Invasive and noninvasive ventilation are essential components in the management of critically ill patients. Some patients require support for respiratory failure or as part of comprehensive management of critical illness, whereas other patients require assistance primarily for airway protection. The reasons for initiating ventilatory support are varied and will influence ventilation strategy, hemodynamics, sedation strategy, and subsequent clinical course.

The decision to intubate is discussed in Chapter 1 and in various other places throughout this textbook in the context of individual conditions. This chapter describes the modalities and techniques of noninvasive and invasive mechanical ventilation.

**PRINCIPLES OF MECHANICAL VENTILATION**

**Physiology of Positive Pressure Breathing**

Spontaneous breathing in normal patients is based on the initiation of negative intrathoracic pressure and is mediated by contraction and relaxation of the diaphragm. Contraction of the diaphragm decreases pressure in the chest cavity and results in inhalation, whereas relaxation of the diaphragm and recoil of the chest wall increase pressure in the chest cavity and result in passive exhalation. The amount of force required to generate adequate inspiration is influenced by the work of breathing; when work of breathing increases, patients may be unable to generate enough negative force to sustain successful respiration and will require ventilatory support. Unlike spontaneous breathing, both invasive and noninvasive mechanical ventilation are based on the delivery of humidified air with positive pressure. The amount of positive pressure required for adequate ventilation is dependent on the patient’s respiratory effort, ranging from mild assistance to full support. “Inhalation” occurs by driving air into the lungs under positive pressure; air is passively exhaled owing to chest wall recoil.

Transition from negative pressure breathing to positive pressure breathing affects cardiovascular and pulmonary physiology and can have significant clinical consequences. Pressure changes in the thoracic cavity directly affect pressures in the chambers of the heart. During spontaneous inspiration, venous return and preload are augmented, cardiac output is increased, and there is an increased pressure gradient between the left ventricle and the aorta. With the initiation of positive-pressure ventilation (PPV), venous return is diminished, cardiac output falls, and there is a decreased pressure gradient between the left ventricle and the aorta. Relative hypotension can occur after ventilatory support has been initiated, and this may be exaggerated in patients with clinical hypovolemia or vasodilatory states.

**Invasive Mechanical Ventilation**

The primary considerations regarding initiation of mechanical ventilation relate to how each breath should be delivered. This includes how a “breath” is defined, how large it should be, how fast it should be delivered, how often it should be delivered, and how to manage the interaction of the patient with the ventilator.

How the ventilator defines a breath is referred to as the control variable. The ventilator can give breaths based on delivery of a set pressure or a set volume, referred to as pressure-controlled ventilation (PCV) and volume-controlled ventilation (VCV), respectively. The amount of time over which the breath is delivered is defined as the inspiratory time, and the rate at which air travels through the circuit is defined as inspiratory flow.

In VCV, a set amount of pressure is applied to the airway to expand the lungs. When the target pressure is reached, gas delivery and passive exhalation is permitted. During PCV, target pressure and inspiratory time are set, whereas the delivered tidal volume and inspiratory flow vary as functions of dynamic lung compliance and airway resistance. Ability to control the pressure delivered to the lungs is particularly useful to prevent barotrauma (see later). In addition, because inspiratory flow is not fixed, PCV may improve ventilator synchrony in intubated patients with a high respiratory drive. A significant disadvantage of PCV is that tidal volume cannot be guaranteed or limited and can change with acute changes in lung compliance. PCV offers advantages over VCV in clinical conditions in which control of airway pressure is strictly mandated. This includes patients with the potential to develop dynamic hyperinflation and intrinsic positive end-expiratory pressure (PEEP), such as patients with severe asthma or those with respiratory failure from chronic obstructive pulmonary disease (COPD).

In VCV a breath is defined by delivery of a set tidal volume to the lungs. Inspiratory flow is fixed, and inhalation ends when a preset tidal volume has been delivered; peak inspiratory pressures (PIPs) and end-inspiratory alveolar pressures vary based on lung compliance and set tidal volume. The main benefit to VCV is the ability to control tidal volume and minute ventilation, but it may be problematic by causing high peak pressures when the compliance of the respiratory system is poor. Clinically, poor respiratory system compliance occurs in conditions that increase lung or chest wall stiffness. Such conditions include pulmonary edema, acute lung injury (ALI) or acute respiratory distress syndrome (ARDS), pneumothorax, and obesity.
When a physician is choosing between pressure-cycled ventilation versus volume-cycled ventilation, it is important to consider the underlying reason for mechanical ventilation. Volume-cycled ventilation should be used when strict control of tidal volume is mandated. Specifically, this includes patients with known ALI or ARDS, in whom low-tidal volume strategies have been proven to reduce mortality.\(^4\) In addition, patients with decreased chest wall compliance should be placed on VCV to ensure that adequate tidal volume is delivered. This includes patients with morbid obesity or severe chest wall burns. Conversely, in conditions in which strict control of airway pressure is desired, pressure-cycled ventilation should be used. As detailed earlier, this includes patients with asthma or COPD. In addition, because inspiratory flow is not limited in pressure-cycled ventilation, this strategy may be preferred to volume-cycled ventilation in patients with a high respiratory drive, such as patients with salicylate overdose. For patients who do not require strict control of either pressure or volume, similar ventilation mechanics can generally be achieved with either pressure-cycled or volume-cycled ventilation (Table 2-1).

