• The patient with severe airflow obstruction often develops hypoperfusion after institution of positive-pressure ventilation as a result of autoPEEP. This response to temporary cessation of ventilation and vigorous volume resuscitation, while measures are employed to reduce airflow obstruction and reduce the total minute ventilation.

• The patient with acute hypoxemic respiratory failure (AHRF) resulting from pulmonary edema benefits from lung-protective ventilation (6 mL/kg ideal body weight and rate approximately 30 breaths/min). The initial FiO2 of 1.0 can be lowered to nontoxic levels by raising positive end-expiratory pressure (PEEP), guided by pulse oximetry or measures of recruitment.

Regardless of the underlying process leading to mechanical ventilation, several principles guide ventilator settings and associated management. This chapter emphasizes preventing complications by using the “ventilator bundle”; avoiding lung injury (through overdistention or autoPEEP); limiting ventilator-induced diaphragm dysfunction (VIDD); understanding cardiopulmonary interactions; choosing modes and settings in relation to the underlying cause of respiratory failure; ensuring synchrony between patient and ventilator; and responding to crises.

Other chapters of this book are complementary to the information presented here. The pathophysiology of respiratory failure is broadly reviewed in Chap. 43; monitoring respiratory system waveforms of pressure and flow is delineated in Chap. 48; noninvasive ventilation is covered in Chap. 44; ventilator-induced lung injury is discussed in Chap. 51; and finally, several chapters (e.g., Chap. 52, Acute Respiratory Distress Syndrome; Chap. 54, Acute on Chronic Respiratory Failure; Chap. 55, Status Asthmaticus; Chap. 58, Restrictive Disease of the Respiratory System) discuss ventilatory support for specific problems.

PREVENTION: THE "VENTILATOR BUNDLE"

Mechanically ventilated patients are at risk for numerous complications related to the presence of the endotracheal tube, most notably ventilator-associated pneumonia (VAP), as well as adverse consequences of sedatives, paralytics, and immobilization. In response, the Institute for Healthcare Improvement promulgated the concept of “bundles,” a structured set of processes that, when performed collectively and reliably, improve outcomes (head of bed elevation; daily sedative interruption and readiness assessment; and steps to prevent venous thromboembolism and gastrointestinal hemorrhage). Ideally, these are evidence-based interventions but, in actual fact, include tactics with an uncertain impact on VAP. Effective preventive measures are more fully discussed in Chaps. 3, 4, 5, and 22, but are briefly summarized here.

Noninvasive ventilation should be used in appropriate candidates, since this reduces the risk of VAP in COPD patients and perhaps those in an immunocompromised state. For intubated patients, the head of the bed should be elevated to 30° to 45°. This intervention may or may not be effective in reducing VAP, but has the virtues of being simple, inexpensive, and logical, since much VAP is thought to arise from aspiration of gastric contents. Ventilator tubing should be changed on a time-based schedule. Sedative use should be limited and patients should be allowed to wake up daily, as discussed below and in Chap. 22. Ideally, the “wake-up” should be coordinated with a spontaneous breathing trial (SBT) to judge readiness for extubation. Prophylaxis against venous thromboembolism is indicated in most patients. Oral care with chlorhexidine is probably effective. There is no consensus regarding the effectiveness of selective decontamination of the digestive tract, subglottic suctioning, or avoidance of antacid therapies for prevention of VAP.
VENTILATOR-INDUCED LUNG INJURY

When the lungs of patients with the acute respiratory distress syndrome (ARDS) are distended excessively, through high tidal volumes or high positive end-expiratory pressure (PEEP), injury follows. Local pulmonary inflammation ensues, including areas of previously healthy lung, and systemic inflammation is seen, potentially causing distant organ failures. Increasing evidence suggests that ventilated patients with normal lungs (having central nervous system failure, eg, or undergoing surgery) may also be at risk when large (12 mL/kg) tidal volumes are used, especially in those with severe left ventricular dysfunction, impeding liberation from the ventilator.16

CHOOSING A VENTILATOR MODE

Technological innovations have provided a plethora of modes by which a patient can be mechanically ventilated. These have been developed with the hope of improving gas exchange, patient comfort, or speed of return to spontaneous ventilation. Aside from minor subtleties, however, nearly all modes allow full rest of the patient, on the one hand, or substantial exercise, on the other, while providing a suitable foundation for maintaining gas exchange and protecting the lung. Thus, in the great majority of patients, choice of mode is merely a matter of patient or physician preference. Noninvasive ventilation should be considered before intubation and ventilation in many patients who are hemodynamically stable and do not require an artificial airway, especially those with acute-on-chronic respiratory failure, postoperative respiratory failure, and cardiogenic pulmonary edema. Management of the patient ventilated noninvasively is discussed thoroughly in Chap. 44.

Choosing a ventilatory mode and settings appropriate for each individual patient depends not only on the physician’s goals (rest vs exercise; lung protection; limitation of autoPEEP; gas exchange), but also on knowledge of the mechanical properties of the patient’s respiratory system. Determining respiratory system mechanics is an integral part of ventilator management and a routine component of examination of the critically ill patient, discussed fully in Chap. 48 (Ventilator Waveforms). The intensivist can combine clinical information, chest radiography, lung ultrasound, and respiratory system mechanical properties to categorize the patient into one of four prototypes: (1) normal gas exchange and mechanics; (2) significant airflow obstruction (as in status asthmaticus or acute exacerbations of COPD); (3) ARDS; and (4) restriction of lung or chest wall. Appropriate initial ventilator settings and subsequent adjustments for each of these four states are discussed later in this chapter.

If full rest of the respiratory muscles is desired, it is incumbent on the physician to ensure that this is indeed achieved. Although some patients are fully passive while being ventilated (those with deep sedation, some forms of coma, metabolic alkalosis, sleep-disordered breathing), most patients will make active respiratory efforts, even on volume assist-control ventilation (VACV), at times performing extraordinary amounts of work. Unintended patient effort can be difficult to recognize but, aside from obvious patient effort, may be signaled by an inspiratory fall in intrathoracic pressure (as noted on a central venous or pulmonary artery pressure tracings or with an esophageal balloon), by triggering of the ventilator, or by a careful analysis of real-time flow and pressure waveforms, discussed more fully in Chap. 48. When there is evidence of unwanted patient effort, ventilator adjustments, psychological measures, pharmacologic sedation, and therapeutic paralysis can be useful. Ventilator strategies to reduce the patient’s work of breathing include increasing the minute ventilation to reduce PEEP (although this may run counter to other goals of ventilation, especially in patients with ARDS or severe obstruction), increasing the inspiratory flow rate, and changing the mode to pressure-targeted ventilation, in pressure-support ventilation (PSV) mode or pressure assist-control ventilation (PACV).

For most patients, however, some degree of triggering and work are desired because this is likely to reduce the degree of VIDD as described above. If some work of breathing on the patient’s part is desired, this can be achieved through any of the existing ventilatory modes. The amount of work done may be highly variable, however, and depends on the specific mode, the settings chosen within each mode, and the interaction between the patient and the ventilator. A recurring question during the time when a patient can carry some of the work of breathing is, “Can this patient breathe without ventilatory assistance?” This issue is more fully developed in Chap. 60, Liberation From Mechanical Ventilation.
## PRESSURE-TARGETED VERSUS VOLUME-TARGETED MODES OF VENTILATION

The terminology describing modes of ventilation can be very confusing and may vary from one company’s ventilator to another. In this chapter, we refer to volume-targeted modes as those in which the physician sets a desired tidal volume that the ventilator delivers, using whatever pressure is required, and pressure-targeted modes, in which the physician sets a desired pressure that the ventilator maintains, delivering a volume that depends on the settings, respiratory mechanics, and patient effort. Some modern modes (dual control modes, see below) attempt to blend pressure and volume targets. Few studies have compared modes directly except with respect to comfort, a measure generally favoring pressure-targeted modes. At the same time, comparative trials are plagued by the details of settings and these are often dissimilar between modes (biasing the study) or are not sufficiently specified in the methods. For this reason, modes are often chosen based on preference, personal experience, or institutional practice, rather than on evidence relating to meaningful outcomes.

