Management of Respiratory Failure
Ventilator Management 101 and Noninvasive Ventilation

Steven D. Pearson,1 Jay L. Koyner,2 and Bhakti K. Patel1

Abstract
Mechanical ventilation is a lifesaving therapy for critically ill patients with respiratory failure, but like all treatments, it has the potential to cause harm if not administered appropriately. This review aims to give an overview of the basic principles of invasive and noninvasive mechanical ventilation. Topics covered include modes of mechanical ventilation, respiratory mechanics and ventilator waveform interpretation, strategies for initial ventilator settings, indications and contraindications for noninvasive ventilation, and the effect of the ventilator on kidney function.

Introduction
Mechanical ventilation is an artificial means of respiratory support to either partially or fully assist respirations and gas exchange. Mechanical ventilation can be delivered both invasively via an endotracheal or tracheostomy tube or noninvasively via a face mask or other interfaces. The most common indications for invasive mechanical ventilation in the intensive care unit (ICU) are refractory hypoxemia, ventilatory failure, shock with metabolic acidosis, and airway compromise due to altered mental status or physical obstruction. The primary goal of mechanical ventilation in patients with respiratory failure is to maintain adequate alveolar ventilation and arterial blood oxygen content, preventing respiratory acidosis and hypoxia (1).

Modes of Mechanical Ventilation
Mechanical ventilation modes can be defined in terms of their control variable and programmed breath sequence (Table 1). The control variable determines the gas volume and flow rate delivered for each breath, while the breath sequence determines the frequency of delivered breaths. As a general principle, ventilators deliver an amount of gas for each breath as either a volume or pressure specified by the control variable, and the patient’s respiratory system compliance determines the other according to their individual pressure-volume relationship. As an example, for a patient with normal lung compliance (change in volume divided by change in pressure) of 100 cc/cm H2O, a volume-controlled breath of 500 cc delivered by the ventilator would result in a gas pressure of 5 cm H2O. For the same patient, a pressure-controlled breath of 10 cm H2O delivered by the ventilator would result in a gas volume of 1000 cc.

For volume-controlled modes, the operator sets a tidal volume (defined as the amount of gas delivered to the lungs with each breath) and inspiratory flow rate. When a breath is initiated, gas flows in at the set flow rate, and the breath is terminated once the specified tidal volume has been achieved. The pressure resulting from the specified flow rate and tidal volume is dependent on patient-specific factors, as discussed later. In pressure-controlled modes, the operator specifies an inspiratory pressure, and the gas flow rate is determined by the preset pressure gradient and resistance to gas flow. The breath terminates either after a set inspiratory time or after the flow rate drops to a prespecified percentage of the initial flow rate. The resulting tidal volume in pressure-controlled modes is dependent on patient-specific factors, which are often changing dynamically in critically ill patients. Pressure-controlled modes are, therefore, unable to guarantee a tidal volume or ensure a minimum minute ventilation (defined simply as the amount of gas breathed per minute); such a guarantee is one advantage of volume-controlled modes. Pressure-controlled ventilation may better simulate spontaneous respiration, improving patient comfort and ventilator synchrony. Despite these differences, the available clinical data do not suggest any difference in patient outcomes between the two (2). In addition to volume- and pressure-controlled modes, dual-control modes combine the desirable features of each by automatically adjusting the inspiratory pressure to achieve a specified target tidal volume. This allows breaths to be delivered as a pressure, which may be more comfortable for patients, while still ensuring an adequate minute ventilation.

In addition to a control variable, a breath sequence must be specified. In continuous mandatory ventilation, also known as assist control, the set rate is the minimum respiratory rate delivered by the ventilator. Additional patient-initiated breaths are delivered by the ventilator according to the set control variable. In the case of volume-controlled continuous mandatory
ventilation, each patient-initiated breath is delivered as the set tidal volume. The most common mode used in the ICU is volume-controlled continuous mandatory ventilation, which is also referred to as volume assist control or simply assist control (3). Pressure-controlled continuous mandatory ventilation is commonly referred to as pressure control or pressure assist control, and dual-control continuous mandatory ventilation is also known as pressure-regulated volume control.

