CHAPTER

10

Mechanical Ventilators

In this chapter, we discuss the relevant physiology, history, and key features of mechanical ventilators. A *mechanical ventilator* is an instrument that replaces, or assists in, spontaneous breathing. Mechanical ventilators are used in a variety of settings, from the operating room (OR) and intensive care unit (ICU) to the home and transport vehicles. This discussion is limited to ICU ventilators (Figure 10.1) used by adult patients recovering from acute lung injuries.

Upon completion of this chapter, each student shall be able to:

1. Understand the mechanisms underlying pulmonary ventilation and blood flow.
2. Derive the relationship between pressure, flow, and volume in a mechanical ventilator.
3. Describe five key features of mechanical ventilators.

FIGURE 10.1 CareFusion Avea® Mechanical Ventilator. CareFusion is the spinoff of Cardinal Health, which purchased Viasys Healthcare in 2007. (CareFusion, San Diego, California).

PULMONARY PHYSIOLOGY

In the pulmonary circulation, deoxygenated blood flows from the right atrium and ventricle, through the pulmonary artery, and to the lungs. The blood flow through the lungs is essentially the cardiac output of 5 L/min. Within the lungs, blood flows to the pulmonary capillaries, where gas exchange takes place within the alveoli (Figure 10.2). Newly oxygenated blood then moves from the pulmonary veins to the left heart and into the aorta.

Pulmonary Blood Flow

Blood flow through the lungs is affected by hydrostatic pressure. In a normal, upright adult, the lung spans a height of approximately 30 cm. This translates into a 23-mmHg vertical pressure gradient, with 15 mmHg above the heart and 8 mmHg below the heart. Due to this pressure difference, the apex of the lung has little flow, while the bottom of the lung has five times as much flow (Figure 10.3).

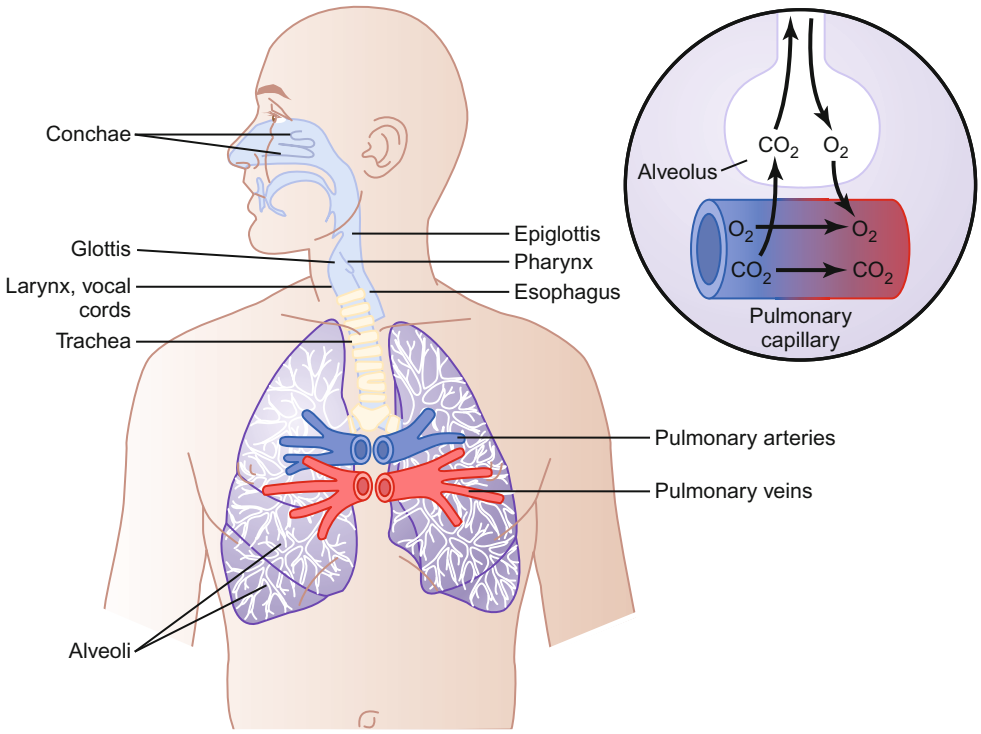

FIGURE 10.2 Respiratory passages. *[Reproduced by permission from Guyton & Hall (2006)].*

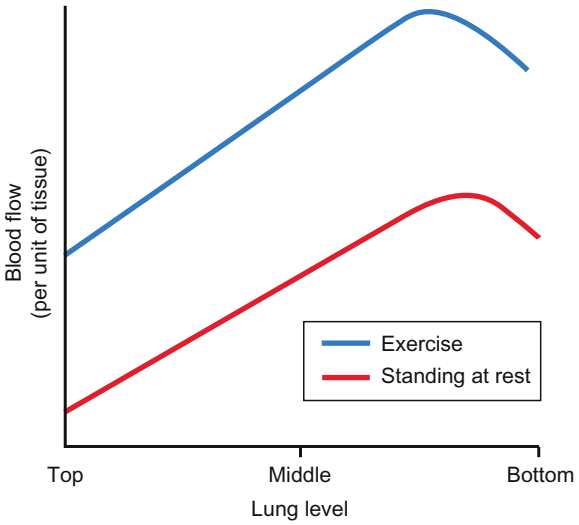

FIGURE 10.3 Blood flow at different levels in the lung of an upright person at rest and during exercise. *[Reproduced by permission from Guyton & Hall (2006)].*

We can explain these differences by considering three zones of local blood flow within the lungs, as a pulmonary capillary interacts with an alveolus (Figure 10.4). In zone 1, the capillary pressure never increases above the alveolar air pressure during any part of the cardiac cycle. This can have greater impact on overall gas exchange as an abnormal condition, which could occur if a subject's pulmonary systolic arterial pressure is very low due to severe blood loss. As a consequence, no blood flows thorough this lung zone. In zone 2, the capillary pressure is greater than the alveolar air pressure only during systole. Capillary pressure is less than alveolar air pressure during diastole. This occurs at the mid-levels of the lung because the pressure is about 10 mmHg during systole, while the alveolar air pressure is 0 mmHg. So now, blood flows intermittently through this lung zone. Finally, in zone 3, the capillary pressure is always greater than the alveolar air pressure. This occurs in the lower regions of the lung, where blood flows continuously.

Pulmonary Ventilation

For pulmonary capillaries to receive oxygen at the alveoli, air must be transported to the alveoli. *Pulmonary ventilation* is the inflow and outflow of air between the atmosphere and the lung alveoli. It is the first of four functions that make up respiration. Air moves to and from the lungs through lung expansion and contraction. During normal quiet breathing, inspiration occurs primarily by contraction of the diaphragm. This contraction pulls the lungs downward. During expiration, the diaphragm relaxes, which causes the lungs to compress through elastic recoil (Figure 10.5).