The differences between volume-targeted and pressure-targeted modes are fewer than often appreciated, since volume and pressure are related through the mechanical properties of the respiratory system, most notably the respiratory system compliance (Crs). For example, a passive patient with a static Crs of 50 mL/cm H2O ventilated on VACV at a tidal volume of 500 cc with no PEEP (or autoPEEP) will have a plateau airway pressure (Pplat; see Chap. 48) of about 10 cm H2O, whereas the same patient ventilated on PACV at 10 cm H2O can be expected to have a tidal volume (VT) close to 500 cc. The difference to the patient between these modes may be quite trivial, often amounting to small differences in inspiratory flow profile. Thus, while physicians’ comfort level with volume-targeted and pressure-targeted modes may be very different, the modes can be similar because they are tied to each other through the patient’s Crs.

### CONVENTIONAL MODES OF VENTILATION

In the following descriptions, each mode is first illustrated for a passive patient, such as following muscle paralysis, and then for the more common situation in which the patient plays an active role in ventilation. On some ventilators tidal volume (VT) can be selected by the physician or respiratory therapist, whereas on others a minute ventilation and respiratory pressure (Pplat) are set by default or user-configured.

**Pressure Targeted Modes:** In pressure-targeted modes, a fixed inspiratory pressure (P) is applied to the patient, whatever the resulting VT. Depending on the particular ventilator, the physician may have to specify the actual level of P, or, alternatively, the increment in pressure over the expiratory pressure (PEEP). Ventilators designed primarily for noninvasive ventilation often require setting P and PEEP independently, while most ICU ventilators require setting the PEEP and an inspiratory pressure increment. For example, the following settings are identical: 1. P = 20 cm H2O; PEEP = 5 cm H2O (noninvasive ventilator); or 2. Pressure increment (eg, “pressure-support” or “pressure-control”) = 15 cm H2O; PEEP = 5 cm H2O. In this chapter, we will specify inspiratory pressure (P) and expiratory pressure (PEEP) pressures to avoid confusion.

In pressure-targeted modes, the VT is predictable (again, passive patient) when the Crs is known:

\[ VT = (P - PEEP) \times Crs \]

assuming time for equilibration between P and alveolar pressure (Pav) and the absence of autoPEEP (both of these assumptions are often not true in patients in the ICU; see below).

Compared with volume-targeted modes, a potential advantage of pressure-targeted ventilation is greater physician control over the maximal alveolar pressure (Pav) in passive patients, although it should be emphasized that a “safe” maximal alveolar pressure is not known. Further, when patients are active, Pav does not represent the transpulmonary pressure, meaning that gross overdistention of lung is possible on pressure-targeted modes despite modest ventilator pressures. In addition, the same reduction in maximal alveolar pressure can be achieved using volume-targeted modes, simply by limiting tidal volume, as has been shown in ALI/ARDS patients. Nevertheless, pressure-targeted modes make such a lung protection strategy easier to carry out by dispensing with the need to repeatedly determine Pplat and periodically adjust the Pav.

Pressure-targeted modes also allow the patient greater control over inspiratory flow rate and therefore potentially increased comfort. On the other hand, during lung protective ventilation, pressure modes (including pressure-regulated volume control, see below) did not reduce work of breathing compared to volume assist-control and did not allow precise control of tidal volume. A disadvantage of pressure-targeted modes is that changes in respiratory system mechanics (eg, increased airflow resistance or lung stiffness) or patient effort may decrease the minute ventilation, necessitating alarms for adequate ventilation. Also, the mechanics cannot be determined readily and partitioned as described in Chap. 48 without switching modes, inserting an esophageal balloon, or using more complex algorithms.

### Pressure Assist-Control Ventilation (PACV)

In the passive patient, ventilation is determined by f, the inspiratory pressure increment (Pav – PEEP), inspiratory to expiratory (I:E) ratio, and Crs. In patients without severe obstruction given a sufficiently long TI, there is equilibration between the ventilator-determined Pav and Pav so that inspiratory flow ceases (Fig. 49-1A). In this situation, tidal volume is highly predictable, based on Pav (= Pav) and the mechanical properties of the respiratory system (Crs). In the presence of severe obstruction or a Tl, is too short to allow equilibration between ventilator and alveoli, VT will fall below that predicted based on Pav and Crs (see Fig. 49-1A). One of the advantages of PACV is that it may facilitate ventilation with a lung protective strategy. For example, alveolar overdistention can be prevented by ensuring that Pav never exceeds some threshold value (this is often taken to be 30 cm H2O, but a truly safe level is unknown) by simply setting Pav (alternatively, PEEP + PSV) to the desired upper limit. Inspiratory activity can raise the transpulmonary pressure well above a safe level, despite a modest Pav, threatening lung protection. During PACV, Tl and Pav can be set by the physician and may not approximate the patient’s Tl and Pav.

When the patient is active, the tidal volume reflects patient effort and the patient may trigger additional breaths. When the patient makes inspiratory efforts synchronized with machine inspiration, the tidal volume is generally greater than that predicted from the Crs and Pav and may exceed targets for lung protection. However, dysynchrony or expiratory effort during machine inspiration may reduce VT below that otherwise expected. Special care must be taken to adjust Tl to the individual patient (Fig. 49-2); otherwise, heavy sedation is typically needed.

When unphysiologic settings are intentionally chosen, as when the physician desires an unusually long Tl (Tl longer than Tl results in inverse ratio ventilation, IRV), deep sedation or therapeutic paralysis is often given.

### Pressure-Support Ventilation (PSV)

The patient must trigger the ventilator in order to activate this mode, so PSV is not applied to passive patients. Ventilation is determined by Pav – PEEP, patient-determined f, patient effort, and the patient’s mechanics. Once a breath is triggered, the ventilator attempts to maintain Pav using whatever flow is necessary to achieve this. Eventually, flow begins to fall due to cessation of the patient’s inspiratory effort combined with increasing elastic recoil of the respiratory system as lung volume rises. The ventilator maintains Pav until inspiratory flow falls an arbitrary amount (eg, to 20% of initial flow) or below an absolute flow rate (set by default or user-configured).

It is useful to first consider what happens if the patient were to trigger the ventilator and then remain passive (an artificial situation). Tidal volume would be determined by Pav and the (largely static) mechanical properties of the respiratory system, as during PACV (see Fig. 49-1B). More typically, the patient makes an effort throughout inspiration, in which case VT is determined, in part, by the degree of effort (see...
When a breath is triggered, \( P_{ao} \) rises to the set level (PI) with flow and VT depending on the PI breath, a more powerful but briefer inspiratory effort is made, shortening the TI but generating a larger VT than during the passive breath. \( P_{ao} \) is airway opening pressure; \( V\dot{ } \) is flow; and \( V \) is volume.

