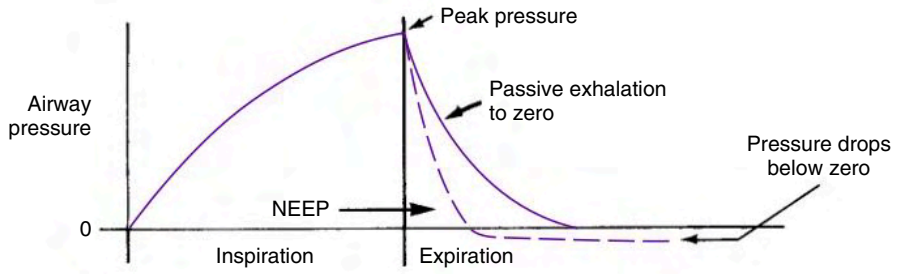

Fig. 3.13 Negative end-expiratory pressure (NEEP). Expiration occurs more rapidly, and the pressure drops below baseline (negative pressure) compared with a normal passive exhalation to zero end-expiratory pressure.

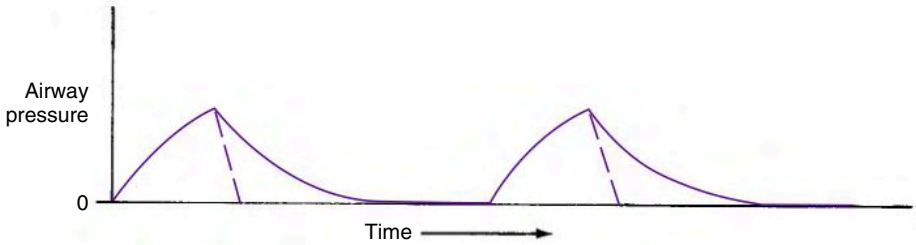

Fig. 3.14 Positive pressure ventilation with expiratory retard *(solid line)* and passive expiration to zero baseline *(dashed line)*. Expiratory retard does not necessarily change expiratory time, which also depends on the patient's spontaneous pattern. However, it increases the amount of pressure in the airway during exhalation.

Time-Limited Expiration

Current mechanical ventilators (e.g., BD Vyaire CareFusion AVEA, Servo-i, Dräger V500, Medtronics Puritan Bennett 840/980) have a mode that allows the clinician to control T_I and expiratory time (T_E). The Dräger Evita was the first ventilator in the United States to provide this mode, which was called *airway pressure release ventilation (APRV)*. During APRV, two time settings are used: Time 1 (T_1) controls the time high pressure is applied, and Time 2 (T_2) controls the *release time,* or the time low pressure is applied. This mode of ventilation limits the expiratory time.

Since the introduction of APRV, other manufacturers of ICU ventilators have chosen to incorporate this mode into their ventilator settings. Interestingly, they use other names for this mode. For example, the Servo-i refers to APRV as Bi-Vent and the Hamilton G5 refers to APRV as Duo-PAP. (APRV is covered in more detail in Chapter 23.)

Continuous Gas Flow During Expiration

Many ICU ventilators provide gas flow through the patient circuit during the latter part of the expiratory phase. When gas flow is provided only during the end of exhalation, resistance to exhalation is minimized. With some ventilators, the clinician sets system flow, whereas in others the system flow is automatically set by the ventilator (e.g., Servo-i). This feature provides immediate inspiratory flow to a patient on demand and in most cases also serves as part of the flow-triggering mechanism.

Expiratory Hold (End-Expiratory Pause)

Expiratory hold, or end-expiratory pause, is a maneuver transiently performed at the end of exhalation. It is accomplished by first allowing the patient to perform a quiet exhalation. The ventilator then pauses before delivering the next machine breath. During this time, both the expiratory and inspiratory valves are closed. Delivery of the next inspiration is briefly delayed. The purpose of this maneuver is to measure pressure associated with air trapped in the lungs at the end of the expiration (i.e., auto-PEEP).

An accurate reading of end-expiratory pressure is impossible to obtain if a patient is breathing spontaneously. However, measurement of the exact amount of auto-PEEP present is not always necessary; simply detecting its presence may be sufficient. Auto-PEEP can be detected in the flow curve on a ventilator that provides a graphic display of gas flow; it is present if flow does not return to zero when a new mandatory ventilator breath begins (see Chapter 9). (NOTE: A respirometer can also be used if a graphic display is not available. The respirometer is placed in line between the ventilator's Y-connector and the patient's endotracheal tube connector. If the respirometer's needle continues to rotate when the next breath begins, air trapping is present [i.e., the patient is still exhaling when the next mandatory breath occurs].)

Expiratory Retard

Spontaneously breathing individuals with a disease that leads to early airway closure (e.g., emphysema) require a prolonged expiratory phase. Many of these patients can accomplish a prolonged expiration during spontaneous breathing by using a technique called *pursed-lip breathing*. Obviously, a patient cannot use pursed-lip breathing with an endotracheal tube in place. To mimic pursed-lip breathing, earlier ventilators provided an expiratory adjunct called *expiratory retard,* which added a degree of resistance to exhalation (Fig. 3.14). Although theoretically expiratory retard should prevent early airway closure and improve ventilation, this technique is not commonly used in clinical practice. It is important to recognize that ventilator

circuits, expiratory valves, and bacterial filters placed on the expiratory side of the patient circuit produce a certain amount of expiratory retard because they cause resistance to flow. This is especially true of expiratory filters, which can accumulate moisture from the patient's exhaled air. The clinician can check for expiratory resistance by observing the pressure manometer and the ventilator pressure—time and flow—time graphics. (Increased resistance is present if pressure and flow return to baseline slowly during exhalation [see Chapter 9].)

Continuous Positive Airway Pressure and Positive End-Expiratory Pressure

Two methods of applying continuous pressure to the airways have been developed to improve oxygenation in patients with refractory hypoxemia: **continuous positive airway pressure** (CPAP) and PEEP.

CPAP involves the application of pressures above ambient pressure throughout inspiration and expiration to improve oxygenation in a spontaneously breathing patient (Fig. 3.15). It can be applied through a freestanding CPAP system or a ventilator. CPAP has been used for the treatment of a variety of disorders, including postoperative atelectasis and obstructive sleep apnea (see Chapter 13 for more details on the use of CPAP).

Like CPAP, PEEP involves applying positive pressure to the airway throughout the respiratory cycle. The pressure in the airway therefore remains above ambient even at the end of expiration. According to its purest definition, the term *PEEP* is defined as positive pressure at the end of exhalation during either spontaneous breathing or mechanical ventilation. In practice, however, clinicians commonly use the term to describe the application of continuous positive pressure when a patient is also receiving mandatory breaths from a ventilator (Figs. 3.16 and 3.17). PEEP becomes the baseline variable during mechanical ventilation.

CPAP and PEEP theoretically help prevent early airway closure and alveolar collapse at the end of expiration by increasing (and

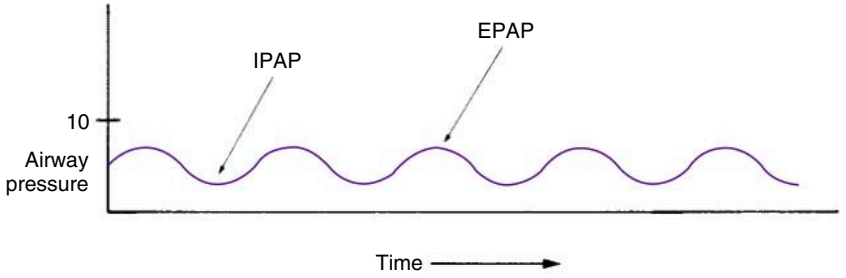

Fig. 3.15 Simplified pressure—time waveform showing continuous positive airway pressure (CPAP). Breathing is spontaneous. Inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) are present. Pressures remain positive and do not return to a zero baseline.

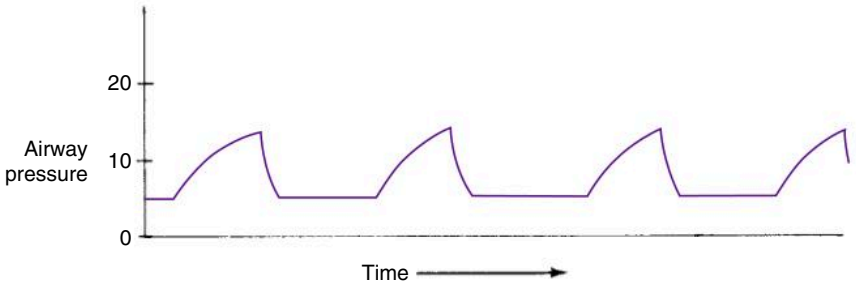

Fig. 3.16 Positive end-expiratory pressure (PEEP) during controlled ventilation. No spontaneous breaths are taken between mandatory breaths, and there are no negative deflections of the baseline, which is maintained above zero.

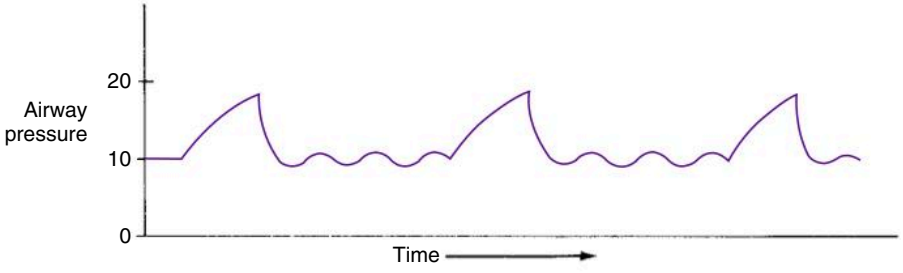

Fig. 3.17 Continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) with intermittent mandatory breaths (also called intermittent mandatory ventilation [IMV] with PEEP or CPAP). Spontaneous breaths are taken between mandatory breaths, and the baseline is maintained above zero. The mandatory breaths are equidistant and occur regardless of the phase of the patient's spontaneous respiratory cycle.

BOX 3.7 Other Names for BiPAP

- Bilevel airway pressure
- Bilevel positive pressure
- Bilevel positive airway pressure
- Bilevel continuous positive airway pressure (CPAP)
- Bilevel positive end-expiratory pressure (PEEP)
- Bilevel pressure assist
- Bilevel pressure support

BOX 3.8 Control Variables, Phase Variables, and Types of Breaths

Control Variables

Control variables are the main variables the ventilator adjusts to produce inspiration. The two primary control variables are pressure and volume.

Phase Variables

Phase variables control the four phases of a breath (i.e., beginning inspiration, inspiration, end inspiration, and expiration). Types of phase variables include:

- *Trigger variable* (begins inspiration)
- *Limit variable* (restricts the magnitude of a variable during inspiration)
- *Cycle variable* (ends inspiration)
- *Baseline variable* (the parameter controlled during exhalation)

Types of Breaths

- *Spontaneous breaths:* Breaths are started by the patient (patient triggered), and tidal volume delivery is determined by the patient (patient cycled).
- *Mandatory breaths:* The ventilator determines the start time for breaths (time triggered) or the tidal volume (volume cycled).
- *Assisted breaths:* Breaths have characteristics of both mandatory and spontaneous breaths (i.e.., all or part of a breath is generated by the ventilator). The ventilator therefore does part of the work of breathing for the patient.

normalizing) the patient's functional residual capacity, which in turn allows for better oxygenation.

Another variation of PEEP and CPAP therapy that is commonly used is bilevel positive airway pressure, or BiPAP. BiPAP is the brand name of a machine manufactured by Philips Respironics (Murrysville, PA), which became popular in the 1980s as a home care device for treating obstructive sleep apnea. The term *BiPAP* has become so commonly used that it is often applied to any device that provides bilevel pressure control (Box 3.7). Fig. 3.18 shows a simplified pressure-time waveform generated by a BiPAP machine.

With bilevel positive pressure, the inspiratory positive airway pressure is higher than the expiratory positive airway pressure. This form of ventilation is patient triggered, pressure targeted, and flow or time cycled. The application of BiPAP in noninvasive ventilation is discussed in Chapter 19.

TYPES OF BREATHS

Three types of mechanical ventilation breaths can be described: spontaneous breaths, mandatory breaths, and assisted breaths. **Spontaneous breaths** are initiated by the patient (i.e., patient triggered), and volume delivery is determined by the patient (i.e., patient cycled). With spontaneous breaths, the volume and flow delivered are based on patient demand rather than a value set by the ventilator operator. During a **mandatory breath,** the ventilator determines the start time (time triggering) or tidal volume (or both). In other words, the ventilator triggers and cycles the breath. **Assisted breaths** have characteristics of spontaneous and mandatory breaths. In an assisted breath, all or part of the breath is generated by the ventilator. As discussed in Chapter 5, the ventilator does part of the breathing for the patient.

Box 3.8 summarizes the main points of control variables, phase variables, and breath types. Fig. 3.19 summarizes the criteria for determining the phase variables that are active during the delivery of a breath.[4]

SUMMARY

- The equation of motion provides a mathematical model for describing the relationships among pressure, volume, flow, and time during a spontaneous or mechanical breath.

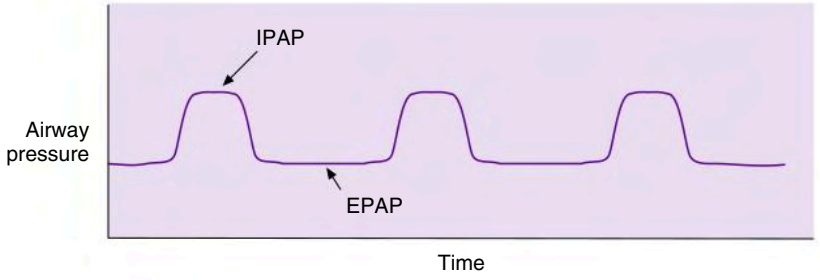

Fig. 3.18 Inspiratory positive airway pressure (IPAP) plus expiratory positive airway pressure (EPAP). IPAP is higher than EPAP when applied to patients. This technique, also called *bilevel positive airway pressure,* or *BiPAP,* is used for noninvasive ventilation in homecare.

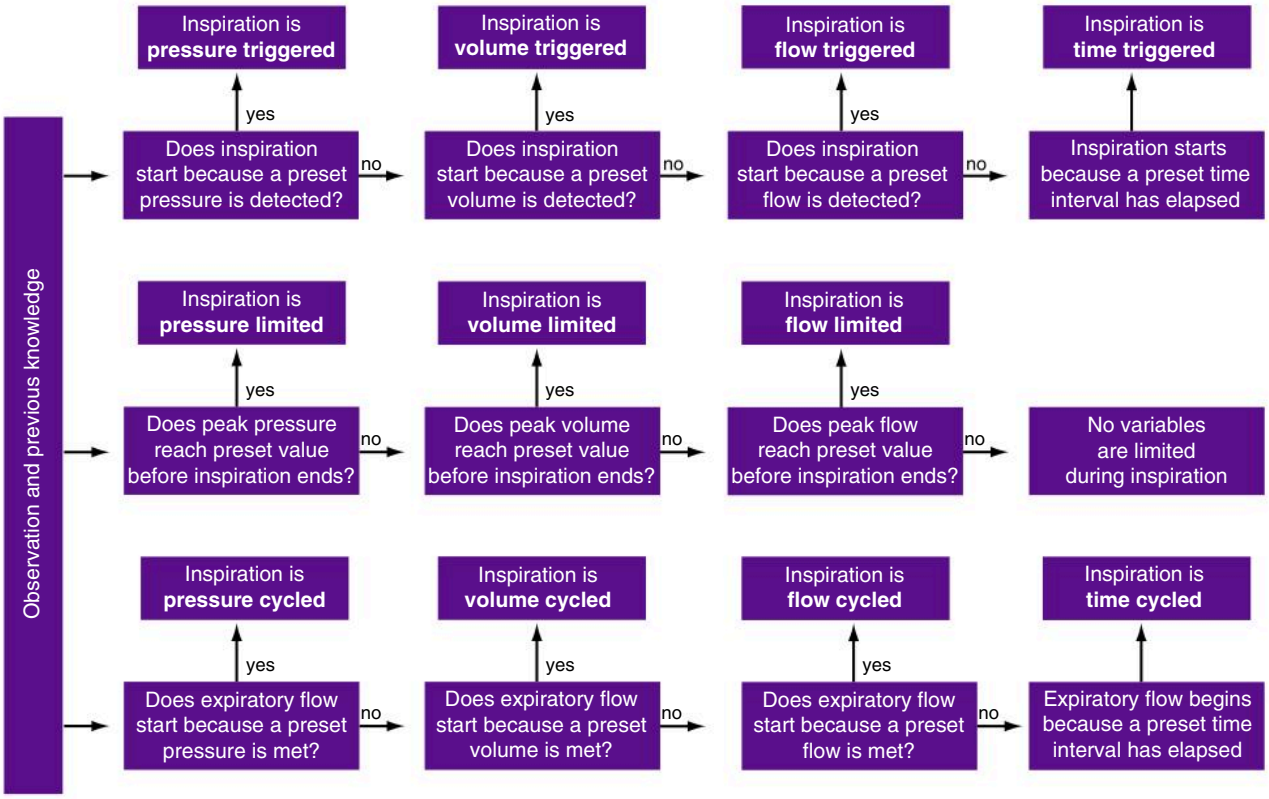

Fig. 3.19 Criteria for determining phase variables during delivery of a breath with mechanical ventilation. (From Kacmarek RM, Stoller JK, Heuer AJ, editors: *Egan's fundamentals of respiratory care,* ed 11, St. Louis, MO, 2017, Elsevier.)

