humidification during NIV application.
6. Describe the factors that will influence the fractional inspired oxygen concentration (F_IO_2) from a portable pressure-targeted ventilator.
7. Identify possible causes of rebreathing CO_2 during NIV administration from a portable pressure-targeted ventilator.
8. Compare the advantages and disadvantages of the various types of interfaces for the application of NIV.
9. List the steps used in the initiation of NIV.
10. Discuss several factors that affect the delivery of aerosols during NIV.
11. Identify several indicators of success for patients on NIV.
12. Make recommendations for ventilator changes based on observation of the patient's respiratory status, acid-base status, or oxygenation status.
13. Recognize potential complications of NIV.
14. Provide optional solutions to complications of NIV.
15. Describe two basic approaches to weaning the patient from NIV.

Noninvasive ventilation (NIV) is defined as the delivery of mechanical ventilation to the lungs using techniques that do not require an endotracheal airway. Before 1960, nearly all patients requiring mechanical ventilation were treated with devices that relied on negative pressure ventilators (i.e., the use of a tank respirator or chest-wrap device) that were able to apply subatmospheric pressure to the body or chest area to ventilate the lungs.

Groundbreaking studies by researchers beginning in the 1950s found that the survival rate for invasive positive pressure ventilation (IPPV) delivered via an endotracheal or tracheostomy tube was higher than that for negative pressure ventilation.[1] As a result, IPPV became the standard of practice for the support and management of patients with acute and chronic respiratory failure.

Invasive ventilation has been shown to be an effective and often necessary method to support alveolar ventilation; however, there are associated risks that often can result in increased mortality and morbidity and a higher financial cost. Advances in technology and results of numerous clinical trials during the past 25 years have demonstrated that NIV is a viable modality for the treatment of acute respiratory failure (ARF) in selected patients. Indeed, clinical application of NIV via a nasal mask, mouthpiece, or full-face mask has been shown to reduce the need for intubation and its related complications, reduce mortality rates, and shorten the hospital stay for selected groups of patients requiring mechanical ventilatory support.[2-7]

TYPES OF NONINVASIVE VENTILATION TECHNIQUES

Three basic methods of applying NIV have been successfully used in the treatment of patients. These methods include negative pressure ventilation and positive pressure NIV, and abdominal-displacement ventilation, which will be discussed in Chapter 21.

Negative Pressure Ventilation

Use of negative pressure ventilators peaked during a worldwide polio epidemic in the 1950s.[8] Negative pressure ventilators, or *body ventilators*, operated on the principle of increasing lung volumes by intermittently applying negative pressure to the entire body below the neck or just to the upper region of the chest. The negative pressure was transmitted across the chest wall, into the pleural space, and into the intraalveolar space. The resulting increase in transpulmonary pressure caused air to enter the lungs. Exhalation was passive and simply depended on the elastic recoil of the lung and chest wall.

The first successful negative pressure ventilator, commonly referred to as the **iron lung**, was designed in 1928 by engineer Phillip Drinker and Dr. Charles McKhann. It consisted of a large metal cylinder that enclosed the patient's entire body below the neck, leaving the head protruding through an airtight rubber neck seal. A simpler and less expensive version of this tank device, which was developed by J. H. Emerson in 1931, became the ventilator that was predominantly used to treat patients paralyzed by polio.

The bulk and lack of portability of the iron lung, along with the difficulty in providing routine nursing care for patients, led to the development of smaller, portable negative pressure devices. The **chest cuirass**, or *shell ventilator*, gained wide popularity during the 1950s. Two versions of this device were primarily used to apply negative pressure to the thorax and upper abdomen. In one version, the patient's chest was covered by a metal shell, which had an air-filled rubber edge that sealed the thorax. Subsequent models used a shell made of plastic, which allowed it to be easier to mold and fit to a patient's chest. Another variation of the chest cuirass used a wraparound piece of plastic over a shell that was powered by a vacuum cleaner motor (Fig. 19.1).