When the patient makes a moderate but prolonged inspiratory effort. The \( P_{ao} \) remains at the set inspiratory level as long as patient effort maintains flow, and a much longer TI and VT result. In the final breath, a more powerful but briefer inspiratory effort is made, shortening the TI, but generating a larger VT, than during the passive breath. \( P_{ao} \) is airway opening pressure; \( V\dot{ } \) is flow; and \( V \) is volume.

### FIGURE 49-1.
A. Pressure-control ventilation of a muscle-relaxed patient showing the effects of changed inspiratory resistance. The left-hand panel shows a pressure-control breath with normal resistance, during which \( P_{r} \) equilibrates with \( P_{av} \) before the inspiratory cycle is terminated (left arrow), flow ceases, and tidal volume can be predicted from the \( P_{r} \) and Crst \( (V_{t} = \text{Cst} \times P_{r} − \text{PEEP}) \). In the right-hand panel, inspiratory resistance is elevated. Note that at the same \( P_{r} \), inspiratory flow is reduced, the tidal volume is not reached until the inspiratory phase is terminated (right arrow), and the tidal volume (solid line) falls below that predicted by Crst and \( P_{r} − \text{PEEP} \) (dotted line). \( P_{ao} \) is airway opening pressure; \( V\dot{ } \) is flow; \( V \) is volume. B. Pressure-support ventilation. When a breath is triggered, \( P_{ao} \) rises to the set level (PI) with flow and \( V\dot{ } \) depending on the \( P_{r} − \text{PEEP} \), respiratory system mechanics, and patient effort. The first breath shown represents a patient who triggers the ventilator and then remains fully passive (a hypothetical circumstance used here for contrast with the usual patient efforts shown in the next two breaths). As long as there is no significant airflow obstruction, \( V \) nearly reaches the volume that would be predicted based on the compliance of the respiratory system \( (V_{t} = \text{Cst} \times P_{r} − \text{PEEP}) \). During the middle breath shown, the patient makes a moderate but prolonged inspiratory effort. The \( P_{ao} \) remains at the set inspiratory level as long as patient effort maintains flow, and a much longer TI and VT result. In the final breath, a more powerful but briefer inspiratory effort is made, shortening the TI, but generating a larger VT, than during the passive breath. \( P_{ao} \) is airway opening pressure; \( V\dot{ } \) is flow; and \( V \) is volume.

Airway opening pressure (\( P_{ao} \)) and lung volume (\( V \)) during VACV ventilation of a patient who is periodically triggering the ventilator. The second breath was set to be delivered at the time marked by the second arrow; instead, the patient lowers the \( P_{ao} \), triggering the ventilator at the time marked by the first arrow, thereby increasing the respiratory rate above the default value, decreasing the expiratory time (TE), and increasing the I:E ratio. D. Airway opening pressure (\( P_{ao} \)), flow (\( V\dot{ } \)) and lung volume (\( V \)) during SIMV. Breath 1 (a mandatory breath) is not triggered by the patient, who remains fully passive. \( V \) and \( V\dot{ } \) are determined by the ventilator, while the \( P_{ao} \) reflects the passive mechanical characteristics of the respiratory system. The shaded rectangle near the second breath denotes the interval during which the ventilator is programmed to synchronize with the patient's inspiratory effort, delivering the mandatory breath slightly ahead of schedule. At the end of this time interval (arrow), a mandatory breath would have been delivered (dotted tracing) if the patient had not triggered the ventilator. The synchronized breath (breath 2) has the same volume and flow as a mandatory breath. The \( P_{ao} \) may not be the same as during a passive breath because of continued patient effort throughout inspiration. The third breath (3) is initiated before the synchronization interval at \( x \) and is therefore not assisted. Flow and tidal volume are totally determined by the patient's effort and mechanics. These breaths are typically shorter and smaller (as indicated) than the mandatory breaths. When the patient fails to trigger another breath within the next synchronization window, another mandatory breath (4) is delivered.

**Volume-Targeted Modes**: During volume-targeted ventilation, a volume is delivered to the patient whatever the pressure required (within the limits of the high pressure alarm). The physician generally also sets an inspiratory flow rate (indirectly determining the TI) as well as f. In volume-targeted modes, the Pplat is predictable (again, passive patient) when the Crs is known:

\[
P_{\text{plat}} = V_{t}/\text{Crs} + \text{PEEP}
\]

where PEEP includes also autoPEEP.

Compared with pressure-targeted modes, a potential advantage of volume-targeted ventilation is greater control over the total minute ventilation, since \( V_{t} \), does not depend on potentially changing patient effort or respiratory system mechanical properties. Also, it is easy to characterize the respiratory system mechanics by measuring Ppk and Pplat, thereby helping to follow the patient's progress or response to therapies.
Volume Assist-Control Ventilation (VACV) The set parameters of the volume assist-control mode are the inspiratory flow rate (V˙), frequency (f), and tidal volume (VT). On some ventilators, one must set the total minute ventilation and rate, thereby determining tidal volume and indirectly determining VT. In the passive patient, the ventilator delivers f [f = 60 s/(TI + TE)] equal breaths per minute, each of VT volume. VT and V˙ determine the inspiratory time (TI), expiratory time (TE), and the inspiratory-expiratory (I:E) ratio. Pplat is related to the VT and the compliance of the respiratory system, whereas Ppk-Pplat includes contributions from V and inspiratory resistance (see Fig. 49-1C).

The active patient can trigger extra breaths by exerting an inspiratory effort exceeding the preset trigger sensitivity, each at the set VT and V. and thereby change TI, TE, and I:E ratio and (potentially) create or increase autoPEEP. Typically, each patient will display a preferred rate for a given VT, and will trigger all breaths when the controlled ventilator frequency is set a few breaths per minute below the patient's rate. In this way, the control rate serves as an adequate support should the patient stop initiating breaths. When high inspiratory effort continues during the ventilator-delivered breath, the patient may trigger a second, superimposed (“stacked”) breath (rarely, a third as well). The total tidal volume of this breath is determined by the point in the first breath at which the second was triggered, so the total VT can range from the set VT to twice VT.21

Typically, the patient performs inspiratory work during a VACV breath.21 This may not be obvious despite careful examination of the patient unless measures of intrathoracic pressure (esophageal pressure, central venous pressure) are available, or the inspiratory pressure waveform is examined carefully (Chap. 48). Effort at the end of the breath will affect the Ppk and Pplat, making determination of respiratory system mechanics unreliable. Lowering f at the same VT generally has no effect on work of breathing (in contrast to SIMV, discussed below) when the patient is initiating all breaths.

Synchronized Intermittent Mandatory Ventilation (SIMV) In the passive patient, intermittent mandatory ventilation (IMV) cannot be distinguished from controlled ventilation in the VACV mode. Ventilation is determined by the mandatory f, VT, and V. However, if the patient is not truly passive, he or she may perform respiratory work during the mandatory breaths. More to the point of the SIMV mode, the patient can trigger additional breaths (see Fig. 49-1D). If this triggering effort comes in a brief, defined interval before the next mandatory breath is due, the ventilator will deliver the mandatory breath ahead of schedule in order to synchronize (SIMV) with the patient's inspiratory effort. If a breath is initiated outside the synchronization window, VT, V, and I:E are determined by patient effort and respiratory system mechanics (see Fig. 49-1D), not by ventilator settings. The spontaneous breaths tend to be of small volume, as depicted in Figure 49-1D, and are highly variable from breath to breath. The SIMV mode has been used historically to gradually augment the patient's work of breathing by lowering the mandatory breath f (or VT), driving the patient to breathe more rapidly in order to maintain adequate ventilation, but this approach appears to prolong “weaning”23,24 Although this mode continues to be used widely (typically with added PSV), there is little rationale for it and SIMV is falling out of favor.