Each lung consists of elastic tissue that floats in the pleural cavity. The pleural cavity is lined by a smooth membrane, the parietal pleura, which is reflected back at its root to become the visceral pleura. The visceral pleura covers the lungs and adjacent structures.

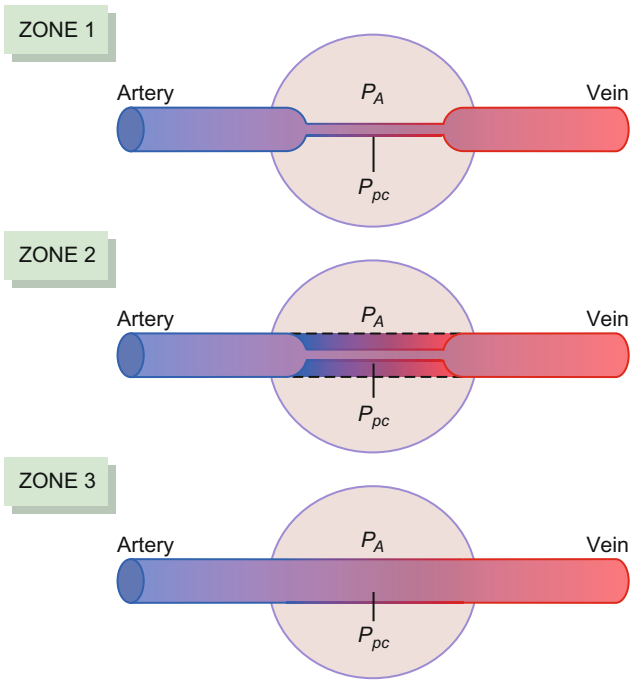

FIGURE 10.4 Mechanics of blood flow in the three blood flow zones of the lung. Zone 1: no flow, zone 2: intermittent flow, zone 3: continuous flow. P_A = alveolar air pressure, P_{pc} = pulmonary capillary pressure. *[Reproduced by permission from Guyton & Hall (2006)].*

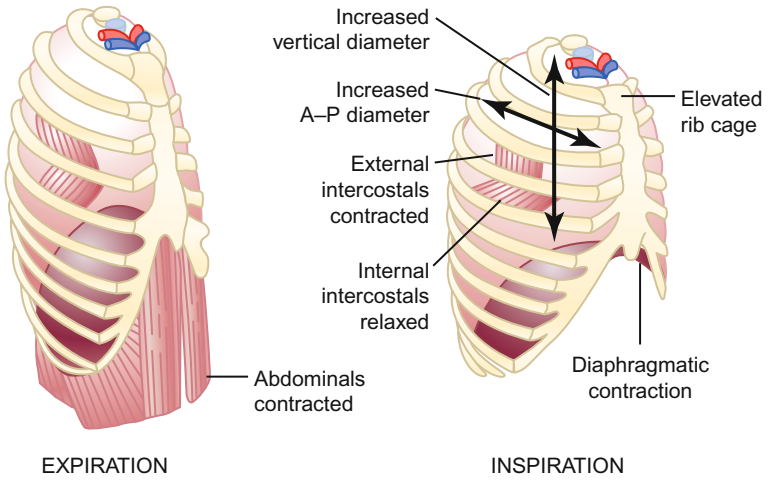

FIGURE 10.5 Contraction and expansion of the thoracic cage during expiration and inspiration. *[Reproduced by permission from Guyton & Hall (2006)].*

Pleural fluid between the two contacting membranes acts as a lubricant to reduce the friction of the lungs' movement within the pleural space.

At the end of expiration, before inspiration begins, the pleural pressure in the pleural fluid is slightly negative, at about $-5\ cmH_2O$, to hold the lungs open at resting level.

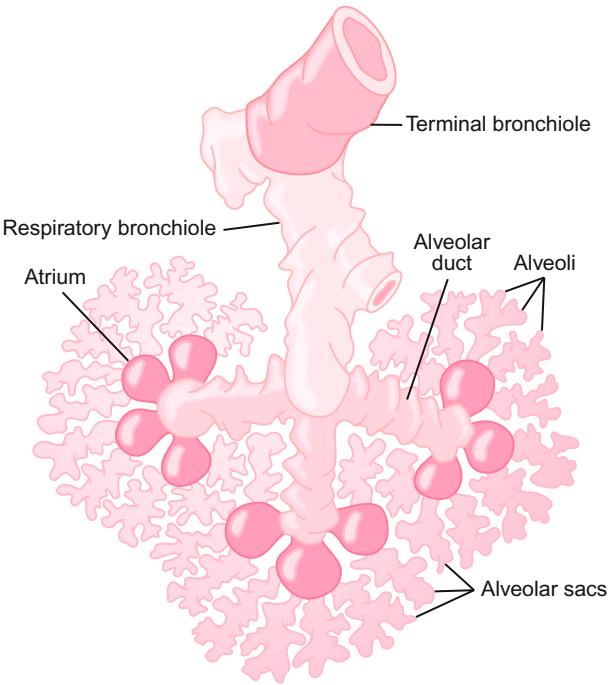

FIGURE 10.6 Respiratory unit. *[Redrawn from Miller (1947). Reproduced by permission from Guyton & Hall (2006)].*

Because the glottis is open and no air is flowing, the pressures in all parts of the respiratory tree, including the alveoli, are equalized to atmospheric pressure, which is 0 cmH_2O.

During inspiration, contraction of the diaphragm and the expansion of the chest cage by the intercostal muscles pull the lungs downward and outward. This creates more negative pleural pressure, with a fall to about -7.5 cmH_2O. This fall causes inward flow of about half a liter of air through the trachea, bronchi, and bronchioles to the alveoli in 2 s of normal quiet breathing (Figure 10.6).

During expiration, the pleural pressure reverses itself, and returns to -5 cmH_2O. The alveolar pressure rises to about $+1$ cmH_2O, which forces half a liter of air out of the lungs (Figure 10.7). The difference between the alveolar and pleural pressures is known as the transpulmonary pressure.

We can measure respiratory excursions over time using a spirometer, which is an instrument that measures inspiration and expiration with a flow sensor. As shown in Figure 10.8, a tidal volume, V_T, of about 500 mL is normally inspired and expired in each breath. A patient may forcefully inspire about 3000 mL above the tidal volume, which is the inspiratory reserve volume, or forcefully expire about 1100 mL beyond the resting lung volume level at the end of a tidal breath, which is the expiratory reserve volume. Even after the most forceful expiration, a residual volume of about 1200 mL remains in the lung. Together, these four volumes make up the total lung capacity.

