Index

Mechanical Ventilation

Chapter 24

PRINCIPLES OF MECHANICAL VENTILATION

" ... an *opening must be attempted in the* trunk *of the trachea, into which* a *tube of reed or cane should be put; you will then blow* into *this,* so *that the lung may rise* again ... and *the heart becomes strong*"
Andreas Vesalius (1555)

Vesalius is credited with the first description of positive-pressure ventilation, but it took 400 years to apply his concept to patient care. The occasion was the polio epidemic of 1955, when the demand for assisted ventilation outgrew the supply of negative-pressure tank ventilators (known as *iron lungs).* In Sweden, all medical schools shut down and medical students worked in 8-hour shifts as human ventilators, manually inflating the lungs of afflicted patients. In Boston, the nearby Emerson Company made available a prototype positive-pressure lung inflation device, which was put to use at the Massachusetts General Hospital, and became an instant success. Thus began the era of positive-pressure mechanical ventilation (and the era of intensive care medicine).

CONVENTIONAL MECHANICAL VENTILATION

The first positive-pressure ventilators were designed to inflate the lungs until a preset pressure was reached. This type of *pressure-cycled* ventilation fell out of favor because the inflation volume varied with changes in the mechanical properties of the lungs. In contrast, *volume-cycled* ventilation, which inflates the lungs to a predetermined volume, delivers a constant alveolar volume despite changes in the mechanical properties of the lungs. For this reason, volume-cycled ventilation has become the standard method of positive-pressure mechanical ventilation.

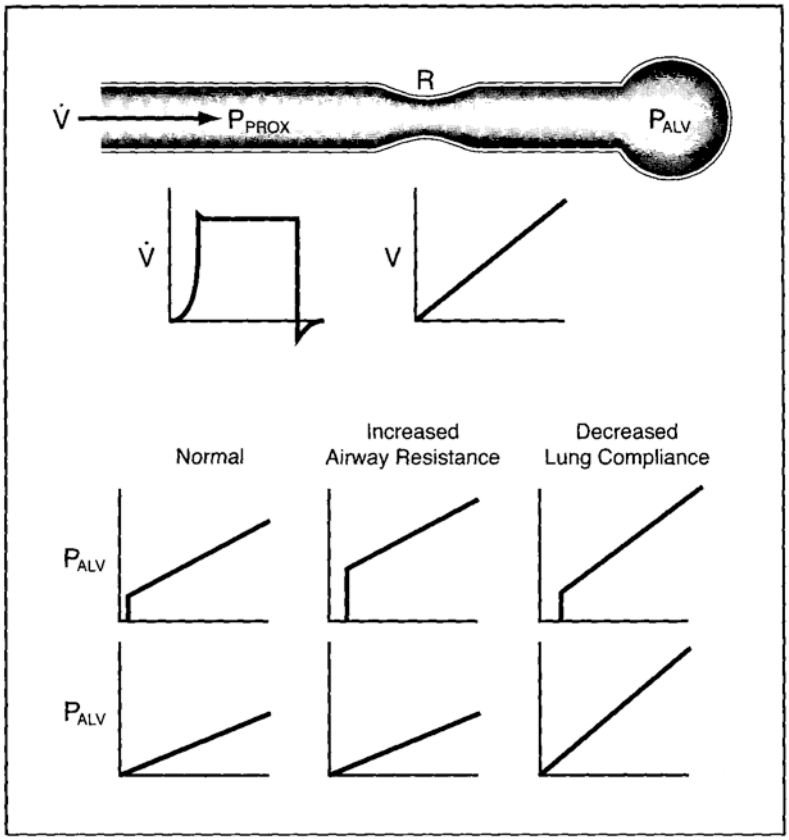

FIGURE 24.1 Waveforms produced by constant-flow, volume-cycled ventilation. $\dot{V}$ = inspiratory flow rate, V = inspiratory volume, R = flow resistance in the airways, P_{prox} = proximal airway pressure, P_{ALV} = alveolar pressure.

Inflation Pressures

The waveforms produced by volume-cycled ventilation are shown in Figure 24.1. The lungs are inflated at a constant flow rate, and this produces a steady increase in lung volume. The pressure in the proximal airways (P ro) shows an abrupt initial rise, followed by a more gradual rise through the remainder of lung inflation. However, the pressure in the alveoli (P_{ALV}) shows only a gradual rise during lung inflation.

The early, abrupt rise in proximal airway pressure is a reflection of flow resistance in the airways. An increase in airways resistance magnifies the initial rise in proximal airway pressure, while the alveolar pressure at the end of lung inflation remains unchanged. Thus, when resistance in the airways increases, higher inflation pressures are needed to deliver the inflation volume, but the alveoli are not exposed to the higher inflation pressures. This is not the case when the distensibility (compliance)

of the lungs is reduced. In this latter condition, there is an increase in both the proximal airways pressure and the alveolar pressure. Thus, when lung distensibility (compliance) decreases, the higher inflation
. pressures needed to deliver the inflation volume are transmitted to the alveoli. The increase in alveolar pressure in noncompliant lungs can lead to pressure-induced lung injury (see later in the chapter).

CARDIAC PERFORMANCE

The influence of positive-pressure ventilation on cardiac performance is complex, and involves changes in preload and after load for both the right and left sides of the heart (4). To describe these changes, it is important to review the influence of intrathoracic pressure on transmural pressure, which is the pressure that determines ventricular filling (preload) and the resistance to ventricular emptying (afterload).

Transmural Pressure

The transmission of intrathoracic pressure into the lumen of intrathoracic blood vessels is described briefly in Chapter 10 (see Fig. 10.1). The influence of lung mechanics on this pressure transmission is illustrated in Figure 24.2. The panel on the left shows what happens when a normal lung is inflated with 700 mL from a positive-pressure source. In this situation, the increase in alveolar pressure is completely transmitted into the pulmonary capillaries, and there is no change in transmural pressure (P_{tm}) across the capillaries. However, when the same lung inflation

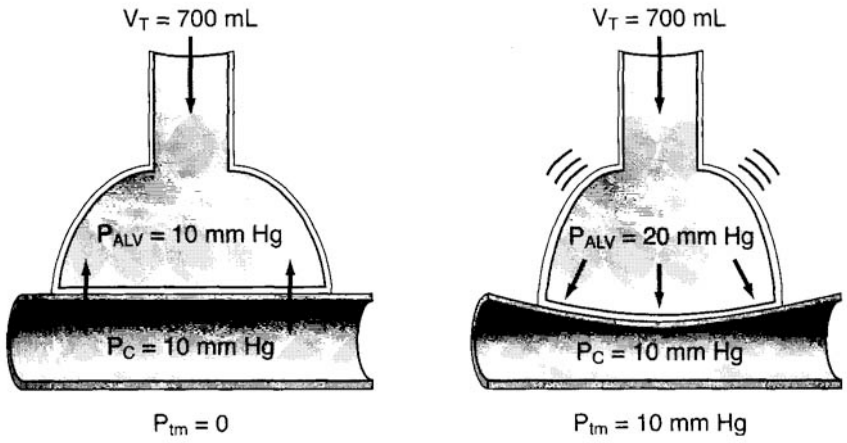

FIGURE 24.2 Alveolar–capillary units showing the transmission of alveolar pressure (P_{ALV}) to the pulmonary capillaries in normal and noncompliant (stiff) lungs. P_c = capillary hydrostatic pressure, P_{tm} = transmural pressure across the capillary wall, V_T = tidal volume delivered by the ventilator.

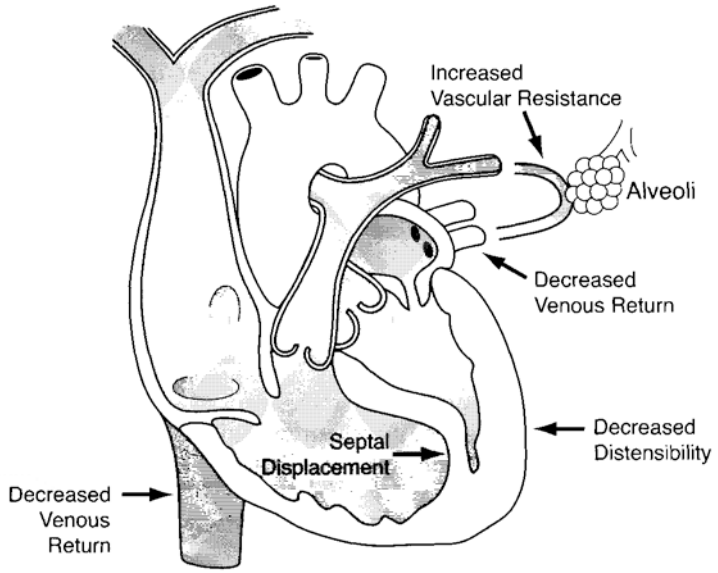

FIGURE 24.3 The mechanisms whereby positive-pressure ventilation can decrease ventricular filling (preload).

occurs in lungs that are not easily distended (panel on the right), the increase in alveolar pressure is not completely transmitted into the capillaries and the transmural pressure increases. This increase in transmural pressure acts to compress the capillaries. Therefore, in conditions associated with a decrease in lung compliance (e.g., pulmonary edema, pneumonia), positive-pressure lung inflation tends to compress the heart and intrathoracic blood vessels (5-7). This compression can be beneficial or detrimental, as described below.

Preload

Positive-pressure lung inflation can reduce ventricular filling in several ways, as indicated in Figure 24.3. First, positive intrathoracic pressure decreases the pressure gradient for venous inflow into the thorax (although positive-pressure lung inflations also increase intra-abdominal pressure, and this tends to maintain venous inflow into the thorax). Second, positive pressure exerted on the outer surface of the heart reduces cardiac distensibility, and this can reduce ventricular filling during diastole. Finally, compression of pulmonary blood vessels can raise pulmonary vascular resistance, and this can impede right ventricular stroke output. In this situation, the right ventricle dilates and pushes the interventricular septum toward the left ventricle, and this reduces left ventricular chamber size and left ventricular filling. This phenomenon, known as *ventricular interdependence,* is one of the mechanisms whereby right heart failure can impair the performance of the left side of the heart (see Fig. 14.4).

Afterload

Whereas compression of the heart from positive intrathoracic pressure impedes ventricular filling during diastole, this same compression *facilitates* ventricular emptying during systole. This latter effect is easy to visualize (like a hand squeezing the ventricles during systole) and can also be explained in terms of ventricular afterload. That is, ventricular afterload, or the impedance to ventricular emptying, is a function of the peak systolic *transmural* wall pressure (see Fig. 1.4). Incomplete transmission of positive intrathoracic pressure into the ventricular chambers will decrease the transmural pressure across the ventricles during systole, and this decreases ventricular afterload.

Cardiac Output

Positive-pressure lung inflation tends to reduce ventricular filling during diastole but enhances ventricular emptying during systole. The overall effect of positive-pressure ventilation on cardiac output depends on whether the effect on preload or afterload predominates. When intravascular volume is normal and intrathoracic pressures are not excessive, the effect on afterload reduction predominates, and positive-pressure ventilation increases cardiac stroke output. The increase in stroke volume causes an increase in systolic blood pressure during lung inflation; a phenomenon known as *reverse pulsus paradoxus.* This is demonstrated in Figure 1.5. The favorable influence of positive intrathoracic pressure on cardiac output is one mechanism that could explain the ability of chest compressions to increase cardiac output during cardiac arrest (see Chapter 15).

The beneficial actions of positive-pressure ventilation on cardiac output are reversed by hypovolemia. When intravascular volume is reduced, the predominant effect of positive intrathoracic pressure is to reduce ventricular preload and, in this setting, positive-pressure ventilation decreases cardiac stroke output. This emphasizes the importance of avoiding hypovolemia in the management of ventilator-dependent patients.

INDICATIONS FOR MECHANICAL VENTILATION

The decision to intubate and initiate mechanical ventilation has always seemed more complicated than it should be. Instead of presenting.the usual list of clinical and physiologic indications for mechanical ventilation, the following simple rules should suffice.

RULE 1. The indication for intubation and mechanical ventilation is thinking of it. There is a tendency to delay intubation and mechanical ventilation as long as possible in the hopes that it will be unnecessary. However, elective intubation carries far fewer dangers than emergency intubation, and thus delays in intubation create unnecessary dangers for the patient. If the patient's condition is severe enough for intubation and mechanical ventilation to be considered, then proceed without delay.

RULE 2. Intubation is not an act of personal weakness. Housestaff tend to apologize on morning rounds when they have intubated a patient during the evening, almost as though the intubation was an act of weakness on their part. Quite the contrary, intubation carries the strength of conviction, and no one will be faulted for gaining control of the airways in an unstable patient.

RULE 3. Initiating mechanical ventilation is not the "kiss of death," The perception that "once on a ventilator, always on a ventilator" is a fallacy that should never influence the decision to initiate mechanical ventilation. Being on a ventilator does not create ventilator dependence; having a severe cardiopulmonary or neuromuscular diseases does.

A NEW STRATEGY FOR MECHANICAL VENTILATION

In the early days of positive-pressure mechanical ventilation, large inflation volumes were recommended to prevent alveolar collapse (8). Thus, whereas the tidal volume during spontaneous breathing is normally 5 to 7 mLIkg (ideal body weight), the standard inflation volumes during volume-cycled ventilation have been twice as large, or 10 to 15 mL/kg. This volume discrepancy is even greater with the addition of mechanical *sighs,* which are 1.5 to two times greater than standard inflation volumes (or 15 to 30 mL/kg) and are delivered 6 to 12 times per hour.

The large inflation volumes used in conventional mechanical ventilation can damage the lungs (3), and can even promote injury in distant organs through the release of inflammatory cytokines (9). The discovery of *ventilator induced lung injury* is drastically changing the way that mechanical ventilation is delivered. This topic has been described in Chapter 22, and will be briefly reviewed here.

Ventilator-Induced Lung Injury

In lung diseases that most often require mechanical ventilation (e.g., acute respiratory distress syndrome [ARDS], pneumonia), the pathologic changes are not uniformly distributed throughout the lungs. This is even the case for pulmonary conditions like ARDS that appear to be distributed homogeneously throughout the lungs on the chest x-ray (see Fig. 22.4) (O). Because inflation volumes are distributed preferentially to regions of normal lung function, inflation volumes tend to overdistend the normal regions of diseased lungs. This tendency to overdistend normal lung regions is exaggerated when large inflation volumes are used.

The hyperinflation of normal lung regions during mechanical ventilation can produce stress fractures at the alveolar-capillary interface (11,12). An example of such a fracture is shown in Figure 24.3 (2). The electron micrographs in this figure are from the lungs of a patient with ARDS who required excessively high ventilatory pressures to maintain adequate arterial oxygenation. These fractures may be the result of excessive alveolar pressures *(barotrauma)* or excessive alveolar volumes *(volutraw/la)* (3). Alveolar rupture like that in Figure 24.4 can have three adverse consequences. The first is accumulation of alveolar gas in

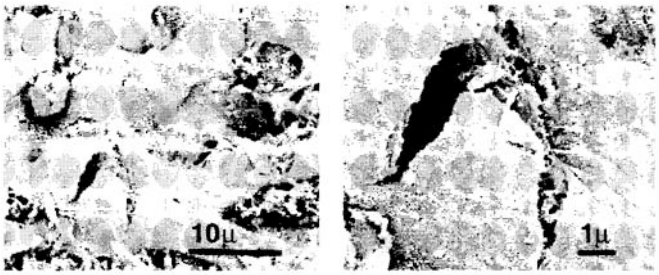

FIGURE 24.4 Electron micrograph showing a tear at the alveolar-capillary interface in a post-mortem specimen from a patient with ARDS. Scales shown in lower right corner of each image. (From Hotchkiss JR, Simonson DA, Marek DJ, et al. Pulmonary microvascular fracture in a patient with acute respiratory distress syndrome. Crit Care Med 2002; 30:2368–2370.) Image digitally retouched.

the pulmonary parenchyma (pulmonary interstitial emphysema), mediastinum (pneumomediastinum), or pleural cavity (pneumothorax). The second adverse consequence is a condition of inflammatory lung injury that is indistinguishable from ARDS (4). The third and possibly worst consequence is multiorgan injury from release of inflammatory mediators into the bloodstream (9). This latter process is known as *biotrauma.*

Lung-Protective Ventilation

The risk of lung injury with large inflation volumes has prompted clinical studies evaluating lower tidal volumes for positive-pressure ventilation. The largest study to date included over 800 patients with ARDS (5), and compared ventilation with tidal volumes of 6 mL/kg and 12 mL/kg using *predicted body weight* (which is the weight at which lung volumes are normal). Ventilation with low tidal volumes was associated with a 9% (absolute) reduction in mortality when the end-inspiratory "plateau pressure" was <30 cm H_2O (this pressure is described later in the chapter).

Low volume or *lllng protective ventilation* is now recommended for all patients with ARDS, but there is evidence that ventilator-induced lung injury also occurs in conditions other than ARDS (6). Therefore, lung protective ventilation with low tidal volumes is considered a beneficial strategy for all patients with acute respiratory failure. A protocol for low volume ventilation is shown in Table 24.1. This protocol is adapted from the protocol for lung protective ventilation in ARDS, which is shown in Table 22.4. This strategy is designed to achieve and maintain a tidal volume of 6 mLlkg (using the patient's predicted body weight).