Mixed Modes: Some ventilators allow combinations of modes, most commonly SIMV plus PSV. There is little reason to use such a hybrid mode, although some physicians use the SIMV mode as a means to add sighs to PSV, an option not otherwise generally available. Since SIMV plus PSV guarantees some backup minute ventilation (which PSV does not), this mode combination may have value in occasional patients at high risk for abrupt deterioration in central drive.

Dual-Control Modes: The sophisticated microprocessors included with modern ventilators allow remarkably complex modes of ventilation. These modes typically try to meld the best features of volume- and pressure-targeted modes. Some cause a switch of modes between breaths (eg, pressure-regulated volume control, PRVC; volume support, VSV) or within a breath (eg, volume-assured pressure support, VAPS). In general, these modes are complex and their effects may vary greatly depending on the details of the patient's effort. None have been shown safer nor more useful than more conventional modes. The greatest problem with such newer modes is that they are very complex, the algorithm describing their function is not usually understood by practitioners, and they change during a breath, or from breath to breath, depending on patient effort, sometimes in ways that can provoke unanticipated effects.

Pressure-Regulated Volume Control (PRVC) This is a pressure-targeted mode with a set VT (ie, it is time-cycled) in which the ventilator compares the VT with a physician-set tidal volume and automatically and gradually adjusts P of subsequent breaths in order to deliver the desired VT. A downside of PRVC is that as patient effort increases, the ventilator reduces support. Further, tidal volume is not controlled as precisely as with a volume-targeted mode.19 Proponents argue that this mode provides the benefits of pressure-targeted modes, while at the same time guaranteeing VT, but any benefits have not been demonstrated.

Volume Support (VSV) Volume support is a pressure-targeted mode in which P is automatically varied to gradually bring VT in line with the desired VT over several breaths, differing from PRVC in that VT is not set but, rather, depends on patient effort as in PSV. It is unknown whether this mode speeds or impedes weaning.

Volume-Assured Pressure Support (VAPS) This mode begins as PSV but, if a desired VT is not met, the ventilator switches to VACV within the same breath in order to guarantee VT. As with many dual-control modes, the physician delegates decision-making to the ventilator. Complex adjustments and their potentially detrimental effects on the patient may come into play at any time of day or night, depending on changes in mechanical properties of the respiratory system, the patient's level of consciousness, comfort, or neuromuscular competence.

Continuous Positive Airway Pressure (CPAP): Continuous positive airway pressure is not a mode of ventilation but rather a means of raising functional residual capacity while allowing the patient to breathe spontaneously. This approach is frequently used when assessing the patient's ability to breathe without ventilatory assistance. Advantages of CPAP over T-piece breathing is that oxygenation may be improved, ventilator alarms (such as low minute ventilation and apnea) remain in place, the patient's spontaneous tidal volumes and rate can be easily read from
A peak inspiratory flow rate (V) of 1 L/s (60 L/min) is a common initial setting, but this may require adjustment upward (for patient comfort or to lengthen expiratory time) or downward. Increased V may have a stimulatory effect on respiratory rate in active patients, paradoxically shortening T$_e$, although the impact on respiratory rate and work of breathing after the first few seconds has not been studied. Lowering the peak V will reduce P$_{pk}$ when there is significant resistance to airflow but in this setting will usually worsen autoPEEP, as described above. One often overlooked adverse consequence of setting peak V at less than the patient wishes is that the patient will actively inspire against the ventilator, increasing the respiratory work.

**Triggered Sensitivity**

In modes that allow the patient to trigger extra breaths, either Pao must be drawn below a preset threshold (pressure-triggering) or flow must be inspired from the circuit (flow-triggering) in order to initiate the breath. Flow triggering has been reported to reduce the work of breathing below that using conventional demand valves, but this is not seen consistently. Further, flow-triggering does not solve the problem of triggering when autoPEEP is present (Fig. 49-4). In several instances we have seen physicians suspect machine malfunction and even change the ventilator when they are confronted with an obviously struggling patient seemingly unable to get a breath despite a “minimal” trigger threshold. The solution for this problem is to eliminate the cause for autoPEEP, sedate the patient, or use externally applied PEEP to counterbalance the autoP-EEP (which only occasionally increases autoPEEP, risking barotrauma and hypotension)20-21 (see Chap. 54). Alternatively, one can use neurally adjusted ventilatory assist (NAVA) to better synchronize patient and ventilator, both for breath initiation and termination.22

**Unconventional Ventilatory Modes**

Airway Pressure-Release Ventilation: Airway pressure-release ventilation (APRV) consists of continuous positive airway pressure (CPAP) that is intermittently released to allow a brief expiratory interval, producing a form of inverse ratio ventilation.25 It has been applied to patients with acute lung injury25 and has proven effective in maintaining oxygenation.
and assisting ventilation. Thus it is typically used as a rescue mode for the patient with refractory hypoxemia due to severe ARDS. Two concerns have arisen. First, tidal volumes are generally much higher than 6 mL/kg, suggesting that APRV may overdistend the lung. A further concern relates to the fact that since the lung cycles tidally below rather than above the volume determined by CPAP, this mode probably encourages repeated recruitment and derecruitment of flooded and collapsed alveoli. Although this point is controversial, the weight of evidence suggests that APRV may amplify ventilator-induced lung injury and worsen outcomes.35,36

Proportional-Assist Ventilation: Proportional-assist ventilation is intended only for spontaneously breathing patients. The goal of this novel mode is to attempt to normalize the relationship between patient effort and the resulting ventilatory consequences.37,38 The ventilator adjusts $P_i$ in proportion to patient effort both throughout any given breath and from breath to breath. This allows the patient to modulate his or her breathing pattern and total ventilation. This is implemented by monitoring instantaneous $V$ and volume $(V)$ of gas from the ventilator to the patient and varying the $P_c$ as follows:

$$P_i = f_1 \times V + f_2 \times V$$

where $f_1$ and $f_2$ are selectable functions of volume (elastic assist) and flow (resistive assist), values which can be estimated from the patient’s respiratory mechanics. Potential advantages of this method are greater patient comfort, lower $P_{pk}$, and enhancement of the patient’s reflex and behavioral respiratory control mechanisms. On the other hand, PAV can amplify instabilities in the patient’s breathing rhythm, as in the common instance of periodic (Cheyne-Stokes) respiration.

High-Frequency Ventilation (HFV): Several modes of ventilation have in common the use of tidal volumes smaller than the dead space volume. Gas exchange does not occur through convection as during conventional ventilation but through bulk flow, Taylor diffusion, molecular diffusion, nonconvective mixing, and possibly other mechanisms. These modes include high-frequency oscillatory ventilation (HFOV) and high-frequency jet ventilation (HFJV). Theoretical benefits of HPV include lower risk of barotrauma due to smaller tidal excursions, improved gas exchange through a more uniform distribution of ventilation, and improved healing of bronchopleural fistulas. Two trials in patients with ARDS have shown that, compared with conventional ventilation (using or encouraging 6 mL/kg), HFOV does not reduce38 mortality and includes, most notably, airway malposition, aspiration, and hypotension. Positive-pressure ventilation may reduce cardiac output, especially in patients with a low mean systemic pressure (eg, hypovolemia, venodilating drugs, decreased sympathetic tone from sedating drugs, neuromuscular disease) or a very high ventilation-related pleural pressure (eg, chest wall restriction, large amounts of PEEP, or obstruction causing autoPEEP). If hypotension occurs, autoPEEP should be sought and intravascular volume should be expanded rapidly while steps are taken to lower the pleural pressure (smaller tidal volumes, less minute ventilation). Meanwhile the FiO2, should be raised to 100%. If these steps do not rapidly restore the circulation, another complicating event (pneumothorax, myocardial ischemia) should be considered.

