

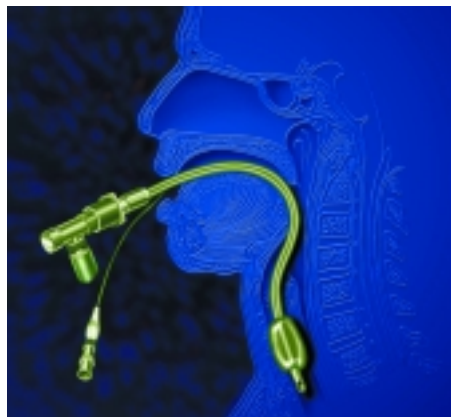
Mechanical Ventilation: A Review and Update for Clinicians

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The ancient Greek physician and philosopher Claudius Galen (129–210) was the first to describe the artificial ventilation of an animal.¹ More than 1000 years later, in the 16th century, this technique was applied to human resuscitation.² Excluding these historical anecdotes, mechanical ventilation did not become a major therapeutic intervention until the poliomyelitis epidemic swept through Europe and the United States in the 1940s and 1950s. Since the middle of the 20th century, a wide variety of ventilatory techniques have been developed for the treatment of patients with respiratory failure. This article reviews the available modalities of mechanical ventilation in terms of clinical indications and practical applications. Ventilator settings, alternative modes of ventilation, ventilation complications, and weaning from mechanical ventilation are also discussed.

CLINICAL GOALS

The basic purpose of mechanical ventilation is to support patients whose respiratory systems have failed until adequate function returns (**Table 1**). Reversal of acute, severe hypoxemia or respiratory acidosis with mechanical ventilation can be life-saving.³ Mechanical ventilation can also relieve respiratory distress in patients for whom the work of breathing has become intolerable. In addition, prevention or reversal of atelectasis and reversal of respiratory muscle fatigue can be accomplished by mechanical ventilation. Even in patients with healthy lungs, mechanical ventilation is often employed when sedation or neuromuscular blockade is necessary (eg, operative anesthesia). By decreasing systemic or myocardial oxygen consumption, mechanical ventilation may also assist patients who experience compromised myocardial function when the work of breathing becomes excessive. Other objectives of



mechanical ventilation include reduction in intracranial pressure by controlled hyperventilation for patients with closed head injury, and stabilization of the chest wall, as in cases of massive flail chest.⁴

Generally, mechanical ventilation supplies only symptomatic relief, not actual therapy for respiratory failure or acute lung injury. A notable exception is the state of congestive heart failure, during which mechani-

cal ventilation may augment cardiac output and therefore be therapeutic. Iatrogenic lung injury may be inflicted during mechanical ventilation, and preventive measures should be taken.

MODES OF MECHANICAL VENTILATION

Studies have demonstrated that suppression of spontaneous breathing and complete dependence on controlled mechanical ventilation lead to rapid respiratory muscle atrophy.⁵ Therefore, modes of mechanical ventilation that allow spontaneous breathing, or patient-triggered modes, are favored when feasible.

Assist/Control Mode Ventilation

Assist/control ventilation (ACV) is a combined mode of ventilation. The mechanical ventilator delivers a positive pressure breath of a predetermined tidal volume in response to each of the patient's inspiratory efforts (termed *assisted ventilation*). However, should a patient fail to initiate a breath within a specific time period, the ventilator automatically delivers a mechanical breath to maintain a minimum or "backup" respiratory rate (termed *controlled ventilation*).⁶ To trigger an

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assisted breath, the patient must lower the airway pressure by a preset amount, called the *trigger sensitivity*. **Figure 1** demonstrates examples of pressure and volume waveforms during volume-cycled ACV.

Intermittent Mandatory Ventilation

Intermittent mandatory ventilation (IMV) is a type of ventilatory support in which mandatory positive pressure breaths are delivered at preset time intervals. Between these breaths, the patient may breathe spontaneously from a separate circuit containing continuously flowing gas. IMV is associated with patient-ventilator dyssynchrony because the mandatory breaths are not delivered in concert with the patient's inspiratory effort. A mechanical breath could therefore be delivered during a spontaneous inspiration, leading to lung overdistention. IMV has largely been abandoned in favor of synchronized intermittent mandatory ventilation (SIMV).

Synchronized Intermittent Mandatory Ventilation

In SIMV, the patient receives a mandatory number of positive pressure breaths per minute, each of which is synchronized to patient effort. The ventilator can detect the initiation of a spontaneous breath by a patient and does not deliver a machine breath during a spontaneous breath. Between mechanical breaths, however, the patient may breathe an indefinite number of times from a reservoir (**Figure 2**). Spontaneous breaths produce no response from the ventilator. **Figure 3** shows typical pressure and volume waveforms during volume-targeted SIMV.