Positive End-Expiratory Pressure

Low tidal volumes can result in airway collapse, particularly at the end of expiration. Repeated opening and closing of airways at the end of expiration can become a source of lung injury (possibly by generating excessive shear forces that can damage the airways epithelium) (7).

TABLE 24.1 Protocol for Lung Protective Ventilation

1. Select assist-control mode and FI02 = 100%.

2. Set initial tidal volume (V_T) at 8 mL/kg using the patient's *predicted body weight* (PBW).

Males: PBW = 50 + [2.3 x (height in inches - 60)]

Females: PBW = 45.5 + [2.3 x (height in inches - 60)]

3. Select respiratory rate (RR) to achieve pre-ventilator minute ventilation, but do not exceed RR = 35/min.

4. Add positive end-expiratory pressure (PEEP) at 5 - 7 cm Hp.

5. Reduce V_T by 1 mUkg every 2 hours until V_T = 6 mUkg.

6. Adjust FI02 and PEEP to keep $Pa0_2$ > 55 mm Hg or $Sa0_2$ > 88%.

7. When V_T is down to 6 mL/kg, measure:

a) Plateau pressure (Ppl)

b) Arterial $PC0_2$ and pH

If Ppl > 30 cm HP or pH < 7.30, follow recommendations in Table 22.4.

Protocol from the ARDS Clinical Network website (www.ardsnet.org).

Airways collapse can be mitigated by adding positive end-expiratory pressure (PEEP). This pressure (which is described in the next chapter) acts as a stent to keep small airways open at the end of expiration.

Permissive Hypercapnia

Another consequence of low volume ventilation is a reduction in CO_2 elimination via the lungs, which can lead to hypercapnia and respiratory acidosis. Allowing hypercapnia to persist in favor of maintaining low volume ventilation is known as *permissive hypercapnia* (8). This is described in Chapter 22.

MONITORING LUNG MECHANICS

During spontaneous breathing, the mechanical properties of the lungs (i.e., elastic recoil of the lungs and resistance to flow in the airways) can be monitored with pulmonary function tests. However, these tests are not easily performed during mechanical ventilation. In this setting, the proximal airways pressures can be used to assess pulmonary function 0,19,20).

Proximal Airway Pressures

Positive-pressure mechanical ventilators have a pressure gauge that monitors the proximal airway pressure during each respiratory cycle. The components of this pressure are illustrated in Figure 24.5.

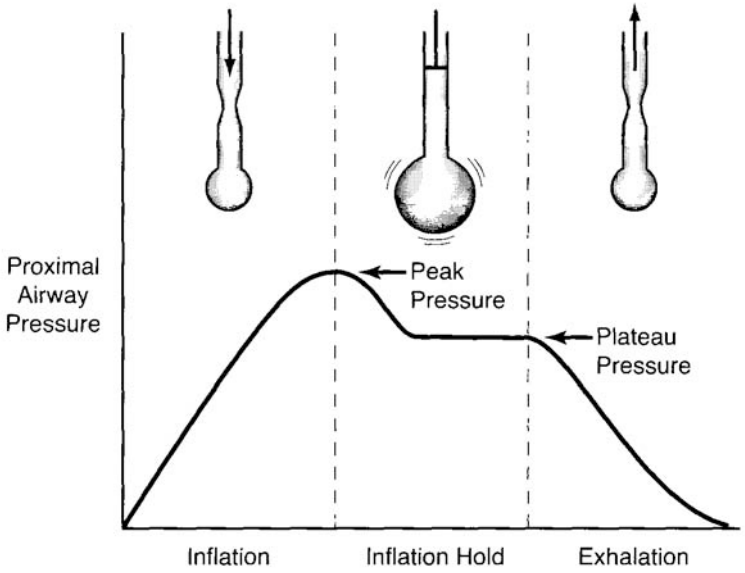

FIGURE 24.5 Proximal airway pressure at the end of a positive-pressure lung inflation (peak pressure) and during an inflation-hold maneuver (plateau pressure), which is performed by occluding the expiratory limb of the ventilator circuit to prevent lung deflation. These pressures can be used to evaluate the mechanical properties of the lungs (see Fig. 24.6).

End-Inspiratory Peak Pressure

The peak pressure at the end of inspiration (P eak) is a function of the inflation volume, the flow resistance in the airw~ys, and the elastic recoil force of the lungs and chest wall. At a constant inflation volume, the peak pressure varies directly with changes in airflow resistance and elastic recoil force (elastance) of the lungs and chest wall

P peak = (Resistance + Elastance) (24.1)

Therefore, when the inflation volume is constant, an increase in peak inspiratory pressure indicates an increase in airway resistance or an increase in the elastic recoil force of the lungs (or both).

End-Inspiratory Plateau Pressure

The contribution of resistance and elastance to the peak inspiratory pressure can be distinguished by occluding the expiratory tubing at the end of inspiration, as shown in Figure 24.5. When the inflation volume is held in the lungs, the proximal airway pressure decreases initially and then reaches a steady level, which is called the end-inspiratory *plateau pressure.* Because no airflow is present when the plateau pressure is created, the pressure is not a function of flow resistance in the airways. Instead,

the plateau pressure (P plateau) is directly related to the elastic recoil force (elastance) of the lungs and chest wall.

P plateau = Elastance

Therefore, the difference between end-inspiratory peak and plateau pressures is proportional to the flow resistance in the airways.

(P peak - P plateau) =Airways Resistance (24.3)

Practical Applications

A common scenario in any ICU is a ventilator-dependent patient who develops a sudden deterioration in cardiopulmonary status (this could include hypotension, hypoxemia, or respiratory distress). The flow diagram in Figure 24.6 demonstrates how the proximal airways pressures can be used to quickly evaluate these patients.

If the peak pressure is increased but the plateau pressure is unchanged, the problem is an increase in airways resistance. In this situation, the major concerns are obstruction of the tracheal tube, airway obstruction from secretions, and acute bronchospasm. Therefore, airways suctioning is indicated to clear secretions, followed by an aerosolized bronchodilator treatment if necessary.

If the peak and plateau pressures are both increased, the problem is a decrease in distensibility of the lungs and chest wall. In this situation, the major concerns are pneumothorax, lobar atelectasis, acute pulmonary edema, and worsening pneumonia or ARDS. Active contraction of the chest wall and increased abdominal pressure can also decrease the distensibility of the thorax. Finally, a patient with obstructive lung disease who becomes tachypneic can develop auto-PEEP, and this increases the peak and plateau pressures as well (see Chapter 26).

If the peak pressure is decreased, the problem may be an air leak in the system (e.g., tubing disconnection, cuff leak). In this situation, the lungs should be manually inflated while listening for a cuff leak. A decrease in peak pressure can also be due to hyperventilation, when the patient is generating enough of a negative intrathoracic pressure to "pull" air into the lungs.

If no change in peak pressure occurs, it does not necessarily mean that there has been no change in lung mechanics. The sensitivity of the proximal airways pressures in detecting changes in lung mechanics is unknown. When the pressures do not change, the evaluation should proceed as it would without the aid of proximal airway pressures.

Bronchodilator Responsiveness

Ventilator-dependent patients often receive aerosolized bronchodilator treatments routinely, without documented need or benefit. The proximal

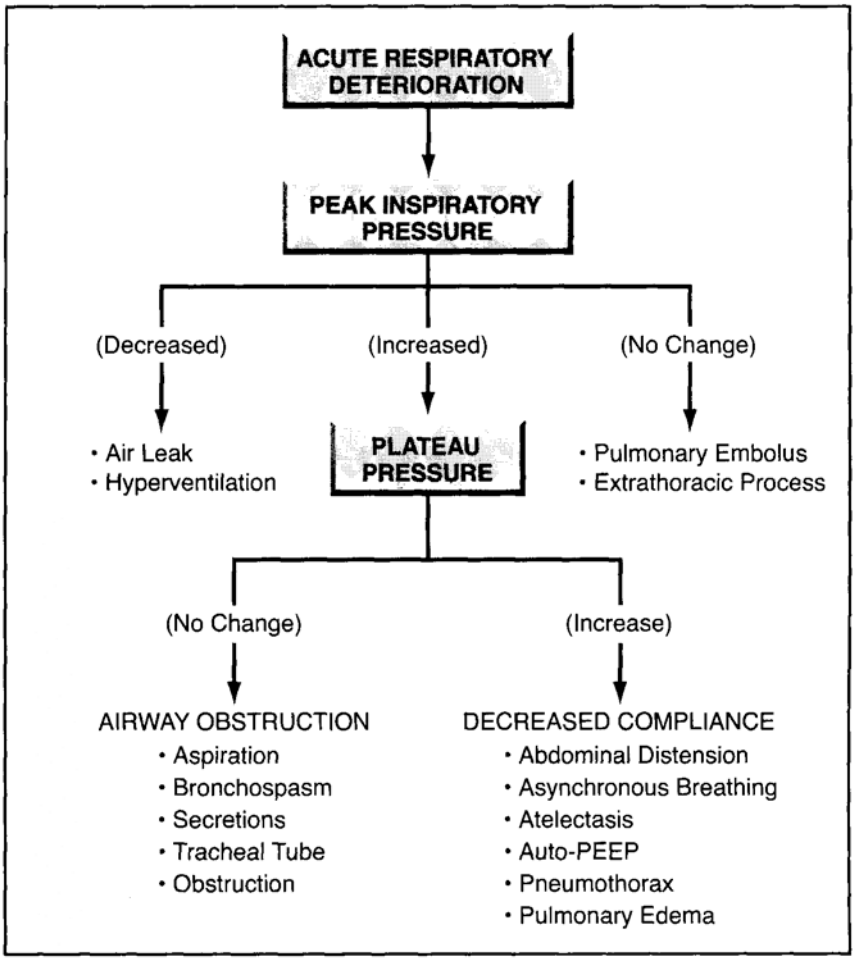

FIGURE 24.6 The use of proximal airway pressures to evaluate a patient with acute respiratory decompensation.

airway pressures can be used to evaluate bronchodilator responsiveness during mechanical ventilation. Bronchodilation represents a decrease in airways resistance, so a positive bronchodilator response should be accompanied by a decrease in the peak inspiratory pressure (assuming lung elastance stays the same). If an aerosol bronchodilator treatment is not accompanied by a decrease in peak inspiratory pressure, then there is little justification for continuing the aerosol treatments.

Thoracic Compliance

Compliance (distensibility) is the reciprocal of elastance. The compliance of the lungs and chest wall (called thoracic compliance) can be

determined quantitatively using the ratio of the tidal volume (V_T) to the elastic recoil pressure (i.e., plateau pressure). Since the plateau pressure is measured in the absence of airflow, the resulting compliance is called a "static" compliance. Thus, the static compliance (C stat) of the thorax is derived as:

$$C\ stat = V / P\ plateau \qquad (244)$$

.

For a tidal volume (V_T) of 500 mL and a plateau pressure (P plateau) of 10 cm H20, the static compliance (Cstat) of the thorax is:

Cstat = 0.5 L/ l0 cm H20
= 0.05 L / cm H20

In intubated patients with no known lung disease, the static thoracic compliance is 0.05 to 0.08 L/ cm H20 (or 50 to 80 mL/ cm H20) (9). In patients with stiff lungs, the thoracic compliance is much lower at 0.01 to 0.02 L/ cm H20 (21). Thus, the compliance determination provides an objective measure of the severity of illness in pulmonary disorders associated with a change in lung compliance (e.g., pulmonary edema and ARDS).

Considerations

The following factors influence the static compliance measurement.

PEEP increases the plateau pressure. Therefore, the level of PEEP (either externally applied or auto-PEEP) should be subtracted from the plateau pressure for the compliance determination.

The connector tubing between the ventilator and patient expands during positive-pressure lung inflations, and the volume lost in this expansion reduces the inflation volume reaching the patient. The volume lost is a function of the peak inflation pressure and the inherent compliance of the tubing. The usual compliance of connector tubing is 3 mL/ cm H20, which means that 3 mL of volume is lost for every 1 cm H20) increase in inflation pressure. Thus, if the inflation volume from the ventilator is 700 mL and the peak inspiratory pressure is 40 cm H20, then (3 x 40) 120 mL will be lost to expansion of the ventilator tubing and the inflation volume reaching the patient will be (700 - 120) 580 mL. Because of this discrepancy, the predetermined volume setting on the ventilator should not be used as the inflation volume for the compliance calculation. Instead, the exhaled volume, which is usually displayed digitally on the ventilator panel, should be used.

Because the proximal airways pressures are transthoracic pressures (i.e., measured relative to atmospheric pressure) and not transpulmonary pressures (i.e., measured relative to intrapleural pressure), the compliance measurement includes the chest

wall as well as the lungs. Because contraction of the chest wall muscles can reduce the compliance (distensibility) of the chest wall, the compliance determination should be performed only during passive ventilation. However during passive ventilation, the chest wall can account for 35% of the total thoracic compliance (20,21).

Airway Resistance

The resistance to airflow during inspiration (Rinsp) can be determined as the ratio of the pressure gradient needed to overtome airways resistance (P peak – P plateau) and the inspiratory flow rate (V Insp):

R insp = (P peak – P plateau) / V insp

This resistance represents the summed resistances of the connector tubing, the tracheal tube, and the airways. Because the resistance of the connector tubing and tracheal tube should remain constant (assuming the tracheal tube is clear of secretions), changes in R insp should represent
changes in airways 'resistance.
A sample calculation of inspiratory resistance is shown below using an inspiratory flow rate of 60 L/min (1.0 L/sec), a peak pressure of 20 cm H_20, and a plateau pressure of 10 cm H_20.
R. insp = (20 - 10) cm H20/ L/sec

= 10 cm H20 /L/ sec (24.7)

The minimal flow resistance in large-bore endotracheal tubes is 3 to 7 cm H_20/L/sec (22), so nonpulmonary resistive elements can contribute a considerable fraction of the total inspiratory resistance.

Limitations

The major limitations of the inspiratory resistance measurement are the contributions of resistive elements not in the lung and the relative insensitivity of the resistance measured during inspiration. Airflow obstruction is usually measured during expiration, when the airways have the greatest tendency to collapse. The distending pressures delivered by the ventilator during lung inflation keep the airways open, and thus the resistance to flow during inspiration does not measure the tendency for the airways to collapse during expiration (20).

A FINAL WORD

After mechanical ventilation was introduced into clinical practice, there was a flurry of interest in developing newer modes of ventilation that would benefit patients with respiratory failure. This type of "more is better" approach was based on a perception that mechanical ventilation is a

type of therapy for patients with respiratory failure (one example of this perception are the claims that positive end-expiratory pressure can drive water out of the lungs in patients with pulmonary edema). However, there is nothing therapeutic about mechanical ventilation. In fact, the most significant discovery about mechanical ventilation since it was first introduced is the fact that it damages the lungs (and indirectly damages other organs as well).
Mechanical ventilation is a technique that opposes the normal physiology of ventilation (by creating positive pressure instead of negative pressure to ventilate the lungs) and, in this sense, it is not surprising that it is problematic. The current trend of using lower tidal volumes during mechanical ventilation is a step in the right direction because a "lesser is better" strategy is the only one that makes sense with a technique that is so unphysiological.

REFERENCES

Chapter 25

MODES OF ASSISTED VENTILATION

Development in most fields,!! medicine appears to occur according to sound scientific principles. However, exceptions can *be found, and the development,!! mechanical ventilatory support is one of them.*
J. Rasanen

In the half century since positive-pressure ventilation first appeared, at least 15 different modes of positive-pressure ventilation have been introduced, each with claims that it will improve gas exchange, prevent complications, or reduce the work of breathing during mechanical ventilation (1-3). Most of these ventilatory modes have failed to make mechanical ventilation more effective, safer, or more tolerable for patients. What they have done instead is make positive-pressure ventilation more complicated than it needs to be.

ASSIST-CONTROL VENTILATION
As described in the last chapter, the most popular method of positive-pressure lung inflation involves the use of a constant inflation volume instead of a constant inflation pressure. This method, which is called *voll/me-cycled ventilation,* allows the patient to initiate or "trigger" each mechanical breath (assisted ventilation) but can also deliver a preset level of minute ventilation if the patient is unable to trigger the ventilator (controlled ventilation). This combination is called *assist-control ventilation.*

Ventilatory Pattern
The upper panel in Figure 25.1 shows the changes in airway pressure produced by assist-control ventilation (ACV). The tracing begins with a

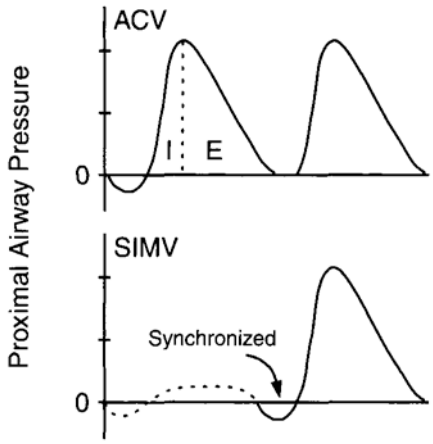

FIGURE 25.1 Airway pressure patterns in assist-control ventilation (*ACV*) and synchronized intermittent mandatory ventilation (*SIMV*). Spontaneous breaths are indicated by dashed lines, and machine breaths are indicated by solid lines. The mechanical breath in the upper panel indicates the period of lung inflation (*I*) and deflation (*E*).

negative-pressure deflection, which is the result of a spontaneous inspiratory effort by the patient. When the negative pressure reaches a certain level (which is usually set at -2 to -3 cm H_2O), a pressure-activated valve in the ventilator opens, and a positive-pressure breath is delivered to the patient. The second machine breath in the tracing is identical to the first, but it is not preceded by a spontaneous ventilatory effort. The first breath is an example of *assisted ventilation,* and the second breath is an example of *controlled ventilation.*

Respiratory Cycle Timing

As mentioned in Chapter 24, volume-cycled ventilation has traditionally employed large inflation volumes (10 to 15 mL/kg or about twice the normal tidal volume during spontaneous breathing). To allow patients sufficient time to passively exhale these large volumes, the time allowed for exhalation should be at least twice the time allowed for lung inflation (see Fig. 25.1) The ratio of inspiratory time to expiratory time, which is called the I:E ratio, should then be maintained at 1:2 or higher 0). This is accomplished by using an inspiratory flow rate that is at least twice the expiratory flow rate. At a normal respiratory rate, an inspiratory flow rate of 60 L/min will inflate the lungs quickly enough to allow the time needed to exhale the inflation volume. However, when a patient has obstructive lung disease and can't exhale quickly, the I:E ratio can fall below 1:2, and an increase in inspiratory flow rate may be needed to achieve the appropriateI:E ratio.