The Patient With Normal Respiratory Mechanics and Gas Exchange: Patients with normal lung mechanics and gas exchange can require mechanical ventilation (1) because of loss of central drive to breathe (eg, drug overdose or structural injury to the brainstem), (2) because of neuromuscular weakness (eg, high cervical cord injury, acute idiopathic myelitis, myasthenia gravis), (3) as an adjunctive therapy in the treatment of shock,39-42 or (4) in order to achieve hyperventilation (eg, in the treatment of elevated intracranial pressure following head trauma). Especially if the patient is at risk for ARDS, and perhaps even if not, tidal volumes (VACV mode) in the range of 6 to 8 mL/kg seem prudent, along with FiO2 of 0.5, respiratory rate around 20 breaths per minute, inspiratory flow rate of 50 to 60 L/min, and PEEP of 5 to 8 cm H2O. Alternatively, if the patient has sufficient drive and is not profoundly weak, PSV can be used. The level of pressure support is adjusted (usually to the range of 10 to 20 cm H2O above PEEP) to bring the respiratory rate down into the low twenties, usually corresponding to tidal volumes of about 450 cc. It is important to realize that PSV is mechanically supported but entirely spontaneous, with no machine “backup,” unless mixed with a mode such as SIMV. Thus hyperventilation may occur despite use of PSV if there is further deterioration of muscle strength or blunting of drive by disease or drugs. If gas exchange is entirely normal, the FiO2 likely can be lowered further based on pulse oximetry or arterial blood gas determinations. However, since right mainstem intubation, aspiration, and bronchospasm are relatively common complications of intubation, it is wise to initiate ventilation with the FiO2 at 0.5 or higher. Should hyperventilation be desired, the initial respiratory rate should be increased above 20, guided by capnography or blood gas analysis.

Soon after the initiation of ventilation, airway pressure and flow waveforms should be inspected for evidence of patient-ventilator dysynchrony or undesired patient effort (see Chap. 48). If the goal of ventilation is full rest, the patient’s drive can often be suppressed by increasing the inspiratory flow rate, frequency, or tidal volume. If such adjustments do not diminish breathing effort, despite hypocapnia, sedation may be necessary. If this does not abolish inspiratory efforts and full rest is essential (as in shock), muscle paralysis can be considered.

Patients With Dominant Airflow Obstruction: Two general types of patients come to mechanical ventilation for significant airflow obstruction; those with status asthmaticus (see Chap. 55) and those with exacerbations of chronic airflow obstruction (see Chap. 54). Rare alternative causes are inhalation injury or central airway lesions, such as tumor or foreign body, not bypassed with the endotracheal tube. In isolated upper airway injuries, assessment of the extent of damage is often possible by bronchoscopy shortly before or at the time of intubation.

Status Asthmaticus: Because the gas exchange abnormalities of airflow obstruction are largely limited to ventilation-perfusion mismatch, an FiO2 of 0.5 suffices in the majority of patients.43 Requirements for a higher FiO2 should prompt a search for an alveolar filling process or for lobar atelectasis. We have had success ventilating these patients with PSV, setting P, at 25 to 30 cm H2O (also with PEEP of 5 cm H2O). Since these patients are typically anxious, we often give narcotics to suppress drive, an approach that, when combined with high-level PSV, often leads to an unusual but stable pattern of breathing: tidal volumes greater than

**MANAGEMENT OF THE PATIENT**

**INITIAL VENTILATOR SETTINGS**

Initial ventilator settings depend on the goals of ventilation (eg, full respiratory muscle rest vs partial exercise), the patient’s respiratory system mechanics, and minute ventilation needs. Although each critically ill patient presents myriad challenges, it is possible to identify four subsets of ventilated patients: (1) the patient with normal lung mechanics and gas exchange, (2) the patient with predominant airflow obstruction, (3) the patient with acute hypoxemic respiratory failure, and (4) the patient with restrictive lung or chest wall disease. Specific recommendations regarding ventilator settings are detailed more fully in Chap. 52 (Acute Lung Injury and the Acute Respiratory Distress Syndrome), Chap. 54 (Acute-on-Chronic Respiratory Failure), Chap. 55 (Status Asthmaticus), and Chap. 58 (Restrictive Disease of the Respiratory System) but are reviewed here briefly, along with guidelines for ventilating patients with normal respiratory system mechanics.

In all patients the initial FiO2 should usually be 0.5 to 1.0 to assure adequate oxygenation, although usually it can be lowered within minutes when guided by pulse oximetry and, in the appropriate setting, applying PEEP. In the first minutes following institution of mechanical ventilation, the physician should remain alert for several common problems.
900 mL, f = 3 – 7. This approach appears to minimize autoPEEP by allowing such a long T I and patients are remarkably comfortable. Alternatively, ventilation can be initiated using the VACV mode with a normal tidal volume (5-7 mL/kg) and respiratory rate of 12 to 15 breaths per minute. A peak flow of 60 L/min is recommended, and higher flow rates do little to increase expiratory time. For example, if the V T is 500, the RR 15, and the V 60 L/min, the expiratory time is 3.5 seconds. Raising V (dramatically) to 120 L/min increases the expiratory time to only 3.75 seconds, a trivial improvement. In contrast, a small reduction in respiratory rate to 14 breaths per minute increases the expiratory time to 3.8 seconds. This example serves to emphasize not only the relative lack of benefit of raising the flow rate but also the importance of minimizing minute ventilation when the goal is to reduce autoPEEP. Finally, if the patient is triggering the ventilator, it is essential that some PEEP be added to reduce the work of triggering. This does not generally worsen the hyperinflation as long as PEEP is not higher than about 85% of the autoPEEP.\textsuperscript{44-46} Ventilatory goals are (1) to minimize alveolar overdistention (keep Pplat < 30) and (2) to minimize dynamic hyperinflation (keep autoPEEP < 10 cm H\textsubscript{2}O or end-inspiratory lung volume < 20 mL/kg), a strategy that largely prevents barotrauma.\textsuperscript{47} Reducing minute ventilation to achieve these goals generally causes the P\textsubscript{aO\textsubscript{2}} to rise above 40 mm Hg, often to 70 mm Hg or higher. Although this requires sedation, such permissive hypcapnia is quite well tolerated except in patients with increased intracranial pressure and perhaps in those with ventricular dysfunction or pulmonary hypertension.

Since peak proximal airway pressure is so high in this patient group, upper-limit alarms of 75 cm H\textsubscript{2}O (sometime higher) are often required when using volume-targeted modes. Changes in flow that have little effect in the patient without airway obstruction can have a dramatic impact in obstructed patients. Specifically, reducing the inspiratory flow or changing to a decelerating flow profile reduces the airway pressures and the amount of ventilator alarming but, by prolonging inspiration, worsens autoPEEP. While the ventilator looks “better,” the patient is worse, but this is only recognized if autoPEEP is regularly sought or if the expiratory flow profile is examined (see Fig. 49-3).