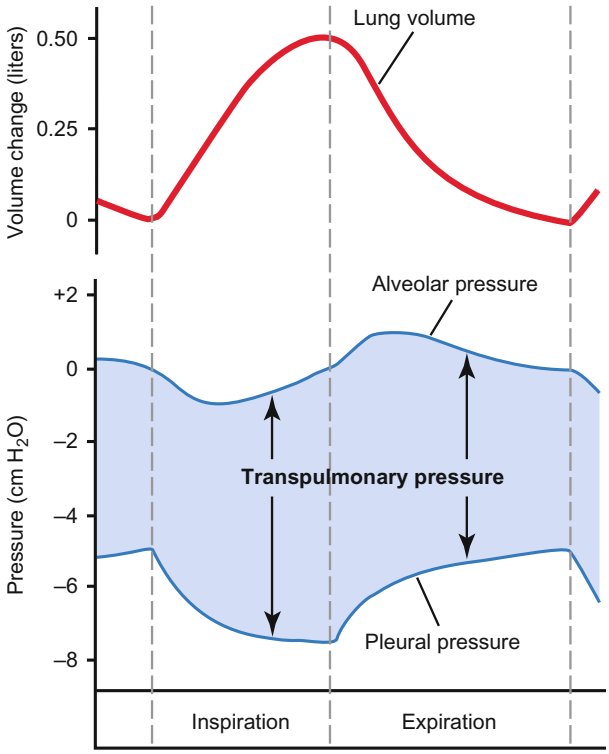

FIGURE 10.7 Change in lung volume, alveolar pressure, pleural pressure, and transpulmonary pressure during normal breathing. *[Reproduced by permission from Guyton & Hall, 2006)].*

With pulmonary ventilation, air moves through the alveoli. The rate of alveolar ventilation, $\dot{V}_A$, or alveolar flow, may be calculated as:

$$\dot{V}_A = f \cdot (V_T - V_D), \tag{10.1}$$

where f is the frequency of respiration, in units min^{-1}, and V_D is the physiologic dead space volume. The physiologic dead space volume refers to air that fills the respiratory passages but that does not participate in gas exchange. When poor blood flow is present, the dead space volume may include the alveolar volume because gas exchange is impaired.

Gas Diffusion

With blood flow to the pulmonary capillaries and air flow to the alveoli, diffusion of gases can occur along a pressure gradient. In the air, the partial pressure of either oxygen (P_{O_2}), nitrogen (P_{N_2}), or carbon dioxide (P_{CO_2}) is equal to its percentage concentration. At sea level, the total pressure of this mixture is 760 mmHg, or 1 atm. Because alveolar air is only partially replaced by atmospheric air with each breath, P_{O_2} decreases from an atmospheric oxygen pressure of 159 mmHg to an alveolar oxygen partial pressure ($P_{A\,O_2}$) of

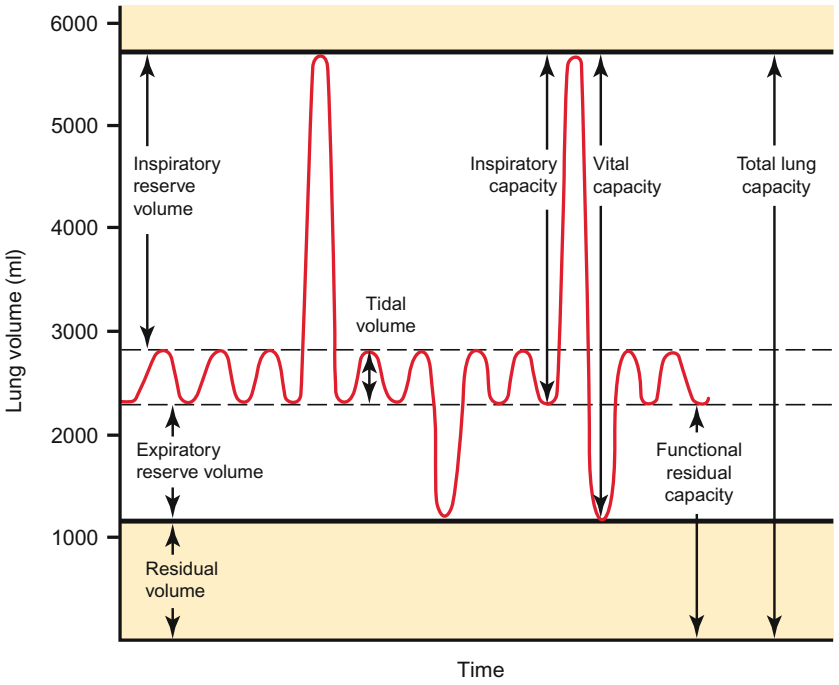

FIGURE 10.8 Diagram of total lung capacity. *[Reproduced by permission from Guyton & Hall (2006)].*

104 mmHg. Conversely, CO_2 increases from an atmospheric P_{CO_2} of 0.3 mmHg to an alveolar CO_2 partial pressure ($P_{A\ CO_2}$) of 40 mmHg.

In a gas dissolved in a fluid, the partial pressure of a gas is determined by the ratio of its concentration to the solubility coefficient of the gas. The blood perfusing the pulmonary capillaries is venous blood returning to the lungs from the systemic circulation. Normal venous blood has a Pv_{O_2} of 40 mmHg and a Pv_{CO_2} of 45 mmHg. With these pressure gradients between the alveoli and venous blood in the pulmonary capillaries, oxygen diffuses from the alveoli to the blood, as CO_2 diffuses from the blood to the alveoli (Figure 10.9). The alveolar capillaries provide about 98% of blood entering the left atrium. The other 2% passes through the bronchial circulation that supplies the deep tissues of the lung but is not exposed to air. This so-called shunt flow decreases the partial pressure of oxygen that enters the left side of the heart to 95 mmHg, which is the partial pressure of arterial blood (Pa_{O_2}) reaching the aorta. The partial pressure of CO_2 in the arterial blood (Pa_{CO_2}) is 40 mmHg (Guyton & Hall, 2006).