Work of Breathing

Acute respiratory failure is often accompanied by a marked increase in the work of breathing, and patients who are working hard to breathe are often placed on mechanical ventilation to rest the respiratory muscles and reduce the work of breathing. However, the assumption that the diaphragm rests during mechanical ventilation is incorrect because the diaphragm is an involuntary muscle that never rests. The contraction of the diaphragm is dictated by the activity of respiratory neurons in the lower brainstem, and these cells fire automatically and are not silenced by mechanical ventilation. Only death can silence the brainstem respiratory centers, and the diaphragm follows suit. This means that the diaphragm does not relax when the ventilator is triggered and delivers the mechanical breath, but it continues to contract throughout inspiration. Because of the continued contraction of the diaphragm, mechanical ventilation may have little impact on the work of breathing (4).

Ventilatory Drive

The activity of the diaphragm is largely dictated by the output from the brainstem respiratory neurons, and this output, which is often referred to as the *ventilatory drive,* is increased as much as three to four times above normal in acute respiratory failure (mechanism unknown) 0,5). Reducing ventilatory drive is the appropriate measure for decreasing the workload of the respiratory muscles. Promoting patient comfort with sedation might help in this regard (6). Altering the mechanism for triggering the ventilator can also help, as described next.

The Trigger Mechanism

The traditional method of assisted ventilation uses a decrease in airways pressure generated by the patient to open a pressure-sensitive valve and initiate the ventilator breath. The threshold pressure is usually set at a low level of -1 to -3 cm H20. Although this does not seem excessive, many ventilator-dependent patients have positive end-expiratory pressure (PEEP), and this adds to the pressure that must be generated to trigger the ventilator. For example, if a patient has +5 cm H20 of PEEP and the trigger pressure is -2 cm H20, a pressure of 7 cm H20 must be generated to trigger a ventilator breath. This may not seem like much, but the diaphragm generates only 2 to 3 cm H20 during quiet breathing in healthy adults, so generating a pressure of 7 cm H20 will require more than twice the normal effort of the diaphragm. The increased effort needed to reach the trigger pressure would explain the observation that about one-third of inspiratory efforts in ventilator-dependent patients fail to trigger the ventilator (7). Inspiratory flow offers advantages over inspiratory pressure as a trigger mechanism because flow triggering (usually at 2 L/min) does not require a decrease in airway pressure, and therefore should require less effort. Studies comparing flow and pressure triggering have been

inconsistent except for patients with chronic obstructive pulmonary disease (COPD), where the work of breathing is less with flow triggering (8). Some ventilators provide flow-triggering as an option (e.g., Bird 8400 ST) but don't expect much.

Disadvantages

Based on an unfounded fear that mechanical ventilation will be accompanied by progressive atelectasis, large tidal volumes have been employed for volume-cycled ventilation. These volumes are about twice the normal tidal volumes in adults 02 to 15 mL/kg vs. 6 to 8 mL/kg, respectively). This practice has changed in recent years, and the preferred tidal volumes for mechanical ventilation have been cut in half to the range of 6 to 8 mL/kg. The conditions that prompted this change are described next.

Ventilator-Induced Lung Injury

The topic of ventilator-induced lung injury (VILI) is described in Chapters 22 and 24, and is only briefly mentioned here. High inflation volumes overdistend alveoli and promote alveolar rupture (see Figure 24.4). This process is known as *volume trauma,* and it incites an inflammatory response in the lungs that can produce a condition of inflammatory lung injury similar to the acute respiratory distress syndrome (ARDS) (9). Inflammatory mediators in the lungs can be released into the systemic circulation and this can lead to inflammatory injury in distant organs (10). This condition of multiorgan injury from local injury in the lungs is known as *biotrauma.*

The discovery of volutrauma led to studies comparing ventilation with conventional tidal volumes (12 to 15 mL/kg) and reduced tidal volumes (6 mL/kg). Some of these studies showed improved outcomes associated with the low-volume *lung protective strategy* (11). As a result, the recommended inflation volumes for volume-cycled ventilation have been cut in half to 6 to 8 mL/kg (see Tables 22.4 and 24.1).

VILI has been described almost exclusively in patients with ARDS, but there is evidence that this condition can occur in any patient with underlying pulmonary disease (2). The recommendation for lowvolume ventilation is thus being applied to all ventilator-dependent patients.

Auto-PEEP

Assist-control ventilation can be problematic for patients who are breathing rapidly or have reduced expiratory airflow because there may not be enough time to exhale the large tidal volumes. The air that remains in the alveoli at the end-of expiration creates a positive end-expiratory pressure (PEEP) that is known as *auto-PEEP.* This pressure can impair cardiac output and can also increase this risk of pulmonary barotrauma (pressure-induced injury). The lower tidal volumes that are now being adopted for volume-cycled ventilation will reduce the risk of auto-PEEp, but will not eliminate this problem. (The deleterious effects of PEEP

are described later in the chapter, and the condition of auto-PEEP is described in Chapter 26.)

INTERMITTENT MANDATORY VENTILATION

The problem of incomplete emptying of the lungs with rapid breathing during assist-control ventilation led to the introduction of *intermittent mandatory ventilation* (IMV). This mode of ventilation was introduced in 1971 to ventilate neonates with respiratory distress syndrome, who typically have respiratory rates in excess of 40 breaths/minute. IMV is designed to provide only partial ventilatory support: it combines periods of assist-control ventilation with periods where patients are allowed to breathe spontaneously. The periods of spontaneous breathing help to prevent progressive lung hyperinflation and auto-PEEP in patients who breathe rapidly, and was also intended to prevent respiratory muscle atrophy from prolonged periods of mechanical ventilation.

Ventilatory Pattern

The lower portion of Figure 25.1 shows the pattern of ventilation associated with IMV. The initial negative and positive deflection in the tracing represents a spontaneous breath (dashed line). The second spontaneous breath triggers an assisted ventilator breath (solid line). Thus, IMV combines assisted ventilation with spontaneous breathing. The ventilatory pattern in Figure 25.1 is called *synchronized* IMV because the assisted breaths are synchronized to coincide with spontaneous inspiratory efforts. When IMY is first set up for a patient, the rate of assisted lung inflations is usually set at 10 breaths/minute. This rate can then be adjusted upward or downward as needed until the patient is breathing comfortably and gas exchange is adequate. The period of spontaneous breathing is poorly tolerated by patients, and it is now a common practice to use pressure-support ventilation (described later in the chapter) at 10 cm H_2O during the spontaneous breathing periods. The addition of pressuresupported breathing has been shown to decrease the work of breathing during IMV (7), and this has improved patient tolerance. However, when IMV is employing both volume-cycled ventilation and pressuresupported ventilation, it is no longer providing partial ventilatory support, and is functioning more like assist-control ventilation than IMV.

Disadvantages

The principal disadvantages of IMV are an increased work of breathing and a decreased cardiac output.

Work of Breathing

The spontaneous breathing during IMV takes place through a high-resistance circuit (the tracheal tube and ventilator tubing) and this can

increase the inspiratory work of breathing. The addition of pressure-supported spontaneous breathing has been effective in minimizing this problem, as described above (13). The increased work of breathing attributed to pressure-based triggering, which was described previously for assist-control ventilation, can also be a problem with IMV, and the use of inspiratory airflow to trigger the ventilator breaths has been shown to reduce the work of breathing during IMV (7).

Cardiac Output

As described in Chapter 24, positive-pressure ventilation can reduce cardiac output by reducing ventricular preload, or can increase cardiac output by reducing ventricular afterload. In patients with a normal intravascular volume, the afterload effect predominates, and positivepressure ventilation usually augments cardiac output (14). This effect is particularly evident in patients with left ventricular dysfunction, where positive-pressure ventilation can serve as a ventricular-assist device 05). IMV has the opposite effect, and decreases cardiac output in patients with left-ventricular dysfunction 05).

Summary

In summary, IMV provides few benefits over assist-control ventilation. The principal benefit of IMV is for the patient who is breathing rapidly because volume-cycled (assist-control) ventilation for these patients creates a risk for hyperinflation, auto-PEEP, and volutrauma. The risk of ventilator-induced lung injury may be lower with IMV than assist-control because less time is devoted to volume-cycled ventilation with IMV. This, however, is unproven. Finally, IMV should be avoided in patients with respiratory muscle weakness or left-ventricular dysfunction.

PRESSURE-CONTROLLED VENTILATION

Pressure-controlled ventilation (PCV) uses a constant pressure to inflate the lungs. This mode of ventilation was frowned upon because the inflation volumes can be variable (this is explained later). However, there has been a resurgence of interest in PCV because the risk for ventilatorinduced lung injury may be lower with PC V, as explained in the next section.
The pattern of ventilation associated with PCV is shown in Figure 25.2. Ventilation with PCV is completely controlled by the ventilator, with no participation by the patient (similar to the control mode in assist-control ven tilation).

Benefits and Risks

The perception that ventilator-induced lung injury may be less of a risk with PCV is based on the tendency for lower inflation volumes and lower airways pressures during PCv. However, there is no evidence to support

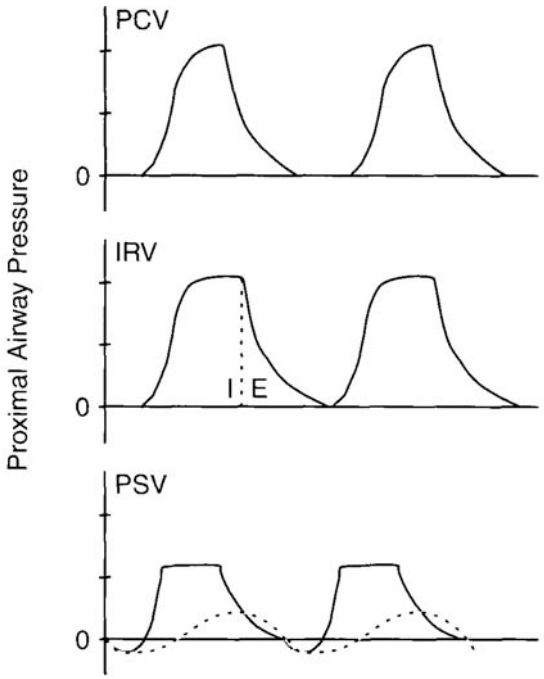

FIGURE 25.2 Airway pressure patterns in pressure-controlled ventilation (*PCV*), inverse ratio ventilation (*IRV*), and pressure-support ventilation (*PSV*). Spontaneous and machine breaths indicated by dashed and solid lines, respectively.

this claim. In fact, in the group of patients who are most likely to develop ventilator-induced lung injury (i.e., those with ARDS), PCV may not provide adequate ventilation.

The major advantage of PCV is the decelerating inspiratory flow pattern. In volume-cycled ventilation, the inspiratory flow rate is constant throughout lung inflation, whereas in pressure-controlled ventilation the inspiratory flow decreases exponentially during lung inflation (to keep the airway pressure at the pre-selected value). This decelerating flow pattern reduces peak airway pressures and can improve gas exchange 06-18).

The major disadvantage of PCV is the tendency for inflation volumes to vary with changes in the mechanical properties of the lungs. This is illustrated in Figure 25.3. The inflation volume increases as the peak inflation pressure increases (dashed lines). However, at a constant peak inflation pressure, the inflation volume decreases as the airway resistance increases or the lung compliance decreases. Therefore, any change in lung mechanics during PCV can lead to a change in inflation volumes. Because of the influence of lung mechanics on inflation volumes during PCV, this method of mechanical ventilation seems best suited for patients with neuromuscular disease (and normal lung mechanics).

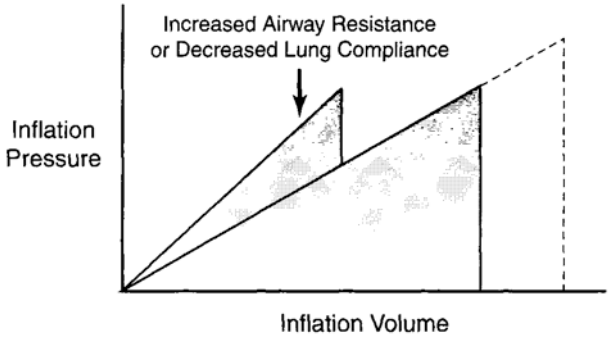

FIGURE 25.3 The determinants of lung inflation volume during pressure-cycled ventilation.

Inverse Ratio Ventilation

When PCV is combined with a prolonged inflation time, the result is *inverse ratio ventilation* (IRV) 09,20). The ventilatory pattern produced by IRV is shown in the middle panel of Figure 25.2. A decrease in inspiratory flow rate is used to prolong the time for lung inflation, and the usual I:E ratio of 1:2 is reversed to a ratio of 2:1. The prolonged inflation time can help prevent alveolar collapse. However, prolonged inflation times also increase the tendency for inadequate emptying of the lungs, which can lead to hyperinflation and auto-PEEP. The tendency to produce autoPEEP can lead to a decrease in cardiac output during IRV (21), and this is the major drawback with IRV. The major indication for IRV is for patients with ARDS who have refractory hypoxemia or hypercapnia during conventional modes of mechanical ventilation (22).

PRESSURE-SUPPORT VENTILATION

Pressure-augmented breathing that allows the patient to determine the inflation volume and respiratory cycle duration is called *pressure-support ventilation* (PSV) (23). This method of ventilation is used to augment spontaneous breathing, not to provide full ventilatory support.

Ventilatory Pattern

The ventilatory pattern produced by PSV is shown in the lower panel of Figure 25.2. At the onset of each spontaneous breath, the negative pressure generated by the patient opens a valve that delivers the inspired gas at a pre-selected pressure (usually 5 to 10 cm H_2O). The patient's inspiratory flow rate is adjusted by the ventilator as needed to keep the inflation pressure constant, and when the patient's inspiratory flow rate falls below 25% of the peak inspiratory flow, the augmented breath is terminated. By recognizing the patient's inherent inspiratory flow rate, PSV allows the patient to dictate the duration of lung inflation and the

inflation volume. This should result in a more physiologically acceptable method of positive-pressure lung inflation.

Clinical Uses

PSV can be used to augment inflation volumes during spontaneous breathing or to overcome the resistance of breathing through ventilator circuits. The latter application is the most popular and is used to limit the work of breathing during weaning from mechanical ventilation. The goal of PSV in this setting is not to augment the tidal volume, but merely to provide enough pressure to overcome the resistance created by the tracheal tubes and ventilator tubing. Inflation pressures of 5 to 10 cm H_20 are appropriate for this purpose. PSV has also become popular as a noninvasive method of mechanical ventilation (24,25). In this situation, PSV is delivered through specialized face masks or nasal masks, using inflation pressures of 20 cm H_20.

POSITIVE END-EXPIRATORY PRESSURE

Collapse of distal airspaces at the end of expiration is a common occurrence in ventilator-dependent patients, and the resulting atelectasis impairs gas exchange and adds to the severity of the respiratory failure. The driving force for this atelectasis is a decreased lung compliance, which is a consequence of the pulmonary disorders that are common in ventilator-dependent patients (i.e., ARDS and pneumonia). To counterbalance the tendency for alveolar collapse at the end of expiration, a positive pressure is created in the airways at end-expiration. This *positive end-expiratory pressure (PEEP)* has become a standard measure in the management of ventilator-dependent patients.

PEEP is created by placing a pressure-relief valve in the expiratory limb of the ventilator circuit, and setting the threshold pressure on the valve to correspond to the desired PEEP. The valve allows exhalation to proceed until the expiratory pressure decreases to the pre-selected pressure on the valve. At this point, airflow will cease (because there is no longer a pressure gradient across the valve) and, in the absence of airflow, the pressure at the valve is equivalent to alveolar pressure, which is the PEEP.

Airway Pressure Profile

The pressure profile with PEEP is shown in Figure 25.4. Note that the entire pressure waveform is displaced upward, resulting in an increase in peak airway pressure and mean airway pressure. The relationship between PEEP, peak intrathoracic pressure, and mean intrathoracic pressure is summarized below.