\textbf{Acute-on-Chronic Respiratory Failure:} \textit{Acute-on-chronic respiratory failure} (ACRF) is a term used to describe exacerbations of chronic ventilatory failure usually occurring in patients with chronic obstructive pulmonary disease (COPD)\textsuperscript{48} (see Chap. 54). Many of these patients are successfully (and preferably) ventilated noninvasively (see Chap. 44). When intubated, they are found to have relatively smaller increases in inspiratory resistance (compared to asthma), their expiratory flow limitation arising largely from loss of elastic recoil.\textsuperscript{49} As a consequence, in the patient with COPD peak airway pressures tend to be only modestly elevated (eg, 30 cm H\textsubscript{2}O), yet autoPEEP and its consequences are common. At the time of intubation, hyperperfusion is common, as manifested by tachycardia and relative hypotension, and typically responds to briefly ceasing ventilation combined with fluid loading.

Since the patient typically has an underlying compensated respiratory acidosis, excessive ventilation risks severe respiratory alkalosis and, over time, bicarbonate wasting by the kidney. Initial ventilator settings of a tidal volume of 5 to 7 mL/kg and a respiratory rate of 16 to 24 breaths per minute, with a VACV mode minimize the risk of producing complications of severe dynamic hyperinflation. Since gas exchange abnormalities are primarily those of ventilation-perfusion mismatch, supplemental oxygen in the range of an F\textsubscript{I\textsubscript{O}} of 0.4 should achieve better than 90% saturation of arterial hemoglobin. Indeed, gas exchange abnormalities requiring an F\textsubscript{I\textsubscript{O}} greater than 0.5 should prompt a search for complicating alveolar filling processes, such as left ventricular failure with pulmonary edema, pneumonia, or lobar collapse. Inspiratory flow rates may be adjusted for patient comfort but usually are in the range of 50 to 60 L/min. PEEP should be used in this phase when the patient is triggering the ventilator since autoPEEP is universally present.

Examination of airway pressure and flow waveforms can be very helpful in identifying patient-ventilator dyssynchrony and suggesting strategies for improving the ventilator settings (Fig. 49-5). Some patients show autoPEEP-induced triggering difficulty, as discussed in Chap. 54 and in Figure 49-4.\textsuperscript{46} Frequently, adding extrinsic PEEP to nearly counterbalance the autoPEEP dramatically improves the patient’s comfort.\textsuperscript{21} An alternative approach is to increase minute ventilation to drive down the P\textsubscript{CO\textsubscript{2}}, but this will worsen autoPEEP and waste bicarbonate. Moreover, full passivity of respiratory muscles is not desired as this may contribute to VIDD. If the patient continues to make significant inspiratory efforts—especially if these efforts are ineffective in actually triggering a machine breath or generating a tidal volume—judicious sedation is in order.

\textbf{Patients With Acute Hypoxemic Respiratory Failure:} Acute hypoxemic respiratory failure (AHRF) is caused by alveolar filling with blood, pus, or edema, the end results of which are impaired lung mechanics and gas exchange (see Chap. 43). The gas exchange impairment results from intrapulmonary shunt that is largely refractory to oxygen therapy. In acute respiratory distress syndrome (ARDS; Chap. 52), the significantly reduced FRC due to alveolar flooding and collapse leaves many fewer alveoli to accept the tidal volume, making the lung appear stiff and dramatically increasing the work of breathing. The ARDS lung should be viewed as a small lung, however, rather than a stiff lung. In line with this current conception of ARDS, it is now clearly established that excessive distention of the ARDS lung compounds lung injury and may induce systemic inflammation.\textsuperscript{52} Ventilatory strategies have evolved markedly in the past decade, changing clinical practice and generating tremendous excitement.

The goals of ventilation are to reduce shunt, avoid toxic concentrations of oxygen, and choose ventilator settings that do not amplify lung damage. The initial F\textsubscript{I\textsubscript{O}} should be 1.0 in view of the typically extreme hypoxemia. PEEP is indicated in patients with diffuse lung lesions but may not be helpful in patients with focal infiltrates, such as lobar pneumonia. In patients with ARDS, PEEP should be instituted immediately, beginning with 15 cm H\textsubscript{2}O, then rapidly adjusted based on oxygenation or measures of recruitment. There is an increasing trend to rely on higher values of PEEP than necessary for oxygenation in order to reduce the prospect of VILI, but this remains controversial.\textsuperscript{53} The tidal volume should be 6 mL/kg (of ideal body weight, IBW) on VACV, since higher tidal volumes are associated with greater mortality.\textsuperscript{54} There is little doubt

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure49-5.png}
\caption{Signs of patient effort during volume-targeted ventilation (volume assist-control [VACV] or intermittent mandatory ventilation [IMV] breaths). In the two breaths of equal tidal volume shown, the left tracing represents a muscle-relaxed patient, while the other breath shows a patient making inspiratory effort. The change in esophageal pressure (Peso) is shown in the bottom tracings. Signs of patient effort in the airway pressure tracing include a fall in pressure at the airway opening (Pao) just before the VACV or synchronized IMV breath (triggering, a), a concave upward rise in Pao during inspiration (b), and a peak airway pressure that is less than it would be if the patient made no effort (c). During the triggered breath, Peso (as an indicator of the pleural pressure) remains more negative than baseline throughout the breath and even after end inspiration (arrow at d).}
\end{figure}
that lung protection can be achieved using pressure-targeted ventilation, for example as practiced in the Lung Open Ventilation Trial.\textsuperscript{44} Whatever the mode, the respiratory rate should be set at 24 to 36 breaths per minute as long as there is no autoPEEP. An occasional consequence of lung protective ventilation is hypercapnia. This approach of preferring hypercapnia to alveolar overdistention ("permissive hypercapnia") is discussed further in Chaps. 51 and 55.\textsuperscript{5,16}

The Patient With Restriction of the Lungs or Chest Wall: A number of restrictive diseases of the lungs or chest wall can lead to respiratory failure, especially when there is a superimposed ventilatory challenge (eg, pneumonia). These conditions are fully discussed in Chaps. 58, 86, and 114 and include lung disease (eg, advanced pulmonary fibrosis or late-stage ARDS), abdominal disease (eg, massive ascites), and other chest wall abnormalities (eg, kyphoscoliosis). Here only the ventilator management is described.

Small tidal volumes (5-7 mL/kg) and rapid rates (18-24 breaths per minute) are especially important in order to minimize the hemodynamic consequences of positive-pressure ventilation and to reduce the likelihood of barotrauma. The FiO\textsubscript{2} is usually determined by the degree of alveolar filling or collapse, if any. Rarely, we have encountered patients with enormous restrictive loads from intraabdominal catastrophes (eg, massive intraperitoneal bleeding) who have a large intrapulmonary shunt yet lack signs of alveolar flooding on the chest radiograph. We speculate that in such patients large numbers of alveolar units may be subserved by airways forced below their closing volume throughout tidal ventilation so that these nonventilated alveoli comprise a large intrapulmonary shunt (see Chap. 114). Reversible contributors to restriction (eg, circumscribed burn eschar, tense ascites) should be identified and treated.