- The work of breathing can be accomplished by contraction of the respiratory muscles during spontaneous breathing or the ventilator during a mechanical ventilatory breath.
- Two factors determine the way that the inspiratory volume is delivered during mechanical ventilation: the structural design of the ventilator and the ventilator mode set by the clinician.
- The primary variable that the ventilator adjusts to produce inspiration is the control variable. Although ventilators can be volume, pressure, flow, and time controlled, the two most commonly used control variables are pressure and volume.
- Determining which variable is controlled can be determined by using graphical analysis. The control variable will remain constant regardless of changes in the patient's respiratory characteristics.
- Pressure and flow waveforms delivered by a ventilator are often identified by clinicians as rectangular, exponential, sine wave, and ramp.
- Phase variables are used to describe variables that (1) begin inspiration, (2) terminate inspiration and cycle the ventilator from inspiration to expiration, (3) can be limited during inspiration, and (4) describe characteristics of the expiratory phase.
- CPAP and PEEP are two methods of applying continuous pressure to the airways to improve oxygenation in patients with refractory hypoxemia.

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

1. Write the equation of motion.

2. Explain the term *elastic recoil pressure* in the equation of motion.

3. Which of the following phase variables is responsible for beginning inspiration?
A. Trigger variable
B. Cycle variable
C. Limit variable
D. Baseline variable

4. List two other names that are used to describe pressure-controlled ventilation.

5. Which of the following variables will remain constant if airway resistance varies during a pressure-controlled breath?
1. Pressure
2. Tidal volume
3. Inspiratory flow
4. Expiratory time
   a. 1 only
   b. 3 only
   c. 2 and 3 only
   d. 1 and 4 only

6. Compare pressure, volume, and flow delivery in pressure-controlled breaths and volume-controlled breaths.

7. What are the two most common patient-triggering variables?
8. What happens in ICU ventilators if the high-pressure limit is exceeded?
   1. Inspiration continues, but pressure is limited.
   2. Inspiration ends, and tidal volume is reduced.
   3. An alarm sounds.
   4. Ventilator function does not change.
      a. 1 only
      b. 4 only
      c. 1 and 3 only
      d. 2 and 3 only
9. Flow triggering gained widespread use by clinicians because:
   A. The respiratory therapist could set it more easily.
   B. It required less work of breathing for the patient.
   C. It was less expensive to manufacture.
   D. It could be used with any mode of ventilation.
10. A patient is on mechanical ventilation. The tidal volume is set at 600 mL and the rate at 7 breaths/min. The low exhaled volume alarm, set at 500 mL, is suddenly activated. The low-pressure alarm is also activated. The volume monitor shows 0 mL. The peak pressure is 2 cm H_2O. On the volume—time waveform, the expiratory portion of the volume curve plateaus and does not return to zero. The most likely cause of this problem is:
    A. Disconnection at the Y-connector
    B. Loss of volume resulting from tubing compressibility
    C. Leakage around the endotracheal tube
    D. Patient coughing
11. Inflation hold increases the inspiratory time.
    A. True
    B. False
12. Which of the following phase variables terminates inspiration?
    A. Limit variable
    B. Trigger variable
    C. Baseline variable
    D. Cycle variable
13. On a pressure-time waveform, the curve during the expiratory phase does not return to the baseline rapidly as it normally would. It eventually reaches the baseline. This may be a result of:
    A. An obstruction in the expiratory line
    B. PEEP set above zero baseline
    C. NEEP
    D. A leak in the circuit
14. Which of the following mechanical ventilation techniques mimics pursed-lip breathing and has been used to prolong the expiratory phase of spontaneously breathing individuals with a disease that leads to early airway closure (e.g., emphysema)?
    A. Setting a high-pressure limit
    B. Inspiratory hold
    C. End-expiratory hold
    D. Expiratory retard
15. Which of the following describes the type of ventilation when the pressure-time waveform does not change during inspiration but the volume-time waveform changes when lung characteristics (i.e., airway resistance and lung compliance) change?
    A. Volume-controlled ventilation
    B. Pressure-controlled ventilation
    C. Time-controlled ventilation
    D. Flow-controlled ventilation

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CHAPTER 4

Establishing the Need for Mechanical Ventilation

OUTLINE

KEY TERMS

- Acute hypercapnic respiratory failure
- Acute hypoxemic respiratory failure
- Acute-on-chronic respiratory failure
- Acute respiratory failure
- Biot respirations
- Cheyne-Stokes respirations
- Functional residual capacity
- Homeostasis
- Paradoxical breathing
- Permissive hypercapnia
- Residual volume
- Respirometer
- Silent chest
- Vital capacity

LEARNING OBJECTIVES

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

1. Differentiate between acute respiratory failure (ARF) and respiratory insufficiency.
2. Describe three categories of disorders that may lead to respiratory insufficiency or ARF.
3. Compare normal values for the vital capacity, maximum inspiratory pressure, maximum expiratory pressure (MEP), forced expiratory volume in 1 second (FEV_1), peak expiratory flow, physiological dead space/tidal volume (V_D/V_T) ratio, alveolar-arterial oxygen pressure difference ($P_{[A-a]}O_2$), and arterial to alveolar partial pressure of oxygen (P_aO_2/P_AO_2) ratio with abnormal values that indicate the need for ventilatory support.

The ability to recognize that a patient requires an artificial airway and mechanical ventilation is an essential skill for clinicians. Although ventilators have been used for more than half a century, surprisingly little evidence and few precise criteria are available to guide clinicians about when to initiate ventilatory support. Originally, mechanical ventilation was instituted because respiratory failure was seen as a "derangement" of gas exchange in the lungs.[1,2] Indeed, clinicians traditionally have relied heavily on arterial blood gas (ABG) analysis to identify the presence of respiratory failure and the need for ventilatory support.[3] More recently, clinicians have used ventilatory measurements (e.g., respiratory muscle strength) to support their decision to initiate mechanical ventilation. Interestingly, many of these threshold measurements actually reflect criteria that clinicians use to determine when to wean a patient from ventilation.

Decisions made in the acute care setting must be supported by evidence-based criteria. The evidence should clearly demonstrate that a particular intervention is beneficial and is associated with effective outcomes, such as improved quality of life, reduced length of stay, or a lower mortality rate.[3] This chapter provides information to help clinicians recognize the signs of respiratory distress and respiratory failure. Specific pathological processes and methods used to identify the need for an artificial airway and mechanical ventilation are discussed. Noninvasive positive pressure ventilation (NIV), an important alternative to invasive positive pressure ventilation, is also reviewed. Five patient case studies

are presented to demonstrate how clinicians can apply various criteria establishing the need for mechanical ventilation in patients with respiratory failure.

ACUTE RESPIRATORY FAILURE

The primary purpose of ventilation is to maintain **homeostasis.** Mechanical ventilation is indicated when a person cannot achieve an appropriate level of ventilation to maintain adequate gas exchange and acid-base balance. Box 4.1 lists the physiological and clinical objectives of mechanical ventilation.[4]

Recognizing the Patient in Respiratory Distress

Left untreated, acute respiratory failure can lead to coma and eventually death. Early recognition of impending respiratory failure can significantly improve the outcomes for these patients. A number of simple and direct observations can be used to identify impending respiratory failure and guide the selection of an appropriate therapeutic strategy.

The initial assessment of the patient in respiratory distress should focus on several physical findings. First, determine the patient's level of consciousness. Is the patient awake or asleep? If the patient is asleep or unconscious, can the patient be awakened and, if so, to what extent? Second, assess the appearance and texture of the patient's skin. Do the nail beds or lips show evidence of cyanosis? Is the patient pale and diaphoretic (sweating)? Third, evaluate the patient's vital signs (e.g., respiratory rate, heart rate, blood pressure, body temperature, oxygenation status).

The sudden onset of dyspnea is typically accompanied by physical signs of respiratory distress. For example, patients experiencing respiratory distress appear anxious, the forehead is furrowed, and the nostrils flared. These patients may be diaphoretic and flushed. They may also try to sit upright or, if seated, lean forward with their elbows resting on a bedside table or their knees. Patients in respiratory or cardiac distress may appear ashen, pale, or cyanotic and using their accessory muscles of respiration (e.g., sternocleidomastoid, scalene, and trapezius muscles). In severe respiratory distress, the intercostal spaces and the supraclavicular notch may appear indented (retracted) during active inspiration. The patient may complain of shortness of breath. Paradoxical or abnormal movement of the thorax and abdomen may be noted, and abnormal breath sounds may be heard on auscultation. Tachycardia, arrhythmias, and hypotension are common findings.[5] Pulse oximetry, which is a quick and cost-effective method of assessing arterial oxygen saturation and pulse rate, can be used to assess the patient's oxygenation status (see Chapter 10). (NOTE: Anemia and reduced cardiac output can compromise oxygen delivery to the tissues. In such cases, reduced pulse pressures and blood flow may prevent the pulse oximeter from accurately estimating the patient's actual arterial oxygen saturation and heart rate.)

It is worth mentioning that in some cases, signs of respiratory distress are the result of the person experiencing a "panic attack." Simply calming the person and questioning him or her about the distress can usually relieve respiratory distress in this type of patient. (The use of both verbal and nonverbal communication with a patient is vital to effective patient assessment.)

Definition of Respiratory Failure

With **acute respiratory failure** (ARF), respiratory activity is absent or insufficient to maintain adequate oxygen uptake and carbon dioxide clearance, in spite of initial therapy. Clinically, ARF may be defined as the inability to maintain P_aO_2, P_aCO_2, and pH at acceptable levels. ARF is generally considered to be present in a patient who demonstrates (1) a P_aO_2 below the predicted normal range for the patient's age under ambient (atmospheric) conditions, (2) a P_aCO_2 greater than 50 mm Hg and rising, and (3) a falling pH of 7.25 and lower.[1-3]

Two forms of ARF have been described: hypoxemic respiratory failure and hypercapnic respiratory failure.[6] Hypoxemic respiratory failure is a result of severe ventilation/perfusion ($\dot{V}/\dot{Q}$) mismatching. It can also occur with diffusion defects, right-to-left shunting, alveolar hypoventilation, aging, and inadequate inspired oxygen. A good working definition of **acute hypoxemic respiratory failure** is acute life-threatening or vital organ—threatening tissue hypoxia.[3] Hypoxemic respiratory failure can be treated with supplemental oxygen or in combination with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) (see Chapter 13). Mechanical ventilation may also be necessary if hypoxemic respiratory failure occurs along with acute hypercapnic respiratory failure and an increased work of breathing.

BOX 4.1 Objectives of Mechanical Ventilation

Physiological Objectives

1. Support or manipulate pulmonary gas exchange:
   - *Alveolar ventilation:* Achieve eucapnic ventilation or allow **permissive hypercapnia** (NOTE: Permissive hypercapnia is sometimes required in the ventilation of patients with a life-threatening asthma exacerbation, acute lung injury [ALI], or acute respiratory distress syndrome [ARDS] to protect the lung by avoiding high ventilation volumes and pressures.)
   - *Alveolar oxygenation:* Maintain adequate oxygen delivery ($C_aO_2 \times$ Cardiac output)
2. Increase lung volume:
   - *Prevent or treat atelectasis with adequate end-inspiratory lung inflation*
   - *Re*store and maintain an adequate **functional residual capacity** (FRC)
3. Reduce the work of breathing

Clinical Objectives

1. Reverse acute respiratory failure
2. Reverse respiratory distress
3. Reverse hypoxemia
4. Prevent or reverse atelectasis and maintain FRC
5. Reverse respiratory muscle fatigue
6. Permit sedation or paralysis (or both)
7. Reduce systemic or myocardial oxygen consumption
8. Minimize associated complications and reduce mortality

Acute hypercapnic respiratory failure, or acute ventilatory failure, occurs when a person cannot achieve adequate ventilation to maintain a normal P_aCO_2. The ventilatory pump consists of the respiratory muscles, thoracic cage, and nerves that are controlled by respiratory centers in the brainstem. The following types of disorders can lead to pump failure (Box 4.2):

- Central nervous system disorders
- Neuromuscular disorders
- Disorders that increase the work of breathing (WOB)

Recognizing Hypoxemia and Hypercapnia

As shown in Table 4.1, the clinical signs of hypoxemia and hypercapnia closely resemble the signs seen in patients with respiratory distress (Key Point 4.1). Tachycardia and tachypnea are early indicators of hypoxia. In some cases of hypoxemic respiratory failure, the patient's condition can be treated successfully by administering enriched oxygen mixtures. However, some hypoxemic conditions, such as severe shunting, are refractory to oxygen therapy (i.e., administering enriched oxygen mixtures does not significantly reduce the level of hypoxemia).

In patients with hypercapnic respiratory failure, P_aCO_2 levels are elevated with accompanying hypoxemia unless the patient is receiving oxygen therapy. Elevation of P_aCO_2 leads to an increase in cerebral blood flow as a result of dilation of cerebral blood vessels. Severe hypercapnia, if left untreated, eventually leads to CO_2 narcosis, cerebral depression, coma, and death.

Untreated hypoxemia, hypercapnia, and acidosis can lead to cardiac dysrhythmias, ventricular fibrillation, and even cardiac arrest.[7] The potential for these consequences underscores the importance of recognizing that a patient is in acute or impending respiratory failure and the need to initiate therapy in a timely manner. The elements required to achieve a successful outcome are (1) use of supplemental oxygen therapy, (2) maintenance of a patent airway, and (3) continuous monitoring of oxygenation and ventilatory status with pulse oximetry and ABG analysis.

PATIENT HISTORY AND DIAGNOSIS

The various types of pathological conditions that increase the risk for a patient developing respiratory failure were mentioned previously (see Box 4.2). The following is a brief discussion of some of these conditions. Several case studies are presented to illustrate the clinical findings associated with respiratory failure.

Central Nervous System Disorders

Central nervous system (CNS) disorders that decrease respiratory drive, such as depression of the respiratory centers induced by drugs or trauma, can lead to significant reductions in minute ventilation ($\dot{V}/\dot{Q}_E$) and alveolar ventilation ($\dot{V}/\dot{Q}_A$) and, ultimately, to hypercapnia and hypoxemia. In otherwise normal individuals, an increase in P_aCO_2 greater than 70 mm Hg has a CNS depressant effect, which reduces respiratory drive and ventilation. Hypoxemia, which accompanies this process, normally acts as a respiratory stimulant (through stimulation of the peripheral chemoreceptors) to increase breathing. However, because the CNS is already compromised, the body's response to hypoxemia is diminished.