Newer ventilators are able to deliver breaths that combine volume and pressure strategies, referred to as dual-control ventilation. A common dual-control method of ventilation is pressure-release volume control (PRVC). A variation of volume control, PRVC is set to deliver a specific tidal volume while simultaneously minimizing airway pressure. Unlike with strict volume control, pressure is measured and modulated by the ventilator with each breath to ensure the delivery of the preset tidal volume. In addition, a pressure limit is set, and the ventilator sounds an alarm when that pressure has been exceeded. Theoretically, this combines the advantages of pressure and volume control to ensure the delivery of a specific tidal volume while the airway pressure is monitored. That said, because the ventilator is set to deliver a specific tidal volume, the disadvantages of volume-cycled ventilation persist. In addition, elevations in airway pressure are still possible and must be addressed if acute changes in respiratory system compliance occur. This mode of ventilation has not been specifically studied but likely does not offer significant advantage over traditional volume- or pressure-cycled ventilation, particularly if strict parameters for airway pressure are desired.

**Ventilator mode** refers specifically to the amount of respiratory support provided by the ventilator but more commonly represents a combination of the type of breath to be given and the way the breath is to be initiated. The most common ventilator modes can be categorized on the basis of how often the ventilator will initiate a breath for the patient and can be divided broadly into continuous mechanical ventilation (CMV), intermittent mechanical ventilation (IMV), and continuous spontaneous ventilation (CSV).\(^5\) CMV and IMV can be delivered via pressure-control or volume-control methods. In CSV, no mandatory breaths are delivered to a patient; the size of the breath is determined by the effort of the patient and can be augmented with applied pressure to the airway. These methods are compared in Table 2-2. Other, more complex modes of ventilation include proportional-assist ventilation (PAV) and airway pressure release ventilation (APRV), though these generally are not used in the emergency department (ED).

CMV is intended to provide full ventilatory support for patients with little or no spontaneous respiratory activity. CMV, also referred to as **assist-control (A/C)** ventilation, provides a preset number of breaths per minute. In addition to preset breaths, A/C will also deliver a breath in response to patient effort. In this mode, patients can trigger a breath at any rate but will always receive at least the preset number of breaths. Notably, when a patient initiates a breath, he or she receives the full breath as set on the ventilator. For the promotion of ventilator synchrony, a spontaneous patient-initiated breath will take priority over a preset breath, meaning that if the ventilator is set to deliver 12 breaths/min, a breath is provided every 5 seconds in the absence of spontaneous inspiratory effort. When the patient makes a spontaneous effort, the ventilator provides an additional breath and the timer resets for another 5 seconds. A/C ventilation is the most useful initial mode of mechanical ventilation in ED patients, as many patients are initially paralyzed and sedated and do not interact with the ventilator. One of the biggest challenges with A/C ventilation, however, is that patient-initiated breaths are not proportional to patient effort; when inspiratory effort is detected, a full-sized breath is delivered. Clinically, this requires adequate sedation of patients when ventilated in A/C mode to prevent spontaneous respiratory efforts that will result in hyperventilation, air trapping, hypotension, and poor ventilator synchrony.\(^6\)

Synchronized intermittent mandatory ventilation (SIMV) provides intermittent ventilatory support to patients by delivering both mandatory and spontaneous breaths. In SIMV, a mandatory breath is given at a preset rate, but the breath is synchronized as much as possible with spontaneous patient effort. Much as with A/C, the patient will receive at least the minimum number of preset mandatory breaths; if the patient provides no effort, the preset number of breaths will be given. If a patient has a rate of spontaneous respirations lower than the set rate, the ventilator will provide the preset number of full breaths but will deliver as many as possible in synchrony with patient effort. In these scenarios there is little difference between A/C and SIMV. If a patient has a

<table>
<thead>
<tr>
<th>SET PARAMETERS</th>
<th>VARIABLE PARAMETERS</th>
<th>CLINICAL IMPLICATIONS</th>
<th>CLINICAL CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure-controlled ventilation (PCV)</td>
<td>Peak pressure target, inspiratory time, RR, PEEP</td>
<td>Tidal volume, inspiratory flow</td>
<td>Controls airway pressure, but tidal volume becomes a function of lung compliance (no guaranteed tidal volume or minute ventilation)</td>
</tr>
<tr>
<td>Volume-controlled ventilation (VCV)</td>
<td>Tidal volume, RR, inspiratory flow pattern, inspiratory time</td>
<td>PIP, end-inspiratory alveolar pressure</td>
<td>Guaranteed delivery of tidal volume, but may result in high or injurious lung pressures End-inspiratory alveolar pressure cannot be reliably estimated and must be measured (plateau pressure)</td>
</tr>
</tbody>
</table>

ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease; PIP, peak inspiratory pressure; PEEP, positive-end expiratory pressure; RR, respiratory rate.
Table 2-2 Selecting Ventilator Strategy: Features of Potential Options