Pressure-Support Ventilation

Pressure-support ventilation (PSV) is a relatively new mode of mechanical ventilation. During PSV, each time the patient initiates a spontaneous breath, the negative pressure or flow in the inspiratory circuit opens a valve. The ventilator then delivers a flow of gas sufficient to maintain a constant inflation pressure. Once the patient's inspiratory flow rate falls below a preset threshold level, the flow of gas terminates. With this mode of ventilation, the patient controls respiratory rate and inspiratory time and flow. Tidal volume and minute ventilation are determined partly by the patient and partly by the ventilator. The tidal volume the patient actually receives depends on the set level of pressure support, patient effort, and the pulmonary mechanics. Pressure support may also be applied to a patient's spontaneous breathing during SIMV. **Figure 4** shows pressure and volume waveforms during PSV.

Table 1. Possible Outcomes of Mechanical Ventilation

Reversal of severe hypoxemia
Reversal of acute, severe respiratory acidosis
Relief of respiratory distress
Prevention or reversal of atelectasis
Reversal of respiratory muscle fatigue
Allowance for sedation or neuromuscular blockade
Decrease in systemic or myocardial oxygen consumption
Reduction of intracranial pressure through hyperventilation
Stabilization of chest wall

VENTILATOR SETTINGS

Tidal Volume

Traditionally, an initial tidal volume of 10 mL/kg has been recommended and remains a valid formula for patients without acute lung injury. Recently, however, a large clinical trial was stopped early when fewer deaths occurred among patients with acute respiratory distress syndrome (ARDS) who received small (6 mL/kg) rather than large (12 mL/kg) tidal volumes.⁷ Therefore, in the ARDS population, an initial tidal volume of 6 mL/kg is recommended.

Research has suggested that, at least in animal models, overdistention of alveoli by large tidal volumes may actually induce lung damage (eg, increased pulmonary microvascular permeability, pulmonary edema, and lung rupture).^{8,9} Excessive lung distention is of major concern, but direct measurement of the actual volume of air that reaches specific alveoli is not possible. However, lung volume is directly related to the pressure that distends the alveoli. This pressure can be approximated by measuring the end-inspiratory plateau pressure. Plateau pressure is measured by occluding the ventilator circuit at the end of inspiration. Largely based on animal data, a plateau pressure of less than 35 cm water (H₂O) has been recommended as safe.⁴ To achieve this target, tidal volume may need to be decreased to below 5 mL/kg, the physiologic value for spontaneous breathing. Low tidal volumes may lead to decreased ventilation and an elevated arterial partial pressure of carbon dioxide (Paco₂), a ventilatory strategy referred to as *permissive hypercapnia*.¹⁰ When using permissive hypercapnia, clinicians are advised to focus on the pH, rather than the Paco₂. Although some authorities recommend the administration of intravenous bicarbonate once the pH is below 7.2, this practice remains controversial.¹¹

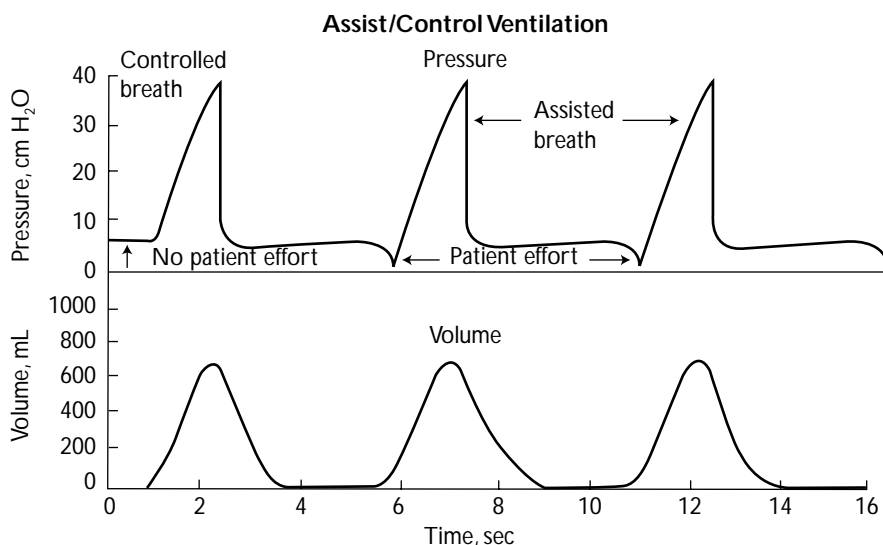


Figure 1. Pressure and volume waveforms during assist/control ventilation. Note that the first breath is provided by the ventilator without any patient trigger (controlled breath) and the second and third breaths are triggered by the patient (assisted breaths). H₂O = water.