Other CNS disorders associated with tumors, stroke, or head trauma can alter the normal pattern of breathing. For example, a head injury might result in cerebral hemorrhage and increased

BOX 4.2 Disorders and Agents Associated With Hypoventilation and Possible Respiratory Failure

Central Nervous System Disorders

Reduced Drive to Breathe

- Depressant drugs (barbiturates, tranquilizers, narcotics, general anesthetic agents)
- Brain or brainstem lesions (stroke, trauma to the head or neck, cerebral hemorrhage, tumors, spinal cord injury)
- Hypothyroidism
- Sleep apnea syndrome caused by idiopathic central alveolar hypoventilation

Increased Drive to Breathe

- Increased metabolic rate (increased CO_2 production)
- Metabolic acidosis
- Anxiety associated with dyspnea

Neuromuscular Disorders

- Paralytic disorders (e.g., myasthenia gravis, tetanus, botulism, Guillain-Barré syndrome, poliomyelitis, muscular dystrophy, amyotrophic lateral sclerosis)
- Paralytic drugs (e.g., curare, nerve gas, succinylcholine, insecticides, nondepolarizing neuromuscular blocking agents [see Chapter 15])
- Drugs that affect neuromuscular transmission (e.g., aminoglycoside antibiotics, long-term adrenocorticosteroids, calcium channel blockers)
- Impaired muscle function (e.g., electrolyte imbalances, malnutrition, peripheral nerve disorders, atrophy, fatigue, chronic pulmonary disease with decreasing capacity for diaphragmatic contraction as a result of air trapping)

Disorders That Increase the Work of Breathing

- Pleura-occupying lesions (e.g., pleural effusions, hemothorax, empyema, pneumothorax)
- Chest wall deformities (e.g., flail chest, rib fracture, kyphoscoliosis, obesity)
- Increased airway resistance resulting from increased secretions, mucosal edema, bronchoconstriction, airway inflammation, or foreign body aspiration (e.g., asthma, emphysema, chronic bronchitis, croup, acute epiglottitis, acute bronchitis)
- Lung tissue involvement (e.g., interstitial pulmonary fibrotic diseases, aspiration, acute respiratory distress syndrome, cardiogenic pulmonary edema, drug-induced pulmonary edema)
- Pulmonary vascular problems (e.g., pulmonary thromboembolism, pulmonary vascular damage)
- Other problems (e.g., increased metabolic rates with accompanying pulmonary problems)
- Postoperative pulmonary complications
- Dynamic hyperinflation (air trapping)

TABLE 4.1 Conditions Seen With Hypoxemia and Hypercapnia

	Mild to Moderate	Severe
Hypoxemia		
Respiratory findings	Tachypnea	Tachypnea
	Dyspnea	Dyspnea
	Paleness	Cyanosis
Cardiovascular findings	Tachycardia	Tachycardia (eventually bradycardia, arrhythmias)
	Mild hypertension	Hypertension (eventually hypotension)
	Peripheral vasoconstriction	
Neurological findings	Restlessness	Somnolence
	Disorientation	Confusion
	Headaches	Delirium
	Lethargy	Blurred vision
		Tunnel vision
		Loss of coordination
		Impaired judgment
		Slowed reaction time
		Manic-depressive activity
		Loss of consciousness
		Coma
Hypercapnia		
Respiratory findings	Tachypnea	Tachypnea (eventually bradypnea)
	Dyspnea	
Cardiovascular findings	Tachycardia	Tachycardia
	Hypertension	Hypertension (eventually hypotension)
	Vasodilation	
Neurological findings	Headaches	Hallucinations
	Drowsiness	Hypomania
	Dizziness	Seizures
	Confusion	Loss of consciousness (eventually coma)
Signs	Sweating	
	Skin redness	

***Key Point* 4.1** Tachycardia and tachypnea are nonspecific and mostly subjective signs that may provide only limited help in deciding when to intubate and ventilate a patient.[3]

intracranial pressure (ICP). If significant bleeding occurs with these types of injuries, abnormal breathing patterns such as Cheyne-Stokes respirations or Biot respirations may occur. In many cases, cerebral abnormalities can also affect normal reflex responses, such as swallowing. In these cases, endotracheal intubation may be required to protect the airway from aspiration or obstruction by the tongue (Case Study 4.1).

There is considerable debate about whether controlled hyperventilation should be used as a ventilatory technique in patients with a closed head injury. Controlled hyperventilation lowers the P_aCO_2 and increases the pH, resulting in reduced cerebral perfusion and reduced ICP. It is important to understand that this effect is temporary, lasting only about 24 hours, because the body eventually adapts to the change through renal compensatory mechanisms.[8] Although controlled hyperventilation is still used by some clinicians to lower sudden increases in ICP, clinicians must keep in mind that the desire to use this technique for patients with traumatic brain injury is not by itself an indication for intubation and mechanical ventilation.[3] Furthermore, patients with traumatic brain injury have a better long-range outcome (3–6 months) when controlled hyperventilation is not used.[8]

Case Study 4.1

Stroke Victim

A 58-year-old man is brought to the emergency department from his home after a suspected stroke (i.e., cerebrovascular accident). Vital signs reveal a heart rate of 94 beats/min, respirations of 16 breaths/min, normal temperature, and systemic arterial blood pressure of 165/95 mm Hg. The patient's pupils respond slowly and unequally to light. Breath sounds are diminished in the lung bases. A sound similar to snoring is heard on inspiration. The patient is unconscious and unresponsive to painful stimuli. What is the most appropriate course of action at this time?

Neuromuscular Disorders

Neuromuscular disorders that can lead to respiratory failure usually are the result of one of the following:

- Motor nerve damage
- Problems with transmission of nerve impulses at the neuromuscular junction
- Muscle dysfunction
- CNS disorders
- Drugs that affect neuromuscular function

The onset of respiratory failure can vary considerably depending on the cause of the neuromuscular dysfunction. Drug-induced neuromuscular failure usually has a rapid onset (Case Study 4.2), whereas the onset of respiratory failure in disease states such as myasthenia gravis may not occur for days or years or it might not happen at all. Regardless of the cause, intubation and mechanical ventilation are indicated if respiratory fatigue occurs rapidly in a patient with a neuromuscular disorder and ARF is imminent.[9]

The maximum inspiratory pressure (MIP) and vital capacity (VC) can be used to assess respiratory muscle strength of patients with neuromuscular disorders. These measurements are noninvasive, relatively easy to obtain, and inexpensive. Respiratory therapists can measure MIP and VC every 2 to 4 hours to monitor changes in respiratory status. Commonly cited threshold values are an MIP of −20 to −30 cm H_2O or less (i.e., 0 to −20 cm H_2O) and a VC lower than 10 to 15 mL/kg. Note that although these measures are often used, their effectiveness in improving outcomes has not been established (Table 4.2).[3] (Techniques for measuring MIP and VC are discussed later in this chapter.)

Case Study 4.2

Unexplained Acute Respiratory Failure

A STAT arterial blood gas evaluation performed on a patient admitted through the emergency department reveals the following: pH = 7.15, P_aCO_2, = 83 mm Hg, P_aO_2, = 34 mm Hg, HCO_3^- = 28 mEq/L on room air. The patient was found unconscious in a nearby park. No other history is available. What is the most appropriate course of action at this time?

Determination of baseline ABG values, along with periodic measurement of oxygen saturation by pulse oximetry (S_pO_2), is also appropriate when caring for patients with neuromuscular problems. Repeating the ABG analysis may be indicated if the patient's clinical status changes significantly. Furthermore, if the patient's condition progressively worsens, the practitioner should *not* wait until an acute situation develops to intervene (Case Study 4.3). Clinicians generally agree that invasive positive pressure ventilation should be initiated before acute respiratory acidosis develops.[3]

Increased Work of Breathing

An increase in the WOB can lead to respiratory failure secondary to respiratory muscle fatigue. WOB normally accounts for 1% to 4% of total oxygen consumption at rest.[10] In patients experiencing respiratory distress, the WOB can increase to as much as 35% to 40% of total oxygen consumption.[11,12] Increased WOB is usually associated with an increased rate or depth of breathing (or both).

Case Study 4.3

Ventilation in Neuromuscular Disorders

Case 1

A 68-year-old woman with a history of myasthenia gravis has been in the hospital for 12 days. She was admitted because her primary disease had worsened. The patient is unable to perform maximum inspiratory pressure (MIP) and slow vital capacity (SVC) maneuvers properly because she cannot seal her lips around the mouthpiece. Her attempts produced the following values: MIP = −34 cm H_2O; SVC = 1.2 L. What should the clinician recommend at this time?

Case 2

A 26-year-old man who is recovering from mycoplasma pneumonia complains of tingling sensations and weakening in his hands and feet. He is admitted to the general medicine service for observation. Over several hours the patient becomes unable to move his legs. A respiratory therapist (RT) is called to assess him. What should the RT recommend at this time?

TABLE 4.2 Indications of Acute Respiratory Failure and the Need for Mechanical Ventilatory Support in Adults

Criteria	Normal Values	Critical Value
Ventilation[a]		
pH	7.35−7.45	<7.25
Arterial partial pressure of carbon dioxide (P_aCO_2) (mm Hg)	35−45	>55 and rising
Ratio of dead space to tidal volume (V_D/V_T)	0.3−0.4	>0.6
Oxygenation[b]		
Arterial partial pressure of oxygen (P_aO_2) (mm Hg)	80−100	<70 (on O_2 ≥0.6)
Alveolar-to-arterial oxygen difference $P_{(A\text{-}a)}O_2$ (mm Hg)	5−20	>450 (on O_2)
Ratio of arterial to alveolar PO_2 (P_aO_2/P_AO_2)	0.75	<0.15
P_aO_2/F_IO_2	475	<200

[a]Indicates the need for ventilatory support.
[b]Indicates the need for oxygen therapy and positive end-expiratory pressure/continuous positive airway pressure.
F_IO_2, fraction of inspired oxygen.

More work is required to move the same V_T when the patient has obstructed airways, a restrictive lung disorder, or both (Case Study 4.4). A patient's tolerance to maintain the increased WOB is limited by the eventual fatigue of respiratory muscles. Increased WOB can induce hypoventilation, respiratory insufficiency, and eventually respiratory failure.

An example of a condition that causes increased WOB is severe chest trauma. Flail chest, pneumothorax, and hemothorax can impair the mechanics of breathing and affect the patient's ability to breathe. A reduction in alveolar ventilation leads to $\dot{V}/\dot{Q}$ mismatching, an increased V_D/V_T, and, ultimately, hypoxemia, hypercapnia, and acidosis. Although the patient's respiratory centers may be intact and responsive to hypercapnia and hypoxemia, his or her ability to maintain the effort to breathe is seriously compromised. Even as the patient initially tries to respond by increasing minute ventilation (i.e., increased respiratory rate and V_T), the P_aO_2 may continue to fall, the P_aCO_2 rises, and the pH decreases despite the patient's compensatory efforts.

In some patients, increased WOB eventually results in rapid, shallow breathing and **paradoxical breathing.**[13] With paradoxical breathing, the abdomen moves out during exhalation and moves in during inhalation while the chest wall moves out during inhalation and moves in during exhalation. This is the reverse of normal breathing, in which the chest wall and abdomen move outward together on inspiration and inward on exhalation. This asynchronous motion of the chest and abdomen is an ominous sign in patients with respiratory distress and usually indicates increased WOB and the onset of respiratory muscle fatigue.[3]

At one time physicians commonly used positive pressure to "splint" the chest and "internally stabilize" the chest wall in patients with flail chest. Current evidence suggests that better outcomes are achieved when patients with flail chest are managed without intubation or invasive positive pressure ventilation (IPPV). IPPV is used for flail chest only when it is associated with imminent respiratory failure.

Case Study 4.4

Asthma Case

A 13-year-old girl is brought to the emergency department for an acute exacerbation of asthma. Continuous nebulizer therapy with a β-2 adrenergic bronchodilator is administered. The patient has been given a high dose of corticosteroids and is receiving oxygen. Four hours after admission, she is alert and responsive. Her respiratory rate is 20 breaths/min. Coarse crackles and end-inspiratory wheezes are heard clearly throughout both lung fields. What recommendation for continuous respiratory care should be made for this patient?

PHYSIOLOGICAL MEASUREMENTS IN ACUTE RESPIRATORY FAILURE

Clinicians often use ventilatory mechanics measurements and ABG results to detect respiratory failure. Unfortunately, valid predictive threshold values for these measurements have not been substantiated.[3] The absence of guidelines can sometimes make it difficult for a novice clinician to know when to intubate or provide ventilation for a patient in distress.

Table 4.3 lists normal adult values for ventilatory mechanics and the suggested critical range that may indicate a need for mechanical ventilation when considered with other assessment criteria. It is worth mentioning that most of these parameters are probably better used as indications for discontinuing mechanical ventilation, with two exceptions: (1) in patients with neuromuscular disorders, the MIP and VC may be beneficial in tracking respiratory muscle strength; (2) in patients with reactive airway disease (e.g., asthma and some patients with chronic obstructive pulmonary disease [COPD]), the forced expiratory volume at 1 second (FEV_1) and the peak expiratory flow (PEF) are helpful for quantifying the degree of airway resistance. FEV_1 is more useful to evaluate small airways function than is the PEF.

Bedside Measurements of Ventilatory Mechanics

Abnormal ventilatory mechanics measurements should alert the clinician to the presence of a potentially serious respiratory problem. As mentioned previously, MIP and VC are the bedside measurements most often used to assess respiratory muscle strength in patients with neuromuscular disease. For patients with acute asthma, PEF is the most frequently used parameter to assess airway resistance. FEV_1 is a better parameter, and bedside spirometry is readily available for measuring FEV_1.

Maximum Inspiratory Pressure

The MIP (or P_{Imax}) is the lowest (i.e., most negative) pressure generated during a forceful inspiratory effort against an occluded airway. MIP is also called *negative inspiratory force (NIF).* At the bedside, MIP is usually measured with a pressure manometer (Fig. 4.1).[14] The device is connected to the patient's airway by means of a mask, mouthpiece, or endotracheal tube adapter. The patient is instructed to inhale forcefully from the device while the

TABLE 4.3 Normal Adult and Critical Range Values

Ventilatory Mechanics	Normal Adult Range	Critical Value That May Indicate the Need for Mechanical Ventilation
Maximum inspiratory pressure (MIP) (cm H_2O)	−100 to −50	−20 to 0
Maximum expiratory pressure (MEP) (cm H_2O)	100	<40
Vital capacity (VC) (mL/kg)	65–75	<10–15
Tidal volume (V_T) (mL/kg)	5–8	<5
Respiratory frequency (f) (breaths/min)	12–20	>35
Forced expired volume at 1 second (FEV_1) (mL/kg)	50–60	<10
Peak expiratory flow (PEF) (L/min)	350–600	75–100

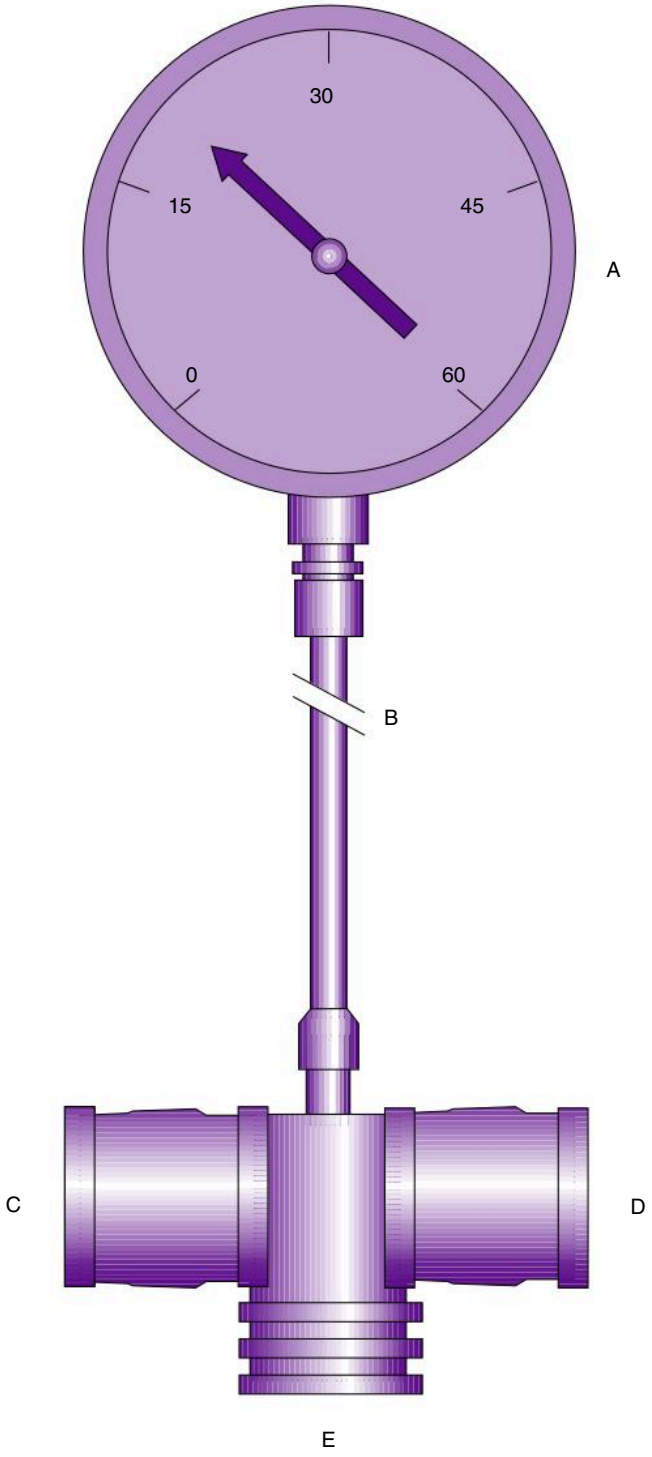

Fig. 4.1 Device for measuring maximum inspiratory pressure. (A) Pressure gauge. (B) Connective tubing. (C) Thumb port, which is occluded during the measurement procedure. (D) One-way valve connection; the valve allows exhalation into the room but does not allow inhalation. (E) Connection to the patient's mouth or endotracheal tube. (Modified from Kacmarek RM, Cycyk-Chapman MC, Young-Palazzo PJ, et al.: Determination of maximum inspiratory pressure: a clinical study and literature review, *Respir Care* 34:868–878, 1989.)