VENTILATOR MECHANICS

We can model the pressures involved in ventilation by using Newton's third law of motion: For every action, there is an equal and opposite reaction. The transrespiratory pressure, $P_{TR}(t)$, is the pressure at the airway opening minus the pressure at the body

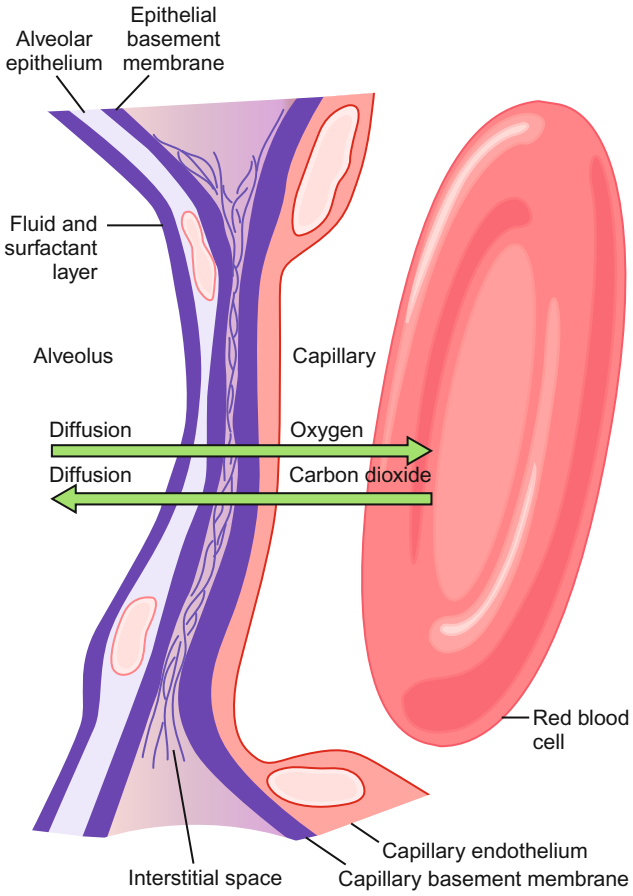

FIGURE 10.9 Diffusion between the alveolus and pulmonary capillary. *[Reproduced by permission from Guyton & Hall (2006)].*

surface. This pressure is equal to sum of the pressure due to the elastic recoil, $P_E(t)$, and the pressure due to resistance to flow, $P_R(t)$:

$$P_{TR}(t) = P_E(t) + P_R(t). \tag{10.2}$$

During spontaneous breathing, the transrespiratory pressure is equal to the pressure resulting from respiratory muscle contraction, $P_{musc}(t)$. But when a patient requires assistance from a mechanical ventilator to breathe, the transrespiratory pressure includes the mechanical ventilator pressure, $P_{vent}(t)$:

$$P_{TR}(t) = P_{musc}(t) + P_{vent}(t). \tag{10.3}$$

The pressure due to elastic recoil is the product of the elastance, E, and lung volume, $V(t)$. Elastance is the change in pressure due to the change in volume, $dP(t)/dV(t)$. For this model, we assume that elastance is constant. As we have already seen in Chapter 6, the pressure due to the resistance to flow is the product of the resistive load, R, and flow, $Q(t)$. Because flow is the derivative of volume with

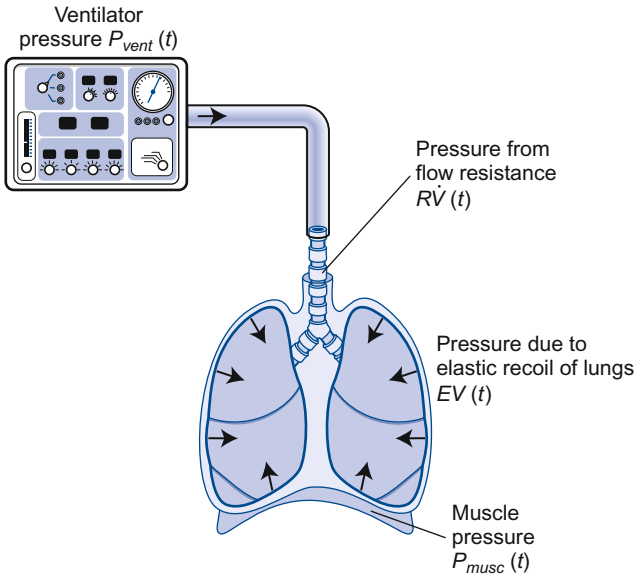

FIGURE 10.10 Model of ventilation representing $P_{musc}(t) + P_{vent}(t) = EV(t) + R\dot{V}(t)$. *[Reproduced by permission from Cairo & Pilbeam (2010)].*

respect to time, we use $\dot{V}(t)$ to represent flow. Combining these relationships with Eqs. (10.2) and (10.3) results in:

$$P_{musc}(t) + P_{vent}(t) = EV(t) + R\dot{V}(t). \tag{10.4}$$

Eq. (10.4) represents the interaction between the patient and ventilator during inspiration and expiration (Figure 10.10). During spontaneous inspiration, $P_{vent}(t) = 0$; so the muscles generate sufficient pressure to offset elastic recoil and resistive pressures. In contrast, a patient may receive controlled mechanical ventilation, in which she does not trigger breathing. Here, $P_{musc}(t) = 0$; so the ventilator must generate sufficient pressure to offset elastic recoil and resistive pressure.

During passive expiration, pressures from the muscles and ventilator are absent, so $P_{musc}(t) = P_{vent}(t) = 0$. Eq. (10.4) becomes

$$0 = EV(t) + R\dot{V}(t) \tag{10.5}$$

and

$$-R\dot{V}(t) = EV(t). \tag{10.6}$$

The negative sign in Eq. (10.6) indicates that the direction of flow is reversed, which you expect for expiration.

When a patient receives mechanical ventilation, this therapy may be based on pressure, volume, or flow control. Because flow is the derivative of volume and because the three variables are related by Eq. (10.4), only one variable acts as the independent variable for control. After the control variable is chosen, the other two follow.

For example, we can assume that the constants in Eq. (10.4) are unity, with $E = R = 1$. With this assumption, we see that the equation has been modified so that flow plus

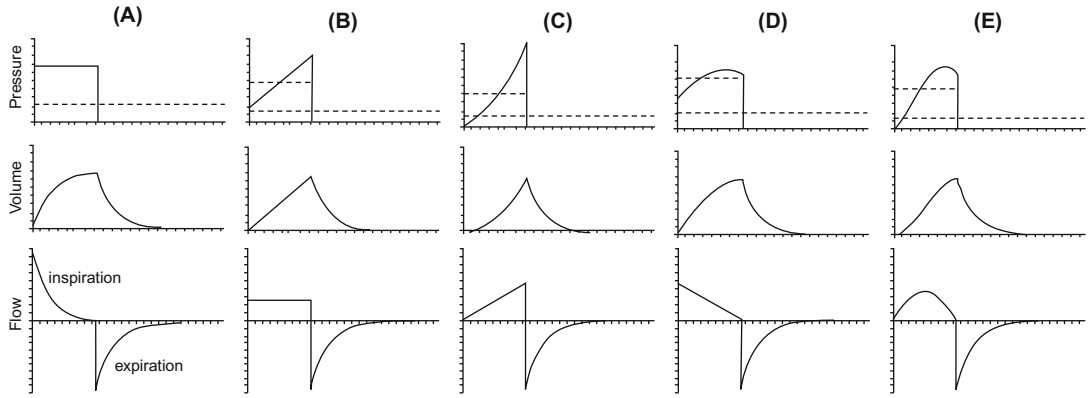

FIGURE 10.11 Idealized ventilator output waveforms. **A**: Constant pressure-controlled inspiration. **B**: Constant flow-controlled inspiration. **C**: Ascending-ramp flow-controlled inspiration. **D**: Descending-ramp flow-controlled inspiration. **E**: sinusoidal flow-controlled inspiration. In all cases, expiration pressure equals zero. *[Reproduced with permission from Chatburn. Figure reproduced with permission from Chatburn (2006)].*

volume equals pressure. This enables us to draw idealized ventilator output waveforms for inspiration and expiration. The waveform sets in Figure 10.11 are based on constant pressure (A), constant flow (B), ascending-ramp flow (C), descending-ramp flow (D), and sinusoidal flow (E) for inspiration. In all cases, the expiration pressure is zero (Chatburn, 2006).