Synchronized Intermittent Mandatory Ventilation

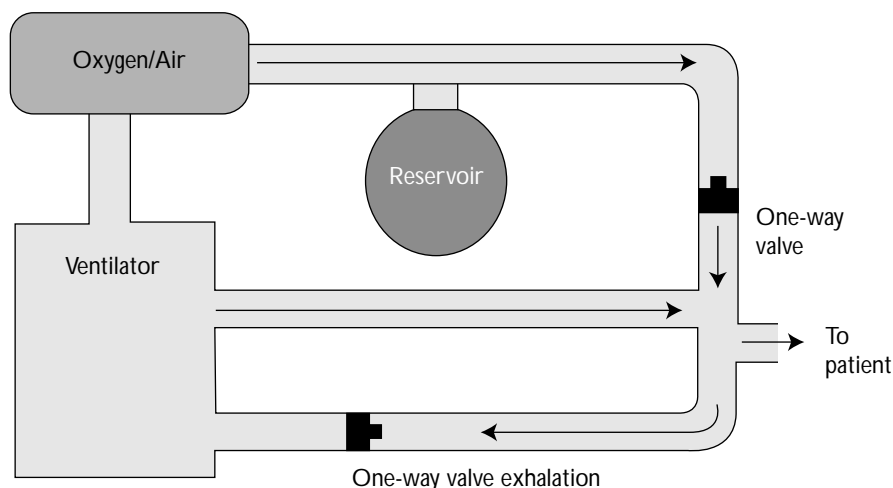


Figure 2. A schematic representation of the circuit employed in synchronized intermittent mandatory ventilation.

Respiratory Rate

The ideal mechanical respiratory rate depends on the particular mode of ventilation. When using ACV, the respiratory rate should be approximately 4 breaths/min less than the patient's spontaneous respiratory rate, thus assuring adequate ventilation if the patient's spontaneous efforts cease.³ If SIMV is employed or if the patient has no spontaneous respirations, the rate should be initially set at 10 breaths/min; respiratory rate should be increased if a higher minute ventilation is needed (as with respiratory acidosis) and decreased gradually as patient tolerance permits. No respiratory rate is set for PSV.

Sensitivity

To trigger an assisted breath during ACV or a pressure-supported breath during PSV, a patient must lower airway pressure by a preset amount to open the "demand valve" that delivers gas. The required change in airway pressure (ie, trigger sensitivity) is typically set from -1 to -3 cm H₂O. The actual negative pressure a patient must generate, however, can be much higher if the demand valve is poorly responsive.¹² Conversely, if the trigger sensitivity is set too low, the ventilator may deliver breaths too frequently (commonly referred to as *auto-cycling*) and produce severe respiratory alkalosis.³ Some ventilators may be triggered by inspiratory flow rather than

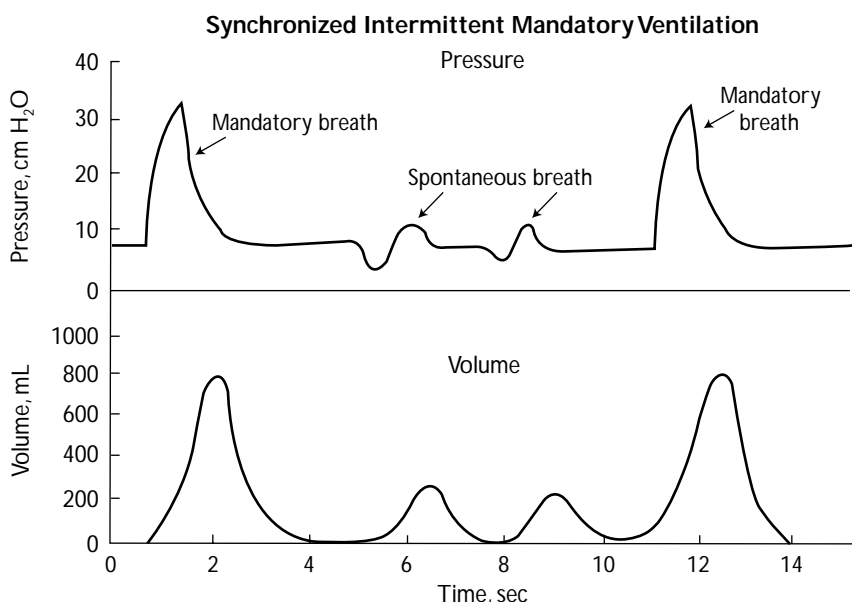


Figure 3. Pressure and volume waveforms during synchronized intermittent mandatory ventilation. Note that the first and last breaths are mandatory and the second and third breaths are spontaneous. H₂O = water.

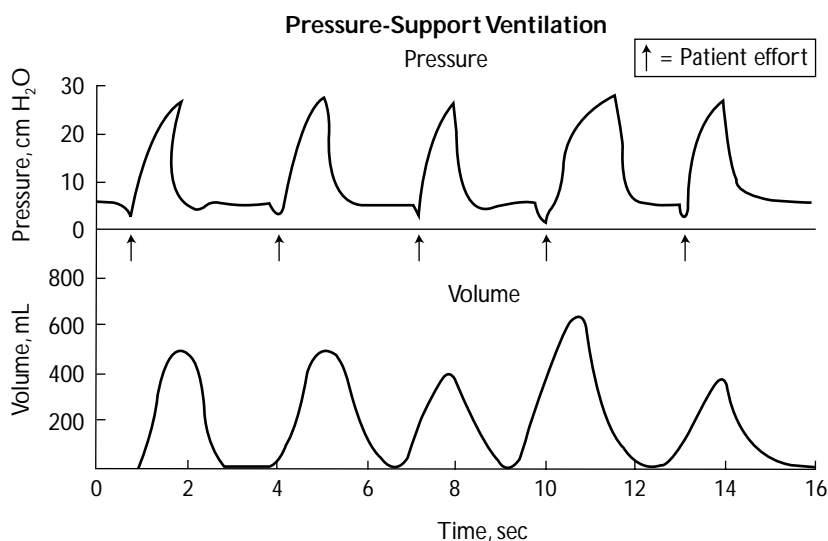


Figure 4. Pressure and volume waveforms during pressure-support ventilation. Note that each breath is triggered by patient effort. The breaths vary in tidal volume and inspiratory time. H₂O = water.