The complications of PEEP are not directly related to the PEEP level, but are determined by the peak and mean airway pressures during ventilation with PEEP.

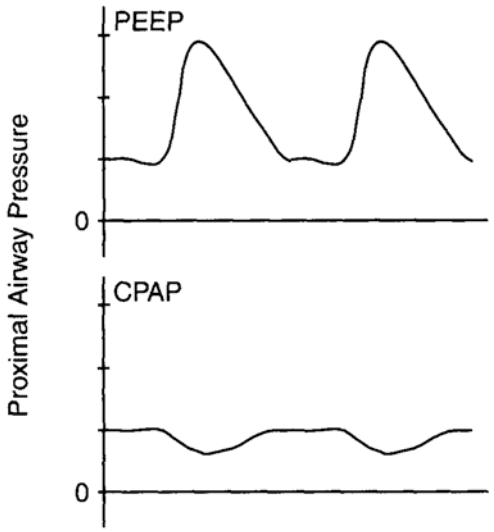

FIGURE 25.4 Airway pressure patterns in volume-cycled ventilation with positive end-expiratory pressure (*PEEP*), and in spontaneous breathing with continuous positive airway pressure (*CPAP*).

The peak airway pressure determines the risk of barotrauma (e.g., pneumothorax). The mean airway pressure determines the cardiac output response to PEEP. When airway pressures are used to evaluate lung mechanics (see Figs. 24.5 and 24.6), the PEEP level should be subtracted from the pressures.

Lung Recruitment

PEEP acts like a stent for the distal airspaces and counterbalances the compressive force generated by the elastic recoil of the lungs. In addition to preventing atelectasis, PEEP can also open collapsed alveoli and reverse atelectasis. This effect is demonstrated in Figure 25.5 (26). The thoracic CT image on the left shows consolidation in the posterior (dependent) regions of the both lungs, and this disappears after the application of PEEP. The PEEP has restored aeration in the area of atelectasis. This effect is known as *lung recruitment,* and it increases the available surface area in the lungs for gas exchange (26,27).

Recruitable Lung

The effect of PEEP shown in Figure 25.5 will result in improved gas exchange in the lungs. However, PEEP does not always have such a beneficial effect, and it can be harmful. In fact, PEEP can result in overdistention of normal lung regions, and this can injure the lungs in a manner similar to ventilator-induced lung injury. The important variable

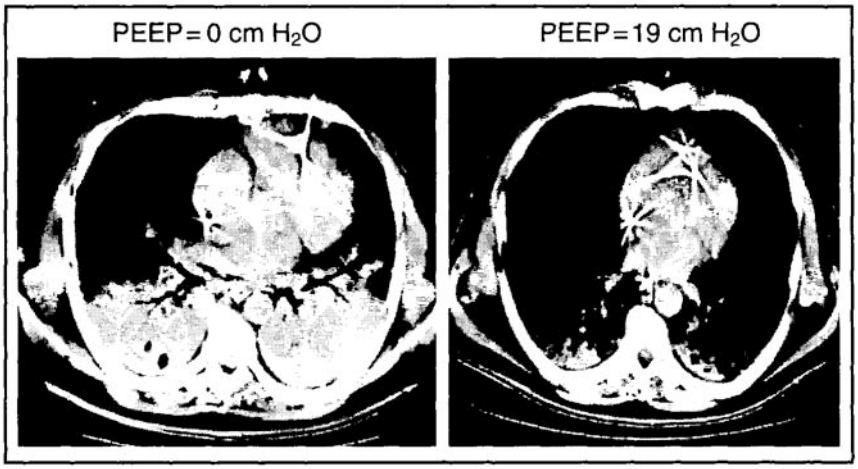

FIGURE 25.5 Thoracic CT images from a patient with ARDS showing resolution of atelectasis (recruitment) in response to PEEP. Images from Barbas CSV. Lung recruitment maneuvers in acute respiratory distress syndrome and facilitating resolution. Crit Care Med 2003;31(suppl):S265–S271.

for determining whether PEEP will have a favorable or unfavorable response is the relative volume of "recruitable lung" (i.e., areas of atelectasis that can be aerated). If there is recruitable lung, then PEEP will have a favorable effect and will improve gas exchange in the lungs. However if there is no recruitable lung, PEEP can overdistend the lungs (because the lung volume is lower if areas of atelectasis cannot be aerated) and produce an injury similar to ventilator-induced lung injury.
Preliminary studies in ARDS show that the volume of recruitable lung varies widely (from 2% to 25% of total lung volume) in individual patients (27). Although it is not possible to measure recruitable lung volume reliably, the appearance of atelectasis on CT images can sometimes help. Areas of atelectasis that contain pockets of aeration are most likely to represent recruitable lung, whereas areas of atelectasis that are airless are unlikely to be recruitable (27).

The $PaO/Fi0_2$ Ratio

The effects of PEEP on lung recruitment can be monitored with the $PaO/FI0_2$ ratio, which is a measure of the efficiency of oxygen exchange across the lungs. The $PaO/FI0_2$ ratio is usually below 300 in acute respiratory failure, and below 200 in cases of ARDS. If PEEP has a favorable effect and converts areas of atelectasis to functional alveolar-capillary units, there will be an increase in the $PaO/FI0_2$ ratio. However, if PEEP is harmful by overdistending the lungs, the $PaO/FI0_2$ ratio will decrease. An example of a beneficial response in the $PaO/FI0_2$ ratio is shown in Figure 25.6.

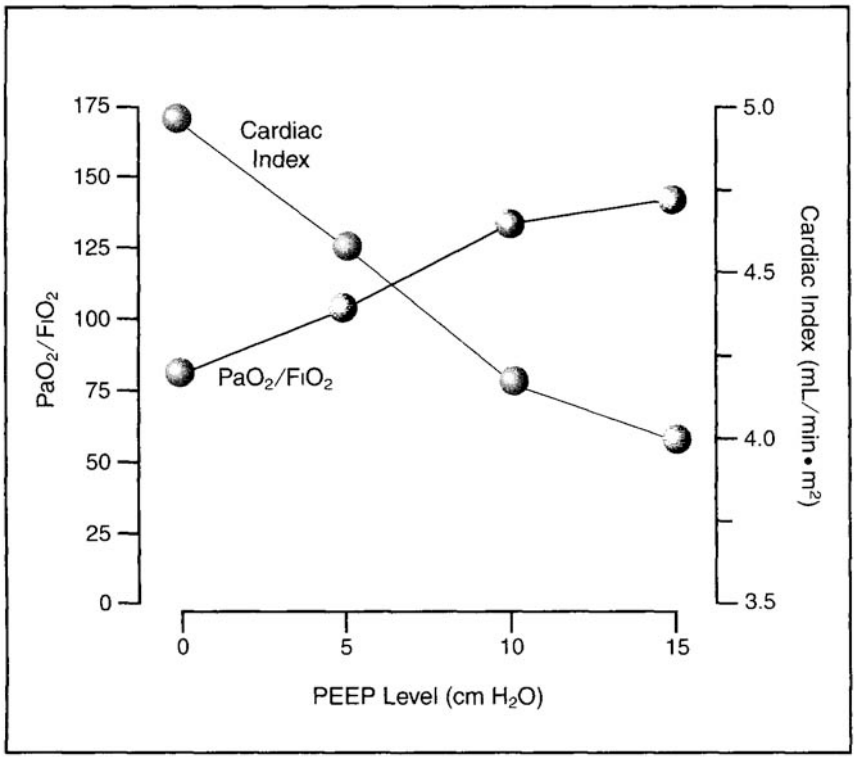

FIGURE 25.6 Effects of positive end-expiratory pressure (*PEEP*) on arterial oxygenation (PaO_2/FiO_2) and cardiac output in patients with ARDS. From Gainnier M, Michelet P, Thirion X, et al. Prone position and positive end-expiratory pressure in acute respiratory distress syndrome. Crit Care Med 2003;31:2719–2726.

Cardiac Performance

PEEP has the same influence on the determinants of cardiac performance as positive-pressure ventilation (described in Chapter 24), but the ability to decrease ventricular preload is more prominent with PEEP. PEEP can decrease cardiac output by several mechanisms, including reduced venous return, reduced ventricular compliance, increased right ventricular outflow impedance, and ventricular external constraint by hyperinflated lungs (28,29). The decrease in cardiac output from PEEP is particularly prominent in hypovolemic patients. PEEP can also increase cardiac output by reducing left ventricular afterload, but the significance of this response is unclear.

Oxygen Transport

The tendency for PEEP to reduce cardiac output is an important consideration because the beneficial effects of PEEP on lung recruitment and gas exchange in the lungs can be erased by the cardiodepressant effects.

The importance of the cardiac output in the overall response to PEEP is demonstrated by the equation for systemic oxygen delivery ($D0_2$):

$$D0_2 = Q \times 1.3 \times Hb \times Sa0_2 \qquad (25.1)$$

Thus, PEEP can improve arterial oxygenation ($Sa0_2$), but this will not improve systemic oxygenation (DO) if the cardiac output (Q) decreases. This situation is shown in Figure 25.6, which is from a study of patients with ARDS (30). In this study, incremental PEEP was accompanied by a steady increase in the $PaO/FI0_2$ ratio, indicating a favorable response in gas exchange in the lungs, but there is also a steady decrease in cardiac output, which means the improved arterial oxygenation is not accompanied by improved systemic oxygenation, and the systemic organs will not share the benefit from PEEP.

Use of PEEP

PEEP is used almost universally in ventilator-dependent patients, presumably as a preventive measure for atelectasis. This practice is unproven, and it creates unnecessary work to trigger a ventilator breath (as described earlier). The few situations where PEEP is indicated are included below:

When the chest x-ray shows diffuse infiltrates (e.g., ARDS) and the patient requires toxic levels of inhaled oxygen to maintain adequate arterial oxygenation. In this situation, PEEP can increase the $PaO/FI0_2$ ratio, and this would permit reduction of the $FI0_2$.

When low-volume, lung-protective ventilation is used. In this situation, PEEP is needed to prevent repeated opening and closing of distal airspaces because this can damage the lungs and add to the severity of the clinical condition(s). PEEP is not recommended for localized lung disease like pneumonia because the applied pressure will preferentially distribute to normal regions of the lung and this could lead to overdistention and rupture of alveoli (ventilator-induced lung injury) (31). In lung-protective ventilation, a PEEP level of 5 to 10 cm H_20 is adequate because higher levels of PEEP are not associated with a better outcome (32).

Misuse of PEEP

Considering that PEEP is used almost universally in ventilator-dependent patients, misuse of PEEP must be common. The following statements are based on personal observations on the misuses of PEEP:

PEEP should not be used routinely in intubated patients because there is no "physiologic PEEP" that is generated by glottic closure. The alveolar pressure at end-expiration is zero in healthy adults. Neonates can generate PEEP by grunting, but this gift is lost by adulthood.

PEEP should not be used to reduce lung water in patients with pulmonary edema. In fact, PEEP increases the water content of the lungs (33,34), possibly by impeding lymphatic drainage from the lungs.

CONTINUOUS POSITIVE AIRWAYS PRESSURE

Spontaneous breathing in which a positive pressure is maintained throughout the respiratory cycle is called *continuous positive airway pressl/re* (CPAP). The airway pressure pattern with CPAP is shown in Figure 25.4. Note that the patient does not have to generate a negative airway pressure to receive the inhaled gas. This is made possible by a specialized inhalation valve that opens at a pressure above atmospheric pressure. This eliminates the extra work involved in generating a negative airway pressure to inhale. CPAP should be distinguished from *spontaneous PEEP.* In spontaneous PEEP, a negative airway pressure is required for inhalation. Spontaneous PEEP has been replaced by CPAP because of the reduced work of breathing with CPAP.

Clinical Uses

The major uses of CPAP are in nonintubated patients. CPAP can be delivered through specialized face masks equipped with adjustable, pressurized valves. CPAP masks have been used successfully to postpone intubation in patients with acute respiratory failure (35,36). However, these masks must be tight-fitting, and they cannot be removed for the patient to eat. Therefore, they are used only as a temporary measure. Specialized nasal masks may be better tolerated. CPAP delivered through nasal masks (nasal CPAP) has become popular in patients with obstructive sleep-apnea (37). In this situation, the CPAP is used as a stent to prevent upper airway collapse during negative pressure breathing. Nasal CPAP has also been used successfully in patients with acute exacerbation of chronic obstructive lung disease (38,39).

A FINAL WORD

Since mechanical ventilation is a support measure and not a treatment modality, nothing that is done with a ventilator will have a favorable impact on the outcome of the primary illness. On the other hand, mechanical ventilation can have a negative impact on outcomes by creating adverse effects. This means that the best mode of mechanical ventilation is the one with the fewest adverse effects. It also means that, if we really want to improve outcomes in ventilator-dependent patients, less attention should be directed to the knobs on ventilators, and more attention should be directed at the diseases that create ventilator dependency.

References

Chapter 26

THE VENTILATORDEPENDENT PATIENT

This chapter describes the practices and common concerns involved in the care of ventilator-dependent patients. The focus in this chapter is on issues that are directly related to artificial airways (endotracheal and tracheostomy tubes) and positive-pressure ventilation. The complicating illnesses that can develop in ventilator-dependent patients, such as nosocomial infections, are described elsewhere.

ARTIFICIAL AIRWAYS

Positive-pressure ventilation is delivered through a variety of plastic tubes that are passed into the trachea through the vocal cords (endotracheal tubes) or are inserted directly into the trachea (tracheostomy tubes) 0-3). These tubes are equipped with an inflatable balloon at the distal end (called a cuff) that is used to seal the trachea and prevent positive-pressure inflation volumes from escaping out through the larynx. The complications created by these tubes are related to

their path of entry into the trachea, and to pressure-induced injury of the tracheal mucosa from the inflated cuffs. The major complications are indicated in Figure 26.1 (2-4).

Endotracheal Intubation

The patient who has been recently intubated will have an endotracheal tube inserted through the nose or mouth. These tubes vary in length from 25 to 35 em, and are sized according to their internal diameter, which varies from 5 to 10 mm (e.g., a "size 7" endotracheal tube will have an internal diameter of 7 mm). The volume capacity of endotracheal tubes is 35 to 45 mL (5), which is about half the volume of the anatomic dead space in adults (1 mg/kg). The decrease in anatomic dead space associated with endotracheal intubation has no apparent significance.

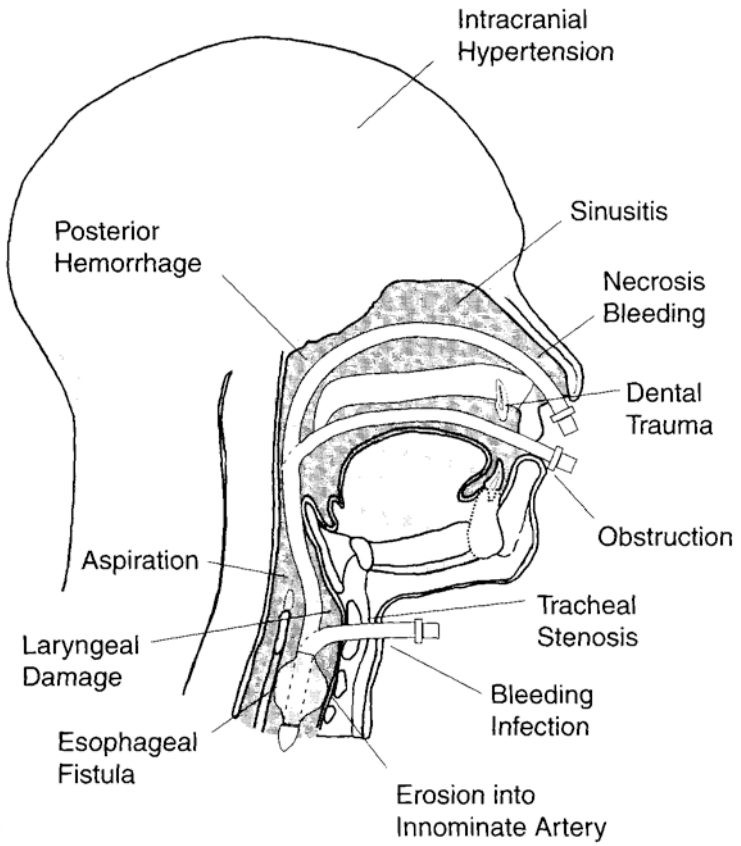

FIGURE 26.1 Complications of endotracheal intubation (nasal and oral routes) and tracheostomy.

The Work of Breathing

Endotracheal tubes increase the resistance to airflow. This resistance is directly related to the length of the tube, and inversely related to the diameter of the tube (e.g., tubes that are longer and narrower will create a greater resistance to airflow). An increased resistance to airflow will require a higher pressure gradient to ensure the same rate of airflow. During spontaneous breathing, a greater negative intrathoracic pressure will be needed to maintain a constant inspiratory flow rate when a subject is breathing through a long and narrow tube. This increases the work of breathing. High flow rates create turbulent flow, and this increases resistance further, so the work of breathing through endotracheal tubes is particularly high when patients are in respiratory distress. The work of breathing created by endotracheal tubes is important when weaning patients from the ventilator. In general, the diameter of endotracheal tubes should be at least 7 mm, and preferably 8 mm, to limit the influence of the tube on the work of breathing. (The process of weaning patients from the ventilator is described in the next chapter.)