<table>
<thead>
<tr>
<th>MODE</th>
<th>PARAMETERS SET BY PROVIDER</th>
<th>CLINICAL SCENARIO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous Mechanical Ventilation (CMV)</td>
<td>Pressure or volume control, RR</td>
<td>Paralyzed or deeply sedated patient, sedated patients with intermittent spontaneous respiratory effort; can lead to hyperventilation</td>
</tr>
<tr>
<td>Intermittent Mandatory Ventilation (IMV)</td>
<td>Pressure or volume control, RR (backup rate)</td>
<td>Patients with regular but poor spontaneous respiratory effort; if used in deeply sedated patients, set RR will need to be higher</td>
</tr>
<tr>
<td>Continuous Spontaneous Ventilation (CSV)</td>
<td>Level of pressure support, PEEP</td>
<td>Spontaneously breathing patients with good respiratory effort requiring minimal ventilatory support</td>
</tr>
<tr>
<td>Continuous positive airway pressure (CPAP)</td>
<td>Level of CPAP</td>
<td>Alert, spontaneously breathing patients with immediately reversible causes of respiratory distress; COPD and ACPE are classic indications for noninvasive ventilation</td>
</tr>
<tr>
<td>Bi-level positive airway pressure (BL-PAP)</td>
<td>IPAP and EPAP</td>
<td>Similar to CPAP</td>
</tr>
</tbody>
</table>

ACPE, acute cardiogenic pulmonary edema; COPD, chronic obstructive pulmonary disease; EPAP, expiratory positive airway pressure; IPAP, inspiratory positive airway pressure; PEEP, positive end-expiratory pressure; RR, respiratory rate.

The decision to intubate carries significant implications for patients, including potentially life-threatening complications related to airway management and subsequent complications related to intensive care unit (ICU) care. Noninvasive positive-pressure ventilation (NPPV) is an appealing option for patients requiring ventilatory assistance with potentially reversible conditions when tracheal intubation is not immediately necessary, or as a therapeutic adjunct for patients with “do-not-intubate” directives. In appropriately selected patients, noninvasive support before intubation. PSV may prove most useful in awake and interactive patients who have been intubated for temporary airway protection rather than for respiratory failure. If PSV is used, careful monitoring and ventilatory alarms are needed to ensure against undetected hypoventilation or apnea.

Positive End-Expiratory Pressure

Regardless of the ventilatory mode chosen, PEEP is often used during invasive mechanical ventilation. PEEP refers to the maintenance of positive airway pressure after the completion of passive exhalation. During acute respiratory failure, lung volumes are typically decreased; application of PEEP increases functional residual capacity (FRC), improves oxygenation, and decreases intrapulmonary shunt. Use of PEEP also reduces portions of nonaerated lung that may contribute to the development of ventilator-induced lung injury. Notably, PEEP increases both intrapulmonary and infrathoracic pressure and may affect pulmonary and cardiovascular physiology. Potential adverse effects of PEEP include decreased cardiac output, lung overdistention, and pneumothorax.

Applied PEEP must be differentiated from intrinsic PEEP (iPEEP, or auto-PEEP), which may result from improper assisted ventilation when adequate time is not allowed between breaths for complete exhalation. This circumstance is discussed later.

Noninvasive Techniques

Noninvasive positive-pressure ventilation (NPPV) is the delivery of CSV via sealed mask rather than endotracheal tube. As with PSV, the ventilator is set to provide a defined level of pressure when a patient takes a breath; inspiratory flow and inspiratory time are completely patient mediated. The most common types of noninvasive ventilation in the ED are continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BL-PAP). BiPAP, a term commonly used for BL-PAP, is the proprietary name of a portable device that uses this method of noninvasive ventilation rather than a term for the ventilation itself (Philips Respironics, Murrysville, Pa.). CPAP provides constant positive pressure throughout the respiratory cycle, whereas BL-PAP alternates between higher pressure during inspiration (IPAP) and lower pressure during expiration (EPAP). Although, strictly speaking, CPAP applies positive pressure to the airway during inspiration, the amount of inspiratory assistance is minimal. Conversely, just as with invasive mechanical ventilation, IPAP augments patient respiratory effort by decreasing the work of breathing during inspiration, whereas EPAP acts as PEEP to maintain FRC and alveolar recruitment. Notably, although PEEP, CPAP, and EPAP all represent positive airway pressure at the end of expiration, PEEP by convention refers to pressure applied during invasive mechanical ventilation, whereas CPAP is the application of positive pressure (invasively or noninvasively) during spontaneous breathing. The terms are occasionally used interchangeably.
NPPV obviates intubation in greater than 50% of cases and improves survival.\(^{11}\) Need for emergency intubation is a contraindication to NPPV, except as a means to improve preoxygenation in preparation for intubation. Other relative contraindications include decreased level of consciousness, lack of respiratory drive, increased secretions, hemodynamic instability, and conditions, such as facial trauma, that would prevent an adequate mask seal.\(^{12,13}\) If NPPV is initiated, patients should be reassessed frequently for progress of therapy, tolerance of the mode of support, and any signs of clinical deterioration that indicate a need for intubation.