BOX 4.3 Alternative Technique for Measuring Maximum Inspiratory Pressure

For accuracy and reproducibility, measurement of the maximum inspiratory pressure (MIP) must begin as closely as possible to the residual volume (RV). If the patient cannot perform a maximum exhalation, a one-way valve (see Fig. 4.1) may be used. This valve allows exhalation but prevents inspiration. To perform the technique, the clinician occludes the thumb port and watches the pressure gauge as the patient takes multiple breaths (because the patient cannot inspire air against the occluded valve, this brings lung volume close to the RV as the patient exhales over several breaths). MIP measurement stops when the lowest negative value is reached (this may take up to 20 seconds). Although the one-way valve technique can provide an accurate MIP value, this type of prolonged measurement is extremely uncomfortable for the patient. (Students can gain considerable insight into that discomfort by trying this technique as a laboratory exercise.)

respiratory therapist occludes the thumb port of the T-piece connector containing two one-way valves.

Values are most accurate and reproducible when MIP is measured from the **residual volume** (RV), that is, after a maximum exhalation.[14-17] At least two measurements should be obtained if possible. Obtaining more than two or three MIP maneuvers from patients in distress may result in erroneous results because the procedure requires significant effort by the patient. MIP is normally −50 to −100 cm H_2O. An MIP of at least −20 cm H_2O is required to generate a V_T large enough to produce a good cough (Box 4.3).[14-18]

Vital Capacity

Vital capacity is the volume of air that can be maximally exhaled following a maximum inspiration. It can provide valuable information about ventilatory function because the patient must be able to take in a large volume of air to produce a cough strong enough to clear the airway. VC is typically 65 to 75 mL/kg of ideal body weight (IBW), but it may be as high as 100 mL/kg IBW. Values less than 10 to 15 mL/kg IBW are considered inadequate to maintain normal ventilation and produce an effective cough.[19] (See Box 6.2 for calculation of IBW.) VC can be measured at the bedside using a pneumotachometer or **respirometer.** Obtaining reliable measurements requires the patient's cooperation, which typically occurs when the patient receives proper instructions from the clinician about how to perform the procedure (Key Point 4.2).

Peak Expiratory Flow and Forced Expiratory Volume in 1 Second

The PEF is a good indicator of airway resistance and a patient's ability to maintain airway patency. Obtaining reliable measurements of PEF is a part of an effective asthma treatment plan for many asthma patients. A peak flowmeter like the one shown

in Fig. 4.2 can be used to measure PEF. Acceptable values range from 350 to 600 L/min. Low PEFs are most often seen in patients having an acute asthma episode. Values less than 75 to 100 L/min are cause for alarm and indicate severe airflow obstruction.

The FEV_1 is another pulmonary function parameter that can be used to assess airway resistance. FEV_1 is normally about 80% of the VC, or about 50 to 60 mL/kg IBW. An FEV_1 less than 10 mL/kg IBW is considered extremely low. Although several portable spirometers are available for measuring FEV_1 at the bedside, it is *not* an appropriate measurement to perform on a patient who is severely short of breath and in acute respiratory distress.

Key Point 4.2 MIP and VC are bedside measurements most often used to assess respiratory muscle strength of patients with neuromuscular disease.

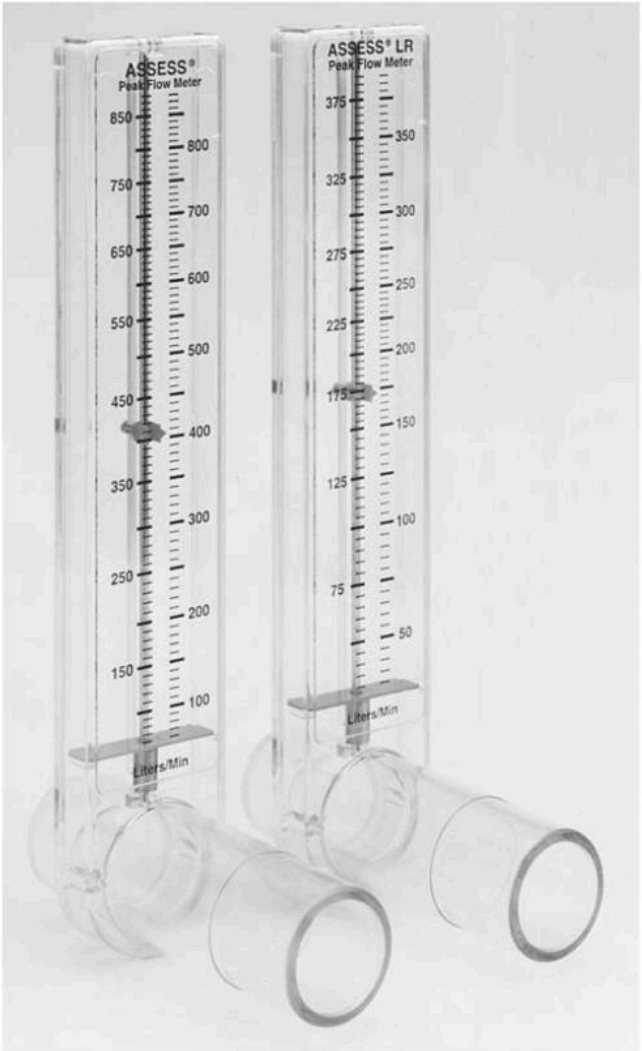

Fig. 4.2 Disposable peak flowmeters. (Used with permission of Philips Respironics, Inc., Murraysville, Pa.)

Respiratory Rate and Minute Ventilation

Two other parameters often mentioned in the assessment of respiratory distress are the respiratory rate (*f*) and minute ventilation ($\dot{V}/\dot{Q}_E$). The respiratory rate is normally about 12 to 20 breaths/min in adults. Respiratory rates exceeding 35 breaths/min for extended periods are a sign of inadequate alveolar ventilation or hypoxemia (or both). As mentioned previously, elevated respiratory rates are an indication of increased WOB, which eventually leads to respiratory muscle fatigue.

Minute ventilation is the product of tidal volume and respiratory rate ($\dot{V}/\dot{Q}_E = \dot{V}/\dot{Q}_t \times f_b$). A normal $\dot{V}/\dot{Q}_E$ is about 5 to 6 L/min, and this value is directly related to the patient's metabolic rate. A $\dot{V}/\dot{Q}_E$ above 10 L/min is cause for concern. In cases in which the patient demonstrates significant pulmonary dysfunction, the $\dot{V}/\dot{Q}_E$ required to maintain a stable P_aCO_2 may become so high that the patient cannot sustain the required work of breathing. Although V_T and $\dot{V}/\dot{Q}_E$ can be measured in unintubated patients with respiratory distress, these measurements are seldom obtained.[3]

Failure of Ventilation and Increased Dead Space

Clinicians generally agree that the best single indicator of adequate ventilation is the P_aCO_2 (Key Point 4.3). A P_aCO_2 of greater than 50 to 55 mm Hg with a decreasing pH (<7.25) indicates acute hypoventilation or acute hypercapnic respiratory failure.

An elevated P_aCO_2 also suggests that dead space (V_D) is increased relative to V_T. The normal V_D/V_T range is 0.3 to 0.4 at normal tidal volumes. Values greater than 0.6 indicate a critical increase in dead space. For example, in a patient with a V_T of 500 mL and a V_D/V_T of 0.6 mL, for each breath taken, only 40% (200 mL) contacts pulmonary blood flow and contributes to alveolar gas exchange; 60% (300 mL) goes to areas of the pulmonary system that are not in contact with the pulmonary capillary bed. Under these conditions, the patient must increase the rate and depth of breathing (i.e., increase $\dot{V}/\dot{Q}_E$) to try to achieve adequate gas exchange. Increases in dead space are associated with $\dot{V}/\dot{Q}$ mismatching (i.e., dead space ventilation is defined as ventilation in excess of perfusion). Common causes of increased dead space ventilation include pulmonary thromboemboli, pulmonary vascular injury, and regional hypoperfusion.

In the past, measurement of the V_D/V_T required the collection of expired gases and simultaneous evaluation of arterial CO_2 tensions. This procedure was time consuming and not well tolerated by patients in severe respiratory failure. V_D/V_T now can be estimated by using less cumbersome, noninvasive methods, such as volumetric capnometry (see Chapter 10).

Key Point 4.3 The best single indicator of the adequacy of ventilation is the P_aCO_2.

Failure of Oxygenation

P_aO_2 is a good indicator of oxygenation status, assuming that abnormal forms of hemoglobin (e.g., carboxyhemoglobin and methemoglobin) are not present. Normal P_aO_2 is 80 to 100 mm Hg when an individual is breathing room air, but this value varies with age and body position. Monitoring S_pO_2 can also provide an easy, noninvasive means of assessing trends in a patient's oxygenation status (Key Point 4.4).[3] A P_aO_2 less than 70 mm Hg (or S_pO_2 <90%) on an oxygen mask (F_IO_2 ≥0.6) indicates refractory hypoxemia or hypoxemic respiratory failure. Additional information about a patient's total oxygen-carrying capacity can be derived from measurements or calculation of his or her arterial oxygen content (C_aO_2) (Box 4.4).

As discussed later in this text (see Chapter 13), the $P_{(A\text{-}a)}O_2$ can be used to determine the cause of altered oxygenation. The normal range for $P_{(A\text{-}a)}O_2$ for patients breathing room air is 5 to 20 mm Hg; the normal range for patients breathing 100% O_2 is 25 to 65 mm Hg. When P_aO_2 is low and $P_{(A\text{-}a)}O_2$ is high, hypoxemia is due to one of the other three general causes: shunt, diffusion defects, and $\dot{V}/\dot{Q}$ mismatching (see Appendix B). (NOTE: In these cases, P_aCO_2 may even be lower than normal, indicating hyperventilation to compensate for hypoxemia.)

The P_aO_2/P_AO_2 ratio is another approach that can be used to evaluate a patient's oxygenation status. P_aO_2/P_AO_2 is normally about 0.75 to 0.95. This range indicates that 75% to 95% of the oxygen available in the alveoli is diffusing into the pulmonary capillaries. For example, a normal P_aO_2 of 90 mm Hg divided by a normal P_AO_2 on room air (100 mm Hg) gives a ratio of 0.90. A value of 0.15 or less is critical (i.e., only 15% of available oxygen is getting into the artery).

Using a P_aO_2/F_IO_2 ratio eliminates the need to calculate the alveolar PO_2 (P_AO_2). Normal values can be calculated as follows: 90 mm Hg/0.21 = 428 (about 430; range, 350–450). A P_aO_2 of 40 mm Hg with an F_IO_2 of 1.0 (40 mm Hg/1.0 = 40) is an example of an extremely severe abnormality (i.e., refractory hypoxemia).[20]

The treatment for hypoxemia is based on its etiology (Table 4.4).[21] For example, when hemoglobin and hematocrit values are low as a result of hemorrhage, the arterial O_2 content is reduced and a blood transfusion is required to improve oxygen content and transport. In some cases, refractory hypoxemic respiratory failure can be treated with PEEP or CPAP (see Chapter 13). When hypoxemia is accompanied by an increase in WOB or a rising P_aCO_2 and a falling pH, mechanical ventilation is required.[3,22] Mask CPAP and oxygen alone are not considered effective treatments for hypoxemia in patients with acute lung injury, and intubation is usually required (Key Point 4.5).[23]

OVERVIEW OF CRITERIA FOR MECHANICAL VENTILATION

The standard criteria for instituting mechanical ventilatory support are listed in Box 4.5. Apnea and impending respiratory failure and arrest are the most obvious indications for invasive ventilation. Current indications for invasive ventilation are shown in Box 4.6.[3]

Reversibility of the underlying disease should always be a consideration. The clinician must consider the patient's medical history, physical assessment, ABG evaluation, lung mechanics measurements, prognosis, and advanced directives (i.e., patient's wishes) when deciding whether to intubate and ventilate a patient's lungs. Thoughtful clinical judgment is essential, as is attention to

TABLE 4.4 Specific Treatments for Arterial Hypoxemia

Cause	Treatment
Hypoventilation	Increase F_IO_2, increase alveolar ventilation
Low ventilation/perfusion ratio	Increase F_IO_2, continuous positive airway pressure (CPAP)
Intrapulmonary shunt	Increase F_IO_2, CPAP
Diffusion defect	Increase F_IO_2, steroids (?), diuretics
Low barometric pressure	Descend (to lower altitude)
Low inspired oxygen concentration (<21%)	Increase F_IO_2

From Downs JB: Has oxygen administration delayed appropriate respiratory care? Fallacies regarding oxygen therapy, *Respir Care* 48:611–620.

Key Point 4.4 P_aO_2 and S_pO_2 are the key indicators of the severity of acute hypoxemic respiratory failure.[3]

Key Point 4.5 No single value for P_aO_2, P_aCO_2, or pH indicates a need for invasive ventilation.[3]

BOX 4.4 Calculation of Arterial Oxygen Content

The arterial oxygen content (C_aO_2) can be calculated with the following equation:

$$C_aO_2([Hb \times 1.34] \times S_aO_2) + (P_aO_2 \times 0.003)$$

where *Hb* is hemoglobin in grams per deciliter of whole blood (g%), S_aO_2 is arterial oxygen saturation, and P_aO_2 is the partial pressure of arterial oxygen in millimeters of mercury (mm Hg).

BOX 4.5 Standard Criteria for Instituting Mechanical Ventilation

- Apnea or absence of breathing
- Acute ventilatory failure
- Impending ventilatory failure
- Refractory hypoxemic respiratory failure with increased work of breathing or an ineffective breathing pattern

BOX 4.6 Indications for Invasive Mechanical Ventilation in Adults With Acute Respiratory Failure

Invasive mechanical ventilation is indicated in any of the following circumstances:

1. Apnea or impending respiratory arrest
2. Acute exacerbation of chronic obstructive pulmonary disease (COPD)* with dyspnea, tachypnea, and acute respiratory acidosis (hypercapnia and decreased arterial pH) plus at least one of the following:
   - Acute cardiovascular instability
   - Altered mental status or persistent uncooperativeness
   - Inability to protect the lower airway
   - Copious or unusually viscous secretions
   - Abnormalities of the face or upper airway that would prevent effective noninvasive positive pressure ventilation
3. Acute ventilatory insufficiency in cases of neuromuscular disease accompanied by any of the following:
   - Acute respiratory acidosis (hypercapnia and decreased arterial pH)
   - Progressive decline in vital capacity to below 10 to 15 mL/kg
   - Progressive decline in maximum inspiratory pressure to below −20 to −30 cm H_2O
4. Acute hypoxemic respiratory failure with tachypnea, respiratory distress, and persistent hypoxemia despite administration of a high fraction of inspired oxygen (F_IO_2) with high-flow oxygen devices *or* in the presence of any of the following:
   - Acute cardiovascular instability
   - Altered mental status or persistent uncooperativeness
   - Inability to protect the lower airway
5. Need for endotracheal intubation to maintain or protect the airway or manage secretions, given the following factors:
   - Endotracheal tube (ET) ≤7 mm internal diameter (ID) with minute ventilation >10 L/min
   - ET ≤8 mm ID with minute ventilation >15 L/min

If any of the conditions listed are not present, emergency intubation and invasive positive pressure ventilation may not be indicated for the following conditions until other therapies have been attempted.

- Dyspnea, acute respiratory distress
- Acute exacerbation of chronic obstructive pulmonary disease
- Acute severe asthma
- Acute hypoxemic respiratory failure in immunocompromised patients
- Hypoxemia as an isolated finding
- Traumatic brain injury
- Flail chest

Modified from Pierson DJ. Indications for mechanical ventilation in adults with acute respiratory failure, *Respir Care* l47:249−262, 2002.