pressure. This method of triggering appears to require less patient effort.¹³

Fraction of Inspired Oxygen

Inhalation of any gas with a fraction of inspired oxygen (FIO₂) higher than the FIO₂ of room air can potentially be toxic. However, 0.6 has been established as the threshold of a toxic concentration of FIO₂.¹⁴ To minimize the risk of toxicity, the lowest FIO₂ that achieves adequate oxygenation should be employed. Acceptable arterial oxygen saturation is usually defined as 90% or greater, which usually corresponds to an arterial partial pressure of oxygen (PaO₂) of approximately 60 mm Hg or more.

Positive End-Expiratory Pressure

In individuals with healthy lungs, the intrathoracic pressure at the end of exhalation closely approximates atmospheric pressure. Intrapleural pressure, however, is slightly negative. This negative pressure keeps a resting volume of air inside the lung at the end of exhalation, called the *functional residual capacity* (FRC). FRC provides a reservoir of gas that maintains a constant PaO₂ and PaCO₂, a crucial physiologic function. Patients with respiratory failure who require mechanical ventilation, however, usually have a reduced FRC as a consequence of surfactant loss and alveolar instability, which leads to alveolar collapse. In turn, alveolar collapse can

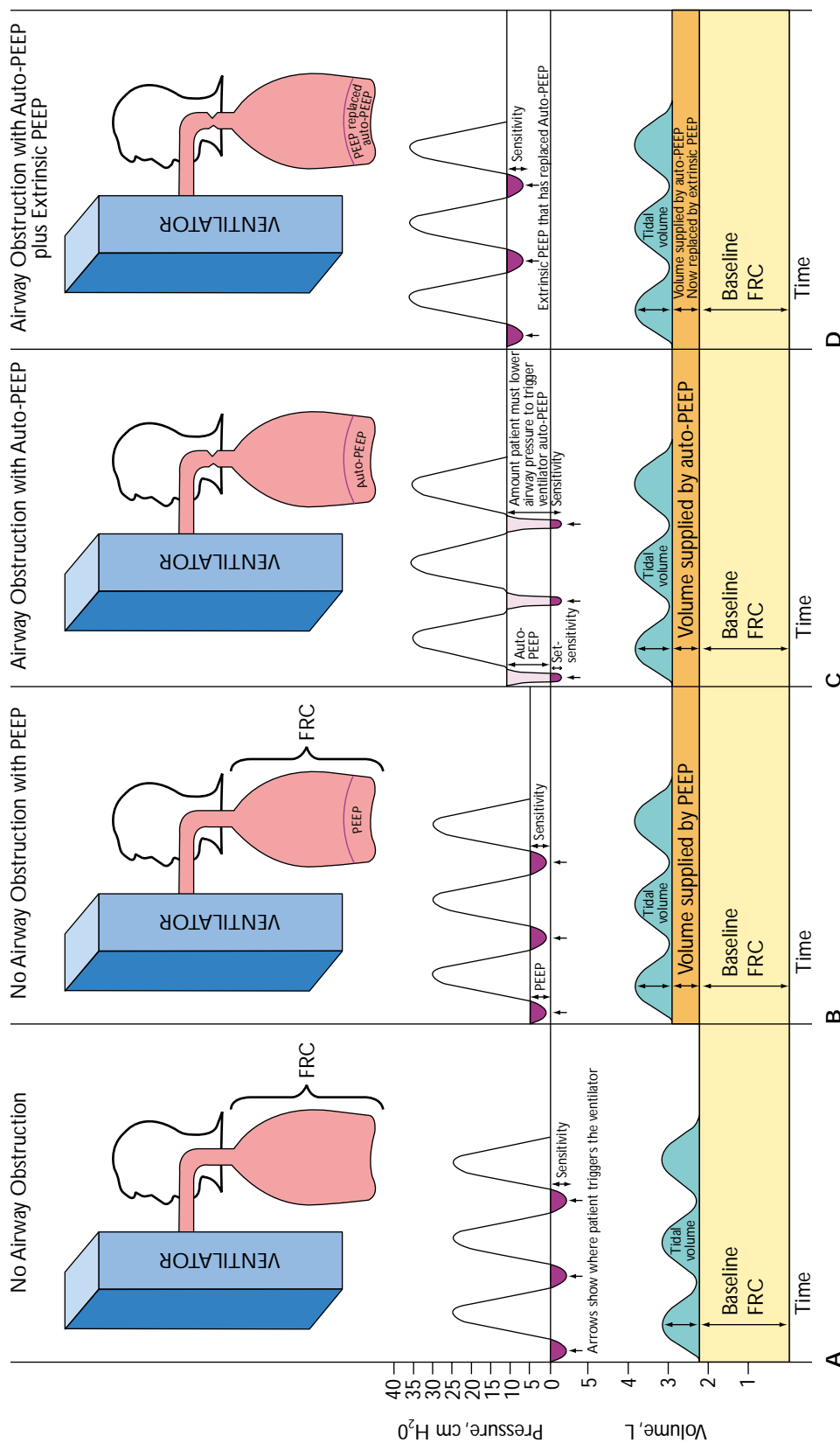


Figure 5. Schematic illustration of positive end-expiratory pressure (PEEP) as a method to elevate mean airway pressure and improve oxygenation. A) During mechanical ventilation without extrinsic PEEP, patients without airway obstruction normally exhale to functional residual capacity (FRC). To trigger the ventilator, patients must lower airway pressure to zero by a level equal to sensitivity. B) During mechanical ventilation with extrinsic PEEP, patients without airway obstruction exhale to a new FRC that equals the baseline FRC plus the volume added by end-expiratory pressure (PEEP). To trigger the ventilator, patients must lower airway pressure below PEEP by a level equal to sensitivity. C) During mechanical ventilation without extrinsic PEEP, patients with airway obstruction may not be able to exhale to FRC, leading to air trapping. The trapped air creates end-expiratory pressure called auto-PEEP. Because the ventilator cannot sense auto-PEEP, spontaneously breathing patients must lower the airway pressure by a level equal to sensitivity plus auto-PEEP to trigger the ventilator, which may take considerable effort. D) In the presence of auto-PEEP, application of extrinsic PEEP approximately equal to auto-PEEP may replace auto-PEEP without increasing lung volume. To trigger the ventilator, patients must lower airway pressure only to a level equal to sensitivity. $H_2O = \text{water}$.

lead to severe and rapid hypoxemia that is reversed by artificially increasing airway pressure at the end of expiration to force the alveoli open and maintain a greater lung volume.