CLINICAL NEED

In the ICU, mechanical ventilators are indicated for a variety of conditions. Some patients present with apnea due to central nervous system damage, and they are not breathing spontaneously. Others have clinical signs of increased work of breathing, with or without laboratory evidence of impaired gas exchange. These patients in respiratory distress may have a 30−50% greater inspiratory resistance and 100% greater dynamic elastance than normal subjects (Laghi & Tobin, 2006). For many of these patients, measurement of blood gases (described in Chapter 11) reveals either a Pa_{O_2} less than 55 mmHg or a Pa_{CO_2} greater than 50 mmHg, with a corresponding pH less than 7.32 (Amitai et al., 2009).

In the OR, mechanical ventilators are also used because the administration of deep general anesthesia suppresses spontaneous breathing.

HISTORIC DEVICES

Scientists have always been interested in resuscitating the dead and dying. Legends purport that both Vesalius and Paracelsus attempted to resuscitate humans in the 1500s.

FIGURE 10.12 Draeger Pulmotor. *[Copyright © the American Society of Anesthesiologist, Inc. This image appears in the Anesthesiology Reflections online collection available at www.anesthesiology.org. Figure reproduced by permission from Bause (2009)].*

Early Devices

More recently, in 1908, the Draeger Oxygen Apparatus Company division in Pittsburgh began to produce Pulmotors to resuscitate miners. The Pulmotor pumped air or oxygen into the airways of victims of fire or mining accidents (Bause, 2009) (Figure 10.12). Because its air supply was not controlled, excessive pressure could damage internal organs.

As an alternative to forcing air into the lungs, scientists began to develop negative-pressure ventilators. A patient's body was placed in an airtight enclosure, with his head outside the enclosure. The body was subjected to cyclical negative and atmospheric pressure. Negative pressure in the enclosure pulled air into the lungs; atmospheric pressure in the enclosure caused exhalation.

The first clinically successful design of this tank respirator was developed by chemical engineer Philip Drinker and physician Louis Agassiz Shaw. Nicknamed the "iron lung" by the general public, this cylindrical device was powered by an electric motor with two vacuum cleaners, which modified pressure within the enclosure. Inventor John Emerson refined the Drinker-Shaw respirator and reduced the cost to almost half of the original design. In the improved design, the patient lay on a movable bed that slid out of the enclosure as needed. Attendants could access the patient through portal windows (Figure 10.13).

The tank respirator was used to treat patients struck down with polio. Poliomyelitis is caused by the poliovirus, which is transmitted by the oral-fecal route or by ingestion of contaminated water. The first known outbreak in the United States occurred in 1894. This epidemic peaked in 1952, when a record 57,628 cases were reported. During the acute stage, the virus destroys motor neurons that control swallowing, breathing, and limb movement. Severe polio patients were placed in tank respirators until they could breathe spontaneously, usually after 1–2 weeks (National Museum of American History, 2009).

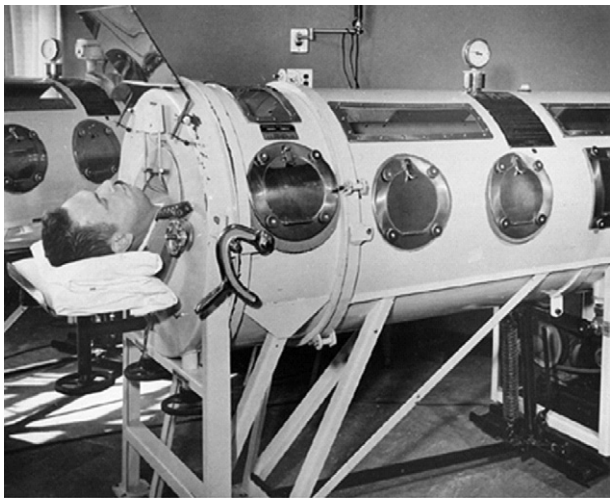

FIGURE 10.13 Man using an Emerson tank respirator equipped with a mirror in the 1950s. Original courtesy of Post-Polio Health International. *[Reproduced by permission from National Museum of American History (2009)].*

Although certainly an improvement in ventilation therapy, the tank respirator's negative pressure was applied to the abdominal wall, as well as to the chest. This created venous pooling in the abdomen and decreased cardiac output.

Enabling Technology: Positive-Pressure Ventilation

During the polio epidemic of 1952 in Copenhagen, Blegdam Hospital, which was the only hospital serving metropolitan Cophenhagen, did not have sufficient respirators for polio patients. At the recommendation of anesthesiologist Bjorn Ibsen, patients began to be treated with manual positive-pressure ventilation. The patients treated by positive-pressure had decreased mortality rates, compared to those treated with conventional respirators (Lassen, 1953).

As a result, positive-pressure ventilation became more popular in Europe, leading to the construction of mechanical positive-pressure ventilators in Europe and later in the United States. In, 1955, physician Forrest Bird introduced the Bird Mark 7 ventilator, which was the first low-cost, mass-produced mechanical ventilator (Figure 10.14).

SYSTEM DESCRIPTION AND DIAGRAM

A modern ICU ventilator is electrically powered and microprocessor controlled. The user inputs the desired breathing parameters, such as respiratory rate, tidal volume, set pressure, and inspiratory time, to the processor module. In a generalized system, which is really a combination of deployed architectures, two 50-psi gas sources for air (an internal compressor compresses external air) and for oxygen (O_2) provide the pressure to deliver inspiratory gas flow to the patient. Control of the inspiratory flow waveform is governed by the processor module, which controls the air and O_2 valves. Two flow sensors provide air and O_2 flow data to the processor module. A rigid accumulator serves as the internal

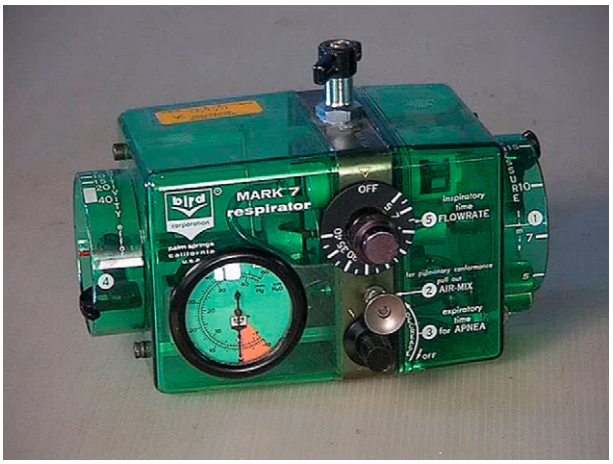

FIGURE 10.14 Bird Mark 7 Ventilator (Photograph taken by PEMED).

reservoir to supply flow on demand to the patient. The flow from the accumulator is controlled by the processor module through the flow valve, with feedback data from a down-the-line flow sensor.