Patients most likely to respond to NPPV in the ED are those with more readily reversible causes of their distress, such as COPD exacerbation or cardiogenic pulmonary edema in which fatigue is a significant factor. Robust evidence suggests benefit of NPPV for both conditions. In patients with acute COPD exacerbations, NPPV decreases the need for subsequent intubation, hospital length of stay, and mortality when compared with standard therapy. Notably, though helpful in most patients with COPD exacerbation, NPPV has been shown to have the largest benefit for patients with hypercapnic acidosis and pH below 7.3.\(^{14-17}\)

Treatment failure, defined as subsequent need for intubation, is predicted by a Glasgow Coma Scale score of less than 11, a sustained arterial pH less than 7.25, and tachypnea greater than 30 breaths/min.\(^{18}\)

In patients with acute cardiogenic pulmonary edema (ACPE), NPPV reduces the work of breathing while simultaneously improving cardiac output; application of NPPV causes elevations in intrathoracic pressure that decrease both left ventricular (LV) ejection pressure and LV transmural pressure. This results in afterload reduction. In addition, decreases in RV preload may improve LV compliance via ventricular interdependence.\(^{1,3,19,20}\) Compared with standard therapy, multiple studies and several meta-analyses have confirmed decreased need for intubation as well as decreased mortality for patients with ACPE treated with NPPV. Benefits were independent of whether patients received CPAP or BL-PAP, and despite suggestions from early clinical data, no increased rate of acute myocardial infarction occurred in patients receiving any form of NPPV.\(^{21-24}\) Though either modality can be used, a recent ED-based study suggested faster clinical improvement with BL-PAP.\(^{25}\) Specific predictors of failure of NPPV in congestive heart failure (CHF) have not been systematically examined.

Use of NPPV in other patients with respiratory compromise, including asthma and pneumonia, is not well studied, though limited preliminary data suggest that NPPV may be beneficial for patients with acute asthma exacerbations.\(^{26-28}\) Studies have failed to establish a role for NPPV in pneumonia. Although no data suggest harm from NPPV, the presence of pneumonia has been shown to be an independent risk factor for failure of noninvasive ventilation.\(^{29-32}\)

**Approach to Initial Ventilator Settings**

Initial settings for noninvasive ventilation should be determined by the amount of ventilatory assistance required by the patient, as well as patient comfort and cooperation with the therapy. The first consideration in the use of NPPV is whether to provide support in the form of CPAP or BL-PAP. This was discussed earlier, and there is no clear benefit of one over the other. Support will be given by a full-face (oronasal) mask or nasal mask; this choice is determined by patient comfort, feelings of claustrophobia, and the need for the patient to effectively cough or speak. Inspiratory support (IPAP) can be initiated at 10 cm H\(_2\)O, and expiratory support (EPAP) can be initiated at 5 cm H\(_2\)O. Subsequent titration of these parameters is based on the patient’s clinical response, particularly pressure tolerance, respiratory rate, and oxygenhemoglobin saturation. Though blood gas analysis is confirmatory, improvement in clinical condition can be observed by decrease in work of breathing, good patient-ventilator synchrony, and patient report. If required, EPAP and IPAP can be adjusted by 1 to 2 cm H\(_2\)O at a time based on clinical response.\(^{33}\) If work of breathing is unchanged, increases in IPAP and EPAP reduce hypercarbia by increasing tidal volume and minute ventilation while increasing oxygenation by combating atelectasis and promoting alveolar recruitment. IPAP greater than 20 cm H\(_2\)O can be uncomfortable and can cause gastric insufflation and should be avoided.\(^{12,33}\)

For the intubated patient, initial ventilator settings should facilitate ventilation that improves gas exchange, promotes ventilatory synchrony, and minimizes the potential for complications. For an apneic or paralyzed patient, full ventilatory support is required; for this reason, A/C is the recommended mode of initial ventilation. Specific required settings depend on whether the patient is receiving PCV or VCV, but the principles underlying selection of settings are similar. Reasonable initial ventilator settings should deliver a tidal volume of 6 to 8 mL/kg of estimated ideal body weight (IBW) at rate of 12 to 14 breaths/min. If VCV is used, tidal volume can be set directly, and if PCV is used, tidal volume is determined by adjusting the targeted pressure to be delivered; initial pressure targets should not exceed 30 cm H\(_2\)O. Initial Fi\(_O_2\) should be set at 1.0 but generally can be adjusted down quickly to maintain an oxygen saturation of 90% or greater. PEEP is routinely given and is set initially at 5 cm H\(_2\)O.\(^{34}\) Settings for specific clinical conditions, such as status asthmatics, are discussed later.

**Ongoing Management**

Mechanical ventilation requires monitoring and regular adjustment to ensure appropriate gas exchange, safe delivery of desired tidal volume, and prevention of metabolic derangement. Changes to ventilator settings are guided dynamically by multiple factors, including pulse oximetry, end-tidal carbon dioxide (ET\(_{CO_2}\)) monitoring, ventilation pressures, and blood gases. For the adequacy of ventilation to be monitored, capnography may be used; arterial blood gases should be measured 15 to 20 minutes after initiation of ventilatory support to correlate ET\(_{CO_2}\) with P\(_{co_2}\). Notably, venous blood gases generally correlate well with pH and P\(_{co_2}\) of arterial samples,\(^{34,35}\) though this correlation may be unreliable in critically ill patients.\(^{36,37}\) Although there is variation in agreement between capnography and blood gas values, capnography generally correlates well with P\(_{co_2}\) of arterial samples and may be useful for ventilator adjustment after initial correlation has been established.\(^{38-41}\) In the event of uncertainty, arterial blood gases remain the definitive test for evaluating P\(_{ao_2}\) and P\(_{co_2}\). Minute ventilation can subsequently be altered by adjusting tidal volume or respiratory rate. To avoid oxygen toxicity, Fi\(_o_2\) should be reduced to the lowest level that provides acceptable (>90%) oxygen saturation. In many instances, increases in PEEP will allow better oxygenation for a given Fi\(_o_2\) but may worsen hypotension or increase intrathoracic pressure.