Improvement in oxygenation correlates with the mean airway pressure, which is defined as the airway pressure averaged over time. Therefore, methods that raise the mean airway pressure also improve oxygenation. Positive end-expiratory pressure (PEEP) is the most commonly employed method to elevate mean airway pressure and improve oxygenation (**Figure 5**). PEEP and continuous positive airway pressure, which is PEEP applied to spontaneous breaths, may enhance oxygenation and allow reduction in FiO_2 with its attendant risk of toxicity. Excessive PEEP, however, may reduce cardiac output and impair systemic oxygen delivery, thus offsetting any improvement in oxygenation. The application of PEEP has traditionally been used to improve oxygenation in patients with respiratory failure caused by various conditions, especially ARDS.

Use in patients with acute respiratory distress syndrome. PEEP improves oxygenation in patients with ARDS by decreasing intrapulmonary shunting.¹⁵ This decrease is accomplished in several ways, including the recruitment of collapsed alveoli, increase in FRC, and redistribution of lung water from the alveoli into the perivascular interstitial space.¹⁶ Patients with ARDS typically have a decreased end-expiratory lung volume, so that tidal breathing takes place on the lower, flat part of the pressure-volume curve.¹⁷ By increasing tidal volume, the application of PEEP moves tidal breathing to a part of the curve with higher compliance and decreases the work of breathing.¹⁷ In one group of patients with ARDS, survival rates at 28 days improved when PEEP was applied to keep the alveoli open as a part of a protective ventilatory strategy.¹⁸

Auto-Positive End-Expiratory Pressure

Some patients with asthma or chronic obstructive pulmonary disease (COPD) may develop air trapping caused by flow limitation and inadequate emptying of the lungs during exhalation. This higher lung volume at the end of exhalation leads to an increased end-expiratory pressure called *auto-PEEP*. Auto-PEEP (**Figure 5**) is measured by occluding the expiratory port of the ventilator during the last 0.5 seconds of exhalation. The auto-PEEP measurement is extremely sensitive to timing. Several trials are usually needed to achieve proper timing with respect to the ventilatory cycle and patient effort.

Modern ventilators measure auto-PEEP automatically. Most older ventilator models cannot sense auto-PEEP, and the patient must decrease airway pressure by an amount equal to auto-PEEP plus the trigger sensitivity in order to trigger the ventilator. Auto-PEEP may therefore cause difficulty triggering the ventilator during spontaneous breaths, which may lead to an excessive work of breathing and respiratory muscle fatigue in some patients.¹⁹ The normal triggering effort during spontaneous breaths can be returned by adding extrinsic PEEP to a level just below the auto-PEEP level.²⁰ Application of extrinsic PEEP is not helpful in patients who are not breathing spontaneously.