During inspiration, the exhalation valve is closed to direct all flow to the patient. A pressure sensor in the patient circuit provides feedback to the processor module for maintaining the positive end-expiratory pressure (PEEP). The LCD provides the ventilator user interface (Figure 10.15). After the user inputs desired breathing parameters for a patient, a ventilation mode is delivered. Ventilation mode is defined by the control type used within and between breaths, as well as by the breath sequence within breaths.

Common Modes of Mechanical Ventilation

As already stated, there are three control variables: pressure, volume, and flow. Because flow is the time derivative of volume, we can simplify control by referring to only pressure and volume control. A third type of control is dual control, in which the control variable switches between pressure and volume, in either order, within a breath.

Breath sequence refers to the combinations of mandatory and spontaneous breaths. The clinical intent may be to provide full ventilator support or partial ventilator support to wean a patient off the ventilator. When spontaneous breaths are not allowed between mandatory breaths, the breath sequence is *continuous mandatory ventilation* (*CMV*). When spontaneous breaths are allowed between mandatory breaths, the breath sequence becomes *intermittent mandatory ventilation* (*IMV*). If no mandatory breaths are allowed, the breath sequence is *continuous spontaneous ventilation* (*CSV*).

Combining control type and breath sequence, Chatburn defines eight ventilation modes:

1. Volume control–continuous mandatory ventilation (VC-CMV)
2. Volume control–intermittent mandatory ventilation (VC-IMV)
3. Pressure control–continuous mandatory ventilation (PC-CMV)

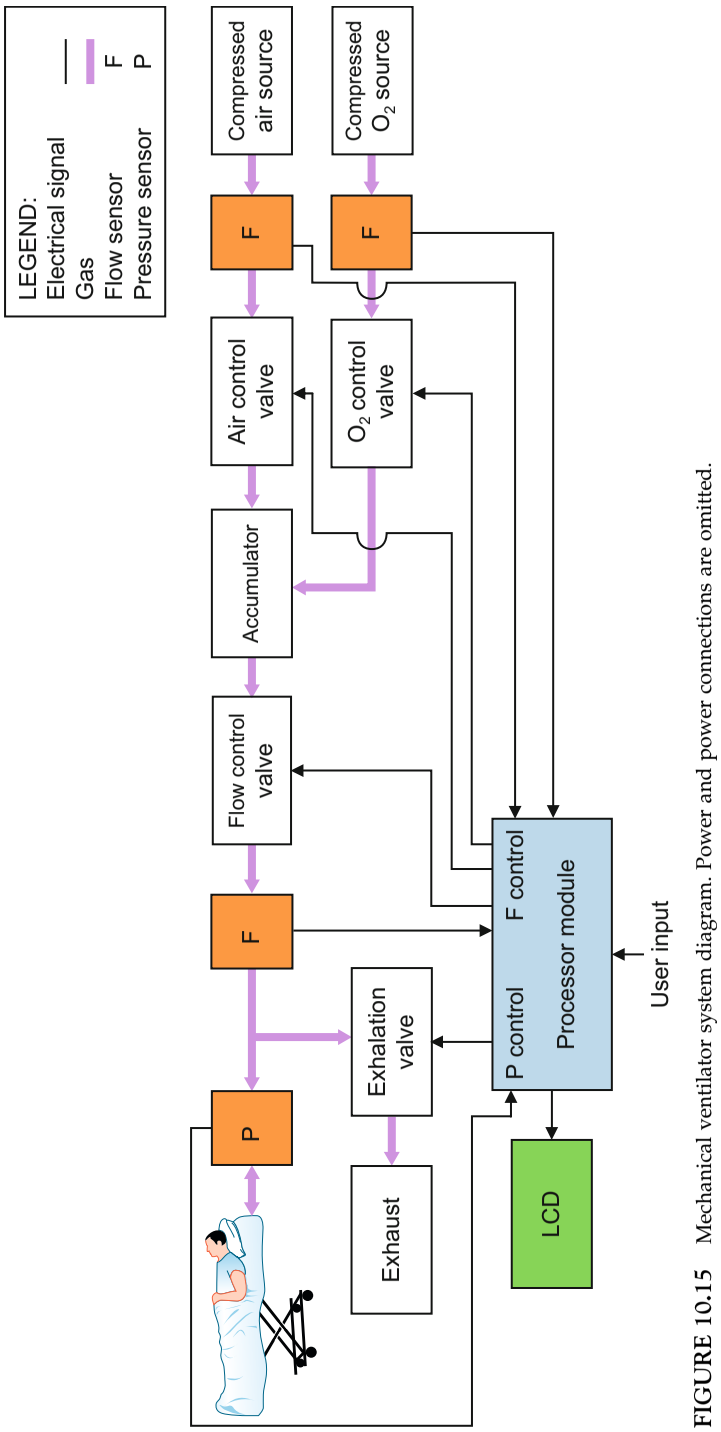

FIGURE 10.15 Mechanical ventilator system diagram. Power and power connections are omitted.

4. Pressure control—intermittent mandatory ventilation (PC-IMV)
5. Pressure control—continuous spontaneous ventilation (PC-CSV)
6. Dual control—continuous mandatory ventilation (DC-CMV)
7. Dual control—intermittent mandatory ventilation (VC-IMV)
8. Dual control—continuous spontaneous ventilation (DC-CSV) (Chatburn, 2007)

Please note that VC-CSV is not possible, because the definition of volume control conflicts with the definition of a spontaneous breath (Chatburn, 2007). We describe four of these ventilation modes in greater detail.

A patient who cannot breathe spontaneously may be placed on controlled mandatory ventilation. Such a patient may be suffering from a drug overdose, a neurologic or neuromuscular disorder, or seizure activity requiring sedation and possibly induced paralysis. Preset volume- or pressure-targeted breaths at set intervals are delivered, according to the control variable. The related pressure, volume, and flow waveforms are shown in Figure 10.16.