The high alveolar pressures typically generated in these patients may lead to increased physiologic dead space (when Palv exceeds the pulmonary artery pressure), especially when large tidal volumes are used. When the restrictive abnormality involves the chest wall (including the abdomen), the large ventilation-induced rise in pleural pressure has the potential to compromise cardiac output. This in turn will lower the mixed venous P\textsub{O}{2} and, in the setting of V/Q mismatch or shunt, the P\textsub{a}{O}{2} as well. If the physician responds to this falling P\textsub{a}{O}{2} by augmenting PEEP or increasing the minute ventilation, further circulatory compromise ensues. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise ensues. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue. A potentially catastrophic cycle of worsening gas exchange, PEES or increasing the minute ventilation, further circulatory compromise can ensue.

\section*{Patient-Ventilator Synchrony}

Initial ventilator settings should be reassessed promptly to assess their appropriateness for the individual patient. Such fine-tuning of the ventilator often means the difference between a patient who is comfortable on the ventilator or who continues to perform fatigueing efforts, leading to deep sedation or therapeutic paralysis. Assessing the patient-ventilator interaction requires substantial skill and experience. In part, the adequacy of ventilator settings is judged by the appearance of the patient (comfortable versus diaphoretic and fighting) and waveform analysis and, much less so, arterial blood gas analysis.

The intensivist should ensure that the patient and ventilator are synchronized, that is, that each attempt by the patient to trigger the ventilator generates a breath. The most common situation in which the patient fails to trigger breaths occurs in severe obstruction when autoPEEP is present (Fig. 49-4). This is recognized at the bedside when the patient makes obvious efforts that fail to produce a breath. Using waveforms, these ineffective efforts cause a temporary slowing of expiratory flow, sometimes halting it completely (Fig. 49-6).

\section*{Response to "Crisis" in the Ventilated Patient}

A vast array of sudden and potentially catastrophic changes in clinical condition can occur in the course of mechanical ventilation (Table 49-1). We will focus on high- and low-pressure alarms, worsened oxygenation, and hypercapnia. Whenever the function of the ventilator or the position and patency of the airway are in question, the patient should be removed from the ventilator and hand-bagged with 100% oxygen. This point is extremely important, since this maneuver immediately circumvents the ventilator (and any malfunction of it), provides the clinician with a direct

\begin{table}[h]
\centering
\begin{tabular}{|l|}
\hline
\textbf{Table 49-1} Ventilator Crises \\
\hline
\textbf{Increased Peak Airway Pressure} \\
Endotracheal tube obstruction, kink, malposition \\
Airway obstruction (eg, bronchospasm, mucous plug) \\
Reduced lung compliance (eg, pulmonary edema) \\
Reduced chest wall/abdomen compliance (eg, pneumothorax, abdominal distention) \\
Patient effort, agitation (eg, coughing, biting, fighting) \\
\hline
\textbf{Reduced Oxygen Saturation} \\
Ventilator/mixer malfunction \\
Endotracheal tube malposition, leak \\
New lung derangement (eg, atelectasis, aspiration, edema) \\
New cardiovascular derangement (eg, shock, pulmonary embolism, fall in hemoglobin concentration) \\
Increased oxygen consumption \\
Change in body position, increasing shunt \\
\hline
\textbf{Rising P\textsub{a}{O}{2}} \\
Ventilator malfunction \\
Endotracheal tube malposition, leak \\
New patient mechanical derangement (eg, bronchospasm, edema) \\
Increased dead space \\
Increased CO\textsub{2} production \\
\hline
\textbf{Patient Distress} \\
Pain, discomfort unrelated to the ventilator or respiratory system (eg, myocardial ischemia) \\
Endotracheal tube malposition \\
Rising work of breathing \\
Rising P\textsub{a}{O}{2} (see above) \\
Oxyhemoglobin desaturation (see above) \\
Shock, pulmonary embolism \\
Inadequate sedation \\
Alcohol or other drug withdrawal \\
\hline
\end{tabular}
\end{table}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig49-6.png}
\caption{Ventilation of an obstructed patient with VACV or IMV. A failed attempt to trigger can be detected in the expiratory flow waveform, where the expiratory flow briefly ceases (due to the patient’s inspiratory effort) but the effort is insufficient to initiate a breath. Often the patient can be seen to make obvious inspiratory efforts between ventilator breaths. V is flow.}
\end{figure}
assessment of respiratory system mechanics, and focuses attention on the patient and not the machine.

High-Pressure Alarm: Aside from alarm or gauge malfunction, increased airway pressure indicates obstruction of the airway, obstruction to gas flow through the ventilator circuit, patient effort against the ventilator, or a change in the mechanics of the respiratory system. If manual bag ventilation is difficult, a suction catheter should be passed immediately through the endotracheal tube. If the catheter cannot be advanced 25 cm or more, obstruction of the airway is likely. If repositioning of the head does not relieve kinking, and if the patient is not biting the airway, reintubation is necessary. If the patient is biting an endotracheal tube, a bite block should be placed, or if this cannot be done, a short-acting neuromuscular blocking drug should be administered. If the airway is patent yet manual ventilation is difficult and the patient is suffering, a sedative should be given. If the patient is now easier to ventilate (implicating vigorous respiratory muscle activity), the cause of the patient’s distress should be sought. Possibilities include hypoxemia, hypercapnia, shock, or a new central nervous system process.

If ventilation remains difficult after deep sedation or muscle paralysis of a patient with a patent endotracheal tube, a new lower airway, pleural, lung, or chest wall process should be sought. Auscultation and bedside ultrasound often identify pneumothorax, collapse, or consolidation. Early portable chest radiography confirms these diagnoses or identifies an alternative cause of the crisis. Placing the patient back on the ventilator and monitoring peak and plateau pressures as well as autoPEEP will further delineate the problem, as described above.

Low-Pressure Alarm: Low-pressure alarms signal machine malfunction, a leak, or inspiratory effort by the patient (usually obvious). Large persistent leaks can occur within the ventilator itself, in the inspiratory limb, at the connection to the Y-adapter and endotracheal tube, around the endotracheal tube cuff, or through a bronchopleural fistula. If normal resistance to ventilation is noted during manual ventilation, the problem lies with the ventilator or tubing. If hand-bagging reveals minimal resistance, an endotracheal tube cuff leak is likely. This can be confirmed by listening over the neck or by placing a hand over the mouth. A large bronchopleural fistula can be identified by inspection of the chest tube and pleural drainage system.

Worsened Oxygenation: When a patient develops hypoxemia, sufficient oxygen should be given immediately to return the saturation to 88%. However, this must be followed by a search for the cause of deterioration. Of course, progression of the primary cause of respiratory failure (ARDS, pneumonia, lung hemorrhage) will impair gas exchange, but this should not be assumed to be the case. Also possible is a new lesion (eg, nosocomial pneumonia, pneumothorax), which may be identified by physical examination, ultrasound, or chest radiograph. However, a systematic approach is useful to identify the myriad (including nonpulmonary) causes of hypoxemia.

From a pathophysiologic perspective, new hypoxemia implies a reduced FiO2 (including ventilator malfunction), hypventilation, ventilation-perfusion mismatching, shunt, or a fall in the mixed venous oxygen saturation. Hypoventilation is usually obvious, being signaled by hypercapnia, and does not cause oxygen-refractory hypoxemia. Ventilation-perfusion mismatch typically causes mild hypoxemia that is easily corrected with supplemental oxygen. Bronchospasm, airway secretions, and airway plugging are common contributors in intubated patients. The combination of worsened ventilation-perfusion matching and an increase in dead space should prompt consideration of pulmonary embolism. Most often, when new hypoxemia develops in a mechanically ventilated patient, shunt or a fall in mixed venous oxygenation can be found. A new shunt (eg, pulmonary edema, pneumonia, or atelectasis) typically can be found on the chest radiograph, while mixed venous desaturation is detected by analyzing a venous blood sample or performing venous oximetry. The causes of venous desaturation include reduced cardiac output or hemoglobin concentration or increased systemic oxygen consumption. These nonpulmonary causes of hypoxemia are particularly common in patients with severe shunt lung disease and may herald life-threatening crises (eg, pneumothorax).