Not all patients with detectable auto-PEEP experience air trapping and hyperinflation. Auto-PEEP may occur without air trapping during forcible exhalation.²¹ Auto-PEEP and air trapping may also occur without flow limitation if the patient's ventilatory requirements are very high and the time for full exhalation is not sufficient.²¹ High resistance to exhalation, as demonstrated in patients with small endotracheal tubes, may also cause auto-PEEP without air trapping.²¹ In such cases, the addition of extrinsic PEEP does not decrease the work of breathing and may be detrimental.²¹

If extrinsic PEEP is applied to patients with true airflow limitation, airway pressures must be monitored. If, after the addition of extrinsic PEEP, the peak and plateau airway pressures do not rise during a passive machine-delivered breath, flow limitation is present and the extrinsic PEEP may be potentially beneficial.²² In other patients, extrinsic PEEP has the potential to worsen hyperinflation and cause volume-related trauma and hemodynamic compromise.²³

Inspiratory Flow Rate

During ACV and SIMV, the inspiratory flow rate is often set at 60 L/min on a mechanical ventilator. Patients with COPD, however, may attain better gas exchange with flow rates as high as 100 L/min. Higher flow rates allow the delivery of a tidal volume in a shorter period of time and leave a longer period of time for exhalation, which favors more complete lung emptying and less gas trapping.²⁴ With higher flow rates, however, peak inspiratory pressure also increases and may exceed the ventilator safety limits, which reduces delivered tidal volume. If the inspiratory flow rate is too slow to meet the ventilatory requirements, the patient generates highly negative intrapleural pressure by inhaling against a closed inspiratory valve.²⁵ The negative intrapleural pressure may lead to muscle fatigue and even pulmonary edema.

ALTERNATIVE MODES OF VENTILATION

Noninvasive Positive Pressure Ventilation

Positive pressure ventilation may be delivered through the endotracheal tube of an intubated patient or through specialized face or nasal masks in patients who are not intubated. This technique is referred to as *noninvasive positive pressure ventilation* (NIPPV). ACV, SIMV, or PSV with or without PEEP or continuous positive airway pressure may all be delivered noninvasively. In patients with acute respiratory failure, studies show that noninvasive ventilation results in effective improvements in gas exchange, fewer complications, and a shorter stay in the intensive care unit when compared with conventional ventilation.²⁶ Similarly, NIPPV reduces the need for intubation, length of hospital stay, and in-hospital mortality in patients with acute exacerbations of COPD.²⁷ Continuous positive airway pressure delivered noninvasively by nasal mask is commonly used in patients with obstructive sleep apnea to prevent upper airway collapse during inspiration.²⁸ NIPPV has also proven useful in the long-term management of neuromuscular diseases.²⁹

Negative Pressure Ventilation

Negative pressure ventilators expose the chest wall to subatmospheric pressure during inspiration, which lowers intrapleural pressure and allows air to enter the lungs. Expiration occurs when the pressure around the chest wall returns to atmospheric levels. Several uncontrolled trials support the benefits of intermittent negative pressure ventilation in patients with chronic respiratory failure caused by neuromuscular, chest wall, or central hypoventilation disorders.³⁰ Similar efficacy was not demonstrated in patients with severe, stable COPD.³⁰ The usefulness of negative pressure ventilation in adult patients with acute respiratory failure remains unproven.³⁰

Airway Pressure Release Ventilation

Airway pressure release ventilation (APRV) is a ventilatory mode that reverses the normal process of breathing. The lungs are kept inflated at a preset pressure level to achieve alveolar distention and recruitment. Exhalation occurs only through cyclic release of the constant airway pressure, which is followed by a rapid restoration. APRV maintains an adequate mean airway pressure; FRC is also maintained without requiring an elevated peak airway pressure. Although APRV appears to be effective as a ventilatory mode and theoretically should be beneficial as a lung-protective strategy, no advantages over conventional mechanical ventilation have been demonstrated.³¹

Mandatory Minute Ventilation

In mandatory minute ventilation, the physician sets a target minute ventilation. If the patient's spontaneous breathing is not adequate to meet that preset need, the ventilator makes up the difference by supplying mechanical breaths. If the patient spontaneously meets or exceeds the target minute ventilation, no support from the ventilator is provided.³²

Pressure-Control Ventilation

Pressure-control ventilation (PCV) delivers a preset airway pressure, as opposed to a preset volume. Pressure-control ventilators that deliver ACV or SIMV breaths are now available. During PCV, the inspiratory pressure and inspiratory time are set by the physician. The actual tidal volume achieved, however, depends on the mechanical properties of the lungs and is not constant. During PCV, the inspiratory flow increases rapidly at first and then decreases exponentially during lung inflation to keep the airway pressure at the preset value. This decreasing inspiratory flow pattern reduces peak airway pressures and can improve gas exchange.³³

SALVAGE MODES OF MECHANICAL VENTILATION

At times in the course of acute respiratory failure, adequate oxygenation with a nontoxic F_{IO_2} or adequate ventilation cannot be achieved with the modalities of ventilatory support previously described. In these cases, "salvage" modes of mechanical ventilation may be employed.