A patient being weaned from the ventilator may be placed on intermittent mandatory ventilation. As with controlled mandatory ventilation, preset volume- or pressure-targeted breaths at a set interval are delivered. Unlike CMV, between mandatory breaths, the patient may breathe spontaneously from the ventilator circuit. A spontaneous breath starts

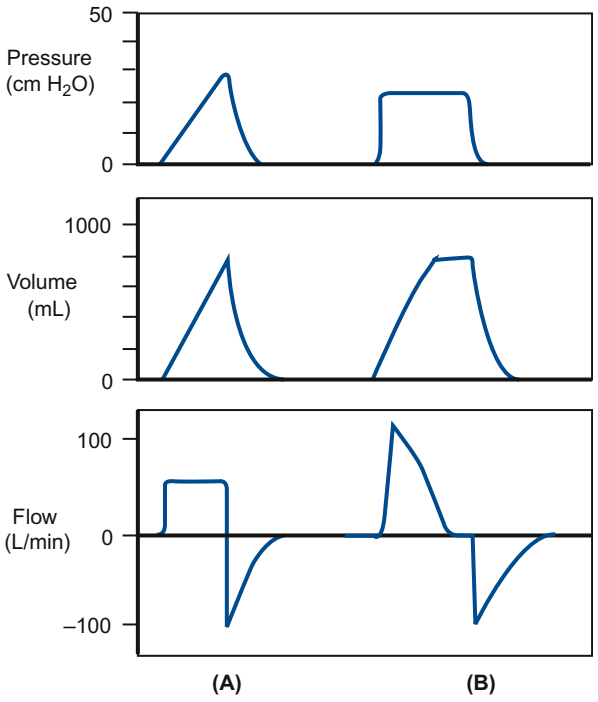

FIGURE 10.16 Pressure, volume, and flow waveforms for two ventilation modes: **A**: VC-CMV; **B**: PC-CMV. *[Reproduced by permission from Cairo & Pilbeam (2010)].*

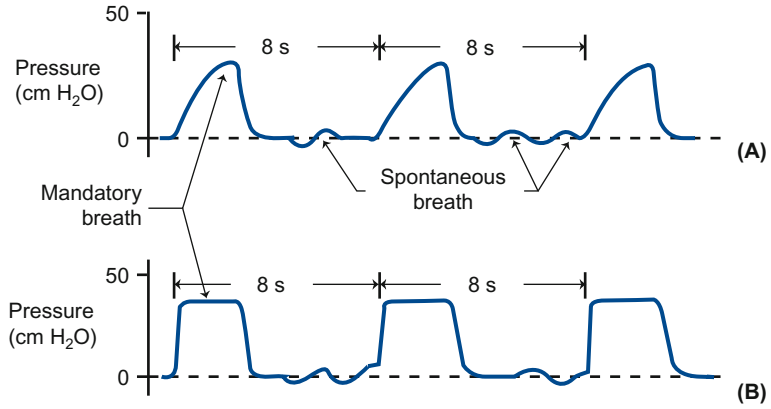

FIGURE 10.17 Pressure-time curves for two ventilation modes: **A**: VC-IMV; **B**: PC-IMV. *[Reproduced by permission from Cairo & Pilbeam (2010)].*

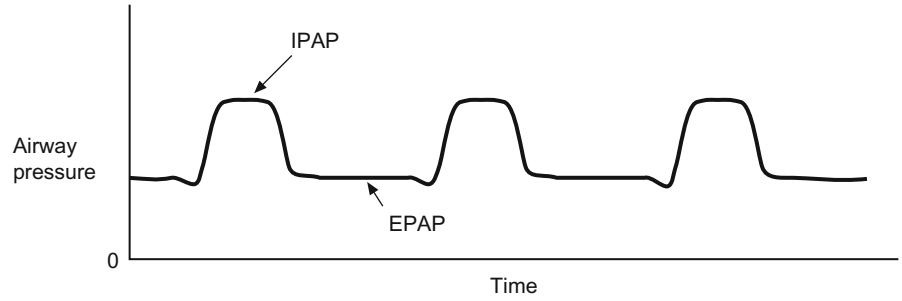

FIGURE 10.18 Bilevel positive airway pressure (BiPAP) showing inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). EPAP is the PEEP. Both pressures are higher than a zero baseline pressure. *[Original figure from Pilbeam (1998). Figure reproduced by permission from Cairo & Pilbeam (2010)].*

from the set baseline, which may be ambient pressure or a positive baseline pressure. For this breath to occur, the patient must assume part of the work of inspiration (Figure 10.17).

A patient receiving noninvasive positive pressure ventilation (NIPPV) through a face mask may have bilevel positive airway pressure administered. This setting, also called BiPAP by one manufacturer, may be used in home ventilators to treat obstructive sleep apnea. With this setting, inspiration and expiration occur at two levels of positive pressure (Cairo and Pilbeam, 2010) (Figure 10.18). Because the patient triggers inspiration, NIPPV is another example of PC-IMV.

Noninvasive Ventilation

Invasive mechanical ventilation requires endotracheal intubation to complete the patient circuit. *Endotracheal intubation* is the insertion of an artificial airway into the trachea by either the oropharyngeal or nasopharyngeal route. Although invasive ventilation is highly

effective and reliable in supporting alveolar ventilation, the intubation procedure is associated with occasional adverse events such as pharyngeal/laryngeal/tracheal tissue damage, self-extubation, and aspiration of gastric contents.

Whenever possible, noninvasive ventilation is a preferable option. The upper airway is intact, airway defense mechanisms are preserved, and patients may eat, drink, verbalize, and expectorate secretions. Because of the convenience in using a face mask, NIPPV is the predominant means of administering noninvasive ventilation (Hill, 2006).

For patients with chronic obstructive pulmonary disease (COPD), NIPPV has been demonstrated to reduce complications, duration of ICU stay, and mortality (Plant et al., 2000). Chronic obstructive pulmonary disease is characterized by airflow limitation that is not fully reversible. We discuss an NIPPV clinical trial for COPD patients in the exercises at the end of the chapter. Noninvasive positive pressure ventilation has not been demonstrated to be more efficacious than invasive ventilation for other types of patients. However, numerous other applications are currently under clinical investigation (Hill, 2006).

KEY FEATURES FROM ENGINEERING STANDARDS

As of 2010, FDA recommends several consensus standards related to various aspects of mechanical ventilators. One of these standards is *IEC 60601-2-12 Medical Electrical Equipment—Part 2:12: Particular Requirements for the Safety of Lung Ventilators—Critical Care Ventilators* (IEC, 2001).

Protection from Interruption of Power Supply

If not being weaned, a patient may be completely dependent on a mechanical ventilator for respiratory support. Should the supply power fall below values specified by the manufacturer, the ventilator must sound an alarm for at least 120 s. While the supply is compromised, the patient must be able to breathe spontaneously from the ventilator.

It is not mandatory that the ventilator switch to an internal electrical power source. However, if this switch occurs, the ventilator must indicate the state of this power source, with an alarm sounded before the internal electrical power source is completely depleted.