Intermittent mandatory ventilation is like continuous mandatory ventilation but differs in how patient-initiated breaths are handled. A set rate determines the minimum respiratory rate, but patient-initiated breaths are not delivered according to the control variable. Instead, the patient is allowed to breathe spontaneously, with the tidal volume determined by the patient’s spontaneous respiratory effort. An additional inspiratory pressure may also be set to support the spontaneous effort. Intermittent mandatory ventilation is most commonly used as volume-controlled synchronized intermittent mandatory ventilation (3). In synchronized intermittent mandatory ventilation, patient-initiated breaths within a certain time of the scheduled breath are delivered according to the control variable, “synchronizing” the patient’s effort with the set ventilator rate.

Spontaneous modes of mechanical ventilation have no ventilator set respiratory rate, and the rate is entirely determined by the patient. The most common spontaneous mode is pressure support, a pressure-controlled spontaneous mode. When the patient triggers a breath, the ventilator increases the airway pressure by a set amount (referred to as the level of pressure support), which increases gas flow beyond what would have been produced by patient-generated negative pleural pressure alone. The breath is terminated when flow drops to a set percentage of the initial inspiratory flow, and airway pressure returns to the set expiratory level, allowing exhalation. Some ventilators allow for automatic adjustment of the pressure support level to achieve a target tidal volume, and this dual-control spontaneous mode is called volume support.

Setting the ventilator to volume assist control with a constant inspiratory flow (typically 60 L/min on the basis of convention) allows for standardization of the pressure-time waveform and rapid assessment of respiratory system mechanics.

The maximum airway pressure at the end of inspiration is called the peak inspiratory pressure (PIP) (Figure 1), and it is a function of three main components: the pressure due to airflow resistance, the elastic recoil pressure of the lung and chest wall, and the positive end expiratory pressure (PEEP). Equation 1 shows the contributing components to PIP in mathematical terms (flow rate \( \dot{V} \), airways resistance \( R_{aw} \), compliance of the lung and chest wall \( C_{lung} \), and tidal volume \( V_T \)):

\[
PIP = \dot{V} \cdot R_{aw} + \frac{V_T}{C_{lung}} + PEEP. \tag{1}
\]

Common problems in ventilator management are the diagnosis and treatment of elevated PIPs, which require assessment of the relative contributions of the airflow resistance and elastic recoil to the PIP. To determine this, an inspiratory hold maneuver is performed to measure the plateau airway pressure (\( P_{plat} \)). The plateau airway pressure is the sum of only the elastic recoil pressure (also called the driving pressure, which is determined by tidal volume and compliance) and the PEEP, as demonstrated in Equation 2:

\[
P_{plat} = \frac{V_T}{C_{lung}} + PEEP. \tag{2}
\]

The relative contributions of these three components to the PIP provide insight to the underlying pathophysiology of the patient. Figure 1 shows the results of an inspiratory hold and graphically displays the three components of the PIP.

The difference between the PIP and the plateau airway pressure can be used to determine whether the airways resistance is normal or elevated (Equations 3 and 4):

\[
R_{aw} = \Delta P = \frac{PIP - P_{plat}}{\dot{V}} \tag{3}
\]

and

\[
C_{lung} = \frac{\Delta V}{\Delta P} = \frac{V_T}{P_{plat} - PEEP}. \tag{4}
\]

A normal airways resistance in a mechanically ventilated patient is <$10$ cm H$_2$O/L per second, which at a gas flow

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**Table 1. Common modes of mechanical ventilation stratified by control variable and breath sequence**