Maintenance of adequate gas exchange is the primary goal of invasive ventilation, but other clinically relevant data are derived from the ventilator once a course of ventilation has been set. Pressures in the ventilator circuit and the lungs reflect potentially detrimental complications of ventilation. PIP and plateau pressure (P\(_{plat}\)) are the main measurements of pressure during ventilation. PIP, sometimes referred to as peak airway pressure, measures the maximum amount of pressure in the ventilator circuit during a breath cycle. It reflects lung compliance and airway resistance, including resistance in the circuit itself. In PCV, because pressure limits are preset, PIP can be estimated as the sum of the set pressure target and PEEP. PIP also generally reflects the amount of pressure in the alveoli at the end of inspiration, an important determinant in the development of ventilator-induced lung injury.
(VILI). In VCV, PIP is influenced greatly by airway resistance and therefore is not reflective of end-inspiratory alveolar pressure. Alveolar pressure at end inspiration in VCV is approximated by the Pplat. The respiratory therapist can measure the Pplat with the ventilator at the end of inspiration by means of an “inspiratory hold.”

Acute increases in PIP indicate increased airway resistance or changes in compliance of the respiratory system (such as those associated with pneumothorax) and can indicate potentially dangerous clinical deterioration. Decreases in PIP, conversely, indicate decreased resistance or decreased air flow in the ventilatory circuit and should prompt investigation of the ventilator circuit for leaks. Large or sudden decreases in PIP suggest disconnection of the ventilator circuit or unintended extubation. For patients with underlying respiratory failure secondary to increased airway resistance, such as in asthma or COPD, more gradual decreases in PIP are associated with clinical improvement.

Other Management Considerations

Aside from specific ventilator management, considerations in the care of the intubated patient include analgesia and sedation, potential neuromuscular paralysis, and secretion management. After intubation, the primary goals of care in the ED are sustained effective ventilation and patient comfort. Intubation causes pain and anxiety for patients, and both analgesia and sedation are required to promote patient comfort and patient-ventilator synchrony. In initiating sedation (see later), sedation should be titrated to comfort and therapeutic goals while avoiding both oversedation and undersedation. Desired level of sedation will differ based on patient tolerance and the clinical scenario: assuming comfort is maintained, lighter sedation may be useful in patients requiring serial neurologic examinations, whereas deep sedation may be more beneficial for patients with severe hypoxemia or ventilator dysynchrony. Several clinical scales have been established for this purpose; the reliability of the Richmond Agitation-Sedation Scale (RASS) has been validated in multiple studies. Sedation should be maintained at the highest RASS score at which the patient is comfortable (between 0 and −5) and should be serially readdressed. Any paralyzed patient should remain deeply sedated (Table 2-3).

After rapid sequence intubation, additional neuromuscular blocking agents (NBMAs) should generally be used only when poor ventilator synchrony interferes with ventilation sedation and analgesia. This may be particularly true in patients with ARDS, in whom the use of NBMAs has been associated with shorter duration of ventilation and improved mortality. With proper sedation and analgesia, however, neuromuscular blockade usually is not required. If needed, longer-acting agents such as rocuronium and vecuronium should be used; note that impaired hepatic or renal function may increase duration of paralysis.

Analgesia is achieved by generous doses of opioid medications; fentanyl and morphine remain the most commonly used agents for analgesia in critically ill patients. Opioids are associated with dose-dependent respiratory depression, a side effect that may be particularly beneficial for patients experiencing ventilator dysynchrony. Both morphine and fentanyl can be used for analgesia, though dosage requirements may vary based on tolerance and drug metabolism. Sedation and analgesia should therefore be titrated with a standard sedation scale, as discussed earlier. Notably, the active metabolite of morphine (morphine-6-glucuronide) is renally cleared and has potent sedative effects. For this reason, fentanyl may be preferred in patients with renal insufficiency. Remifentanil is an ultra-short-acting opiate that is metabolized by nonspecific plasma esterases. The predictable metabolism and short half-life of remifentanil make it an emerging choice for sedation, and ICU data demonstrate decreased duration of ventilation with remifentanil compared with morphine when used for analgesia in the ICU.

Sedation after intubation can be accomplished via multiple pharmacologic modalities, and though there are limited data about ED sedation practices, lorazepam and midazolam are the most commonly used agents in the ICU. Benzodiazepines exert dose-dependent clinical effects by binding γ-aminobutyric acid (GABA) receptors, first producing anxiolysis, then sedation and hypnosis. Benzodiazepines also cause respiratory depression, which is potentiated by concomitant opioid administration. For this reason, a sedation regimen of both opioids and benzodiazepines may improve ventilator dysynchrony while providing an anxiolytic and amnestic effect. Benzodiazepines can be administered either as repeated boluses or by continuous infusion, though in critically ill patients, benzodiazepines have altered pharmacokinetics that result in tissue accumulation and prolonged sedation. This is particularly true in obese patients and in patients with renal or hepatic insufficiency. For this reason, sedation with benzodiazepines should be attempted with intermittent bolus administration before a continuous infusion is used.