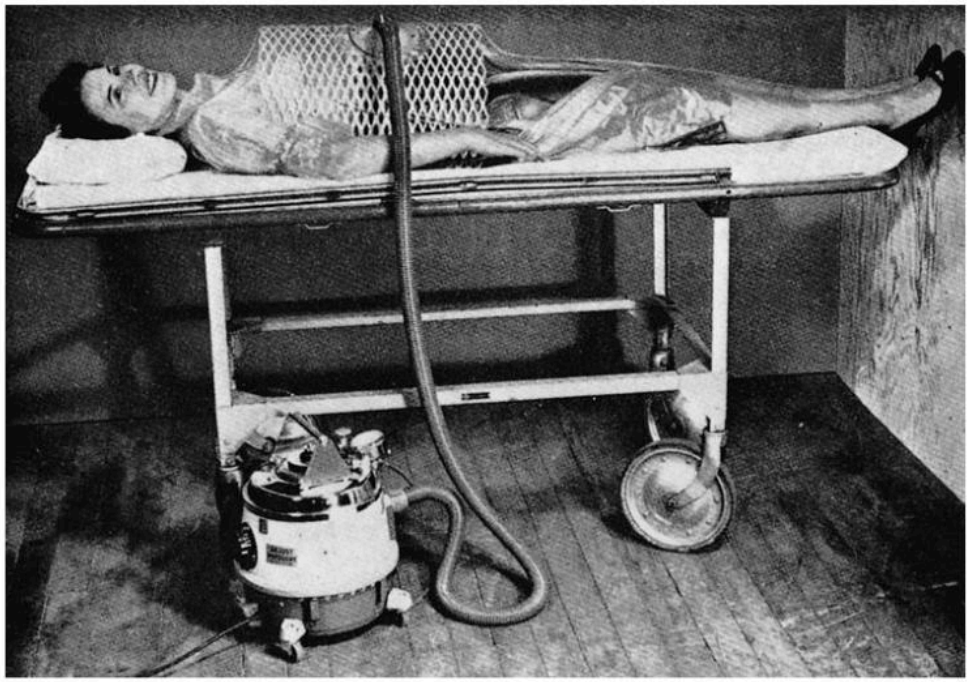

Fig. 19.1 The chest respirator developed by Emerson. (Courtesy J. H. Emerson, Cambridge, Mass.)

Positive Pressure Ventilation

The use of positive pressure ventilation can be traced as far back as 1780, when the first bag-mask apparatus was designed for resuscitative efforts. Positive pressure ventilation with a mask was first used clinically in the mid-1940s by Motley and colleagues[9] to treat patients with ARF. **Intermittent positive pressure ventilation** (IPPV), which used a pressure-targeted ventilator (PTV) and a mask, was later used primarily to treat ARF complicated by chronic obstructive pulmonary disease (COPD) and asthma.

Intermittent positive pressure breathing (IPPB), which used a mask or mouthpiece, became a means of simply delivering aerosolized medication periodically with positive pressure breaths. Clinical studies in the 1980s began to show that the benefits of IPPB were often overstated and could be accomplished using other simpler and more cost-effective therapies.[10] As the routine use of IPPB diminished in clinical practice, nasal-mask continuous positive airway pressure (CPAP) began to emerge as a highly effective therapy in the treatment of **obstructive sleep apnea** (OSA).[11,12] Researchers found that application of low levels of continuous airway pressure through a mask interface created a pneumatic splint that prevented airway collapse during sleep.[11]

The use of NIV was soon reported to be successful in the treatment of chronic ventilatory insufficiency and muscle weakness in patients with various neuromuscular illnesses.[13-15] In 1989, Meduri and colleagues[16] successfully treated a small sample of patients with ARF using pressure support ventilation through a face mask.