These requirements are verified by simulating a drop below the supply rates of the manufacturer and checking for the power failure alarm and enablement of spontaneous breathing. If an internal electrical power source is present, the internal power source is depleted to below the minimum value specific by the manufacturer. The internal power failure alarm is then verified (IEC, 2001).

Maximum Pressure to Patient

The patient must be protected from excess pressure. When the pressure at the patient connection port exceeds 125 cmH_2O during normal use, this pressure must not reach the patient. (Pressure is usually set to a lower limit by the clinician.) The respiratory pressure

at the patient connection port must be displayed and accurate within ±2% of the full-scale reading +4% of the actual reading. The site of actual pressure measurement may be anywhere at the ventilator breathing system, but the displayed pressure must reference the patient connection port.

These requirements are tested by the administration of various pressures of unspecified magnitude, with "visual inspection and verification of accuracy" (IEC, 2001).

Active Pressure Limit

The pressure limit in the breathing system for each type of breath must be operator adjustable, or specified by an active breathing algorithm, or some combination of both. When an active limit value is reached, the ventilator must act to reduce the pressure to a level at or below the PEEP value within 200 ms. The method of testing this requirement is unspecified (IEC, 2001).

Oxygen Monitor

Adequate oxygen must be delivered to the patient. The ventilator is required to be equipped with an oxygen monitor for the measurement of inspiratory oxygen concentration, such as in the inspiratory limb or at the patient connection port. This oxygen monitor must comply with separate standard ISO 7767, which details specific requirements and testing for monitoring patient breathing mixtures. The oxygen monitor must be provided with a high alarm limit (IEC, 2001)

Protection from Breathing System Leakage

The patient must be adequately ventilated. Leakage from the ventilator breathing system must not exceed 200 mL/min at 50 hPa for ventilators providing tidal volumes greater than 300 ml or 100 mL/min at 40 hPa for tidal volumes between 300 mL and 30 ml. Testing is conducted by sealing all ports, connecting a pressure measuring device, and introducing air until the appropriate pressure is reached. The air flow is then adjusted to stabilize the pressure, with leakage flow recorded (IEC, 2001).

SUMMARY

A mechanical ventilator is an instrument that replaces or assists in spontaneous breathing. Mechanical ventilators are used in a variety of settings, from the operating room and intensive care unit to the home and transport vehicles. ICU ventilators are used by adult patients recovering from acute lung injuries.

In the pulmonary circulation, deoxygenated blood flows from the right atrium and ventricle, through the pulmonary artery, and to the lungs. The blood flow through the lungs is essentially the cardiac output of 5 L/min. Within the lungs, blood flows to the pulmonary capillaries, where gas exchange takes place with the alveoli. Atmospheric air moves

to and from the lung alveoli through lung expansion and contraction. Newly oxygenated blood then moves from the pulmonary veins to the left heart and into the aorta.

We can model the pressures involved in ventilation as $P_{musc}(t) + P_{vent}(t) = EV(t) + R\dot{V}(t)$.

When a patient receives mechanical ventilation ($P_{musc}(t) = 0$), this therapy may be based on pressure, volume, or flow control. Because flow is the derivative of volume and because the three variables are related by this equation, only one variable acts as the independent variable for control. After the control variable is chosen, the other two follow.

Early ventilators were based on negative pressure, but are currently based on positive pressure. Key ventilator features include protection from interruption of power supply, maximum pressure to patient, active pressure limit, oxygen monitoring, and protection from breathing system leakage.

Exercises

10.1 Expired respiratory gases are 74% N_2, 16% O_2, 4% CO_2, and 6% H_2O. What are the corresponding partial pressures of expired O_2 and CO_2 at sea level? At the beginning of expiration, what are the pleural, alveolar, and transpulmonary pressures?

10.2 Mechanical ventilators once used a piston to increase gas pressure. Why is this design no longer needed?

10.3 Sketch the corresponding flow waveforms for the volume and pressure waveforms given in Figure 10.19.

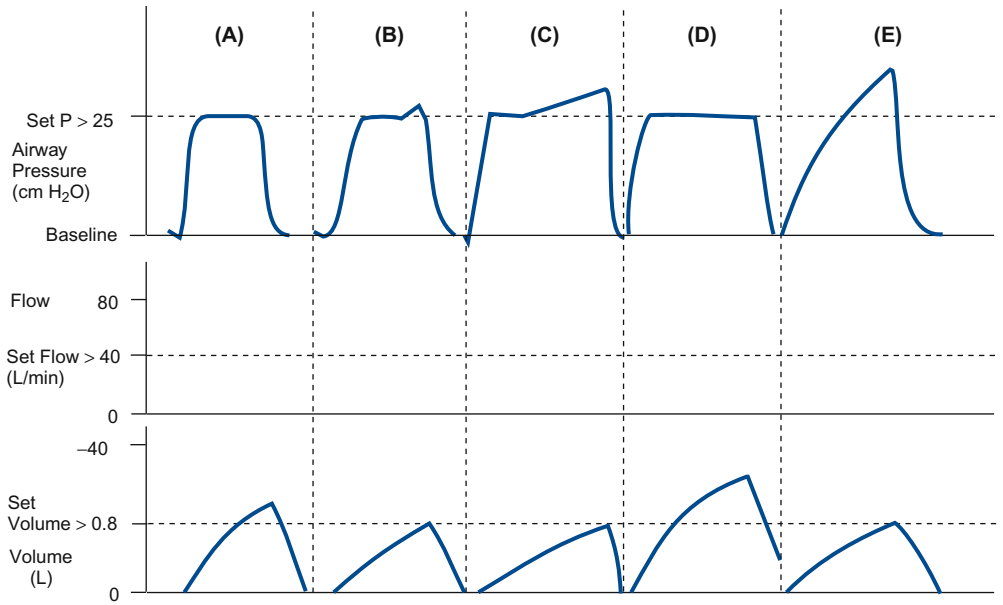

FIGURE 10.19 Various waveform combinations achievable with volume-controlled ventilation. *[Original from Pilbeam & Cairo (2006). Figure reproduced by permission from Cairo & Pilbeam (2010)].*

10.4 Why is BiPAP used to treat obstructive sleep apnea?

Read (Lassen, 1953):

10.5 What was the mortality rate of polio patients during the first month of the epidemic? How was respiratory insufficiency treated in these patients? How did a tank respirator work?

10.6 Describe the replacement therapy for the tank and cuirass respirators in detail. What was the purpose of using soda lime?

Read Plant et al. (2000):

10.7 What is a multicenter randomized controlled clinical trial? What was the null hypothesis? What is *power* as the term is used in this study? How was the sample size calculated?

10.8 What are the normal ranges for arterial pH, partial pressure of oxygen, and partial pressure of carbon dioxide? What were the primary and secondary outcome measures? Is intubation necessary in this type of patient group?

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