Hypercapnia: A rising PaCO2 often elicits a change in the ventilator orders (increased frequency or tidal volume). However, a pathophysiologic approach is useful here too. From the equation for PaCO2:

\[ \text{PaCO}_2 = \left( \frac{V_{\text{CO}2} \times k}{V_t \times f \times (1 - V_{d}/V_t)} \right) \]

Where \( V_{\text{CO}2} \) is carbon dioxide production; \( k \) is a constant; and \( V_d \) is the dead space, it can be seen that in addition to a fall in minute ventilation, rising CO2 production (eg, fever, shivering, agitation) or increasing dead space (eg, hypovolemia, pulmonary embolism, PEEP) may account for new hypercapnia. Responding to hypercapnia by simply raising the minute ventilation is dangerous because causes of increased \( V_{\text{CO}2} \) and dead space may be important to diagnose in their own right. In addition, augmenting minute ventilation has the potential to (paradoxically) decrease alveolar ventilation if the increase in \( V_d \) or \( f \) worsens dead space (such as when autoPEEP is present). Indeed, in this setting, the \( \text{PaCO}_2 \) may rise when minute ventilation is increased and fall when minute ventilation is reduced. These issues are further discussed in Chap. 55.

Liberation of the Patient from Mechanical Ventilation

We refer to the discontinuation of mechanical ventilation not as weaning (which implies the withdrawal of a nurturing life-support system) but rather as liberation (connoting freedom from a confining, noxious, and dangerous circumstance). Since liberation from the ventilator is fully discussed in Chap. 60, we make here only a few points relevant to all ventilated patients. Patients recover the ability to breathe spontaneously because central drive is regained, neuromuscular competence is restored, or respiratory system load is reduced. Once these are achieved, the ventilator is no longer needed. Gradual adjustments of IMV rates or pressure-support levels that are too slow for the patient’s needs simply serve to prolong the duration of mechanical ventilation, as shown in large trials of weaning strategies. On the other hand, if drive, strength, and load are not repaired, no amount of ventilator technology will allow the patient to breathe on his or her own. Most often, when physicians believe they are “weaning” the patient, they are simply allowing time for their other therapies to treat the respiratory failure; ventilator changes are prescribed coincidentally but are irrelevant. Accordingly, effective liberation of each patient begins at intubation and stabilization on the ventilator, with the measurement of respiratory mechanics to assist in evaluating the reversible features of the patient’s abnormally increased load; as soon as is clinically relevant, the respiratory muscle strength is evaluated for reversible causes of weakness; and frequent attempts are made to identify the earliest point at which the patient has regained the capacity to breathe.

Key References

- Girard TD, Kress JP, Fuchs BD, et al. Efficacy and safety of a paired sedation and ventilator weaning protocol for mechanically ventilated patients in intensive care (Awakening and


**REFERENCES**

Complete references available online at [www.mhprofessional.com/hall](http://www.mhprofessional.com/hall)

**CHAPTER 50**

**Novel Modes of Mechanical Ventilation**

Mashael Al-Hegelan

Neil R. MacIntyre

**KEY POINTS**

- Mechanical ventilators are support devices, not therapeutic devices. The clinical goal is thus to support gas exchange without causing harm.
- A number of challenges face clinicians in providing safe and effective mechanical ventilatory support. Two of the most important are (1) supporting gas exchange without causing injury from applied pressure or FiO₂; (2) providing comfortable interactive support as the lung recovers.
- Innovations need to focus on addressing clinical challenges. Moreover, to be accepted as “standard of care,” an innovation must be shown to improve an important clinical outcome.
- Recent innovations focusing on supporting gas exchange in a “lung protective” fashion include airway pressure release ventilation, high frequency ventilation, and adaptive support ventilation.
- Recent innovations focusing on improving patient-ventilator synchrony include various feedback controls on variable flow-pressure-targeted breaths, proportional assist ventilation, and neutrally adjusted ventilatory assistance.
- While all of these innovations have conceptual appeal and supporting observational data, none as yet have convincing randomized control trial data demonstrating improved clinical outcomes.

**INTRODUCTION**

The overarching goal of positive pressure mechanical ventilation is to provide adequate gas exchange support while not causing harm. Indeed, positive pressure mechanical ventilators are only support technologies, not therapeutic technologies. As such they cannot be expected to “cure” disease; they can only “buy time” for other therapies (including the patient’s own defenses) to work.

Conventional approaches to positive pressure ventilation involve applying ventilatory patterns mimicking normal through either masks or artificial airways. This is usually done with modes of support incorporating assist/control breath triggering mechanisms, gas delivery patterns governed by either a set flow or pressure, and breath cycling based on either a set volume, a set inspiratory time or a set flow. Often this support includes positive end expiratory pressure (PEEP) and supplemental oxygen. In recent decades a number of novel or unconventional approaches to providing mechanical ventilatory support have been introduced. For these to be considered of value, however, it would seem reasonable that they address important clinical challenges and be shown to improve important clinical outcomes (eg, mortality, duration of ventilation, sedation needs, complications). The remainder of this chapter will focus on challenges facing clinicians in providing mechanical ventilatory support and assess several novel approaches introduced over the last two decades in the context of these challenges.

**CLINICAL CHALLENGES FACING CLINICIANS PROVIDING MECHANICAL VENTILATORY SUPPORT**

**VENTILATOR INDUCED LUNG INJURY**

Probably the most important challenge facing clinicians providing mechanical ventilatory support today is managing the balance between providing adequate gas exchange and avoiding lung injury associated with positive airway pressure and oxygen exposure. On the one hand, patients in respiratory failure need adequate tissue oxygenation and acid/base balance; on the other hand, the lungs are fragile structures easily injured by excessive stretch, alveolar collapse-reopening and high oxygen exposure. This challenge is made more difficult by the fact that lung injury is usually heterogeneous and thus what may benefit gas exchange in one region (eg, higher pressure) may cause worse injury in another.¹

Lung injury from mechanical ventilatory support is often termed ventilator associated lung injury, or more commonly, ventilator-induced lung injury (VILI).²,³ Pathologically, VILI resembles in many ways the inflammatory response seen in other forms of acute lung injury and the acute respiratory distress syndrome (ALI/ARDS).²,³ The principal cause of VILI is alveolar injury induced by alveolar overstretch at end inspiration (overdistension), extended periods of tidal breath delivery above normal physiologic values, and cyclic atelectasis-recruitment that occur during positive pressure ventilation (Fig. 50-1).²,³ In general, the

**FIGURE 50-1.** Ventilator-induced lung injury during positive pressure ventilation comes from several factors. Depicted is the sigmoidal-shaped pressure-volume relationship seen in the acutely injured lung. Injury can occur from end inspiratory overdistension (upper right region), repetitive excessive tidal breath delivery (middle region), and repetitive collapse-reopening of alveolar units (lower left region).

---

1/3/2015 2:19:23 PM