Similar to benzodiazepines, propofol binds to the GABA receptor to induce sedation. Propofol is lipophilic, and the ability of the drug to rapidly penetrate the blood-brain barrier and distribute into peripheral tissues is responsible for both the rapidity and the short duration of clinical effect. Unlike benzodiazepines, the clearance of propofol is minimally affected in critically ill patients. In addition, propofol can precipitate hypotension by increasing venous capacitance, a side effect that is exaggerated in hypovolemic patients. For these reasons, propofol should be given as an infusion rather than as a bolus, initiated at low doses (0.1 mg/kg/min) and titrated to desired level of sedation. In comparison to benzodiazepines, continuous infusions of propofol have been demonstrated to decrease duration of mechanical ventilation, suggesting that propofol may confer benefit when compared with benzodiazepines in the sedation regimen for mechanically ventilated patients.

### Table 2-3 The Richmond Agitation-Sedation Scale (RASS)

<table>
<thead>
<tr>
<th>SCORE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Comatose</td>
</tr>
<tr>
<td>+3</td>
<td>Very agitated</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated</td>
</tr>
<tr>
<td>+1</td>
<td>Restless</td>
</tr>
<tr>
<td>0</td>
<td>Calm</td>
</tr>
<tr>
<td>−1</td>
<td>Drowsy</td>
</tr>
<tr>
<td>−2</td>
<td>Light sedation</td>
</tr>
<tr>
<td>−3</td>
<td>Moderate sedation</td>
</tr>
<tr>
<td>−4</td>
<td>Deep sedation</td>
</tr>
<tr>
<td>−5</td>
<td>Unarousable</td>
</tr>
</tbody>
</table>

Other medications for the sedation of ventilated patients in the ED include dexmedetomidine and haloperidol. Dexmedetomidine is a centrally acting alpha2-agonist with sedative and analgesic properties, largely distinguished from other sedative agents by a negligible impact on respiratory drive, even with simultaneous opioid administration. It is administered as a loading dose followed by a continuous infusion and can precipitate bradycardia and relative hypertension.65 Studies have demonstrated dexmedetomidine to be beneficial in facilitating use of noninvasive ventilation, as well as awake fiberoptic intubations.58–60 When dexmedetomidine was compared with continuous infusion of midazolam, a large, multicenter evaluation demonstrated that dexmedetomidine was associated with shorter duration of mechanical ventilation, as well as decreased sedation-associated delirium.61 Although not systematically studied in the ventilated ED patients, dexmedetomidine is an emerging alternative for sedation strategy for critically ill patients and may be considered as an alternative to traditional modalities in clinical settings in which agitation or anxiety limit therapeutic goals.

Haloperidol, commonly used as a sedative for agitated patients, can also be used as an adjunct to traditional sedation regimens in mechanically ventilated patients. The use of haloperidol may be particularly useful in patients who remain acutely agitated after large doses of other sedative medications, especially because it does not affect hemodynamics. Recent limited data have suggested a mortality benefit for mechanically ventilated patients who receive haloperidol, though this requires further study.62 Notably, however, haloperidol does not have any analgesic or amnestic properties and cannot be used as a single therapy for sedation in critically ill patients.

Other ED considerations in the care of the ventilated patient include secretion management and steps to reduce potential ventilator-associated pneumonia (VAP). Management of secretions is achieved via regular endotracheal suctioning, recognizing a balance between secretion clearance and the disruption of ventilation. In addition, a nasogastric or orogastric tube should be placed for gastrointestinal decompression. Finally, evidence demonstrates benefit in the prevention of VAP from placing the patient in the semirecumbent position by elevating the head of the bed.63 Limited data suggest that use of VAP care “bundles,” including elevation of the head of the bed, have decreased incidence of VAP in the ICU; this may warrant further study to determine benefit in the ED.64 A recent meta-analysis also demonstrated a decrease in the incidence of VAP with continuous aspiration of subglottic secretions. This is done via a specialized endotracheal tube for this purpose, and, though not routinely used in the ED, this technique may be a direction for future investigation.65

Complications of Positive-Pressure Ventilation

Although initiated as a lifesaving intervention, PPV carries the risk of significant complications. As highlighted earlier, initiation of PPV and elevated intrathoracic pressure can be associated with relative hypotension, and any subsequent changes in end-inspiratory alveolar pressures or PEEP during the management of ventilation may have hemodynamic consequences.

PPV also has a direct impact on the lungs. Whether delivered as a set volume or set pressure, invasive PPV forcibly distends the lung and can be injurious. Injuries from elevated lung volume or lung pressure are known as volutrauma and barotrauma, respectively, and contribute to the development of VILI. VILI is mitigated by limiting pathologic stretch on the alveoli; current data support that maximum “safe” end-inspiratory alveolar pressures are 30 to 32 cm H2O,68–69 though this continues to be actively researched. Barotrauma can also manifest overtly with pneumothorax or pneumomediastinum, but this is relatively uncommon.69

Another potential complication of PPV is the development of intrinsic PEEP (iPEEP or auto-PEEP). Particularly problematic in patients with obstructive lung disease, iPEEP is accumulation of end-expiratory volume and end-expiratory pressure that occurs when exhalation cannot be fully completed. In patients with obstructive lung disease, expiratory flow is limited secondary to small airway obstruction and diminished elastic recoil. Time required for full exhalation may be significantly longer than normal, and in patients receiving mechanical ventilation, exhalation may not be complete before the next delivered breath. This phenomenon, known as “breath stacking,” results in dynamic hyperinflation. Intrinsic PEEP results in unexpectedly high PIPs, difficulty in ventilation, hypotension, and potential circulatory collapse. Ventilation difficulty caused by iPEEP can be improved by decreasing the respiratory rate or decreasing the inspiratory time, both of which facilitate increased time for exhalation.