Pressure-Control Inverse Ratio Ventilation

During normal spontaneous breathing, the ratio of inspiratory to expiratory time is 1:2 to 1:3. During pressure-control inverse ratio ventilation (PC-IRV), the inspiratory time is prolonged, lasting up to 50% to 75% of the respiratory cycle, which yields an inspiratory to expiratory time ratio of 1:1 to 3:1. This inversion holds the lungs at peak inflation pressure for a longer time period than conventional ventilation, increasing mean airway pressure without increasing peak inspiratory pressure. Because oxygenation correlates with lung volumes, which derive from mean airway pressure, PC-IRV, like PEEP, has the potential to improve oxygenation. PC-IRV is uncomfortable for the patient, and heavy sedation may be required. In addition, this mode of ventilation can lead to auto-PEEP, worsen hypercapnia, and precipitate barotrauma. PC-IRV should be applied judiciously and only by experienced clinicians.

High-Frequency Jet Ventilation

High-frequency jet ventilation employs a nozzle or

injector to deliver jets of gas directly into the lung at high rates. Although high-frequency jet ventilation has been advocated in patients with severe ARDS or bronchopleural fistulae, no advantage has been demonstrated in prospective comparisons with conventional mechanical ventilation.³⁴ A study comparing "ultrahigh frequency"-jet ventilation (ie, respiratory rates greater than or equal to 180 breaths/min) and conventional ventilation in patients with ARDS suggested improvement in oxygenation in the ultrahigh frequency arm.³⁵ No randomized trial has confirmed these findings.

High-Frequency Oscillation

High-frequency oscillators rhythmically apply pressure to the airway at very high rates using pistons or microprocessor gas controllers.³⁶ In a preliminary evaluation, high-frequency oscillation was shown to be safe and effective in improving gas exchange in adults with severe ARDS who failed conventional mechanical ventilation.³⁷

Partial Liquid Ventilation

Partial liquid ventilation utilizes perfluorocarbon liquids. These liquids do not mix with surfactant and have a low surface tension and a high solubility coefficient for oxygen and carbon dioxide.³⁸ During partial liquid ventilation, liquid perfluorocarbon is instilled into the lungs until the level approximates FRC; standard mechanical ventilation is then superimposed. The preliminary results of a multicenter, randomized, controlled study of the safety and efficacy of partial liquid ventilation in patients with ARDS found no significant differences in mortality or ventilator-free days in comparison with conventional mechanical ventilation.³⁹

Extracorporeal Membrane Oxygenation

Although technically not a mode of mechanical ventilation, extracorporeal membrane oxygenation (ECMO) has been utilized to support oxygenation and ventilation in severe respiratory failure patients who have little or no contribution from the lungs. ECMO utilizes external membranes for oxygenation and removal of carbon dioxide from the blood. This technique is expensive and has a high morbidity. Trials have not demonstrated any survival benefit of ECMO in the treatment of ARDS.^{40,41}

COMPLICATIONS OF MECHANICAL VENTILATION

Mechanical ventilatory support is by no means risk free, and one of the principal goals of mechanical ventilation is avoidance of complications. Close monitoring is necessary to identify patients at risk for or in the early phases of iatrogenic complications. Patients should also

be continuously tested to detect when they are able to discontinue mechanical ventilatory support, because the incidence of most problems is correlated to the duration of mechanical ventilation.³

Complications can be related to the endotracheal tube (ie, laryngeal injury, tracheal stenosis, tracheomalacia, or sinusitis).³ The toxic effects of oxygen have been mentioned previously. Barotrauma, such as pneumothorax, subcutaneous emphysema, or pneumomediastinum, occurs in approximately 10% to 20% of mechanically ventilated patients.⁴² Barotrauma is probably related to asymmetric alveolar overdistention in addition to high airway pressures. Because mechanically ventilated patients are usually supine, the classic apical or apicolateral presentation of pneumothorax occurs less frequently than in nonventilated patients and the diagnosis is often missed.³ Positive pressure ventilation may cause diminished venous return and, in turn, decrease cardiac output in patients with normal myocardial function. Patients with impaired myocardial contractility, however, can experience increased cardiac output because left ventricular afterload is decreased when intrathoracic pressure increases and, conversely, transmural aortic pressure decreases.³

Ventilator-Associated Pneumonia

Ventilator-associated pneumonia is an ominous development that carries a crude mortality rate of approximately 30%.⁴³ Nonpharmacologic strategies to prevent ventilator-associated pneumonia include:⁴³

- Rigorous hand washing by caregivers
- Semirecumbent positioning of the patient to prevent aspiration
- Adequate nutritional support
- Avoidance of gastric distention
- Early removal of endotracheal and nasogastric tubes
- Continuous subglottic suctioning

Although maintenance of adequate pressure in the endotracheal tube cuff is also recommended, cuff pressure above 25 mm Hg may affect perfusion of the tracheal mucosa and result in injury.³ The use of sucralfate in place of histamine₂-receptor antagonists or antacids for stress-ulcer prophylaxis is associated with lower rates of ventilator-associated pneumonia.⁴⁴ Other pharmacologic interventions that have been advocated for prevention of ventilator-associated pneumonia include: avoidance of unnecessary antibiotics, use of chlorhexidine oral rinse, and use of prophylactic antibiotics and