Intubation Routes

Endotracheal tubes can be inserted through the nose or mouth. The nasal route is usually preferred for patients who are awake and cooperative, while the oral route is used in patients who have an altered mental status or are uncooperative. The complications associated with each route are indicated in Figure 26.1. The complications of nasotracheal intubation include epistaxis, paranasal sinusitis, and necrosis of the nasal mucosa. The complications of orotracheal intubation include dental trauma, pressure necrosis of the mucosa at the back of the mouth (where the tubes bend at an acute angle and exert a torque) and obstruction of the tubes by patients who bite down on the tubes. Laryngeal damage is a complication of endotracheal (translaryngeal) intubation, and occurs with both nasal and oral routes of intubation.

Proper Tube Position

Evaluation of endotracheal tube position is mandatory after intubation, and should be repeated on a regular basis until the patient is extubated or receives a tracheostomy. Some useful radiographic landmarks for evaluating endotracheal tube position are shown in Table 26.1 (6). The vocal cords are usually situated at the interspace between the fifth and sixth cervical vertebrae (C5-C6). If not visible, the main carina is usually over the interspace between the fourth and fifth thoracic vertebrae (T4-T5) on a portable chest x-ray film. When the head is in a neutral position, the tip of the endotracheal tube should be 3 to 5 cm above the carina, or midway between the carina and vocal cords. The head is in the neutral position when the inferior border of the mandible projects over the lower cervical spine (C5-C6). Note that flexion or extension of the head and

TABLE 26.1 Radiographic Evaluation of Tracheal Intubation

	Radiographic Location
Anatomic Structures	
Vocal Cords	Usually over the C4-C5 interspace
Carina	Usually over the T 4- T5 interspace
Head and Neck Position	
Neutral Position	Inferior border of mandible is over C5-C6
Flexion	Mandible is over T1- T2
Extension	Mandible is above C4
Tracheal Tube Position	
Head in Neutral Position	Tip of the tube should be midway between the vocal cords and carina, or 3-5 cm above the carina
Head Flexed	Tip of tube will descend 2 cm
Head Extended	Tip of tube will ascend 2 cm

From Goodman LR, Putman cE, eds. Critical care imaging. 3rd ed, Saunders. 1992:35-56.

neck causes a 2 cm displacement of the tip of the endotracheal tube (6). The total displacement of 4 cm with changes in head position represents about one-third of the length of the trachea.

Migration of Tubes

Endotracheal tubes can migrate in either direction along the trachea (from manipulation of the tube during suctioning, turning, etc.), and when tubes move distally into the lungs, they often end up in the right mainstem bronchus (which runs a straight course down from the trachea). What can happen next is shown in Figure 26.2; i.e., selective ventilation of one lung results in progressive atelectasis in the non-ventilated lung. Delivering the entire tidal volume to one lung can also result in alveolar rupture and pneumothorax in the ventilated lung (which is not the case in Figure 26.2).

There are two measures that can reduce the incidence of endotracheal tube migration into one of the mainstem bronchi. First, for orotracheal intubations, don't allow the tip of the endotracheal tube to advance further than 21 em from the teeth in women and 23 cm from the teeth in men (7). Second, monitor the position of the endotracheal tube on

regularly-scheduled chest x-rays (which is a common practice in ventilator-dependent patients), and keep the tip of the tube at least 3 em above the main carina (where the trachea bifurcates to form the right and left mainstem bronchi.

Paranasal Sinusitis
Nasotracheal (and nasogastric) tubes can obstruct the ostia that drain the paranasal sinuses, and this predisposes to a purulent sinusitis (8,9). The maxillary sinus is almost always involved (see Fig. 39.3). The presence of an air-fluid level by sinus radiography suggests the diagnosis, but confirmation requires aspiration of infected material from the involved sinus. The significance of paranasal sinusitis is unclear. It may be an important consideration when all other causes of fever have been eliminated (9), but in the routine evaluation of fever in the ICU, sinusitis is often not evaluated and there are no apparent adverse consequences. This complication has also been reported with orotracheal tubes (9). See Chapter 39 for more information on sinusitis in the ICU.

Laryngeal Damage
The risk for laryngeal injury with endotracheal tubes is a major concern, and is the principal reason for performing tracheostomies when prolonged intubation is anticipated. The spectrum of laryngeal damage includes ulceration, granulomas, vocal cord paresis, and laryngeal edema. Some type of laryngeal damage is usually evident after 72 hours of translaryngeal intubation, and laryngeal edema is reported in 5% of cases. Fortunately, most cases of laryngeal injury do not result in significant airways obstruction, and the injury resolves within weeks after extubation (1). The problem of laryngeal edema after tracheal decannulation is described in Chapter 27.

Tracheostomy
Tracheostomy is preferred in patients who require prolonged mechanical ventilation. There are several advantages with tracheostomy, including greater patient comfort, more effective clearing of secretions, red uced resistance for breathing, and reduced risk oflaryngeal injury (2). Tracheostomy patients can ingest food orally and can even vocalize using specially designed tracheostomy tubes.

Trachcostomy Timing
The optimal time for performing a tracheostomy can vary widely in individual patients. However, the following recommendation can be used as a general guideline (2): After 5 to 7 days of endotracheal intubation, assess the likelihood of extubation in the following week: if the likelihood is low, proceed to tracheostomy. This recommendation is based on the observation that the clinical course in the first week is predictive of the final outcome in patients with ventilator-dependent respiratory failure (13).

Techniques

Tracheostomy can be performed as an open surgical procedure, or by percutaneous placement of the tracheostomy tube at the bedside. There are three percutaneous techniques; each involves puncturing the anterior wall of the trachea with a needle and then passing a guidewire through the needle into the trachea. The guidewire is then used to advance the tracheostomy tube into the trachea. The original technique, *percutaneous dilatational tracheostomy,* uses dilator catheters advanced over the guidewire to create a tract for the tracheostomy tube (4), similar to the Seldinger technique used to insert vascular catheters (see Fig. 6.3). In experienced hands, each of the percutaneous techniques can be performed successfully and safely (2).

The technique known as *cricothyroidotomy* is used only for emergency access to the airway. The trachea is entered through the cricothyroid membrane, just below the larynx, and there is high incidence of laryngeal injury and subglottic stenosis. Patients who survive following a cricothyroidotomy should have a regular tracheostomy (surgical or percutaneous) as soon as they are stable (2).

Complications

The morbidity and mortality associated with tracheostomy has declined in recent years. Combining surgical and percutaneous tracheostomy, the mortality rate is less than 1 %, and major adverse events occur in 5 to 10% of cases (2). The acute complications of most concern are bleeding and infection. A comparison of surgical and percutaneous tracheostomy from pooled studies showed less bleeding and fewer infections with the percutaneous technique (5). However, the results of individual studies are conflicting, and the local expertise with each procedure will probably determine the technique with the fewest complications at each hospital.

One acute complication that deserves mention is accidental decannulation. If the tracheostomy tube is dislodged before the stoma tract is mature (which takes about one week) the tract closes quickly, and blind reinsertion of the tube can create false tracts. To minimize the risk of a bad outcome in this situation, the patient should be reintubated orally before attempting to reinsert the tracheostomy tube.

The most feared complication of tracheostomy is tracheal stenosis, which is a late complication that appears in the first 6 months after the tracheostomy tube is removed. Most cases of tracheal stenosis occur at the tracheotomy site, and are caused by tracheal narrowing after the stoma closes. The incidence of tracheal stenosis ranges from zero to 15% in individual reports (2), but most cases are asymptomatic.

Cuff Management

Positive-pressure ventilation requires a seal in the trachea that prevents gas from escaping out through the larynx during lung inflation, and this seal is created by inflatable balloons (called cuffs) that surround the distal portions of endotracheal tubes and tracheostomy tubes. An illustration

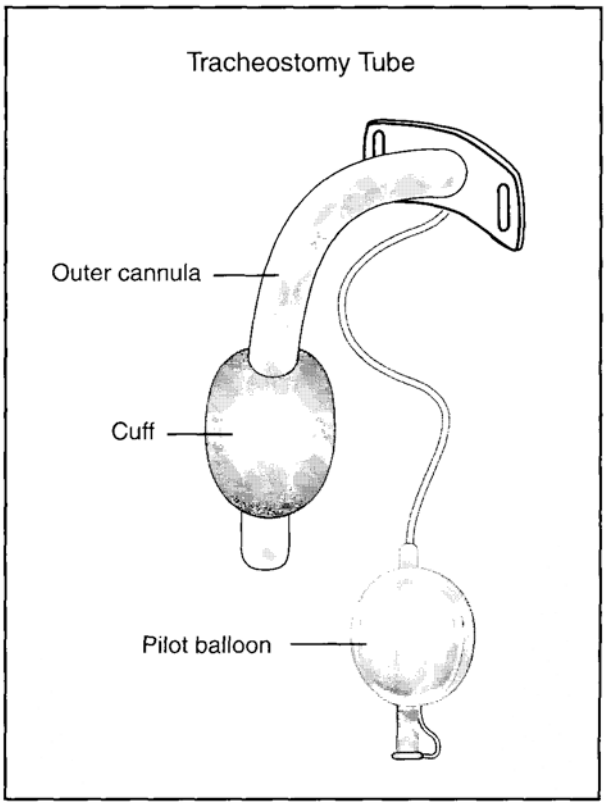

FIGURE 26.3 Illustration of a tracheostomy tube with an inflated cuff.

of a tracheostomy tube with an inflated cuff is shown in Figure 26.3. The cuff is attached to a pilot balloon that has a one-way valve. Cuff inflation is accomplished by attaching a syringe to the pilot balloon and injecting air. When the cuff is inflated, the pilot balloon will also inflate. Air is injected until there is no evidence of a leak around the cuff (i.e., there is no difference between inspiratory and expiratory volumes). The pressure in the cuff (measured with a pressure gauge attached to the pilot balloon) should not exceed 25 mm Hg (35 cm H_20) (4). This pressure limit is based on the assumption that the capillary hydrostatic pressure in the wall of the trachea is 25 mm Hg, and thus external (cuff) pressures above 25 mm Hg can compress the underlying capillaries and produce ischemic injury and tracheal necrosis. The cuffs are elongated, which allows for greater dispersion of pressure, and this high-volume, low-pressure design allows for a tracheal seal at relatively low pressures.

The risk of pressure-induced tracheal injury can be virtually eliminated by using tracheal tubes that have a foam rubber cuff that is normally inflated at atmospheric pressure (an example is the Bivona Fome-Cuf tracheostomy tube). When the tube is inserted, the cuff must be manually deflated by applying suction from a syringe. Once in place, the cuff

is allowed to inflate and create a tracheal seal. This cuff design allows the trachea to be sealed at atmospheric (zero) pressure, which eliminates the risk of pressure-induced injury in the wall of the trachea.

Aspiration

Contrary to popular belief, cuff inflation to create a tracheal seal does not prevent aspiration of mouth secretions and tube feedings into the lower airways. Aspiration of saliva and liquid tube feedings has been documented in over 50% of ventilator-dependent patients with tracheostomies and, in over three-fourths of the cases, the aspiration is clinically silent (6). Considering that approximately 1 L of saliva is normally produced each day (the normal rate of saliva production is 0.6 mL/min) and that **each microliter of saliva contains approximately 1 billion microorganisms,** the danger associated with aspiration of mouth secretions is considerable (7). This emphasizes the value of oral decontamination described in Chapter 4.

Cuff Leaks

Cuff leaks are usually detected by sounds generated during lung inflation (created by gas flowing through the vocal cords). When a cuff leak becomes audible, the volume of the leak can be estimated as the difference between the desired inflation volume and the exhaled volume recorded by the ventilator. Cuff leaks are rarely caused by disruption of the cuff (8), and are usually the result of nonuniform contact between the cuff and the wall of the trachea.

If a cuff leak is apparent, the patient should be separated from the ventilator and the lungs should be manually inflated with an anesthesia bag. If the cuff leak involves an endotracheal tube, the tube should be replaced (or a fiberoptic instrument can be advanced through the tube to make sure it is still in the lungs). Air should never be added to the cuff blindly because the tube may be coming out of the trachea, and cuff inflation will damage the vocal cords. If the cuff leak involves a tracheostomy tube, air can be added to the cuff in an attempt to produce a seal. If this eliminates the leak, the cuff pressure should be measured. If the cuff pressure is above 35 cm H_2O, or the leak persists despite adding volume, the tracheostomy tube should be replaced.

CLEARING SECRETIONS

Routine suctioning to clear secretions is a standard practice in the care of intubated patients. This section will focus on measures that are used to liquify respiratory secretions to aid in their removal.

Physicochemical Properties

The respiratory secretions create a blanket that covers the mucosal surface of the airways. This blanket has a hydrophilic (water soluble) layer, and a hydrophobic (water insoluble) layer. The hydrophilic layer faces

inward, and keeps the mucosal surface moist. The hydrophobic layer faces outward, towards the lumen of the airways. This outer layer is composed of a meshwork of mucoprotein strands (called mucus threads) held together by disulfide bridges. This meshwork traps particles and debris in the airways, and the combination of the mucoprotein meshwork and the trapped debris is what determines the viscoelastic behavior of the respiratory secretions.

Saline Instillation

Saline is often instilled into the trachea to facilitate the clearing of secretions, but this practice is ill-advised for two reasons. First, according to the physicochemical properties of respiratory secretions just described, the layer that contributes to the viscoelastic properties of respiratory secretions is not water soluble, which means that saline cannot liquify or reduce the viscosity of respiratory secretions. Adding saline to thick, respiratory secretions is like pouring water over grease.

The second problem with saline instillation is the risk for infection.

As mentioned in Chapter 7, bacteria form biofilms on prosthetic devices (see Fig. 7.2), and bacterial biofilms have been demonstrated on the inner surface of endotracheal tubes and tracheostomy tubes (19). Saline injections into tracheal tubes can dislodge these bacterial biofilms. In a study using endotracheal tubes from extubated patients, the results showed that injection of 5 mL of saline can dislodge up to 300,000 colonies of viable bacteria from the inner surface of the tubes (20). Therefore, saline injection provides a vehicle for transporting bacteria into the lower airways. This observation deserves much more attention than it receives.

Mucolytic Therapy

The accumulation of tenacious secretions can result in a condition like the one shown in Figure 26.4, where a waterless "plug" is completely obstructing a major airway. In this situation, a mucolytic agent like N-acetylcysteine (Mucomyst) can help to disrupt the plug and relieve the obstruction. N-acetylcysteine (NAC) is a sulfhydryl-containing tripeptide that is better known as the antidote for acetaminophen overdose, but is primarily a mucolytic agent that acts by disrupting the disulfide bridges between mucoprotein strands in sputum (21). The drug is available in a liquid preparation 10 or 20% solution) that can be given as an aerosol spray, or injected directly into the airways (Table 26.2). Aerosolized NAC should be avoided when possible because it is irritating to the airways and can provoke coughing and bronchospasm (particularly in asthmatics). Direct instillation of NAC into the tracheal tube is preferred, especially when there is an obstruction.

If intratracheal injection of NAC does not relieve an obstruction, bronchoscopy should be performed (the NAC is then applied directly to the mucous plug). Following relief of the obstruction, NAC can be instilled two or three times a day for the next day or two. Daily use of NAC is not advised because the drug solution is hypertonic (even with the saline additive) and can provoke bronchorrhea.

ALVEOLAR RUPTURE

The problem of ventilator-induced lung injury described in Chapters 22 and 24 has its ultimate expression in clinically apparent alveolar rupture, which occurs in up to one-fourth of patients receiving positive-pressure ventilation (22).

Clinical Presentation

Escape of gas from the alveoli can produce a variety of clinical manifestations. The alveolar gas can dissect along tissue planes and produce *pulmonary interstitial emphysema,* and can move into the mediastinum and produce *pneumomediastinum.* Mediastinal gas can move into the neck to produce *subcutaneous emphysema,* or can pass below the diaphragm to produce *pneumoperitoneum.* Finally, if the rupture involves the visceral pleura, gas will collect in the pleural space and produce a *pneumothorax.* Each of these entities can occur alone or in combination with the others (22,23).

Pneumothorax

Radiographic evidence of pneumothorax occurs in 5 to 15% of ventilator-dependent patients (22,23). Risk factors include high inflation pressures

TABLE 26.2 Mucolytic Therapy with N-acetylcysteine (NAC)

Aerosol therapy:	• Use 10% NAC solution. • Mix 2.5 mL NAC with 2.5 mL saline and place mixture (5 mL) in a small volume nebulizer for aerosol delivery. • *Warning:* this can provoke bronchospasm, and is not recommended in asthmatics.
Tracheal injection:	• Use 20% NAC solution. • Mix 2 mL NAC with 2 mL saline and inject 2 mL aliquots into the trachea. • *Warning:* Excessive volumes can produce bronchorrhea.

and inflation volumes, positive end-expiratory pressure (PEEP), and diffuse lung injury.

Clinical Presentation

Clinical manifestations are either absent, minimal, or nonspecific. Themost valuable clinical sign is subcutaneous emphysema in the neck and upper thorax, which is pathognomonic of alveolar rupture. Breath sounds are unreliable in ventilator-dependent patients because sounds transmitted from the ventilator tubing can be mistaken for airway sounds.