<table>
<thead>
<tr>
<th>Breath Sequence</th>
<th>Volume Controlled</th>
<th>Pressure Controlled</th>
<th>Dual Controlled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous mandatory</td>
<td>Volume assist; other temporarily used equivalent terms and acronyms: volume control, AC, A/C, AC-VC</td>
<td>Pressure assist; other temporarily used equivalent terms and acronyms: pressure control, PCV</td>
<td>Pressure-regulated volume control</td>
</tr>
<tr>
<td>Intermittent mandatory</td>
<td>Synchronized intermittent mandatory ventilation</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Continuous spontaneous</td>
<td>n/a</td>
<td>Pressure support, continuous positive airway pressure</td>
<td>Volume support</td>
</tr>
</tbody>
</table>

AC, assist control; AC-VC, assist control-volume control; PCV, pressure control ventilation; n/a, not applicable.
rate of 60 L/min corresponds to a peak to plateau pressure gradient of 10 cm H₂O. Values above this are considered abnormal and can be due to obstruction anywhere from the ventilator circuit to the small airways as outlined in Figure 2 and Table 2. It is important to note the gas flow rate when measuring the peak to plateau pressure gradient, as the pressure gradient is dependent on flow rate as well as airways resistance; increasing the gas flow rate without a change in airways resistance will increase the peak to plateau pressure gradient.

Figure 1. | Pressure-time and flow-time ventilator waveforms for a passive patient receiving volume assist control mode ventilation with a constant inspiratory flow. An inspiratory hold maneuver is performed during the second breath. Positive end expiratory pressure (PEEP), peak inspiratory pressure (PIP), and plateau airway pressure (Pplat) are labeled.

Figure 2. | Graphic representation of a patient and ventilator circuit illustrating the sites responsible for increased airways resistance and compliance. ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease; ETT, endotracheal tube.
Selecting Ventilator Settings

When initiating invasive mechanical ventilation, in addition to selecting the appropriate initial mode, the operator must carefully select appropriate settings for that mode. For continuous mandatory modes of mechanical ventilation, the most common initial modes, the operator must set a tidal volume (or pressure), respiratory rate, PEEP, and fraction of inspired oxygen (FiO2). For volume-controlled and dual-control modes of mechanical ventilation, the target tidal volume is set directly, whereas in pressure-controlled modes, the level of pressure support must be titrated to achieve the desired tidal volume.

### Tidal Volume

The goal of selecting an appropriate tidal volume is to ensure adequate alveolar minute ventilation while preventing injury from the stress and strain of excessive tidal volumes (5). In patients with acute respiratory distress syndrome (ARDS) as defined by the Berlin criteria (Table 3), tidal volumes of 6 ml/kg predicted body weight (PBW) or less are lung protective and improve mortality compared with larger tidal volumes (6,7). Additionally, patients with ARDS being ventilated with tidal volumes of 6 ml/kg PBW who have driving pressures (plateau airway pressure minus PEEP) above 15 cm H2O may benefit from further reductions in tidal volume (8,9). This must, however, be balanced against the need to maintain adequate alveolar minute ventilation. Patients with ARDS and very low lung compliance may benefit from advanced respiratory support, such as extracorporeal membrane oxygenation, which is beyond the scope of this review (10).

In patients without ARDS, the optimal tidal volume is less certain, as these patients receive less benefit from lower tidal volumes. In medical patients without ARDS receiving mechanical ventilation, a randomized controlled trial comparing tidal volume of 6 versus 10 ml/kg PBW showed no difference in mortality or ventilator-free days (11). Observational data also suggest that the benefit of low tidal volumes increases with lower lung compliance (8). In surgical patients, a large randomized controlled trial comparing intraoperative tidal volumes of 6 versus 10 ml/kg PBW showed no difference in postoperative pulmonary complications (12), although one smaller prior study comparing low tidal volumes combined with the application of PEEP and recruitment maneuvers (periodic lung hyperinflation) suggested a benefit (13). Given the lack of conclusive data for mechanically ventilated patients without ARDS, tidal

### Selecting Ventilator Settings

Increases in the PIP with a normal peak to plateau pressure gradient are a result of increased plateau pressure. As PEEP and tidal volume are set by the operator, increases in the plateau airway pressure are most commonly due to decreased compliance of the lung and/or chest wall. Figure 2 and Table 2 summarize common causes of decreased lung and chest wall compliance.