*Also applies to life-threatening asthma if respiratory acidosis or airflow obstruction has worsened despite aggressive management with bronchodilators and other therapy.

Key Point 4.6 Protecting the patient's airway is critical with conditions such as stroke, drug overdose, cerebral damage, and copious or viscous secretions.

the goals of therapy for a mechanically ventilated patient[3,4] (Key Point 4.6):

1. Support the pulmonary system so that it can maintain an adequate level of alveolar ventilation
2. Reduce the work of breathing until the cause of respiratory failure can be identified and treated
3. Restore arterial and systemic acid−base balances to levels that are normal for the patient
4. Increase oxygen delivery to and oxygenation of body organs and tissues
5. Prevent complications associated with mechanical ventilation

POSSIBLE ALTERNATIVES TO INVASIVE VENTILATION

In some cases, using a more conservative approach may avoid the need for invasive ventilation. Providing supplemental oxygen through standard oxygen therapy devices or through more specialized respiratory therapy devices (e.g., high-flow nasal cannula) may help relieve hypoxemia. For example, high-flow nasal cannula therapy allows the delivery of high gas flows through a narrow tube (e.g., nasal or transtracheal cannula).[18]

Placing the patient in an upright or Fowler position and providing appropriate medications can also be used to avoid the need for mechanical ventilation. However, the clinician must always keep in mind that hypoxemia can arise from a variety of causes, and these causes should be the focus of therapy. Oxygen therapy, repositioning the patient, and administration of medications such as bronchodilators and mucolytics are palliative measures that usually provide only transient relief of hypoxemia. They do not eliminate the cause.

Noninvasive Positive Pressure Ventilation

In some patients, NIV is a viable alternative to invasive positive pressure ventilation and may be a means of avoiding endotracheal intubation.[23] NIV is the treatment of choice for **acute-on-chronic respiratory failure** unless cardiovascular instability is also a factor. The use of NIV for acute-on-chronic respiratory failure has been shown to reduce the need for intubation, reduce complications of ventilation, shorten the hospital stay, and reduce hospital mortality rates.[23-25] It is important to recognize that the patient must meet the criteria for NIV and must not have any of the exclusionary factors (Box 4.7).[21,26-29] Invasive ventilation with intubation is indicated if any of the contraindications for NIV listed in Box 4.7 are present.[27,28]

Although this approach is controversial, NIV may be beneficial for patients with ARF associated with cardiogenic pulmonary edema.[26] Acute-on-chronic hypercapnic respiratory failure in patients with musculoskeletal problems (e.g., kyphoscoliosis and postpolio syndrome) may be well suited to NIV. NIV is also an alternative to invasive ventilator support for patients with hypercapnic acute exacerbation of COPD and considered a standard of

BOX 4.7 Indications and Contraindications for Noninvasive Positive Pressure Ventilation in Adults[29,30]

Indications

At least two of the following factors should be present:

- Respiratory rate >25 breaths/min
- Moderate to severe acidosis: pH, 7.25 to 7.30; $PaCO_2$, 45 to 60 mm Hg
- Moderate to severe dyspnea with use of accessory muscles and paradoxical breathing pattern

Contraindications

Any of the following factors alone is a contraindication.

Absolute Contraindications

- Respiratory arrest
- Cardiac arrest
- Cardiovascular instability (hypotension, dysrhythmias, acute myocardial infarction)
- Nonrespiratory organ failure (e.g., severe encephalopathy, severe gastrointestinal bleeding or surgery, hemodynamic instability with or without unstable cardiac angina)
- Patent tracheoesophageal fistula
- Inability to protect the airway or high risk for aspiration (or both)
- Uncooperative patient (impaired mental status, hypersomnolence)
- Facial or head surgery or trauma

Relative Contraindications

- Copious or viscous secretions
- Fixed nasopharyngeal abnormalities
- Extreme obesity

BOX 4.8 Circumstances in Which Noninvasive Positive Pressure Ventilation Should Be Changed to Invasive Ventilation

- Respiratory arrest
- Respiratory rate >35 breaths/min
- Severe dyspnea with use of accessory muscles and possibly paradoxical breathing
- Life-threatening hypoxemia: PaO_2 <40 mm Hg or PaO_2/FIO_2 <200
- Severe acidosis (pH <7.25) and hypercapnia ($PaCO_2$ >60 mm Hg)
- Hypersomnolence, impaired mental status
- Cardiovascular complications (hypotension, shock, heart failure)
- Failure of noninvasive positive pressure ventilation
- Other circumstances (e.g., metabolic abnormalities, sepsis, pneumonia, pulmonary embolism, barotrauma, massive pleural effusion)

care for these patients (Box 4.8).[29,30] In fact, NIV is an effective method of resting the ventilatory muscles and averting invasive ventilation in this latter group of patients. However, patients with neuromuscular disorders who develop acute respiratory acidosis and evidence of muscle weakness that progressively worsens (decreasing MIP and VC) require prompt intubation to prevent ARF and protect the airway.[3]

Invasive ventilation is also indicated for patients with severe refractory hypoxemia associated with increased WOB. These patients may suffer adverse events if mask CPAP is used for treatment rather than invasive ventilation.[29,30] Even with appropriately selected patients and skilled clinicians, NIV has been shown to be ineffective and endotracheal intubation is required.[27,28,30] (Chapter 19 discusses many additional important aspects of NIV.)

Intubation Without Ventilation

Some adult patients must be intubated because of an airway obstruction or to protect the airway or facilitate the removal of secretions (Key Point 4.6). If no other indications for ventilatory support are present and if a 7-mm endotracheal tube or larger is used, it is reasonable to conclude that IPPV is not needed. WOB related to the resistance of the endotracheal tube is considered significant only in small tubes (<7 mm) and when the minute ventilation is high (over 10 L/min)[3,31] (see Chapter 17 for additional information on the work of breathing through an artificial airway).

Ethical Considerations

Ethical considerations must be part of the decision-making process before initiating mechanical ventilation. First and foremost, does the patient want to be on ventilatory support? If the patient is unable to answer, it is important to determine whether the patient has a designated surrogate. Does the patient have a living will, advance directive, or similar legal document indicating his or her wishes regarding life support procedures? These significant concerns must be addressed. Invasive ventilation is contraindicated when it is contrary to the patient's advance directives. It also might be considered contraindicated if its use would be medically pointless and futile. If no reasonable chance exists that ventilation would extend a person's quality of life in a meaningful way, it is probably contraindicated.[3]

Clinical Scenario: Drug Overdose

A 23-year-old woman is taken to the hospital after ingesting an unknown amount of drugs and alcohol. Friends found her unconscious in her apartment. On admission to the emergency department, she is unconscious and unresponsive to verbal commands. Her pulse is 124 beats/min and blood pressure is 85/50 mm Hg. Her respiratory rate is 15 breaths/min, and respirations are shallow. ABG values on room air are as follows: $P_aO_2 = 60$ mm Hg, $P_aCO_2 = 69$ mm Hg, and pH = 7.24. Breath sounds reveal bilateral coarse crackles, especially in the bases.

Drugs and alcohol are known to depress the respiratory centers of the brain and also reduce the normal glottic response. The patient's shallow breathing is most likely caused by the substances ingested. Crackles heard over the right middle lobe may be because of aspiration. Weakening of the normal glottic response causes failure of the glottis to protect the airway, which is important to recognize because drug overdose and alcohol can cause nausea and vomiting.

The ABG values reported indicate ARF. The clinician's first priority is to protect the airway through intubation and provide ventilation to normalize blood gases. With drug overdose the patient can sometimes be treated pharmacologically, depending on the types of drugs ingested. For example, narcotic overdoses can be treated with naloxone hydrochloride (Narcan), an opioid antagonist. (NOTE: Narcan is used only to reverse the CNS and ventilatory depression caused by a narcotic overdose.) Determining the type and quantity of drugs ingested can provide valuable information to guide the immediate treatment of the patient.

Clinical Scenario: Guillain-Barré

A 30-year-old man is admitted to the hospital emergency department complaining of weakness of the limbs, tingling of the hands and feet, and increasing lack of coordination. Two weeks previously he had been treated for a flulike illness. The respiratory care practitioner obtains a baseline ABG evaluation, which is within normal limits. MIP is −70 cm H_2O, and VC is 4.3 L (predicted, 4.8 L).

Over a 36-hour period, VC, MIP, V_T, S_pO_2, and respiratory rate are monitored every 3 to 4 hours. Values progressively decrease to a VC of 2.1 L (44% of predicted [23 mL/kg]) and an MIP of −32 cm H_2O. A repeat ABG evaluation on room air shows the following: $P_aO_2 = 70$ mm Hg, $P_{aCO_2} = 48$ mm Hg, and pH = 7.34.

The diagnosis of Guillain-Barré syndrome was made on the basis of the patient's history and physical findings. Given the patient's diagnosis and clinical findings, the physician decides to intubate him and begin respiratory support. Guillain-Barré syndrome is a rapidly progressive, ascending, bilateral, flaccid muscle paralysis. Once it begins, it can progress to affect the respiratory muscles and other skeletal muscles.

Often, in neuromuscular disorders, an effective drug therapy is not available to reverse the progression of paralysis. When respiratory mechanics begin to deteriorate, patients are often intubated or provided with invasive respiratory support before the respiratory mechanics reach a critical value. Interestingly, ABG values and oxygen saturations may often be within normal limits. However, the clinician must remember that it is better to act early than to wait for respiratory arrest.

Clinical Scenario: Asthma

A 15-year-old girl with a life-threatening exacerbation of asthma has been treated in the emergency department over several hours. Administration of oxygen, corticosteroids, heliox, and aerosolized β-2 adrenergic bronchodilators and ipratropium bromide, although appropriate,[32] has not been effective in reducing airway obstruction and WOB. The patient has a respiratory rate of 37 breaths/min and her breathing is labored. Bilateral inspiratory and expiratory wheezes are present in both lungs. PEF is 70 L/min and FEV_1 is 0.75 L. ABG values obtained while the patient is breathing 50% oxygen are as follows: $P_aO_2 = 73$ mm Hg, $P_aCO_2 = 28$ mm Hg, $HCO_3- = 19$ mEq/L, pH = 7.46. P_aO_2/P_AO_2 is 0.23.

Hyperventilation in moderate or severe attacks of asthma is probably localized to areas of the lung where resistance to flow is lowest. Other areas of the lung are typically underventilated, resulting in shunting and reduced oxygenation. The length of time that a patient can tolerate this amount of work and hypoxemia varies considerably. If the persistent bronchospasm and mucus plugging cannot be alleviated, P_aCO_2 will rise despite a high minute ventilation. In some cases patients may begin to fatigue and progress to ARF. (A good indication that ARF is imminent is a marked decrease in breath sounds [i.e., **silent chest**].)

Continuous nebulization is started, and after 2 hours of treatment, the patient's ABG values are as follows: $P_aO_2 = 75$ mm Hg on 80% oxygen, $P_aCO_2 = 56$ mm Hg, $HCO_3- = 28$ mEq/L, pH = 7.31. $\dot{V}/Q_E$ is 18 L/min.

Serious consideration must be given to intubating the patient under mild sedation and initiating mechanical ventilatory support. This decision is often difficult to make for a fully conscious, distressed patient. Even the use of sedation is controversial. Unfortunately, invasive ventilation may be the only alternative. If no intervention is taken, a reduction in previously strong respiratory efforts sometimes occurs. Rates and tidal volumes may start to fall. The patient may become stuporous or uncontrollably agitated, both of which are often signs of fatigue.

This situation is an example of impending respiratory failure in which aggressive care is required to prevent respiratory failure or possibly cardiopulmonary arrest. NIV may be contraindicated or insufficient for these patients.[33] If NIV is initiated, and the patient's P_aCO_2 rises and a severe acidosis develops during NIV, a strong argument can be made for intubation and ventilation.[32] Some clinicians start ventilatory support when the P_aCO_2 rises to normal in the presence of high minute ventilation (this would indicate an increased V_D/V_T); they do not wait for the P_aCO_2 to exceed 45 mm Hg. Other clinicians use evidence of cardiac failure and a drop in pulse intensity in addition to the presence of dysrhythmias as criteria for intubation and initiating mechanical ventilation. Deterioration in mental status and exhaustion can also be important indicators for intubation and mechanical ventilation.[33]

Clinical Scenario: Chronic Restrictive Disorder

An 83-year-old woman with severe kyphoscoliosis is admitted to the hospital from a long-term care facility. She is diagnosed with pneumonia. Evaluation of the patient reveals that she is weak and pale and has decreased skin turgor (Key Point 4.7). Blood pressure is 110/72 mm Hg, and heart rate is 110 beats/min. Respiratory rate is 28 breaths/min, and breaths are shallow. Breath sounds reveal bilateral crackles scattered throughout both lungs. After 3 days of hospitalization and antibiotic therapy, the patient's condition appears to be progressively worsening. ABG results while the patient receives supplemental oxygen via a nasal cannula (2 L/min) are as follows: $P_aO_2 = 58$ mm Hg, $P_aCO_2 = 68$ mm Hg, pH = 7.24. The decision is made to intubate the patient and begin ventilatory support.

The combination of her severe, chronic, restrictive disorder and the unresolved pneumonia have contributed to the deteriorating gas exchange, which indicates ARF and the need for invasive ventilation.[3] No advance directives were available to indicate the patient's wishes regarding invasive ventilation.

Clinical Scenario: Chronic Obstructive Pulmonary Disease and Congestive Heart Failure

A 78-year-old man with a history of COPD and chronic congestive heart failure is admitted to the hospital. He has been admitted to the hospital three times during the past 9 months. The patient has a history of noncompliance with his medications and continues to smoke. He has a long and continued history of alcohol abuse.[25]

The patient is given oxygen therapy via nasal cannula (2 L/min). His respiratory rate is 18 breaths/min. Expirations are prolonged, and he demonstrates pursed-lip breathing and use of his accessory muscles of breathing. The patient is pale and appears anxious. Heart rate and blood pressure are elevated. Auscultation of his chest reveals breath sounds with scattered wheezes and crackles. Sputum is thin and frothy. Current ABG values on an estimated F_IO_2 of 0.28 are as follows: $P_aO_2 = 55$ mm Hg, $P_aCO_2 = 74$ mm Hg, $HCO_3- = 34$ mEq/L, pH = 7.28.

With an increasing P_aCO_2, moderately severe respiratory acidosis, and an apparently increased WOB, this patient demonstrates all the signs of respiratory distress. On the basis of the patient's history, the attending physician undoubtedly would have to determine which medications would be appropriate for both the patient's heart failure and his respiratory problems. Blood gas values and respiratory assessment support the need for intervention to improve the patient's ventilation and reduce WOB before his condition becomes more critical.

The presence of acute congestive heart failure is one of the exclusionary criteria for NIV in patients with acute-on-chronic COPD. This patient probably needs invasive ventilation. Several measures can be attempted, but they should not preclude intubation if the patient's condition worsens. Two simple measures that can be attempted are continuing oxygen therapy and maintaining the patient in an upright or semi-Fowler position.[23] Determining the patient's wishes is also important. Does he want aggressive support if it becomes necessary? If the patient does not want to be intubated, NIV could be used, particularly considering the alternative if no ventilation is provided.

Key Point 4.7 A simple test for dehydration involves gently pinching the skin on the back of the patient's hand. If the skin quickly returns to its normal position, skin turgor is normal. If the skin remains puckered, skin turgor is decreased and the patient is probably dehydrated.

SUMMARY

- The ability to recognize that a patient requires an artificial airway and mechanical ventilation is an essential skill for practitioners.