Troubleshooting the Ventilator

When a patient’s condition suddenly deteriorates during mechanical ventilation, a systematic approach should be applied to immediately assess for life-threatening conditions (Fig. 2–1). The first step in evaluating the ventilated patient who has a change in clinical status is to assess vital signs. Patients with acute hemodynamic compromise or acute hypoxia should be removed from the ventilator and bagged manually on 100% oxygen. Tension pneumothorax, increased iPEEP, and accidental extubation are the most life-threatening concerns in this situation and must be immediately addressed. While the patient is bagged, the chest should be examined to ensure bilateral breath sounds. Changes in breath sounds may indicate a pneumothorax or a migration of the endotracheal tube. Clinical examination, oxygen saturation, and ETCO2 monitoring can be used as surrogates for tube placement, but suspicion of inadvertent extubation should prompt direct visualization. Acute hypotension can be precipitated by extreme elevations in intrathoracic pressure; compromise from iPEEP will improve once the patient has been disconnected from the ventilator, whereas hypotension from a tension pneumothorax will not. If the patient’s condition remains unstable after he or she has been disconnected from the ventilator circuit, a tension pneumothorax should be treated presumptively with needle decompression and eventual chest tube placement. Although unlikely, it is possible that a patient could sustain bilateral pneumothoraces, and this should be considered. If the patient remains unstable after chest decompression, sources for compensation unrelated to the ventilator must be pursued.

Acute distress without hemodynamic changes can be precipitated by multiple factors, both mechanical and physiologic (Table 2–4). Again, the initial evaluation should begin by confirming position and patency of the endotracheal tube before other diagnoses are investigated, including evaluation of the tracheal balloon. Once tube placement has been confirmed, the next step in evaluation of ventilator-related causes of distress should focus on airway pressures. Acute decreases in PIP indicate discontinuity in the ventilator circuit, which could include inadvertent extubation. Patients with increased PIP can be considered in two categories: those with concomitant increases in Pplat, and those with unchanged Pplat. If both PIP and Pplat acutely increase, this suggests decreased compliance of the respiratory system. Elevated PIP with unchanged Pplat indicates problems with increased airway resistance, either in the lungs or the ventilator circuit. Specific conditions that cause decreased respiratory system compliance or increased airway resistance are detailed in Figure 2–1.
EVALUATION OF THE DISTRESSED PATIENT ON MECHANICAL VENTILATION

**No**  
Confirm ETT placement, evaluate airway pressures  
Elevated PIP and Pplat (indicates decreased respiratory system compliance)  
**Yes**  
Remove from ventilator and manually bag with 100% oxygen, examine patient, and confirm ETT placement. Life-threatening causes include iPEEP and tension pneumothorax.  
Presumptive treatment for tension pneumothorax with needle decompression. If patient remains unstable, other diagnoses should be pursued, including PE.  
Likely iPEEP: resume mechanical ventilation with decreased RR and increased expiratory time.

**Table 2-4** Troubleshooting the Ventilator: Potential Causes of Acute Respiratory Distress

<table>
<thead>
<tr>
<th>MECHANICAL</th>
<th>PHYSIOLOGIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endotracheal tube migration into bronchus</td>
<td>Worsening lung compliance</td>
</tr>
<tr>
<td>Endotracheal tube obstruction</td>
<td>Worsening airway obstruction</td>
</tr>
<tr>
<td>Endotracheal tube cuff leak</td>
<td>Abdominal distention</td>
</tr>
<tr>
<td>Inadvertent extubation</td>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Discontinuity in ventilator circuit</td>
<td>Pain or inadequate sedation</td>
</tr>
</tbody>
</table>

**Special Clinical Circumstances**

Although generalizations can be made regarding ventilatory management in the ED, certain clinical circumstances merit specific discussion.

**Acute Exacerbation of Chronic Obstructive Pulmonary Disease**

Strategies for managing intubated patients with COPD focus on improving gas exchange while minimizing iPEEP. Reduction of iPEEP is achieved by decreasing airway resistance with bronchodilators and corticosteroids and ensuring adequate expiratory time during mechanical ventilation. Adequate expiratory time is achieved by decreasing respiratory rate, tidal volume, and inspiratory time. Adequate oxygenation (saturation of 90%) is achieved while minimizing barotrauma by deliberately reducing minute ventilation, so-called "permissive hypercapnia." No data suggest the advantage of PCV versus VCV, and either method can be used. The ideal ratio of inspiratory-to-expiratory time (I/E ratio) is variable, but the ratio should initially be set at 1:4. Data in asthmatics suggest that expiratory times longer than 4 seconds have minimal impact on airflow. IPEEP can also result in poor patient-ventilator synchrony, causing inadequate gas exchange. Initially, deep sedation and analgesia (or sometimes paralysis) are required to prevent ventilator asynchrony and permit effective ventilation. Corticosteroids often are indicated (see Chapter 74). NBMAs are avoided if possible, as patients receiving both NMBA and corticosteroids are at higher risk for "polymyopathy of critical illness" and subsequent increased mortality.