In cases of expiratory airflow obstruction, prolonged expiration can cause inspiration to begin before expiration is complete, leading to dynamic air trapping and auto-PEEP. This is most common in patients with status asthmaticus or exacerbations of chronic obstructive pulmonary disease (COPD), but it can also occur with central airway obstruction or very high respiratory rates. The presence of auto-PEEP can be detected by examining the flow-time waveform, where negative flow (exhalation) continues up to the initiation of the next breath. In cases of extreme airflow obstruction, very small values of negative flow can be missed on the waveform display, in which cases an end expiratory hold maneuver can be performed to detect and quantify the presence of auto-PEEP (4). During an end expiratory hold, the ventilator circuit is closed at the time the next breath would be initiated, and the resulting airway pressure is measured. If the pressure measured during the hold is greater than the set extrinsic PEEP, auto-PEEP is present and is quantified as the difference between the measured pressure and the set PEEP. Figure 3 shows a characteristic flow-time waveform and the results of an end expiratory hold for a patient demonstrating auto-PEEP. If auto-PEEP is present, adjustments, such as decreasing the respiratory rate or increasing the inspiratory flow, should be made to increase the exhalation time. If severe auto-PEEP is present, high intrathoracic pressure can impede venous return, leading to obstructive shock and hypotension, in which case the mechanical ventilation circuit should be temporarily disconnected from the patient to allow complete exhalation.

### Table 2. Causes of increased peak airway pressure

<table>
<thead>
<tr>
<th>Increased Peak to Plateau Pressure Gradient</th>
<th>Increased Plateau Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kinked ventilator tubing</td>
<td>ARDS</td>
</tr>
<tr>
<td>Biting on the endotracheal tube</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Obstructed endotracheal tube</td>
<td>Pulmonary edema or hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Interstitial lung disease</td>
</tr>
<tr>
<td></td>
<td>Atelectasis</td>
</tr>
<tr>
<td>Mucus plug</td>
<td>Decreased pleural space compliance</td>
</tr>
<tr>
<td>Central airway mass or foreign body</td>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Increased resistance in the large airways</td>
<td>Pleural effusion</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>Decreased chest wall compliance</td>
</tr>
<tr>
<td>Asthma</td>
<td>Obesity</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>Musculoskeletal restriction</td>
</tr>
<tr>
<td></td>
<td>Abdominal distension</td>
</tr>
</tbody>
</table>

ARDS, acute respiratory distress syndrome.

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For continuous mandatory modes of mechanical ventilation, the most common initial modes, the operator must set a tidal volume (or pressure), respiratory rate, PEEP, and fraction of inspired oxygen (FiO2). For volume-controlled and dual-control modes of mechanical ventilation, the target tidal volume is set directly, whereas in pressure-controlled modes, the level of pressure support must be titrated to achieve the desired tidal volume.
volumes of 6–8 ml/kg PBW are reasonable, with further increases being acceptable if needed to maintain alveolar minute ventilation.

**Respiratory Rate**

All modes other than spontaneous modes require a set rate, which is the minimum respiratory rate delivered by the ventilator in the absence of patient-initiated breaths. The optimal respiratory rate and resulting minute ventilation are highly dependent on patient-specific factors such as dead space fraction and acid-base status. In general, an initial set respiratory rate of 12–16 breaths per minute is reasonable, but pH and PaCO₂ levels should be monitored, followed by adjustments to the rate as needed. When the set minute ventilation is too low, most patients will trigger additional breaths to maintain a low normal pH in the absence of heavy sedation, paralytics, or severe neurologic impairment. Additionally, patients may overbreathe the set rate due to pain, anxiety, or hypoxemia, resulting in respiratory alkalosis, in which case, the underlying cause of tachypnea should be addressed. When adjusting the respiratory rate, the clinician should be mindful of both the set and actual respiratory rate, as they may differ significantly.