- Decisions made in the acute care setting must be based on evidence-based criteria that clearly demonstrate that a particular technique is beneficial and associated with good outcomes, such as improved quality of life, reduced length of stay, or a lower mortality rate.
- ARF is defined as an inability to maintain adequate oxygen uptake and carbon dioxide clearance.
- Two types of ARF are hypoxemic respiratory failure and hypercapnic respiratory failure.
- Hypoxemic respiratory failure can be treated with oxygen or in combination with PEEP or CPAP.
- Acute hypercapnic respiratory failure occurs when a person cannot maintain adequate ventilation to maintain P_aCO_2.
- The primary physiological objectives of mechanical ventilation should include supporting or improving pulmonary gas exchange, increasing lung volume, and reducing the WOB.
- Simple and direct observations can provide valuable information about the cause of respiratory distress and serve as a guide for selecting an appropriate therapeutic strategy.
- Recognizing the clinical signs of hypoxemia and hypercapnia is the first step to successful treatment of a patient in respiratory distress.
- The onset of respiratory failure can vary considerably depending on the cause of the neuromuscular dysfunction.
- The MIP and VC can be used to assess the respiratory muscle strength of patients with neuromuscular disorders. Determination of baseline ABGs along with periodic measurement of S_pO_2 is also appropriate for the management of these patients.
- WOB normally accounts for 1% to 4% of total oxygen consumption at rest. Increased WOB is typically associated with an increased rate or depth of breathing (or both).
- VC, PEF, respiratory rate, $\dot{V}/\dot{Q}_E$, and V_D/V_T can provide valuable information about a patient's status and alert the clinician to impending respiratory dysfunction.
- Standard criteria for initiation of mechanical ventilation include apnea, impending or confirmed ARF, and refractory hypoxemia characterized by an increased WOB or an ineffective breathing pattern.
- NIV offers a viable alternative to invasive mechanical ventilation in select patients.

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

1. Which of the following suggests the presence of respiratory insufficiency and the need for ventilatory support?
1. MIP of −17 cm H_2O
2. VC of 2.1 L in a 70-kg man
3. P_aCO_2 of 81 mm Hg and pH of 7.19
4. P_aO_2 of 65 mm Hg on room air
5. 1 and 3 only
6. 2 and 4 only
7. 1, 3, and 4 only
8. 1, 2, 3, and 4

2. Blood gas results on room air from an unconscious patient brought to the emergency department are as follows: pH = 7.23, P_aCO_2 = 81 mm Hg, bicarbonate = 33 mEq/L, P_aO_2 = 43 mm Hg, and $S_{a}O_2$ = 71%. With no other data available, which of the following forms of therapy is indicated?
A. Oxygen with a nonrebreathing mask
B. CPAP mask
C. Aerosol treatment with albuterol
D. Mechanical ventilatory support

3. A 30-year-old woman is seen in the emergency department. She demonstrates paralysis of the lower extremities that is progressively worsening. After several hours of frequent monitoring, her VC has decreased to 12 mL/kg and MIP is −30 cm H_2O. The results of blood gas evaluations are not yet available. What type of therapy would you suggest for this patient?
A. Aerosolized bronchodilator administered with a metered-dose inhaler
B. Place the patient on NIV
C. Incentive spirometry to improve muscle strength
D. Narcotic-blocking agent

4. A 28-year-old man with botulism poisoning is beginning to develop progressive paralysis. The respiratory therapist has been monitoring the patient's MIP and VC every 2 hours. The most recent results show that the patient continues to deteriorate: MIP = −27 cm H_2O, VC = 32 mL/kg. Which of the following could be appropriately recommended?
A. Gastric lavage
B. Oxygen therapy
C. Medication to reverse the paralysis
D. Mechanical ventilatory support

5. A 34-year-old man is brought to the emergency department by emergency medical services (EMS) after a motor vehicle accident. He is unconscious and unresponsive. ABGs obtained while the patient is receiving oxygen via a nonrebreathing mask show the following: P_aO_2 = 47 mm Hg, P_aCO_2 = 93 mm Hg, pH = 7.09, and bicarbonate = 27 mEq/L. Which of the following would the therapist recommend?
A. Recheck vital signs
B. Intubate and ventilate
C. Change to a Venturi mask and coach the patient to breathe
D. Begin cardiopulmonary resuscitation (CPR)

6. A 68-year-old man with a history of COPD and CO_2 retention is brought to the emergency department by ambulance. He is receiving oxygen through a nasal cannula (2 L/min). He is conscious and cooperative but in distress. He is leaning forward and using accessory muscles to breathe. His vital signs are as follows: heart rate = 100 beats/min, blood pressure = 128/78 mm Hg, temperature = 37.8° C, respiratory rate = 20 breaths/min with prolonged expiration through pursed lips. Breath sounds reveal bilateral crackles and wheezes. Which of the following is most appropriate?
A. Change to a Venturi mask and coach the patient to breathe
B. Intubate and ventilate
C. Change to a nonrebreather mask
D. Evaluate for NIV

7. A 43-year-old man who weighs 165 lb (75 kg) and has myasthenia gravis is beginning to develop progressive weakness of the muscles. The respiratory therapist has been monitoring MIP and VC every 4 hours. The most recent results show that

the patient continues to deteriorate despite treatment with anticholinesterase drugs: MIP = −35 cm H_2O, VC = 23 mL/kg. Which of the following would be appropriate to recommend?
A. Incentive spirometry to improve muscle strength
B. Oxygen therapy
C. Mechanical ventilatory support
D. Administer a neuromuscular blocking agent

8. A 48-year-old woman admitted to the emergency department demonstrates tachypnea and tachycardia. She appears pale. Breath sounds reveal bilateral crackles. ABG results with the patient using a nonrebreathing mask are as follows: P_aO_2 = 45 mm Hg, P_aCO_2 = 32 mm Hg, pH = 7.49, and bicarbonate = 24 mEq/L. Which of the following is the most appropriate initial treatment for this patient?
A. Increase the flow to the oxygen mask
B. Begin ventilatory support
C. Provide CPAP by mask
D. Administer bronchodilator therapy

9. A 60-year-old man was admitted to the hospital yesterday for a suspected myocardial infarction. Current ABG values on room air are as follows: P_aO_2 = 57 mm Hg, P_aCO_2 = 33 mm Hg, pH = 7.47, and bicarbonate = 25 mEq/L. Which of the following is the most appropriate form of respiratory therapy for this patient?
A. Oxygen therapy
B. Noninvasive mechanical ventilation
C. CO_2/O_2 therapy (5/95)
D. CPAP by mask

10. Which of the following are goals of mechanical ventilation?
1. Provide support to the pulmonary system to maintain an adequate level of alveolar ventilation
2. Reduce the work of breathing until the cause of respiratory failure can be eliminated
3. Restore arterial blood gas levels to normal
4. Increase respiratory therapy department revenue

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

11. A 74-year-old man with COPD who has acute-on-chronic respiratory failure is supported with NIV. The patient is becoming more confused. Blood gas values are as follows: P_aO_2 = 45 mm Hg, P_aCO_2 = 58 mm Hg, pH = 7.21, and bicarbonate = 23 mEq/L. Which of the following would be appropriate treatment for this patient?
A. Increase oxygen delivery
B. Ask the patient if he is comfortable with the mask
C. Ask the physician for a sedative
D. Switch to invasive ventilation

12. A 14-year-old boy who previously had been diagnosed with mild persistent asthma has a PEF of 100 L/min. This indicates which of the following?
A. Increased airway resistance
B. Heart failure
C. Increased lung compliance
D. Inability to take in a deep breath and cough

13. After oxygen is administered, a patient's heart rate changes from 110 beats/min to 85 beats/min. The initial tachycardia was most likely caused by which of the following?
A. Anxiety
B. Hypoxemia
C. Hypercapnia
D. Pain

14. A 34-year-old patient who was in a motor vehicle crash is admitted to the hospital with crushed chest injuries and a fractured tibia. Two days later the P_aO_2 is 56 mm Hg while he is breathing 80% oxygen, and the respiratory rate is 30 to 34 breaths/min. On the basis of the history and these findings, which of the following does the patient most likely need?
A. Invasive positive pressure ventilation
B. Noninvasive positive pressure ventilation
C. 100% oxygen via a nonrebreathing mask
D. High-flow nasal cannula

15. Which of the following arterial blood gas parameters is considered the best indicator of a patient's ventilatory status?
A. pHa
B. P_aCO_2
C. P_aO_2
D. S_{aO_2}

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CHAPTER 5

Selecting the Ventilator and the Mode

OUTLINE

KEY TERMS

- Assisted breaths
- Assisted ventilation
- Bilevel PAP
- Continuous mandatory ventilation
- Controlled ventilation
- Full ventilatory support
- Mandatory breaths
- Partial ventilatory support
- Patient-ventilator asynchrony
- Pressure augmentation
- Spontaneous breaths

LEARNING OBJECTIVES

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

1. Select an appropriate mechanical ventilator, breath type, and mode of ventilation on the basis of clinical findings derived from patient assessment data.
2. Describe how continuous positive airway pressure (CPAP) and noninvasive positive pressure ventilation (NIV) can be used in the management of patients with various cardiopulmonary disorders.
3. Discuss the advantages and disadvantages of volume-controlled and pressure-controlled ventilation.
4. Explain the differences in function among continuous mandatory ventilation, spontaneous ventilation, and intermittent mandatory ventilation.
5. Describe the functions of the *trigger, cycle,* and *limit* variables as they are used in volume-controlled continuous mandatory ventilation, pressure-controlled continuous mandatory ventilation, volume-controlled intermittent mandatory ventilation, pressure-controlled intermittent mandatory ventilation, and pressure support ventilation.
6. Define each of the following terms: *pressure augmentation, pressure-regulated volume control, volume support, mandatory minute ventilation, airway pressure release ventilation, bilevel positive airway pressure,* and *proportional assist ventilation.*

Once the need for mechanical ventilation has been established, the clinician must select the type of ventilator, ventilator mode, and breath type for the patient. This chapter focuses on factors that affect the choice of ventilator, patient interface (i.e., artificial airway or mask), control variable (volume or pressure), breath type, and ventilator mode.

Selecting the appropriate ventilator and mode of ventilation can be challenging even for an experienced clinician. The following

questions provide a framework for making the selection process manageable:

- Why does the patient need ventilatory support? (Indication)
- Does the ventilatory problem require a special mechanical ventilation mode? (Pathology)
- What therapeutic goals can be achieved by using a specific ventilator? (Treatment goals)
- Does the patient need to be intubated, or can noninvasive ventilation be used? (Patient interface)
- Will the ventilatory support be provided in the intensive care unit, the patient's home, or an extended care facility? (Location)
- Will ventilatory support be required for a brief period or long term? (Duration)
- How familiar are the staff with the ventilator (or ventilators) under consideration? (Staff training)

If a change is to be made from one type of ventilator to another, the respiratory therapist must know whether the change is being made because a different mode or feature is needed. For example, a patient may have acute respiratory distress syndrome (ARDS) and may need a ventilator that can provide an advanced modality.

If a patient is to be discharged from the hospital to his or her home or an extended care facility, a ventilator that would be appropriate in that setting should be selected. For example, a patient with amyotrophic lateral sclerosis may be familiar and comfortable with noninvasive ventilation. A patient with postpolio syndrome may be more familiar with a chest cuirass or tank ventilator (negative pressure ventilator [NPV]). (Although NPV is not discussed here, it is important to mention that it is sometimes used in the homecare or alternative-care setting. In rare cases, NPV has been used for ventilation of acutely ill infants.[1,2] See Chapter 21 for additional information on NPV.)

This chapter focuses on positive pressure ventilation. Positive pressure ventilators can function in various types of settings and provide a variety of modes, features, monitors, and alarms.

NONINVASIVE AND INVASIVE POSITIVE PRESSURE VENTILATION: SELECTING THE PATIENT INTERFACE

A patient can be connected to a positive pressure ventilator using either a positive pressure mask or an artificial airway. Face and nasal masks are used to administer NIV. Artificial airways, which include translaryngeal airways (oral or nasal endotracheal tubes [ETs]) and tracheostomy tubes, are required for invasive ventilation. Historically, about 75% of patients receiving invasive positive pressure ventilation are intubated (95% with an oral ET and 5% with a nasal ET). Tracheostomy tubes are used in the remaining 25% of patients receiving invasive positive pressure ventilation.[3]

Noninvasive Positive Pressure Ventilation

There are two methods of providing noninvasive positive pressure ventilator support:

- Continuous positive airway pressure (CPAP)
- Noninvasive positive pressure ventilation (NIV)

As previously mentioned, CPAP and NIV are most commonly administered via a face or nasal mask. Ensuring that the mask fits properly will minimize patient discomfort and help prevent air leaks. Mask interfaces are discussed in more detail in Chapter 19.

Continuous Positive Airway Pressure

As previously described, CPAP involves administering a continuous level of pressure throughout the respiratory cycle. In hospitalized patients, CPAP has been shown to be an effective method to improve oxygenation. It also is an accepted method used to treat obstructive sleep apnea, especially in the home. In addition, CPAP can be used to assist patients with chronic obstructive pulmonary disease (COPD) who have difficulty breathing. The use of CPAP in COPD patients has received some attention because air trapping can occur in a spontaneously breathing individual with increased airway resistance (R_{aw}). Air trapping in patients with COPD or in patients with acute asthma can lead to an increase in the functional residual capacity (FRC). In the past, CPAP was considered contraindicated in patients with COPD because these patients already had an increased FRC. The concern among some clinicians was based on the assumption that external CPAP, or positive end-expiratory pressure (PEEP), would further increase the FRC and not benefit the patient.

Patients with COPD often have difficulty generating the pressure difference between the alveoli and the mouth to begin inspiratory gas flow. The air trapped in the lungs (called *intrinsic PEEP* or *auto-PEEP*) creates a positive alveolar pressure (P_{alv}). If the pressure in the lungs is positive at end exhalation, the pressure must drop below the pressure at the mouth (atmospheric pressure) to start gas flowing into the lungs for inspiration. For example, if auto-PEEP is greater than +5 cm H_2O, the patient must exert an effort of at least −5 cm H_2O to drop the lung pressure below zero. Once alveolar pressure drops below zero, inspiratory gas flow can start.

Externally applied CPAP may reduce the pressure difference between the mouth and alveoli when flow limitation (increased R_{aw}) is the cause of auto-PEEP. The patient therefore does not have to work as hard to drop the P_{alv} so that inspiratory gas flow enters the lungs. In other words, externally applied CPAP can reduce inspiratory work.[4] Mask CPAP set at 80% to 90% of the measured auto-PEEP (usually about 4–10 cm H_2O) reduces diaphragmatic work and dyspnea, improves gas exchange, and does not worsen hyperinflation.[5-7] As discussed in Chapter 4 and later in this chapter, many clinicians often prefer using noninvasive positive pressure ventilator support in the form of bilevel positive airway pressure (**bilevel PAP**) rather than CPAP for patients with R_{aw}-induced auto-PEEP. Bilevel PAP is a method often used to treat acute-on-chronic respiratory failure. An example of an acute-on-chronic respiratory condition is a patient with chronic bronchitis who develops pneumonia (an acute condition).

Noninvasive Positive Pressure Ventilation

Numerous investigators have examined the use of NIV for patients with respiratory failure caused by various neuromuscular disorders, chest wall deformities, COPD, central ventilatory control abnormalities, and acute cardiogenic pulmonary edema (Box 5.1).[8-12] Their findings suggest that NIV can reduce the need for intubation in 60% to 75% of these patients.[9,10,13] Two types of ventilators can be used to provide NIV:

- Pressure-triggered, pressure-limited, flow-cycled devices designed specifically for mask ventilation, for example, BiPAP (Philips Respironics, Murrysville, PA)[9]
- Critical care ventilators that have a variety of available modes, including NIV in many cases

Box 5.2 lists some of the advantages and disadvantages of NIV compared with invasive ventilation.[4,11,13] Box 4.7 lists accepted indications and contraindications for NIV. If patients are not

BOX 5.1 Cardiopulmonary Conditions Managed With Noninvasive Positive Pressure Ventilation

- Chronic respiratory failure
- Chest wall deformities
- Neuromuscular disorders
- Central alveolar hypoventilation
- Chronic obstructive pulmonary disease (COPD)
- Cystic fibrosis
- Bronchiectasis
- Acute respiratory failure (ARF)
- Acute respiratory distress syndrome (ARDS)
- Pneumonia
- Postoperative complications
- Asthma
- Cardiogenic pulmonary edema
- Heart failure
- Postextubation failure in difficult-to-wean patients
- Obstructive sleep apnea

From Liesching T, Kwok H, Hill NS: Acute applications of noninvasive positive pressure ventilation, *Chest* 124:699–713, 2003.

BOX 5.2 Advantages and Disadvantages of Noninvasive Positive Pressure Ventilation in Acute Respiratory Failure

Advantages
- Avoids complications associated with artificial airways
- Provides flexibility in initiating and removing mechanical ventilation
- Reduces requirements for heavy sedation
- Preserves airway defense, speech, and swallowing mechanisms
- Reduces need for invasive monitoring

Disadvantages
- Can cause gastric distention, skin pressure lesions, facial pain, dry nose, eye irritation (conjunctivitis), discomfort, claustrophobia, and poor sleep and mask leaks can occur

excluded by the presence of contraindications, NIV offers the clinician an excellent option for a number of acute and chronic conditions requiring mechanical ventilation.[14]

Invasive Positive Pressure Ventilation

As mentioned previously, a high percentage of patients who need mechanical ventilation require invasive positive pressure ventilation via an artificial airway. Once the appropriate ventilator has been chosen, the clinician must select a mode of ventilation that is most advantageous for the patient's condition.