Radiographic Detection

The radiographic detection of pleural air can be difficult in the supine position, because pleural air does not collect at the lung apex when patients are supine (23,24). Figure 26.5 illustrates this difficulty. In this case of a traumatic pneumothorax, the chest x-ray is unrevealing, but the CT scan reveals an anterior pneumothorax on the left. Pleural air will collect in the most superior region of the hemithorax and, in the supine position, this region is just anterior to both lung bases. Therefore, basilar and subpulmonic collections of air are characteristic of pneumothorax in the supine position (24).

Redundant Skin Folds

When the film cartridge used for portable chest x-ray examinations is placed under the patient, the skin on the back can fold over on itself, and the edge of this redundant skin fold creates a radiographic shadow that can be mistaken for a pneumothorax. The radiographic appearance of a redundant skin fold is shown in Figure 26.6. Note that there is a gradual increase in radiodensity that suddenly ends as a wavy line. The increase in density is produced by the skin that is folded back on itself. A pneumothorax would appear as a sharp white line with dark shadows (air) on either side.

When a redundant skin fold is suspected, a repeat chest film should be obtained (it may be wise to alert the x-ray technician to make sure the film cartridge is flush on the patient's back). The shadow should disappear if it is due to a redundant skin fold.

Pleural Evacuation
Evacuation of pleural air is accomplished by inserting a chest tube through the fourth or fifth intercostal space along the mid-axillary line. The tube should be advanced in an anterior and superior direction (because this is where pleural air collects in the supine position). The pleural space is drained of fluid and air using a three-chamber system like the one shown in Figure 26.7 (25).

Collection Chamber
The first bottle in the system collects fluid from the pleural space and allows air to pass through to the next bottle in the series. Because the inlet

of this chamber is not in direct contact with the fluid, the pleural fluid that is collected does not impose a back pressure on the pleural space.

Water-Seal Chamber
The second bottle acts as a one-way valve that allows air to escape from the pleural space but prevents air from entering the pleural space. This one-way valve is created by submerging the inlet tube under water. This imposes a back-pressure on the pleural space that is equal to the depth that the tube is submerged. The positive pressure in the pleural space then prevents atmospheric air (at zero pressure) from entering the pleural space. The water thus "seals" the pleural space from the surrounding atmosphere. This water-seal pressure is usually 2 cm H_20.
Air that is evacuated from the pleural space passes through the water in the second bottle and creates bubbles. Thus, the presence of bubbles in the water-seal chamber (called bubbling) is used as evidence for a continuing bronchopleural air leak.

Suction-Control Chamber
The third bottle in the system is used to set a maximum limit on the negative suction pressure that is imposed on the pleural space. This maximum pressure is determined by the height of the water column in the air inlet tube. Negative pressure (from wall suction) draws the water down the air inlet tube, and when the negative pressure exceeds the height of the water column, air is entrained from the atmosphere. Therefore, the pressure in the bottle can never become more negative than the height of the water column in the air inlet tube.

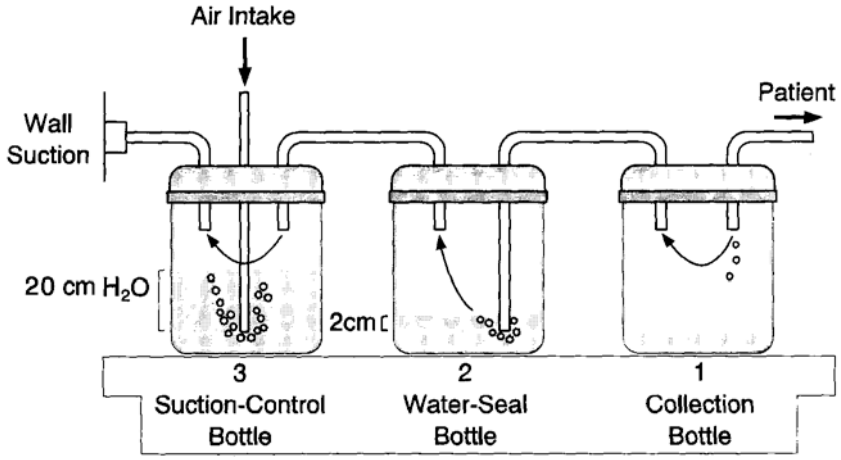

FIGURE 26.7 A standard pleural drainage system for evacuating air and fluid from the pleural space.

Water is added to the suction-control chamber to achieve a water level of 20 em. The wall suction is then activated and slowly increased until bubbles appear in the water. This bubbling indicates that atmospheric air is being entrained, and thus the'maximum negative pressure has been achieved. The continuous bubbling causes water evaporation, so it is imperative that the height of the water in this chamber is checked periodically, and more water is added when necessary.

Why Suction?
The practice of using suction to evacuate pleural air is unnecessary and potentially harmful. Although there is a perception that suction will help the lungs reinflate, the lungs will reinflate without the use of suction. Furthermore, creating a negative pressure in the pleural space also creates a higher transpulmonary pressure (the pressure difference between alveoli and the pleural space), and this increases the rate of the air flowing through the bronchopleural fistula. Thus, applying suction to the pleural space increases bronchopleural air leaks, and this can keep bronchopleural fistulas patent. If a persistent air leak is present when suction is applied to the pleural space, the suction should be discontinued, Any air that collects in the pleural space will continue to be evacuated when the pleural pressure becomes more positive than the water-seal pressure.

INTRINSIC PEEP

As mentioned in Chapter 25, mechanical ventilation with high inflation volumes and rapid rates can (and probably often does) produce PEEP as a result of incomplete alveolar emptying during expiration (26-28). The illustration in Figure 26.8 helps explain this phenomenon.

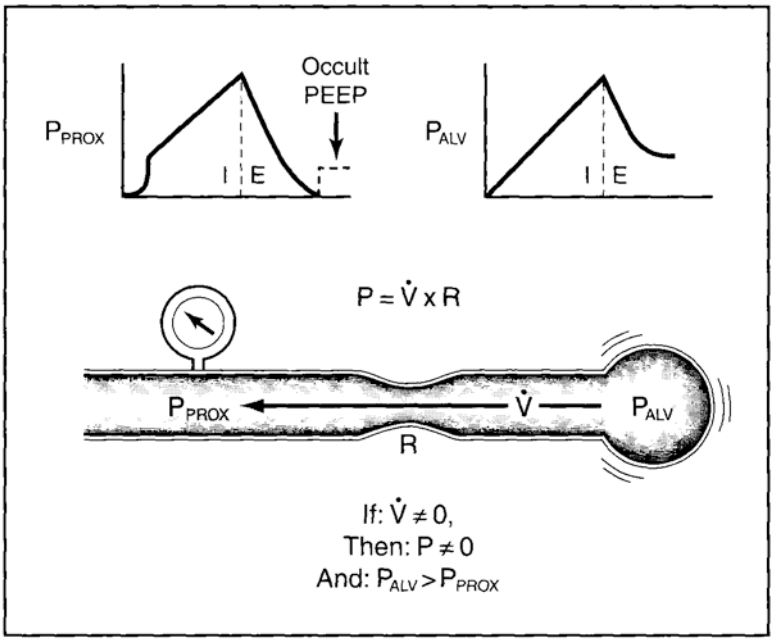

FIGURE 26.8 The features of intrinsic PEEP resulting from inadequate alveolar emptying. The presence of airflow ($\dot{V}$) at end-expiration indicates a pressure drop from the alveolus (P_{ALV}) to the proximal airways (P_{prox}). As shown at the top of the figure, the proximal airway pressure returns to zero at end-expiration while the alveolar pressure remains positive; hence the term *occult* PEEP. The upper left panel illustrates the end-expiratory occlusion method for detecting occult PEEP.

Pathogenesis

Under normal circumstances, there is no airflow at the end of expiration, and thus end-expiratory pressure is the same in the alveoli and proximal airways. On the other hand, when alveolar emptying is incomplete during expiration, airflow is present at the end of expiration, and this creates a pressure drop from the alveoli to the proximal airways at end-expiration. Thus in this situation, alveolar pressure will be positive relative to atmospheric (zero) pressure at end-expiration (i.e., PEEP). As shown in the upper portion of Figure 26.8, the positive alveolar pressure is not evident on the proximal airways pressure tracing, and hence this is an *occult* PEEP (26). More popular terms for this pressure are *intrinsic PEEP, autoPEEP,* and *dynamic hyperinflation* (27). The latter term is usually reserved for PEEP produced by obstructive airways disease (see below).

Predisposing Factors

The factors that predispose to hyperinflation and intrinsic PEEP can be separated into ventilation-associated and disease-associated factors. The ventilatory factors that promote hyperinflation include high inflation

volumes, rapid breathing, and a relative decrease in exhalation time relative to inhalation time. The disease-related factor that promotes hyperinflation is airways obstruction (as occurs in asthma and chronic obstructive pulmonary disease [COPD]. All three factors are operative in ventilator-dependent patients with obstructive airways disease.
In patients with asthma and COPD, intrinsic PEEP is probably universal during conventional volume-cycled mechanical ventilation (27-29). Intrinsic PEEP is also common in ventilator-dependent patients with ARDS (30), but the absolute level is low (<3 cm HP).

Consequences

Cardiac Performance

Intrinsic PEEP has the same effects on cardiac performance as extrinsic PEEP, which are described in Chapter 25. However, because intrinsic PEEP is often overlooked, the cardiac suppression from intrinsic PEEP can be life-threatening. Intrinsic PEEP from overly aggressive ventilation has been implicated as an occult cause of cardiac arrest (31), and failed cardiopulmonary resuscitation (32).

Alveolar Rupture

Intrinsic PEEP increases peak inspiratory airway pressures, and this will predispose to pressure-induced alveolar rupture (barotrauma). Hyperinflation-induced alveolar rupture and pneumothorax are particular concerns in ventilator-dependent asthmatic patients (29).

Work of Breathing

Intrinsic PEEP increases the work of breathing in several ways. To begin with, PEEP increases the pressure that must be generated to trigger the ventilator (e.g., if the ventilator breath is triggered at a pressure of -2 cm H_2O and there is an end-expiratory pressure of 5 cm H_2O, then the total pressure that must be generated to trigger the ventilator is 7 cm H,O). In addition, hyperinflation places the lungs on a flatter portion of the pressure-volume curve, so higher pressures are needed to inhale the tidal volume. (To appreciate this effect, take a deep breath, and then try to breathe in further.) Finally, hyperinflation flattens the diaphragm and this (according to the Law of LaPlace; T = Pr) will increase the muscle tension needed to generate a given change in thoracic pressure. The sum of all of these factors can lead to a considerable increase in the work of breathing, and this could impede efforts to wean from mechanical ventilation (see Chapter 27).

Thoracic Compliance

Intrinsic PEEP increases the end-inspiratory pressures in the proximal airways (both peak and plateau pressures). When intrinsic PEEP goes undetected, the increase in plateau pressure is misinterpreted as a decrease in the compliance of the lungs and chest wall. Therefore, failure to consider intrinsic PEEP can result in an underestimation of thoracic

compliance. When thoracic compliance is calculated (see Chapter 24), the level of intrinsic PEEP should be subtracted from the measured plateau pressure and the resulting pressure should be used to calculate thoracic compliance.

Cardiac Filling Pressures

As mentioned in Chapter 10, the presence of PEEP can cause a spurious increase in cardiac filling pressures: central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP). This effect is due to transmission of PEEP into the lumen of intrathoracic vessels, resulting in an increase in intravascular pressure with little or no increase in transmural pressure (the physiologically important pressure).

A simple method for correcting the CVP and PAOP in the presence of intrinsic PEEP is to measure the level of intrinsic PEEP with the end-expiratory occlusion method and then subtract this pressure from the CVP or PAOP measured at end-expiration. Although PEEP is not always fully transmitted into vessels (33), the degree of transmission of intrinsic PEEP into vessels is difficult to determine because of the dynamic nature of this pressure.

Monitoring Intrinsic PEEP

Intrinsic PEEP is easy to detect but difficult to quantify. The easiest method of detection is listening for the presence of airflow at the end of expiration, which has been shown to be a sensitive but not specific method of detection (34). Expiratory flow tracings can also be used to detect intrinsic PEEP. The most accurate method of measuring intrinsic PEEP is to measure intraesophageal (pleural) pressure at end-expiration. The most popular method of monitoring intrinsic PEEP is the end-expiratory occlusion method.

End-Expiratory Occlusion

During controlled ventilation (i.e., when the patient is completely passive), intrinsic PEEP can be uncovered by occluding the expiratory tubing at the end of expiration (27). This maneuver blocks airflow and allows the pressure in the proximal airways to equilibrate with alveolar pressure. Thus, a sudden rise in proximal airways pressure with end-expiratory occlusion is evidence of intrinsic PEEP (see the upper left panel in Fig. 26.8). Unfortunately, accuracy requires that the occlusion occur at the very end of expiration, just before the next breath (because expiratory flow can persist until the end of expiration), and this is not possible on a consistent basis when the occlusion is performed manually.

Response to Extrinsic PEEP

The application of extrinsic PEEP normally increases the peak inspiratory airway pressure by an equivalent amount. However, in the presence of intrinsic PEEP, the peak inspiratory pressure may not change when extrinsic PEEP is applied (this is explained below). Therefore, failure

of extrinsic PEEP to produce an increase in peak inspiratory airway pressure is evidence of intrinsic PEEP (35-37). Furthermore, the level of extrinsic PEEP that first produces a rise in peak inspiratory pressures can be taken as the quantitative level of occult PEEP (35).

Management
The methods used to prevent or reduce hyperinflation and occult PEEP are all directed at promoting alveolar emptying during expiration. Ventilator-induced hyperinflation can be minimized by avoiding excessive inflation volumes, and by optimizing the time allowed for exhalation. These measures are described in Chapters 24 and 25.

Extrinsic PEEP
When hyperinflation is due to small airway collapse at end-expiration, as occurs in patients with asthma and COPD, the application of extrinsic PEEP can help keep the small airways open at end-expiration. The level of extrinsic PEEP must be enough to counterbalance the pressure causing small airways collapse (the *critical closing pressure)* but should not exceed the level of intrinsic PEEP (so that it does not impair expiratory flow) (36). To accomplish this, the level of extrinsic PEEP should match the level of intrinsic PEEP. The appropriate level of extrinsic PEEP is difficult to determine because of inaccuracies in measuring intrinsic PEEP. An alternative method is to apply external PEEP and measure the changes in end-expiratory plateau pressure. If this pressure decreases with external PEEP, this indicates that external PEEP has effectively decreased internal PEEP (37). The clinical significance of all of this, however, is unclear.

A FINAL WORD
The patient's clinical course in the first few days of mechanical ventilation will give you a fairly accurate indication of what is coming. If the patient is not improving, proceed to tracheostomy as soon as it can be done safely. (Tracheostomies are more comfortable for patients, and they allow more effective airway care.) Most of the day-to-day management in the ICU is aimed at preventing other adverse events (such as pneumothorax), and remaining vigilant for adverse events so they can be treated or corrected quickly. You will learn that, in many cases, you are (unfortunately) not the one controlling the course of the patient's illness.

REFERENCES

Chapter 27

DISCONTINUING MECHANICAL VENTILATION
Discontinuing mechanical ventilation is a rapid and uneventful process for most patients, but for one of every four or five patients, the transition to spontaneous breathing is a prolonged process that can consume almost half of the total time on a ventilator. This chapter describes the process of discontinuing mechanical ventilation, and the principal causes of difficulty in the transition to spontaneous breathing (1-4).
READINESS CRITERIA
The management of patients who require mechanical ventilation requires constant vigilance for signs indicating that ventilatory support may no longer be necessary. When patients begin to show evidence of clinical improvement, the criteria listed in Table 27.1 can be used to identify possible candidates for removal of ventilatory support. In general, oxygenation should be adequate while breathing non-toxic levels of inhaled oxygen, and patients should be hemodynamically stable with minimal or no vasopressor support. Patients should be aware of their surroundings when not sedated, and should be free of any reversible conditions (e.g., sepsis or electrolyte abnormalities) that can add to the difficulty of removing ventilatory support.
When the criteria in Table 27.1 have been satisfied, the patient should be removed from the ventilator briefly to obtain the measurements listed in Table 27.2. These measurements have been used to predict the likelihood that the patient will tolerate a trial of spontaneous breathing. Unfortunately,

none of these measurements, when used in isolation, can predict with certainty which patients are ready to resume spontaneous breathing (1,5,6). However, when taken together, these measurements provide an impression of how difficult it will be for the patient to resume

Table 27.1 Checklist for Identifying Patients who Can be Considered for a Trial of Spontaneous Breathing

Respiratory Criteria:

- ☑ $PaO_2 \geq 60$ mm Hg on $F_IO_2 \leq 40–50\%$ and PEEP $\leq 5–8$ cm H_2O
- ☑ $PaCO_2$ normal or baseline (except for permissive hypercapnia).
- ☑ Patient is able to initiate an inspiratory effort.

Cardiovascular Criteria:

- ☑ No evidence of myocardial ischemia.
- ☑ Heart rate $\leq$ 140 beats/minute.
- ☑ Blood pressure normal without vasopressors or with minimum vasopressor support (e.g., dopamine < 5 μg/kg/min).