For patients with ARDS managed with low tidal volume ventilation, a high respiratory rate is often required to maintain an adequate alveolar minute ventilation. Conversely, in patients with obstructive lung disease and auto-PEEP, decreasing the respiratory rate allows for longer time in exhalation and can decrease or prevent air trapping. In both cases, some amount of permissive hypercapnia is acceptable as long as the pH remains >7.2 (14).

**Positive End Expiratory Pressure**

The goal of ventilator-applied PEEP, or extrinsic PEEP, is to recruit atelectatic or consolidated lung regions and prevent end expiratory lung collapse, thereby improving gas exchange and lung compliance and protecting against
ventilator-induced lung injury. Inappropriately high levels of PEEP, however, can lead to lung overdistension, barotrauma, and hemodynamic compromise. A reasonable initial PEEP setting for most patients is 5 cm H2O, although much higher levels are frequently required to improve oxygenation in ARDS. The objective when titrating the PEEP is to balance the benefit of increased lung compliance with the harm of overdistension and high intrathoracic pressures (barotrauma). The exact method of PEEP titration in ARDS is an area of active investigation with many negative studies, although a reasonable approach is to choose the PEEP that results in the best lung compliance while limiting the plateau airway pressure to a maximum of 30–35 cm H2O (15–18).

Fraction of Inspired Oxygen

The FiO2 should be set to the lowest value needed (ideally 60% or less) to maintain acceptable oxygenation targets to prevent the consequences of hyperoxia and oxygen toxicity (19). For most critically ill patients, a peripheral arterial oxygen saturation of 90%–96% is a reasonable target, with a lower SpO2 target of 88%–92% for those with or at risk of hypercapnia (20). In patients with ARDS, an arterial oxygen tension of 55 or above and an peripheral arterial oxygen saturation of 88% or above are acceptable, and trials comparing these conservative oxygenation targets with more liberal targets have had conflicting results, with neither strategy showing a clear benefit in terms of survival or ventilator-free days (6,21).

Discontinuing Mechanical Ventilation

Protocolized daily spontaneous breathing trials are the most effective means of identifying patients who can be successfully liberated from mechanical ventilation (22). Patients who show an improvement in their underlying condition, have adequate gas exchange, are hemodynamically stable, and are able to initiate an inspiratory effort are considered ready to participate in a spontaneous breathing trial (23). Volume status should be optimized to reduce any additional load on the respiratory system during the spontaneous breathing trial. Additionally, pairing daily spontaneous breathing trials with an interruption of sedatives reduces the duration of mechanical ventilation and improves long-term mortality compared with spontaneous breathing trials alone (24,25). For the initial spontaneous breathing trial, shorter 30-minute trials with low levels of pressure support (5–8 cm H2O) result in a higher rate of successful extubation than longer 2-hour trials on T piece (a device that supplies supplemental oxygen alone through the endotracheal tube without any additional ventilatory support) (26). The optimal method of conducting a spontaneous breathing trial for those who fail their initial trial or have had a prolonged course of mechanical ventilation is less clear, and it should be decided on an individualized basis (27). During the spontaneous breathing trial, vital signs should be monitored along with monitoring for signs of respiratory distress or agitation, and ultimately, the spontaneous breathing trial should be deemed a success or failure on the basis of clinical judgment. For patients who pass their spontaneous breathing trial, the safety of extubation should be assessed next, which includes level of consciousness, endotracheal secretion volume, cough strength, and airway patency (28). For patients at high risk of failing extubation, such as those with cardiopulmonary comorbidities or hypercapnia, application of noninvasive ventilation immediately following extubation may prevent the development of postextubation respiratory failure and reduce the need for reintubation (27).