FULL AND PARTIAL VENTILATORY SUPPORT

The terms **full ventilatory support** and **partial ventilatory support** describe the extent of mechanical ventilation provided. With full ventilatory support the ventilator provides all the energy necessary to maintain effective alveolar ventilation.[15] Full ventilatory support results in arterial partial pressure of carbon dioxide (P_aCO_2) values less than 45 mm Hg or a P_aCO_2 that is normal for the patient (i.e., eucapnic breathing). Full ventilatory support is provided when ventilator rates are high (8 breaths/min or more) and tidal volume (V_T) is adequate for the patient. (See Chapter 6 for information on V_T settings and ideal body weight.)[16] Full ventilatory support is typically provided using a ventilator mode that provides a preset volume or pressure when a breath is delivered. The mode must be set so that the patient receives adequate alveolar ventilation regardless of whether the person can breathe spontaneously.[15] For example, full ventilatory support might include fully controlled and assisted positive pressure ventilation.

Partial ventilatory support is any degree of mechanical ventilation in which set machine rates are lower than 6 breaths/min and the patient participates in the work of breathing (WOB) to help maintain effective alveolar ventilation. A variety of ventilator modes can be used for partial ventilatory support; however, by definition the patient must actively participate in ventilation to maintain adequate levels of P_aCO_2. Partial ventilatory support modes might include intermittent mandatory ventilation (IMV), pressure support ventilation (PSV), volume support ventilation (VSV), proportional assist ventilation (PAV), and mandatory minute volume (MMV). These modes are described later in this chapter.

Strategies for partial ventilatory support are appropriate when attempts are made to discontinue ventilator support. Partial ventilatory support should be avoided in patients with ventilatory muscle fatigue and when a patient has a high WOB level.

When treating patients with acute respiratory failure, the initial goal of mechanical ventilation is to supply all the necessary ventilation (i.e., full ventilatory support) while the ventilatory muscles are given time to rest.[17,18] Ideally, after several hours to several days of full ventilatory support, the patient's condition stabilizes and the patient begins to recover. Maintaining complete rest after just a few days on full ventilatory support can result in muscle wasting or atrophy; for this reason, some clinicians object to full ventilatory support and provide partial support from the beginning. This is a matter of clinician preference and patient history. Whether full ventilatory support is used or not, the patient assumes part of the WOB within a short time, once other factors have been stabilized, to prevent muscle wasting.

BREATH DELIVERY AND MODES OF VENTILATION

The breath type (i.e., source of energy used to deliver the breath) and pattern of breath delivery during mechanical ventilation constitute the mode of ventilation. The mode is determined by the following factors:

- Type of breath (mandatory, spontaneous, assisted)
- Targeted control variable (volume or pressure)
- Timing of breath delivery (continuous mandatory ventilation [CMV], IMV, or continuous spontaneous ventilation [CSV])

Type of Breath Delivery

Mandatory Breaths

As described in Chapter 3, **mandatory breaths** are breaths for which the ventilator controls the timing, V_T, or inspiratory

Case Study 5.1

What Type of Breath Is It?

1. A patient receives a breath that is patient triggered, volume targeted, and time cycled. What type of breath is it?
2. A patient breathes spontaneously at a baseline pressure of +8 cm H_2O. The pressure stays at +8 cm H_2O during inspiration and exhalation. What type of breath is it?

pressure. For example, a patient-triggered, volume-targeted, volume-cycled breath is a mandatory breath. The ventilator controls V_T delivery.

Spontaneous Breaths

For **spontaneous breaths,** the patient controls the timing and V_T. The volume or pressure (or both) delivered is not set by the clinician but rather based on patient demand and patient lung characteristics.

Assisted Breaths

Assisted breaths have characteristics of both mandatory and spontaneous breaths. In an assisted breath, all or part of the breath is generated by the ventilator, which does part of the WOB for the patient. If the airway pressure rises above baseline during inspiration, the breath is assisted.[19] For example, during the pressure support mode the clinician sets the target pressure but the patient initiates the breath (patient triggered). The ventilator delivers the set pressure above baseline pressure to assist the patient's breathing effort. The patient cycles the breath (Case Study 5.1).

Targeting Volume as the Control Variable

By choosing either volume or pressure ventilation, the clinician determines the control variable that will be used to establish gas flow to the patient.[19,20] Control variables are independent variables; in volume-controlled ventilation, for example, the volume selected is constant and independent of what happens to pressure when the patient's lung characteristics (lung compliance and R_{aw}) change or when the patient's effort changes. The selection of using volume-controlled or pressure-controlled ventilation is based on whether consistency of V_T delivery is important or limiting of pressure delivery is important.

The primary advantage of volume-controlled ventilation is that it guarantees a specific volume delivery and volume of expired gas (V_E), regardless of changes in lung compliance and resistance or patient effort.[21] The goal of volume-controlled ventilation is to maintain a certain level of P_aCO_2.

The main disadvantage of volume-controlled ventilation becomes evident when the lung condition worsens. This can cause the peak and alveolar pressures to rise, leading to alveolar overdistention (see Critical Care Concept 5.1 and Fig. 5.1*). Box 5.3 summarizes how changes in lung compliance and airway resistance can affect peak and plateau pressures during volume-controlled ventilation. It is worth noting, however, that these changes are reversible. When the lung condition improves, less pressure is required to deliver the volume and ventilating pressures decline.

CRITICAL CARE CONCEPT 5.1

Volume-Controlled Breaths With Changing Lung Characteristics

Use Fig. 5.1 to answer the following questions:

1. What is the approximate inspiratory time? Does it change?
2. What type of flow scalar is shown in this figure?
3. What is the approximate tidal volume delivery for each breath?
4. What are the peak inspiratory pressures (PIPs) in A, B, and C?
5. What types of lung or thoracic abnormalities can result in reduced compliance?
6. What would happen to the PIP if compliance remained unchanged but airway resistance increased?

Other disadvantages of volume-controlled breaths are related to flow and sensitivity settings. Specifically, the delivery of flow is fixed on some ventilators and may not match patient demand. Similarly, if the sensitivity level is not set appropriately for the patient, it can make it more difficult for the patient to trigger inspiration. Both situations can lead to **patient-ventilator asynchrony** and patient discomfort.[22,23] The operational controls available for volume ventilation vary depending on the ventilator manufacturers. Operational controls typically include V_T, respiratory rate, inspiratory flow, and a flow pattern. With some ventilators, the practitioner can set the V_T, respiratory rate, and inspiratory time; however, the flow pattern is not adjustable. (Chapter 6 reviews guidelines for setting the appropriate volume and rate in volume ventilation.)

Targeting Pressure as the Control Variable

Pressure-controlled ventilation allows the clinician to set pressure as the independent variable; that is, the pressure remains constant, but volume delivery (the dependent variable) changes as lung characteristics change. Volume delivery therefore must be closely monitored (Critical Care Concept 5.2 and Fig. 5.2).

Pressure-controlled ventilation has several advantages. First, it allows the clinician to set a maximum pressure, which reduces the risk for overdistention of the lungs by limiting the amount of positive pressure applied to the lung. Second, the ventilator delivers a decelerating flow pattern during pressure-controlled ventilation (Fig. 5.3). It has been suggested that limiting the peak pressure spares more normal areas of the lungs from overinflation.[21] Pressure-controlled ventilation is therefore considered a component of protective strategies for the lung.[16] It may also be more comfortable for patients who can breathe spontaneously. When the patient makes an inspiratory effort, the negative pressure produced at the upper airway causes the ventilator to vary gas flow to match the patient's need. This can reduce WOB, particularly in patients with ARDS, compared with volume-controlled ventilation.[24] Disadvantages of pressure-controlled ventilation include the following:

- Volume delivery varies as the patient's lung characteristics (i.e., lung compliance and airway resistance) change.
- V_T and V_E decrease when lung characteristics deteriorate (Box 5.4)

*Figs. 5.1 and 5.2 introduce ventilator graphics so that the reader can become familiar with this tool. Ventilator graphics are discussed in greater detail in Chapter 9.

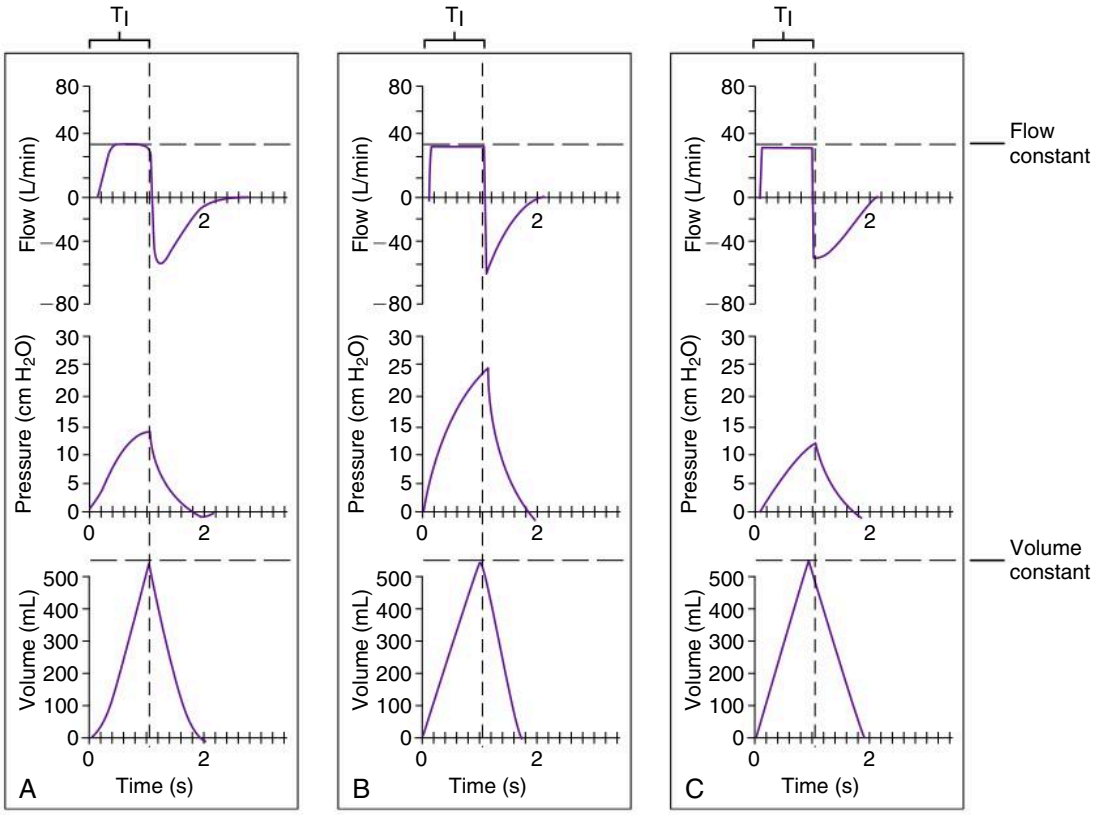

Fig. 5.1 Graphs for constant-flow, volume-controlled ventilation (constant volume) with normal compliance (A), reduced compliance (B), and increased compliance (C). Note that inspiratory flow is above the *x*-axis and expiratory flow is below the *x*-axis. (See text for more information.)

BOX 5.3 Factors That Affect Pressures During Volume-Controlled Ventilation

Patient Lung Characteristics

- Reductions in lung or chest wall compliance produce higher peak and plateau pressures; increased compliance produces lower peak and plateau pressures.
- Increased airway resistance produces a higher peak pressure; reductions in airways resistance produce lower peak pressures.

Inspiratory Flow Pattern

- Peak pressure is higher with a constant flow and lower with a decelerating flow pattern. Decelerating flow pattern has a higher mean airway pressure; constant flow generates the lowest mean airway pressure.
- High inspiratory gas flow creates a higher peak pressure.

Volume Setting

- High volumes produce higher peak and plateau pressures; low volumes produce lower peak and plateau pressures.

Positive End-Expiratory Pressure

- Increasing PEEP increases the peak and mean pressures.

Auto-PEEP

- Increases in auto-PEEP increase the peak inspiratory pressure.

PEEP, Positive end-expiratory pressure.

CRITICAL CARE CONCEPT 5.2

Pressure-Controlled Breaths With Changing Lung Characteristics

Use Fig. 5.2 to answer the following questions:

1. What type of pressure curve is delivered in A, B, and C?
2. What type of flow waveform is present during inspiration in A, B, and C?
3. Compare the flow-time curve during inspiration in C with that in A. What is the difference between the two?
4. Look at the dotted line in C that starts at the flow waveform just when flow drops to zero during inspiration. Look at the volume-time curve C. What do you notice about this volume-time curve compared with those in A and B? Why is it flat at the top?
5. Why is volume delivery higher in B than A?

Clinical studies comparing pressure-controlled ventilation with volume-controlled ventilation are divided over which method is superior. Pressure-controlled ventilation and volume-controlled ventilation are equally beneficial in patients who are not spontaneously breathing when a targeted flow pattern is used.[21] On the other hand, it has been suggested that for spontaneously breathing patients, pressure-controlled ventilation may lower the WOB and improve patient comfort to a greater extent than volume-controlled ventilation, thereby reducing the need for sedatives and neuromuscular blocking agents.[21] Clinician preference and institutional protocol are also important criteria that can influence the selection of pressure-controlled versus volume-controlled ventilation (Case Study 5.2).

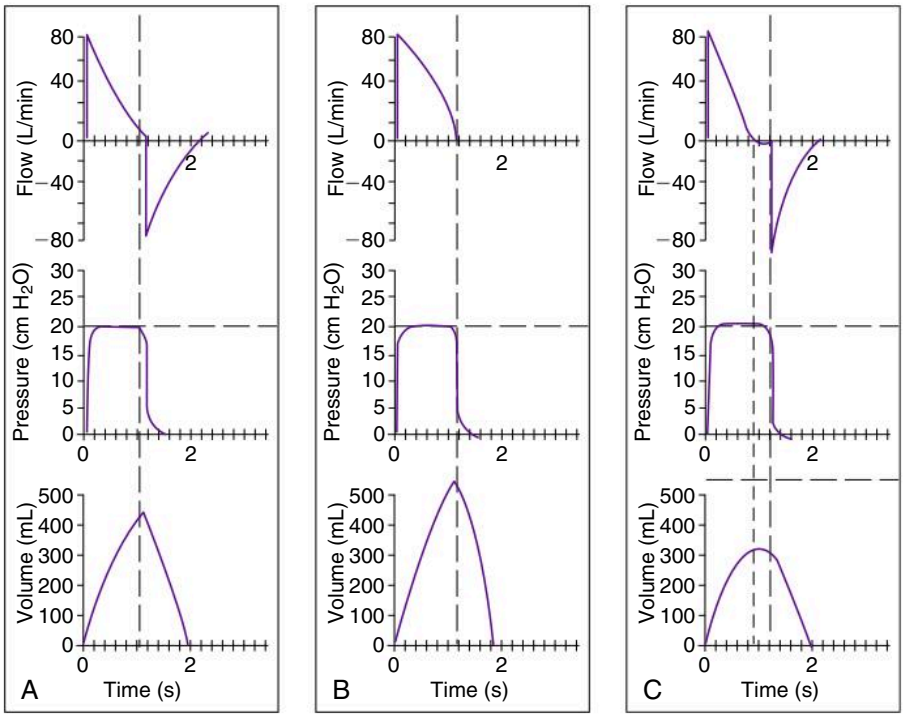

Fig. 5.2 Graphs for pressure-targeted ventilation (constant pressure) with normal compliance (A), increased compliance (B), and decreased compliance (C). (See text for more information.)