Adequate Mental Status:

- ☑ Patient is arousable, or Glasgow Coma Score ≥ 13.

Absence of Correctible Comorbid Conditions:

- ☑ Patient is afebrile.
- ☑ There are no significant electrolyte abnormalities.

From Reference 1.

spontaneous breathing. Two of the more predictive measurements are described next.

Rapid-Shallow Breathing Index

Rapid and shallow breathing is common in patients who do not tolerate spontaneous breathing (6). An index of rapid and shallow breathing is provided by the ratio of respiratory rate to tidal volume (RR/V *T)'* which is also called the *rapid-shallow breathing index.* This ratio is normally 40 to

Table 27.2 Measurements Used to Identify Patients Who Will Tolerate a Spontaneous Breathing Trial (SBT)

Measurement*	Reference Range in Adults	Threshold for Successful SBTf
Tidal Volume (V_T)	5-7 ml/kg	4-6 ml/kg
Respiratory Rate (RR)	10-18 bpm	30-38 bpm
Total Ventilation (V E)	5-6 L/min	*10-15 L/min*
RRN_T Ratio	40-50/L	60-105/L
Maximum Inspiratory Pressure (Plmax)	-90 to -120 cm H20	-15 to -30 cm H20

'All measurements should be obtained during spontaneous breathing.
tFrom Reference 1

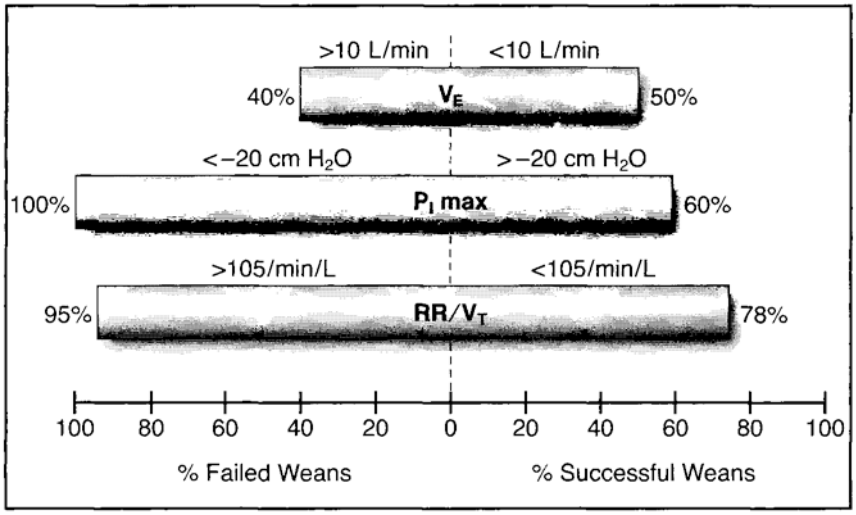

FIGURE 27.1 The predictive value of selected measurements obtained during spontaneous breathing. V_E = minute ventilation, *Pimax* = maximum inspiratory pressure, RR/V_T = ratio of respiratory rate to tidal volume. (Data from Yang K, Tobin MJ. A prospective study of indexes predicting the outcome of weaning from mechanical ventilation. N Engl J Med 1991;324:1445 – 1450.)

50/L, and is often above 100/L in patients who do not tolerate spontaneous breathing.

Predictive Value

The original study of the predictive value of the RR/V_T ratio is shown in Figure 27.1 (6). When the RR/V_T ratio was above 105 /L, 95% of the attempts to discontinue (wean) mechanical ventilation failed. However when the RR/V_T ratio was below 105, 80% of the wean attempts were successful. Therefore, the results of this study establish a threshold RR/V T ratio of approximately 100/L for predicting success or failure to resume spontaneous breathing. Other studies have shown that that the threshold RR/V T ratio may differ in specific groups of patients (2), and that serial measurements of the RR/V T ratio during a spontaneous breathing trial may be more predictive than measurements obtained at the onset of the trial (7). Nevertheless, the threshold RR/V_T ratio of 100/L remains useful because it can identify which patients might have difficulty during the spontaneous breathing trial.

Maximum Inspiratory Pressure

The strength of the diaphragm and other muscles of inspiration can be evaluated by having a patient exhale to residual lung volume and then inhale as forcefully as possible against a closed valve (8). The airway

pressure generated by this maneuver is called the *maximum inspiratory pressure* (PImax). Healthy adults can generate a negative PImax of 90 to 120 cm H_2O (see Table 27.2), with men generating higher pressures than women. The threshold PImax for predicting return to spontaneous breathing is 20 to 30 cm H_2O (negative pressure). ICU patients may have difficulty performing the maneuver needed to measure the PImax reliably, and this limits the utility of the measurement.

Predictive Value

The predictive value of the PIma x in one large study is shown in Figure 27.1 (6). When the PImax was less negative than 20 cm H_2O, no patient was successfully weaned. However, 40% of the patients with a PImax that was more negative than 20 cm H_2O also did not wean. Therefore, a PImax that is below threshold is valuable for identifying patients who will not tolerate spontaneous breathing, but a PImax that is above threshold has little predictive value.

THE SPONTANEOUS BREATHING TRIAL

The spontaneous breathing trial (SBT) can be conducted while the patient is still connected to the ventilator circuit, or the patient can be removed completely from the ventilator and allowed to breathe from an independent source of oxygen. The latter technique is often referred to as a *T-piece trial* because the breathing circuit is shaped like the letter *T* (see Figure 27.3). The advantages and disadvantages of each method are described next. There is no evidence that either method is superior to the other for allowing patients to be removed from the ventilator permanently (1).

Breathing Through the Ventilator

Spontaneous breathing trials are usually conducted while the patient is attached to the ventilator. The advantage of this method is the ability to monitor the tidal volume and respiratory rate during spontaneous breathing to detect the rapid and shallow breathing that often signals failure to sustain spontaneous breathing. The drawback with this method is an increased work of breathing, which is due to: 1) the negative pressure that must be generated to open an actuator valve in the ventilator and receive the inhaled oxygen mixture, and 2) the resistance created by the ventilator tubing between the patient and the ventilator. To counteract this increased work of breathing, *pressure-support ventilation* (described in Chapter 25) is used routinely during spontaneous breathing trials.

Pressure-Support Ventilation

The goal of pressure-support ventilation (PSV) during a spontaneous breathing trial is to add enough inspiratory pressure to reduce the work of breathing through the endotracheal tube and ventilator circuit without augmenting the spontaneous tidal volume. A positive pressure of

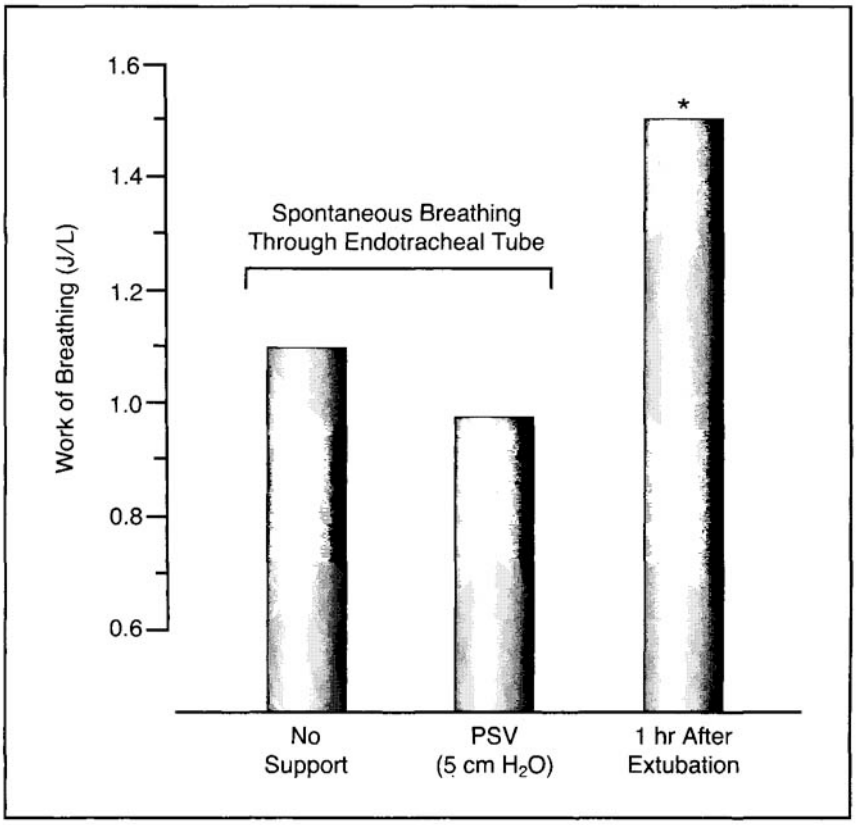

FIGURE 27.2 The work of breathing during spontaneous breathing trials conducted with and without the aid of pressure-support ventilation (PSV) at 5 cm H_2O, and one hour after extubation. Work is expressed in joules per liter (J/L). The asterisk indicates a significant difference from the other conditions ($p < 0.05$). All patients in this study were removed from ventilatory support without incident. (Adapted from Mehta S, Nelson DL, Klinger JR, et al. Prediction of post-extubation work of breathing. Crit Care Med 2000; 28:1341–1346, with permission.)

5-7 cm H_20 is routinely used for this purpose. The effect of this practice is shown in Figure 27.2. In this group of patients (who tolerated spontaneous breathing), the use of pressure support ventilation (PSV) at 5 cm H_20 is associated with a small but insignificant decrease in the work of breathing. Note also that the work of breathing is much higher after the endotracheal tubes are removed! (This observation is discussed again at the end of the chapter.)
The graph in Figure 27.2 indicates that: 1) inspiratory pressure support is not necessary during spontaneous breathing trials, and 2) breathing through endotracheal tubes and ventilator circuits is not associated with an increased work of breathing, at least in comparison to the early time period following extubation. These conclusions may only apply to

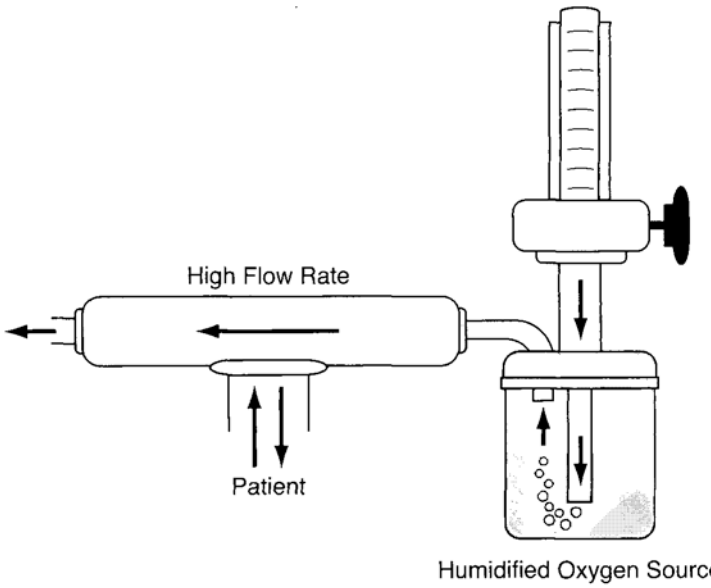

FIGURE 27.3 Diagram of the T-shaped circuit used for spontaneous breathing trials when the patient is disconnected from the ventilator.

patients who can sustain spontaneous breathing (like the patients used in the study in Figure 27.2), but they may apply to all patients as well.

Breathing Through the T-Piece

Spontaneous breathing trials can be conducted with the patient disconnected from the ventilator using the T-shaped circuit design shown in Figure 27.3. The inhaled gas is delivered at a high flow rate (greater than the patient's inspiratory flow rate) through the upper arm of the apparatus. The high flow rate serves two purposes. First, it creates a "suction effect" that carries the exhaled gas out of the apparatus and prevents rebreathing of exhaled gas. Second, it prevents the patient from inhaling room air from the exhalation side of the apparatus.

The disadvantage ofT-piece trials is the inability to monitor the patient's spontaneous tidal volume and respiratory rate. The assumed advantage is less work of breathing when compared to spontaneous breathing while connected to the ventilator. T-piece trials are often preferred in patients who are breathing rapidly or have a high minute ventilation.

Protocol

The recommended protocol is to allow 30 to 120 minutes for the initial trial of spontaneous breathing (1). Although not mentioned, it is wise to increase the $FI0_2$ by 10% for the period of spontaneous breathing. Success or failure is judged by a combination of patient appearance (comfortable

versus labored breathing), breathing pattern (Le., the presence or absence of rapid, shallow breathing) and gas exchange (e.g., ability to maintain $Sa0_2$ > =90% and end-tidal $PC0_2$ normal or constant throughout the trial).
About 80% of patients who tolerate 30 to 120 minutes of spontaneous breathing can be permanently removed from the ventilator (1). For patients with brief periods of ventilatory support (e.g., cardiac surgery patients), a successful one-hour period of spontaneous breathing is enough to consider extubation. In patients who have received prolonged mechanical ventilation (one week or longer), longer periods of spontaneous breathing are suggested before considering extubation. Our practice for patients who have been ventilator-dependent for one week or longer is to permit at least 8 hours (and sometimes up to 24 hours) of spontaneous breathing before deciding to remove the ventilator from the room.

An Approach to Rapid Breathing

Patients who are being removed from ventilatory support often experience anxiety and a sense of dyspnea, and these patients often breathe rapidly even though they are ventilating adequately (9,10). Therefore, for patients who begin to breathe rapidly during the spontaneous breathing trials, it is important to distinguish anxiety from ventilatory failure requiring continued mechanical ventilation. The flow diagram in Figure 27.4 shows a simple approach to this problem using the patient's spontaneous tidal volume. Anxiety is accompanied by hyperventilation, where the tidal volume is usually increased, whereas ventilatory failure is usually accompanied by a decrease in tidal volume (rapid shallow breathing). Therefore, an increase in tidal volume suggests anxiety, whereas a decrease in tidal volume suggests continued need for ventilatory support. When the tidal volume is unchanged or increased, the arterial $PC0_2$ can also be helpful i.e., a decrease in arterial $PC0_2$ indicates that ventilation is adequate and suggests that anxiety is the problem. In this case, judicious use of sedation should be considered. On the other hand, **if** the arterial $PC0_2$ is normal or increasing, the patient should be returned immediately to ventilatory support. (Remember that a normal arterial $PC0_2$ in the face of a high minute ventilation is a sign of ventilatory failure.)

Abdominal Paradox

The respiratory movements of the abdomen can be a useful sign of success or failure to tolerate spontaneous breathing. Normally, abdominal movements during breathing provide information about the functional integrity of the diaphragm (11). When the diaphragm contracts, it descends into the abdomen and increases intraabdominal pressure. This pushes the anterior wall of the abdomen outward. However, when the diaphragm is weak, the neg'ltive intrathoracic pressure created by the accessory muscles of respiration pulls the diaphragm upward into the thorax. This decreases the intraabdominal pressure and causes a paradoxical inward displacement of the abdomen during inspiration. This

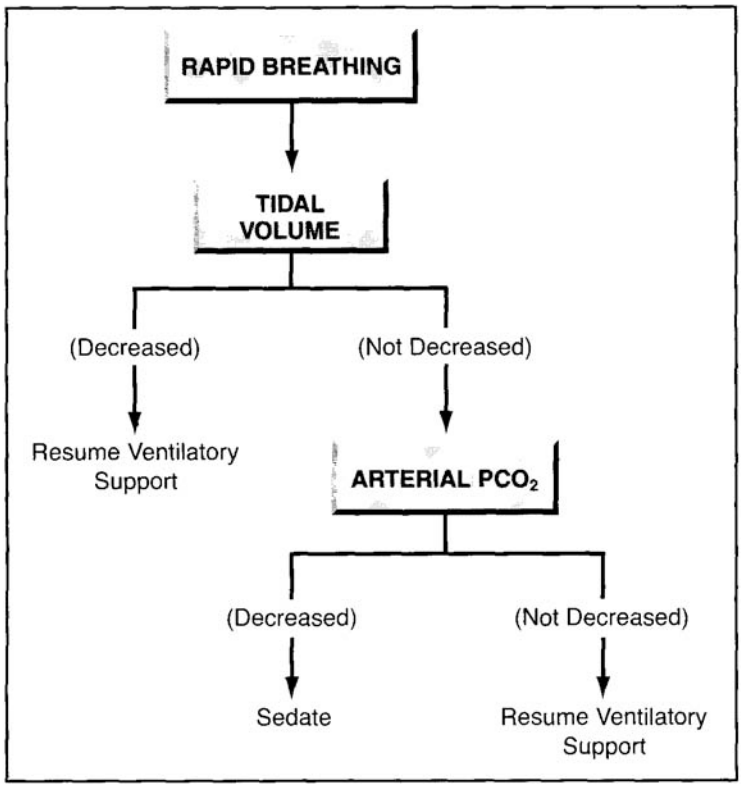

FIGURE 27.4 An approach to the patient who is breathing rapidly during a spontaneous breathing trial.

phenomenon is called *abdominal paradox,* and it is sign of a diaphragmatic weakness if it occurs during quiet breathing.