Noninvasive Ventilation

Noninvasive ventilation is an effective means of respiratory support for select patients with acute respiratory failure and avoids many of the negative consequences of invasive mechanical ventilation. Noninvasive ventilation can be delivered as both continuous positive airway pressure, which splits open the upper airway and provides PEEP, as well as bilevel positive airway pressure (BPAP), which augments inspiratory effort in addition to providing PEEP. The most common noninvasive ventilation interface for critically ill patients is the face mask, although other interfaces, such as the helmet, are available (29). Noninvasive ventilation should be avoided in patients with cardiac or respiratory arrest, inability to clear secretions or protect the airway, facial trauma or surgery, and recent esophageal anastomosis.

Chronic Obstructive Pulmonary Disease

For patients with acute exacerbations of COPD resulting in respiratory acidosis, noninvasive ventilation is the first-line method of respiratory support in the absence of contraindications as it has been shown to reduce the need for intubation and decrease mortality in randomized controlled clinical trials (30,31). BPAP is the preferred modality for these patients, with titration of the inspiratory pressure to offload the respiratory muscles and adjustment of the PEEP to assist initiating breaths in those with auto-PEEP. Robust clinical data to support the use of noninvasive ventilation in acute exacerbations of asthma are lacking, although observational evidence suggests that noninvasive ventilation is a safe and effective modality of respiratory support for these patients as well (32).

Acute Cardiogenic Pulmonary Edema

Noninvasive ventilation, delivered as either continuous positive airway pressure or BPAP, offers several physiologic benefits to patients with cardiogenic pulmonary edema. The increased intrathoracic pressure resulting from noninvasive ventilation works to offload the left ventricle by reducing preload and afterload, and increased alveolar pressure redistributes pulmonary edema to help improve gas exchange. In addition to its physiologic benefits, noninvasive ventilation redistributes the need for intubation and decreases mortality in patients with acute cardiogenic pulmonary edema (30,33).

Acute Hypoxemic Respiratory Failure

For patients with acute hypoxemic respiratory failure, the use of noninvasive ventilation is controversial due to conflicting and inconclusive evidence (30). Although noninvasive ventilation may avoid the need for endotracheal
intubation in some, those who fail noninvasive ventilation may suffer worse outcomes compared with initial management with invasive mechanical ventilation (34). An alternative method of noninvasive oxygen support for patients with hypoxemia is oxygen delivered by high-flow nasal cannula. High-flow nasal cannula devices supply heated and humidified oxygen through a specialized nasal cannula at flows of up to 60 L/min at a specified FiO2. The physiologic benefits of oxygen delivered by high-flow nasal cannula include reliable delivery of FiO2 with less entrainment of room air, low levels of effective PEEP, and washout of the anatomic dead space (35,36). One landmark randomized trial showed high-flow nasal cannula to be superior to noninvasive ventilation by face mask for hypoxemic respiratory failure, although a subsequent network-level meta-analysis showed a benefit of both noninvasive ventilation and high-flow nasal cannula over standard oxygen therapy in these patients (37). Additionally, the selection of interface appears to play an important role, with multiple studies suggesting that the helmet interface is superior to the face mask for hypoxemic respiratory failure (29,37). For patients with hypoxemic respiratory failure who are selected for a trial of noninvasive ventilation, close monitoring is required to avoid delays in endotracheal intubation in those who fail to respond or worsen.

Lung and Kidney Interactions

For many patients suffering from respiratory failure in the ICU, AKI commonly develops as a component of multiorgan system dysfunction (38). The presence of kidney dysfunction further complicates the care of patients with respiratory failure, especially in relation to the management of acid-base disorders and volume status.