Timing of Breath Delivery

Three types of breath delivery timing or sequence are available on current intensive care unit (ICU) ventilators:

1. Continuous mandatory ventilation (CMV)
2. Intermittent mandatory ventilation (IMV)
3. Continuous spontaneous ventilation (CSV)

With CMV, either time-triggered or patient-triggered breaths are mandatory breaths; the patient is not generating any spontaneous breaths. During IMV, the patient receives a set number of mandatory breaths each minute but is also allowed to breathe spontaneous breaths between mandatory breaths. Thus the patient breathes spontaneously, and the ventilator intermittently delivers a mandatory breath. In CSV, all breaths are spontaneous and therefore patient triggered. These spontaneous breaths may be assisted (e.g., PSV) or unassisted (e.g., CPAP). Note that PSV may also refer to pressure-controlled CSV (PC-CSV).[20]

MODES OF VENTILATION

As mentioned previously, the breath type, targeted control variable, and timing of breath delivery determine the mode of ventilation. Modes of ventilation are often identified using abbreviations (e.g., volume-controlled CMV [VC-CMV], PC-IMV, volume support ventilation [VSV], PSV, and MMV). The following sections focus on describing five basic modes of ventilation (VC-CMV, PC-CMV, VC-IMV, PC-IMV, and PC-CSV) and also briefly introduce several spontaneous modes of ventilation used commonly in the ICU: BiPAP, dual-control modes, and other closed-loop modes of ventilation (e.g., MMV, airway pressure release ventilation [APRV], and PAV).

Ventilator manufacturers often use proprietary terms to describe a mode of ventilation that has been developed for their device. This variability in terminology can sometimes be confusing for clinicians unfamiliar with the similarities and differences of modes offered on most current-generation ICU ventilators. Chatburn and colleagues have advocated for an updated system for classifying modes of ventilation used in the clinical setting. Additional details regarding this classification system can be found in the references listed at the end of this chapter.[19,20]

History of Intermittent Positive Pressure Breathing and Intermittent Positive Pressure Ventilation: Understanding the Terminology

Mechanical ventilation historically involved delivering a positive pressure breath intermittently during mechanical ventilation. For this reason, in the past this type of ventilation was called *intermittent positive pressure breathing (IPPB)* or *intermittent positive pressure ventilation (IPPV)*. IPPV implied continuous mechanical support of breathing in an apneic patient; every breath was mandatory. In current terminology, the abbreviation *CMV* (which stands for continuous mandatory ventilation) is most often used to describe a mode in which every breath is mandatory. A minimum breathing rate is set, but patients can trigger more breaths if they are able. (NOTE: Another commonly used name for CMV is *assist/control [A/C] ventilation;* this term usually means that a set volume or pressure is delivered with every breath.)

In the early development of mechanical ventilation, small, portable IPPV devices were adapted to deliver aerosolized medications with IPPB. Consequently, in modern usage, IPPB implies an IPPB aerosol treatment. If correctly adapted, the same equipment (e.g., Bird Mark 7, CareFusion Corp., San Diego, Calif.) can also be used to provide ventilatory support.

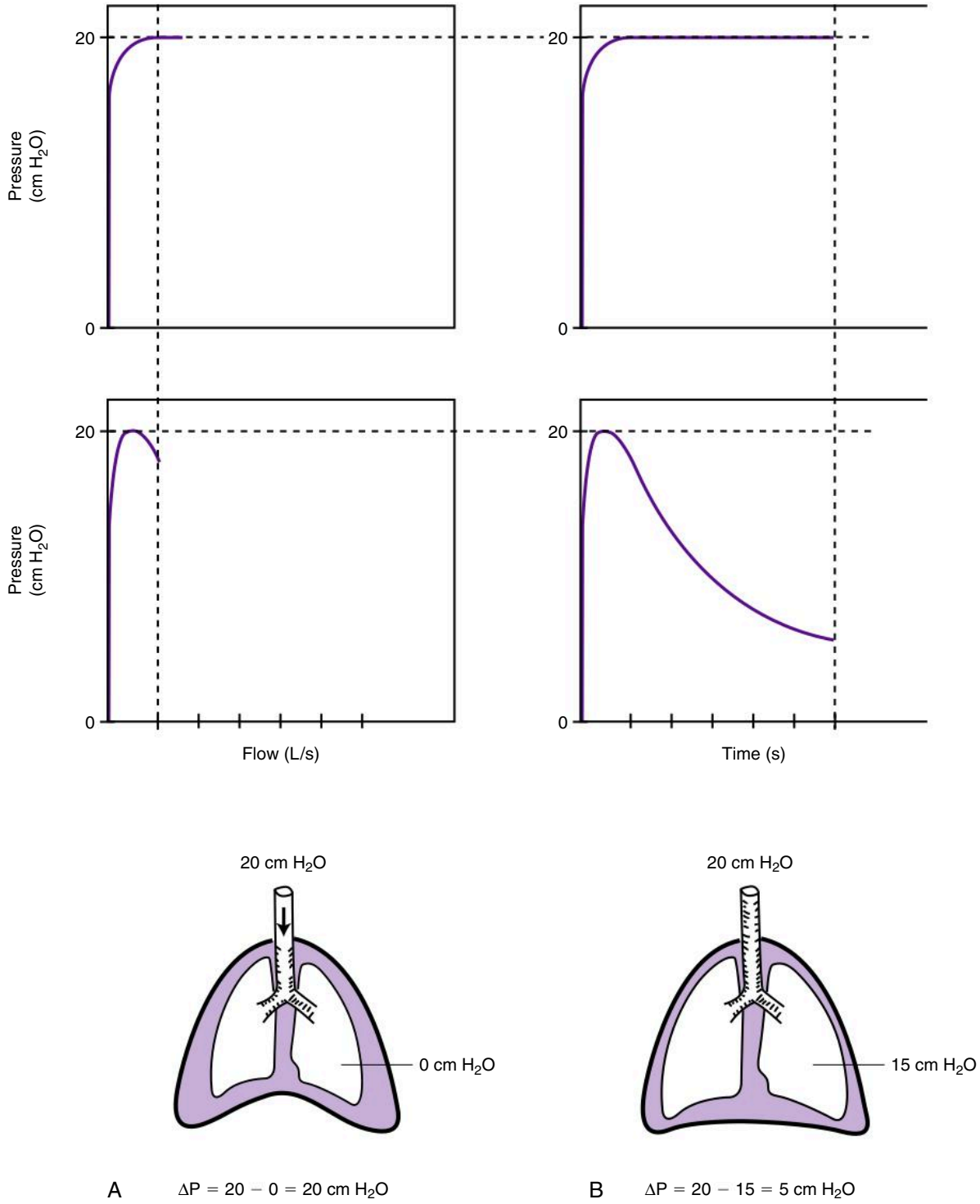

Fig. 5.3 Pressure-controlled ventilation produces a descending waveform. When the ventilator starts gas flow (inspiration), the pressure at the patient's upper airway builds quickly to the set pressure. This produces a pressure (ΔP) gradient between the upper airway and alveoli (i.e., P_{alv} of 0 cm H_2O or slightly lower). The gradient is highest at the beginning of inspiration (A); as pressure builds inside the alveolus, the gradient slowly decreases, as does flow (B).

Continuous Mandatory Ventilation

With **continuous mandatory ventilation** (CMV), all breaths are mandatory and can be volume or pressure targeted. Breaths can be time triggered or patient triggered. When the breaths are time triggered, the breaths are described as **controlled**

Key Point 5.1 Historically, clinicians have differentiated between controlled and assisted ventilation by the trigger used to initiate a breath. The control mode is time triggered, and the patient makes no spontaneous effort, whereas during assisted ventilation the breath can be either time triggered or patient triggered.

BOX 5.4 Factors That Affect Volume Delivery During Pressure-Controlled Ventilation

Pressure Setting
- Higher pressure settings produce larger volumes, whereas lower pressure settings produce lower volumes. In other words, increasing the peak inspiratory pressure (PIP) while maintaining a constant end-expiratory pressure (EEP) increases volume delivery (and vice versa).

Pressure Gradient
- Increasing EEP (PEEP + auto-PEEP) while keeping PIP constant reduces the pressure gradient (PIP − EEP) and lowers volume delivery (and vice versa).

Patient Lung Characteristics
- Reduced compliance results in lower volume; increased compliance results in increased volume for a given inspiratory pressure.
- Increased airway resistance (R_{aw}) results in lower volume delivery if active flow is present; reductions in airway resistance result in higher volume delivery if active flow is present.

Inspiratory Time
- When the inspiratory time (T_I) is extended, volume delivery increases. Notice that this is true as long as flow is present during inspiration (i.e., the flow—time curve shows flow above zero when inspiration ends). However, if flow returns to zero before inspiration ends, further increases in T_I can decrease volume delivery if adequate time is not provided for exhalation.

Patient Effort
- Active inspiration by the patient can increase volume delivery.

Case Study 5.2

Pressure-Controlled or Volume-Controlled Ventilation

1. A physician wants to ensure that a patient's P_aCO_2 remains at the person's normal level of 50 mm Hg. Would volume-controlled ventilation (VC-CMV) or pressure-controlled ventilation (PC-CMV) best meet this requirement?
2. Ventilation pressure can become high in patients with acute respiratory distress syndrome. To prevent excessive pressures, what independent variable would be most appropriate, volume or pressure?

ventilation or the *control mode* (see Fig. 3.16). When the breaths are patient triggered during CMV, the breaths are described as **assisted ventilation** (Key Point 5.1).

Controlled Ventilation

Controlled (time-triggered) ventilation is appropriate only when a patient cannot make an effort to breathe. Patients who are obtunded because of drugs, cerebral malfunction, spinal cord or phrenic nerve injury, or motor nerve paralysis may be unable to make voluntary efforts; therefore controlled ventilation is appropriate for these patients.

Controlled ventilation may be difficult to use unless the patient is sedated or paralyzed with medications or is deliberately hyperventilated to suppress spontaneous breathing efforts. For example, sedation and paralysis are used if seizure activity or tetanic contractions cannot be prevented. Sedation and sometimes paralysis are recommended during inverse ratio ventilation and with permissive hypercapnia because these conditions are uncomfortable and not well tolerated by conscious patients.

Deliberate (iatrogenic) hyperventilation is used occasionally to temporarily induce respiratory alkalosis and thereby reduce intracranial pressure (ICP) in patients with a closed head injury and severely elevated ICP. Iatrogenic hyperventilation has also been used in Reye syndrome and after neurosurgery when ICP is elevated, but, as mentioned in Chapter 4, its use is controversial. When iatrogenic hyperventilation is used, it is typically employed for short periods until a more effective strategy can be implemented.

Adequate alarms and monitors must be used to safeguard patients. "Locking out" a patient by making the ventilator totally insensitive to patient effort is rarely advisable.[25] It is important to understand that regardless of the mode selected, sensitivity should be set so that the ventilator responds to even minimal patient effort (Key Point 5.2).

Key Point 5.2 The operator sets the trigger threshold by setting the ventilator's sensitivity. An appropriate sensitivity setting does not require an excessive amount of patient effort to initiate a breath, but it is also not so sensitive that accidental triggering or ventilator self-triggering occurs.

Assisted Ventilation

Assisted ventilation is a term used by many clinicians to describe a time-triggered or patient-triggered CMV mode in which the operator sets a minimum breathing rate, sensitivity level, and type of breath (volume or pressure). Although the patient can trigger breaths at a faster rate than the set mandatory rate, the set volume or pressure is delivered with each breath.

With CMV, every breath (time triggered or patient triggered) is a machine breath. Patient triggering occurs because the ventilator is sensitive to pressure or flow changes that occur as the patient attempts to take a breath. When the ventilator senses a slightly negative pressure (−1 cm H_2O) or a decrease in flow (1—2 L/min below the expiratory bias flow), the inspiratory cycle begins (see the section on patient triggering in Chapter 3). The clinician must keep in mind that with CMV, a minimum breathing rate is set on the ventilator to guarantee a minimum V_E.

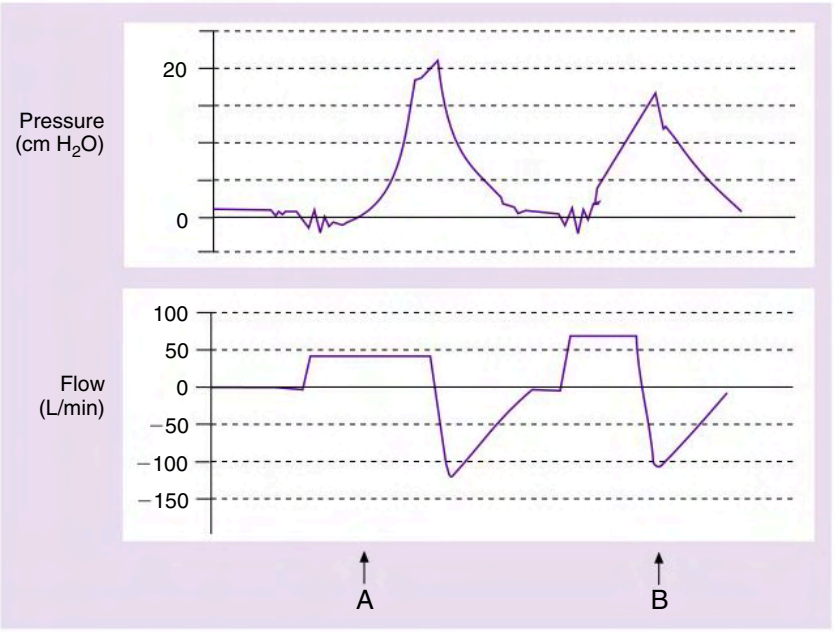

Fig. 5.4 Graphs of volume-controlled continuous mandatory ventilation showing a constant flow waveform. (A) Graphic display indicates that the flow, which is set at 50 L/min, is too low for patient demand. Note the concave appearance of the pressure curve; flow begins at one point, but pressure does not rise with the flow curve until much later. This indicates that the machine is not responding to patient effort. (B) Flow has been increased to 75 L/min. The pressure curve is normal, but sensitivity must also be increased so that the flow and pressure curves begin to rise almost simultaneously. In addition, inspiration time is shortened.

Several problems can arise with patient-triggered modes. These problems involve the ventilator's sensitivity setting and response time. It is worth noting if the machine is overly sensitive to patient effort, the machine triggers rapidly (autotriggering). This can be corrected easily by adjusting the machine's sensitivity so that it responds to a greater patient effort (−1 to −2 cm H_2O). Conversely, if an inspiratory effort shows a pressure reading of −3 to −5 cm H_2O or more below the baseline before an inspiration is initiated, the machine is too insensitive to the patient's effort and WOB increases.[26] In this case the sensitivity level is set too low and must be increased (see Fig. 3.6).

Historically, another common problem with patient-triggered modes is related to response time. Response time is the time increment between when a patient effort is detected and when flow from the ventilator to the patient begins. Manufacturers of ICU ventilators have made significant strides to improve response times in ventilators used in critical care.

Preventing respiratory alkalosis may be difficult to avoid in some patients on CMV unless respiratory depressants, muscle relaxants, or sedatives are used. The P_aCO_2 may reach the apneic threshold (32 mm Hg) in some patients.[27] It is unknown whether this is caused by the mode of ventilation, patient-ventilator asynchrony, or a change in the drive to breathe. Sometimes even switching to IMV on similar settings does not significantly change the arterial blood gas value.[28,29]

Volume-Controlled Continuous Mandatory Ventilation

VC-CMV is also referred to as *volume-targeted continuous mandatory ventilation*. Although VC-CMV was once thought to minimize WOB during mechanical ventilation, studies have shown that patients receiving this mode of ventilation may actually perform 33% to 50% or more of the work of inspiration.[23,26] This is especially true when inspiration is active and the set gas flow does not match the patient's inspiratory flow demand. Clinically this can be observed by watching the pressure manometer or the pressure-time curve on the graphic display. If the pressure does not rise smoothly and rapidly to peak during inspiration, flow is inadequate. A concave pressure curve indicates active inspiration. Flow must be increased until the patient's demand is met and the curve assumes a slightly convex shape (Fig. 5.4).

Pressure-Controlled Continuous Mandatory Ventilation

PC-CMV is also called *pressure-targeted CMV* or simply *pressure-controlled ventilation* (PCV). With PC-CMV all breaths are time or patient triggered, pressure targeted, and time cycled. The ventilator provides a constant pressure to the patient during inspiration (Fig. 5.5A). The operator sets the length of inspiration, pressure level, and backup rate of ventilation. The V_T delivered by the ventilator is influenced by the compliance and resistance of the patient's lungs, patient effort, and set pressure.[30] Several studies have shown that the decelerating ramp flow curve associated with PC-CMV (see Fig. 5.2 and Fig. 5.5) may improve gas distribution and allows the patient to vary inspiratory gas flow during spontaneous breathing efforts.[31-33]

The maximum pressure limit during PC-CMV is typically set at approximately +10 cm H_2O above the target or set pressure because the set pressure level is not the maximum pressure possible on most ventilators. Active coughing can increase circuit