**Status Asthmaticus**

Concerns in ventilating the acute asthmatic generally parallel those for patients with COPD, with small notable differences. In acute asthma, respiratory failure is both a result of airway obstruction and airway inflammation. Furthermore, unlike COPD, airway obstruction is much less dynamic and occurs predominantly in large airways. In addition, acute inflammatory changes throughout the lung contribute to decreased lung compliance, which has a direct impact on lung pressures during ventilation. Strategies should focus on low respiratory rates with emphasis on maximizing expiratory time. The use of PEEP is debated and is largely thought to contribute to increased lung pressure. Though no data definitively exist supporting VCV over PCV, decreased lung compliance and potential iPEEP may make the delivery of adequate tidal volumes with PCV difficult. This is especially problematic for patients with severe, acute respiratory acidosis for whom adequate ventilation is essential. Recommendations for ventilator settings include VCV with tidal volumes of 6 to 8 mL/kg IBW, respiratory rate of 10 to 15 breaths per minute, and no PEEP. Decreased inspiratory time allows greater expiratory time and is achieved by increasing the inspiratory flow rate.

**Acute Lung Injury and Acute Respiratory Distress Syndrome**

ALI and ARDS are on a spectrum of inflammatory lung disease characterized by heterogeneous noncardiogenic pulmonary
edema, hypoxia, and diffuse lung consolidation. Strictly, the difference between these conditions is defined by the ratio of arterial oxygen concentration to the fraction of inspired oxygen (Pao2/Fio2), with ALI being less severe than ARDS. ALI and ARDS can be caused by pulmonary or extrapulmonary inflammation, including VILI. Though epidemiologic data suggest that ALI is not common on initial presentation to the ED, incidence of ALI in ventilated patients is not uncommon. The impact of ventilation in the ED on development of lung injury or ARDS is unclear. Nonetheless, development of VILI has been associated with lung overdistention and alveolar injury, and attention to ventilation strategy in the ED is warranted. Studies confirm that decreased tidal volumes are of clear benefit in the management of patients with ALI and ARDS. The majority of studies examining low–tidal volume ventilation strategies, including the landmark ARDSnet trial in 2000, used 6 to 7 mL/kg tidal volumes based on IBW, though studies with 7 mL/kg did not demonstrate a difference in mortality. A meta-analysis of these data concluded that tidal volumes below 7 mL/kg and Pplat above 31 cm H2O conferred mortality benefit in patients with ALI or ARDS, though more recent work suggests that low–tidal volume ventilation may improve outcome for patients without lung injury as well. Level of PEEP in patients with ALI and ARDS continues to be actively researched. Therefore in patients with ALI or ARDS (Pao2/Fio2 < 300), low–tidal ventilation strategy should be used. Initial ventilator settings should be volume cycled, with tidal volumes based on 6 mL/kg of IBW. IBW can also be estimated by height.

OUTCOMES

Because of the heterogeneity of ventilation strategies and clinical reasons for respiratory failure, no data exist regarding the superiority of one ventilation method over another; considerations in initiating mechanical ventilation must be individualized and serially reevaluated. Nonetheless, as detailed previously, certain conclusions regarding outcome can be drawn. Data clearly indicate the effectiveness of NPPV in preventing intubation for patients with COPD and ACPE, and these benefits have resulted in decreased admission to the ICU and decreased mortality. In addition, increased alveolar volumes and pressures have been shown to contribute to VILI and to increase mortality in patients with ALI and ARDS. The implication of decreased tidal-volume ventilation on the prevention of lung injury in patients with normal lungs is not definitive, and the role of ventilation strategy in the ED on subsequent clinical course of critically ill patients remains uncertain.

Lastly, although the treatment of mechanically ventilated patients usually extends beyond the ED, delays in ICU admission can have significant implications on ED management of the critically ill ventilated patient as the role of emergency physicians extends beyond acute stabilization toward ongoing clinical management. In addition, when boarding times are long, patients intubated solely for airway protection may be candidates for extubation in the ED if the initial insult has been reversed.

KEY CONCEPTS

- Primary goals of mechanical ventilation are improved gas exchange and decreased work of breathing while maximizing patient comfort and minimizing the potential for complications. Strategies for approaching mechanical ventilation are individualized, dependent both on the patient and the underlying cause of respiratory failure.
- Noninvasive ventilatory support is often adequate for reversal impending respiratory failure and should considered as the first-line therapy for patients with exacerbations of chronic obstructive pulmonary disease and acute cardiogenic pulmonary edema in whom immediate intubation is not required.
- Invasive mechanical ventilation is not without consequence and requires dynamic, ongoing management. After intubation, blood gas analysis should be performed to confirm appropriate ventilation and provide correlation with noninvasive monitoring modalities. In addition, hemodynamic consequences as a result of positive pressure should be anticipated. Elevated lung pressures can be deleterious and should be actively avoided.
- Sudden difficulty in the treatment of patients receiving mechanical ventilation should prompt a quick and systematic evaluation for life-threatening conditions, followed by assessment of the ventilator settings, the ventilator circuit, airway pressures, and physiologic problems. In hemodynamically unstable patients, this starts with disconnecting the patient from the ventilator and temporary use of a bag-mask with 100% oxygen.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References