Table 2. Criteria for Weaning a Patient from Mechanical Ventilation

Hemodynamic and cardiopulmonary stability
$\text{PaO}_2 \geq 60$ mm Hg, $\text{FiO}_2 \leq 40\%$, and positive end-expiratory pressure ≤ 5 cm H_2O
Adequate and stable hemoglobin
Stable renal function
Conscious and alert status
No need for vasoactive or sedative agents, or alert on a stable dose of vasoactive or sedative agents
Afebrile, or febrile and hemodynamically stable status
Intact respiratory drive
Ability to protect airway and clear secretions

FiO_2 = fraction of inspired oxygen, H_2O = water, PaO_2 = arterial partial pressure of oxygen.

granulocyte colony-stimulating factor in ventilated patients with neutropenic fever.⁴³ Routine antibiotic prophylaxis in non-neutropenic patients, however, is not recommended and may be detrimental.⁴³

WEANING FROM MECHANICAL VENTILATION

The term *weaning* is commonly used to describe the process of removing a patient from mechanical ventilation and restoring spontaneous breathing. In some cases (eg, patients recovering from drug overdose or operative anesthesia), ventilatory support may simply be removed when clinical observations suggest that the patient is capable of resuming spontaneous breathing. In other cases, especially in patients recovering from ARDS or severe COPD, weaning can be a labor-intensive and time-consuming endeavor.

Anticipation of Weaning

Discontinuation of mechanical ventilation is generally not considered until the patient has achieved hemodynamic and cardiopulmonary stability. Additionally, many authors recommend that the patient should at least have a PaO_2 greater than or equal to 60 mm Hg while breathing, an FiO_2 less than or equal to 40%, and a PEEP less than or equal to 5 cm H_2O .³ Ideally, the patient should be awake and alert, have adequate and stable hemoglobin and renal function, and be free of the need for vasoactive or sedative agents. The patient should also be afebrile or febrile and hemodynamically stable, have an intact respiratory drive, and have the ability to protect the upper airway and adequately clear secretions. **Table 2** lists the criteria for weaning a patient from mechanical ventilation.

Prediction of Weaning Success

Successful discontinuation of mechanical ventilation depends in large part on the ability of the respiratory muscles to tolerate a workload that is usually elevated as the lungs recover.⁴⁵ Parameters that have been associated with successful weaning include a minute ventilation less than 10 L/min, a vital capacity greater than or equal to 10 mL/kg, and a negative inspiratory pressure less than or equal to -15 cm H_2O .⁴⁶ The rapid shallow breathing index (ie, the ratio of respiratory frequency to tidal volume) has also been advocated. Values less than 105 breaths/min/L are highly predictive when identifying patients who are able to tolerate discontinuation of mechanical ventilation.⁴⁷

Methods of Weaning

Weaning is practiced in a rather empiric manner and little standardization of technique exists. Some clinicians recommend alternating trials of spontaneous breathing, lasting from a few minutes to a few hours, with full ventilatory support. The trial of spontaneous breathing may last up to 2 hours, although recent evidence suggests that a 30-minute trial is equally as efficacious in predicting successful weaning.⁴⁸ Spontaneous breathing can be achieved by removing patients from the ventilator and placing them on an open breathing circuit that provides a continuous flow of gas (ie, "T-tube"). The patient may also breathe spontaneously while still attached to the ventilator, receiving 5 to 7 cm H_2O of pressure support. At the end of the trial, if the patient remains clinically stable with an acceptable arterial oxygen saturation, extubation may be performed based on the physician's clinical judgment. If the patient does not tolerate the trial, full ventilatory support is resumed at the first sign of distress. Trials of spontaneous breathing may be attempted again later, usually at a rate of one trial per day.⁴⁹

Other methods of weaning include gradual reduction in the number of SIMV breaths, which slowly shifts the work of breathing to the patient. A patient may also be gradually removed from PSV; pressure support levels must reach 5 to 7 cm H_2O prior to extubation. In many patients, clinicians combine SIMV with PSV. When weaning is considered in this population, the number of mandatory SIMV breaths is first decreased to zero and then the amount of pressure support is lowered to between 5 and 7 cm H_2O . If the patient tolerates a period of breathing at this level of support, extubation is attempted.

Trials comparing different modes of weaning have produced conflicting results.^{49,50} Therefore, the optimal weaning technique remains unknown. Regardless

of method, weaning should be stopped and mechanical ventilatory support should be restarted if signs of fatigue, distress, respiratory acidosis, or hypoxemia occur. After extubation, patients should be closely monitored for the development of respiratory distress, fatigue, or stridor.

SUMMARY

In conclusion, mechanical ventilation has been a life-saving development during this century. Many methods of mechanical ventilation exist, but which method is superior when employed in particular disease states has yet to be determined. Like all therapeutic modalities, mechanical ventilation is not without complications. Physicians must be familiar with the mechanisms and potential hazards of mechanical ventilation in order to care for the patients who require it.

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