Kidney Effects of Positive Pressure Ventilation

Positive pressure ventilation itself has deleterious effects on kidney perfusion and function (39). The increased intrathoracic pressures resulting from positive pressure ventilation can decrease venous return, subsequently decreasing cardiac output (40). This, in turn, results in decreased renal blood flow and GFR (41). Additionally, patients with right ventricular dysfunction are particularly susceptible to the increases in right ventricular afterload resulting from increased intrathoracic pressure.

Beyond the direct hemodynamic effects on cardiac output, positive pressure ventilation can cause alterations in several neurohormonal pathways. Some of these effects include increases in antidiuretic hormone release, activation of the renin-angiotensin-aldosterone axis, and suppression of atrial natriuretic peptide (41–43). The composite effect of these pathways is to further decrease renal blood flow and GFR and, ultimately, result in oliguria with fluid retention, all of which may further worsen pulmonary gas exchange.

Acid-Base Considerations

In patients with ARDS, permissive hypercapnia is often used to allow for lung-protective low tidal volume ventilation as previously discussed. Little data exist to guide the exact degree of acidosis that may be safely allowed and when a bicarbonate buffer or KRT should be administered. Given a lack of specific guidelines, a reasonable strategy is to follow the general approach outlined in the original trial of low–tidal volume ventilation (6). This protocol specified that, for a pH of <7.30, the respiratory rate should be first increased to 35 or until a PaCO2 of below 25 was achieved. For a pH of <7.15 due to respiratory acidosis despite the above, tidal volumes could be incrementally increased above 6 ml/kg PBW until a pH of 7.15 or above is achieved.

Although there are no randomized controlled trials on the subject, patients with combined respiratory and metabolic acidosis may benefit from alkali therapy if indicated for correction of the metabolic component while simultaneously providing respiratory compensation by increasing alveolar minute ventilation (44). Additionally, many patients with chronic lung disease, such as COPD, have a chronic respiratory acidosis and resulting compensatory metabolic alkalosis. A reasonable acid-base management strategy in these patients is to target their baseline plasma bicarbonate level (if known) in order to reduce the load on the respiratory system and facilitate liberation from mechanical ventilation; this can be done using bicarbonate infusion or using higher bicarbonate baths for those receiving dialysis (45).

Volume Management Considerations

Fluid balance is a crucial component in the management of patients with acute lung injury, and a large randomized controlled trial showed that a conservative fluid strategy resulted in more ventilator-free days and fewer ICU days in these patients when compared with a liberal strategy (46). However, this conservative fluid strategy did not provide any mortality benefit, and it did not improve kidney-focused outcomes (including need for dialysis). In fact, patients with kidney injury requiring KRT were excluded from the study, and enrolled patients were taken off protocol if they developed AKI requiring dialysis. Unfortunately, high-quality data are lacking to guide the optimal strategy for fluid management in patients with acute lung injury who also require KRT, and an extensive discussion around fluid balance and kidney injury is beyond the scope of this review.

Conclusion

Mechanical ventilation is a lifesaving therapy for critically ill patients with respiratory failure, although it has the potential to cause harm if not managed properly. Ventilator-induced lung injury can be minimized in patients with ARDS by carefully selecting the appropriate initial ventilator settings to avoid alveolar distension and barotrauma. In patients with obstructive lung disease, identification and prevention of auto-PEEP can prevent disastrous hemodynamic consequences. A wealth of information is available in the ventilator waveforms. Additionally, timely application of noninvasive ventilation in the appropriate patient may prevent the need for endotracheal intubation altogether.

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study; J.L. Koyner and S.D. Pearson were responsible for resources;
B.K. Patel was responsible for visualization; J.L. Koyner and B.K. Patel
provided supervision; S.D. Pearson wrote the original draft; and
J.L. Koyner, B.K. Patel, and S.D. Pearson reviewed and edited
the manuscript.

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