These early successes stimulated the production of a variety of interfaces and small pressure- and volume-targeted ventilators that were lightweight, easy to operate, and ideal for home use. Over the past two decades, the use of NIV has significantly increased and it has been used to treat both acute and chronic respiratory failure in a variety of clinical settings.

GOALS OF AND INDICATIONS FOR NONINVASIVE POSITIVE PRESSURE VENTILATION

The goals of **noninvasive positive pressure ventilation** (NIV) and the indications for its use are derived from clinical experiences and systematic research. The following sections review the evidence that supports the use of NIV in various disorders associated with acute and chronic respiratory failure.[17]

Acute Care Setting

NIV is considered by most clinicians to be a lifesaving application for ARF. It offers a number of benefits over IPPV (Box 19.1). The most significant benefit is the avoidance of intubation. Endotracheal intubation is associated with complications such as airway trauma, increased risk for aspiration, ventilator-associated pneumonia, and considerable patient discomfort, typically requiring the use of sedatives. Such complications can lead to a longer hospital stay, higher mortality rate, and increased health care costs. Evidence has established that NIV can safely support ventilation, without endotracheal intubation, until the condition leading to the ARF has been reversed. In addition, evidence strongly indicates that NIV reduces the mortality rate, reduces the duration of ventilator use, and shortens the hospital stay in appropriately selected patients. As such, the primary goal of NIV in the acute care setting is to avoid the need for endotracheal intubation and invasive ventilation.

BOX 19.1 Clinical Benefits of Noninvasive Ventilation

Acute Care Setting
- Reduces the need for endotracheal intubation
- Reduces incidence of ventilator-associated pneumonia
- Shortens stay in the intensive care unit
- Shortens hospital stay
- Reduces mortality
- Preserves physiological airway defenses
- Improves patient comfort
- Reduces need for sedation

Chronic Care Setting
- Alleviates symptoms of chronic hypoventilation
- Improves duration and quality of sleep
- Improves functional capacity
- Prolongs survival

The physiological goal of NIV in ARF is to improve gas exchange by resting the respiratory muscles and increasing alveolar ventilation. NIV reduces diaphragmatic pressure swings, which suggests that the respiratory muscles are being rested. In addition, when positive end-expiratory pressure (PEEP) is applied during pressure support ventilation (PSV), PEEP helps offset auto-PEEP, thereby reducing the work required to initiate inspiration.[17] Likewise, pressure support facilitates inspiration, thus increasing the tidal volume (V_T). Resting of the respiratory muscles and improved V_T lead to a lower arterial partial pressure of CO_2 (P_aCO_2), better oxygenation, and decreased respiratory rates.

Acute Exacerbation of Chronic Obstructive Pulmonary Disease

During an acute exacerbation of COPD (i.e., acute-on-chronic hypercapnic respiratory failure), increased airway resistance and an increased respiratory rate lead to hyperinflation, development of auto-PEEP (air trapping), and alveolar hypoventilation. As hyperinflation worsens, respiratory muscle activity increases, significantly increasing the O_2 cost of breathing. This becomes a vicious circle of increased demand for ventilation, further air trapping, hypoventilation, and muscle fatigue. Without intervention, ventilatory failure and death may occur. Conventional medical therapy for these patients has included bronchodilators, anti-inflammatory agents, judicious use of supplemental O_2, and antibiotics. If these measures failed, endotracheal intubation and invasive ventilation were the next therapeutic steps. Studies of patients with COPD who have experienced acute exacerbations have shown that NIV reduces inspiratory muscle activity and the respiratory rate and increases V_T and minute volume, allowing for better gas exchange and respiratory muscle rest.[18-21] NIV may help reverse the acute condition when used in conjunction with conventional medical therapy.[18]

The use of NIV in the treatment of ARF caused by COPD exacerbation has been successful and has been studied more than any other disorder leading to ARF. The strongest evidence from randomized control trials (RCTs) has confirmed that use of NIV with a face mask significantly reduces the need for intubation, shortens the duration of mechanical ventilation, shortens the