During labored breathing, contraction of the accessory muscles of inspiration can overcome the contractile force of the diaphragm and pull the diaphragm up into the thorax during inspiration. This results in abdominal paradox in the face of an actively contracting diaphragm. Because quiet breathing may be uncommon during a spontaneous breathing trial, the appearance of abdominal paradox during weaning is not necessarily a sign of diaphragm weakness. However, it is a sign of labored breathing, and should prompt immediate return to full ventilatory support.

FAILURE OF SPONTANEOUS BREATHING

Failure to sustain spontaneous breathing is usually a sign that the pathologic condition requiring ventilatory support needs more time to improve. Therefore, patients who require continued ventilatory support should have a daily trial of spontaneous breathing to identify when the condition

has improved enough to permit sustained spontaneous breathing. This method allows the earliest possible detection of when spontaneous breathing will be tolerated (12-14). The following conditions can add to the difficulty of discontinuing mechanical ventilation, and each of these conditions should be considered in patients who fail spontaneous breathing trials.

Low Cardiac Output

The transition from positive-pressure ventilation to negative-pressure spontaneous breathing can result in a decrease in cardiac output (caused by an increase in left-ventricular afterload) (15). This can add to the difficulty of spontaneous breathing by promoting pulmonary congestion (which reduces lung compliance) and also by impairing diaphragm function. The diaphragm depends heavily on cardiac output (like the heart, the diaphragm maximally extracts oxygen), and a drop in cardiac output can decrease the strength of diaphragmatic contractions (16). Low cardiac output can also promote hypoxemia through a decrease in mixed venous 0z saturation (see Figure 19.5 in Chapter 19). These deleterious effects of a low cardiac output should be considered in patients with cardiac dysfunction who do not tolerate spontaneous breathing. Some simple methods of detecting a low cardiac output are described next.

Detecting a Low Cardiac Output

The following are two measurements can be used to detect a low cardiac output in patients who are not tolerating spontaneous breathing. Neither method requires a pulmonary artery catheter. Each measurement should be performed at the beginning of a spontaneous breathing trial, and repeated when the patient is experiencing difficulty.

02 EXTRACTION (Sa02-Sv02) A decrease in cardiac output will increase 0z extraction in the systemic capillaries, and this will be reflected in an increase in the (SaO_2 - $Sv0_2$) difference (see Chapter 2). The $Sa0_2$ is easily monitored with a pulse oximeter, and the SvO_z can be measured with a central venous catheter in the superior vena cava. (See Chapter 20 for a description of the difference between "mixed venous" and "central venous" 02 saturation.) The (SaO_z - SvO_z) is normally about 25%, and it will increase to 50% in low output states.

ARTERIAL-END TIDAL $PC0_2$ GRADIEINT: ($PaC0_2$ - $PnC0_2$). The arterial and end-tidal PCO? are equivalent in healthy subjects. A decrease in cardiac output will de-crease end-tidal PCO_z relative to arterial PCO_z. and this will be reflected in an increase in the ($PaCO_z$ - $PETCO_z$) difference. An increase in dead-space ventilation from lung disease will also increase the ($PaCO_z$ - $PETCO_z$) gradient, so it is important to monitor changes in the gradient during the spontaneous breathing trial. (See Chapter 20 for a description of the end-tidal PCO_z measurement.)

Myocardial Ischemia

In patients with coronary artery disease, failure to tolerate spontaneous breathing is occasionally associated with acute myocardial ischemia (17).

The ischemia is often silent, and will not be detected without an electrocardiogram. Therefore, an ECG should be obtained in patients with coronary artery disease who do not tolerate spontaneous breathing.

Continuous Positive Airway Pressure

For patients who show evidence of a reduced cardiac output during spontaneous breathing, continuous positive airway pressure (CPAP) can help by eliminating the increased afterload caused by negative intrathoracic pressures. (See Chapter 25 for a description of CPAP.) Noninvasive ventilation with CPAP has been used effectively in patients with acute cardiogenic pulmonary edema, and can reduce the need for intubation and mechanical ventilation in these patients (18). The same benefits can be gained by adding CPAP to facilitate removal from mechanical ventilation. In patients who show a favorable response, CPAP can be continued after extubation (by face mask) if necessary.

Overfeeding

An increase in the daily intake of calories is associated with an increase in metabolic CO_2 production, as demonstrated in Figure 27.5 (19). If the daily intake of calories exceeds the daily caloric requirements (the resting

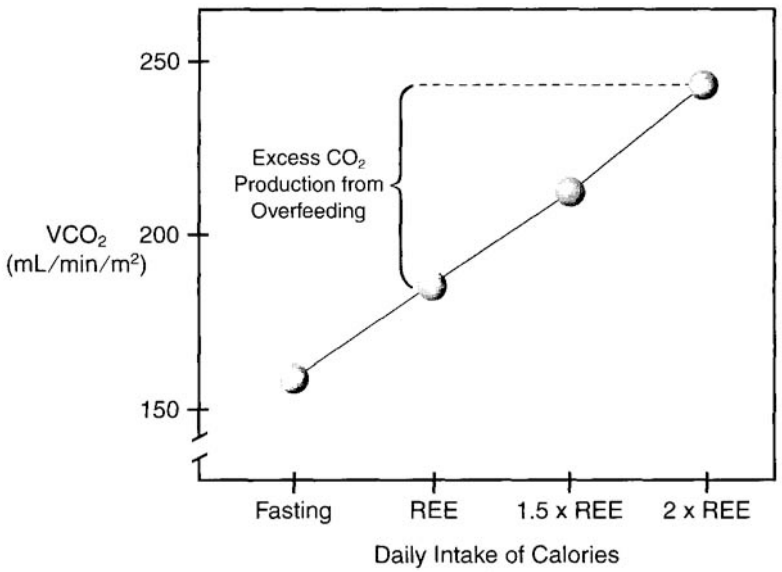

FIGURE 27.5 The influence of daily caloric intake on metabolic CO_2 production (VCO_2) in mechanically ventilated patients. REE is the resting energy expenditure (in kcal/24 hours). Each data point represents a mean value. (Data from Talpers S, Romberger DJ, Bunce SB, et al. Nutritionally associated increased carbon dioxide production. Chest 1992;102:551.)

energy expenditure in Figure 27.5), the excess CO_2 that is produced must be removed by the lungs. This requires an increase in minute ventilation, and this can add to the difficulty of sustaining spontaneous breathing in patients who are ventilator-dependent (19). To avoid this problem, ventilator-dependent patients should have their daily energy needs measured with indirect calorimetry (not estimated with conventional formulas) and their daily caloric intake should be matched to daily needs (see Chapter 45 for a description of how to measure daily energy needs).

Respiratory Muscle Weakness

The role of respiratory muscle weakness in failure to discontinue mechanical ventilation is unclear. The general belief is that the diaphragm becomes weak during mechanical ventilation (20), like muscles that are immobilized. However, the diaphragm is not a voluntary muscle that will stop contracting during mechanical ventilation. The diaphragm is controlled by the respiratory neurons in the lower brains tern, and these cells fire automatically throughout life. In fact, the output of these respiratory neurons to the diaphragm (called *ventilatory drive)* is increased in respiratory failure (21), and the activity of the diaphragm will likewise be increased.

Critical Illness Polyneuropathy and Myopathy

There are two conditions that can produce respiratory muscle weakness in critically **ill** patients: *critical illness polyneuropathy and myopathy.* These conditions often accompany cases of severe systemic sepsis with multiorgan failure, and they can prolong the need for mechanical ventilation (22). These conditions are described in Chapter 51. They are often first recognized when patients show a continued need for ventilatory assistance at a time when the primary condition that prompted mechanical ventilation is subsiding. The diagnosis is usually made by exclusion (although nerve conduction studies and electromyography can secure the diagnosis). There is no treatment for these disorders, and recovery takes weeks to months.

Electrolyte Depletion

Depletion of magnesium and phosphorous can impair respiratory muscle strength (23,24), and deficiencies in these electrolytes should be corrected for optimal performance during the spontaneous breathing trials.

TRACHEAL DECANNULATION

For patients who have proven that they no longer require ventilatory support, the next step is to remove the tracheal tube. There are 2 concerns prior to removing these tubes. First, is the patient able to protect the airway and clear secretions? Second, is there any evidence of laryngeal injury that would compromise breathing after the tube is removed?

Protecting the Airway

Patients should be alert and able to follow commands prior to removing tracheal tubes. The ability to protect the airway is determined with the gag and cough reflexes, and also by the volume of respiratory secretions. Cough strength can be assessed by placing a file card or piece of paper 12 cm from the end of the tracheal tube and asking the patient to cough. **If** wetness appears on the card, the cough strength is considered adequate (25). The absence of a cough or gag reflex will not prevent removal of tracheal tubes, but it will identify the patients who need to be watched closely after the tubes are removed.

Laryngeal Edema

Severe laryngeal edema is reported in as many as 40% of cases of prolonged translaryngeal intubation (26), and about 5% of patients can develop significant upper airway obstruction following extubation (27). Laryngeal edema is a particular concern in cases where the intubation was traumatic or required multiple attempts, or when multiple reintubations were necessary because of self-extubation. However, virtually any patient subjected to translaryngeal intubation can develop significant laryngeal edema.

The Cuff-Leak Test

The cuff-leak test is designed to identify patients with severe laryngeal edema prior to removal of the endotracheal tube. The test is performed while the patient is receiving volume-cycled ventilation, and it involves measuring the volume of gas exhaled through the endotracheal tube before and after deflating the cuff. **If** there is no obstruction at the level of the larynx, cuff deflation will cause exhaled gas to flow around the endotracheal tube, and the volume exhaled through the tube will decrease. Therefore, a decrease in exhaled volume after cuff deflation is used as evidence against the presence of a significant obstruction at the level of the larynx. However, there is no agreement about the magnitude of the volume change that is significant. Different studies have used volumes of 110 mL, 140 mL, and a 25% change in volume as the threshold that separates the presence and absence of significant upper airways obstruction (26-28).

One problem with the cuff-leak test is the lack of agreement on the volume change after cuff deflation that signifies a patent upper airway. This may not be possible because factors other than upper airway patency can influence the volume change after cuff deflation. One source of variability in exhaled volumes after cuff deflation is an inspiratory volume leak (29). Air that escapes out of the lungs during positive-pressure lung inflation will result in a decrease in exhaled volume through the endotracheal tube, and this volume will vary according to lung compliance and airways resistance. Therefore, the mechanical properties of the lungs are a source of individual variation in results of the cuff-leak test. Another source of variability is the diameter of the endotracheal tube relative to the diameter of the trachea. Tubes that fit snugly in the trachea can create

an obstruction to expiratory flow after cuff deflation that will be misinterpreted as indicating a significant upper airway obstruction.
Thus, there are multiple factors that can influence the results of the cuff-leak test, and this creates doubt about the predictive value of the test.

Fenestrated Tracheostomy Tubes
Laryngeal obstruction is also possible after removal of tracheostomy tubes (30). In this case, the laryngeal damage can be the result of the endotracheal tube that was present prior to tracheostomy, or can be caused by ischemic injury to the larynx during the tracheostomy procedure. Detection of laryngeal obstruction is sometimes possible using a fenestrated (windowed) tracheostomy tube like the one shown in Figure 27.6. When the inner cannula is removed, the fenestration allows airflow through the larynx. When the opening of the tube is capped and the cuff is deflated, the patient must breathe entirely through the mouth, and any difficulty in breathing in this situation could be the result of obstruction at the level of the larynx. When this occurs, direct laryngoscopy can be used to identify a source of laryngeal obstruction. Fenestrated tracheostomy tubes are also available without a cuff: these tubes can be used to test the patency of the upper airways, but they must be replaced by cuffed tubes if the patient is placed back on the ventilator.

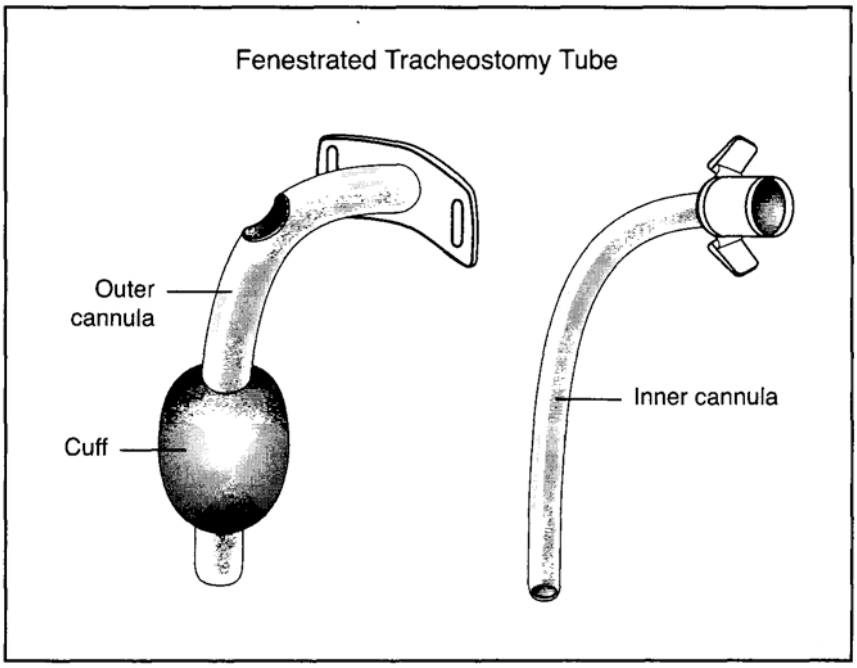

FIGURE 27.6 A fenestrated tracheostomy tube. When the inner cannula is removed, the fenestration (window) allows air to flow through the larynx, and this can be used to test the patency of the larynx before tracheal decannulation.

Steroids for Anythino That Swells?

The antiinflammatory actions of steroids (see Table 23.3) is somehow used to justify the use of steroids for cases of traumatic edema. One example of this *steroids for anything that swells* philosophy (31) is the use of steroids for traumatic cerebral edema, and another example is the use of steroids to treat intubation-induced laryngeal edema. Few studies have evaluated the effects of steroids on post-extubation laryngeal edema in adults, and the results of the available studies are inconclusive. One study shows no effect when intravenous methylprednisolone (40 mg) is given one hour before extubation (32), while another study shows a beneficial effect when intravenous methylprednisolone is given 24 hours before extubation (either as a single dose of 40 mg or in repeated doses of 40 mg every 6 hrs) (28). Further study is required, but it seems unlikely that one dose of steroids (or one day of therapy) will reverse the cumulative effects of days of trauma to the larynx.

The Postextubation Period

The Work of Breathing

The cross-sectional area of the glottis (the narrowest portion of the upper airway) is 66 mm^3 in the average-sized adult, whereas the cross-sectional area of an average-sized adult tracheal tube (8 mm internal diameter) is 50 mm^3 (33). The narrower cross-sectional area of the tracheal tube creates an increased resistance to airflow, so the work of breathing should be greater when breathing through endotracheal tubes. However, as shown in Figure 27.2, removal of endotracheal tubes is accompanied by an *increased* work of breathing (at least in the early period following removal). This is a consistent observation in patients who otherwise do well after extubation, and it is unexplained.

There is a popular perception that patients who do not tolerate spontaneous breathing while intubated might breathe easier after the tube is removed. The observation in Figure 27.2 proves otherwise, and shows that endotracheal tubes should never be removed based on the assumption that **it** will be easier for the patient to breathe.

Postextuhation Stridor

Laryngeal obstruction following extubation is signaled by the onset of stridor. Because the obstruction is extra thoracic, the stridor occurs during inspiration (i.e., the negative intrathoracic pressure is transmitted to the larynx and causes inspiratory narrowing). Postextubation stridor is not always an indication for immediate reintubation. If the patient is not *in extremis,* aerosolized epinephrine (2.5 mL of 1 % epinephrine) is a popular intervention. This is effective in treating post-extubation stridor in children (34), but similar efficacy in adults is unproven. A racemic mixture of epinephrine (which has equal amounts of the l-isomer and d-isomer) has been popular in this situation, but the standard (l-isomer) epinephrine preparation is equally effective (33).

Breathing a helium-oxygen (heliox) gas mixture has also been used in patients with postextubation stridor (27). Helium is less dense than air, and it flows more readily through regions of reduced cross-sectional area, where flow is turbulent. Although heliox has theoretical advantages in patients with postextubation laryngeal edema, the influence of heliox breathing in preventing reintubation has not been studied.

A FINAL WORD

The most important issue in discontinuing mechanical ventilation is not how to do it, but when to do it; i.e., recognizing when a patient is ready to try spontaneous breathing. A trial of spontaneous breathing is usually indicated when the patient is afebrile, alert, hemodynamically OK, and able to oxygenate while breathing 50% oxygen or less with a minimum of applied PEEP. Once these criteria are satisfied, start the spontaneous breathing trial and continue it as long as tolerated. If the patient does well, then consider extubation, but make sure the extubation can be done safely before proceeding. For patients who do not tolerate periods of spontaneous breathing, be patient and repeat the spontaneous breathing trial every day.

For patients who require prolonged ventilatory support, there is nothing you can do to make the patient less ventilator-dependent (other than treating the primary disease process). Your principal task is to provide general support while the primary disease process improves, and to use whatever measures are available to reduce the risk of complications that can keep the patient on a ventilator (e.g., acute pulmonary embolism).

REFERENCES