Conventional mechanical ventilation

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INTRODUCTION — Conventional mechanical ventilation refers to the delivery of full or partial ventilatory support by a volume-cycled mechanical ventilator or by pressure support. It also can include the maintenance of positive airway pressure at the end of exhalation, ie, positive end-expiratory pressure (PEEP) [1]. (See "Physiologic and pathophysiologic consequences of positive pressure ventilation").

Different approaches to volume-cycled mechanical ventilation, including alternate methods of mechanical ventilation, such as inverse ratio, high-frequency, or airway pressure release ventilation, and issues relating to permissive hypercapnia and the discontinuation of mechanical ventilation are discussed separately. (See "Alternate modes of mechanical ventilation" and see "High-frequency ventilation" and see "Permissive hypercapnic ventilation" and see "Methods of discontinuing mechanical ventilation").

Partial ventilatory support, which can be delivered as noninvasive positive pressure ventilation, is often sufficient in select circumstances, such as cardiogenic pulmonary edema or hypercapnic respiratory failure due to chronic obstructive pulmonary disease (COPD). (See "Noninvasive positive pressure ventilation in acute respiratory failure"). In other cases, full ventilatory support is provided via an artificial airway such as an endotracheal tube or a tracheostomy tube. (See "Endotracheal tube management" and see "Overview of tracheostomy").

MECHANISM OF BENEFIT — Mechanical ventilation has two beneficial effects: it improves gas exchange and it decreases the work of breathing. The application of positive pressure to the respiratory system can improve ventilation-perfusion (V/Q) matching and decrease intrapulmonary shunting, both of which relieve hypoxemia and diminish hypercapnia, if present.

Alterations in lung mechanics, such as increased airways resistance and decreased lung compliance, result in increased work of breathing for the ventilatory muscles, particularly the diaphragm. Anaerobic metabolism may occur, resulting in lactic acidosis [2]. The initiation of mechanical ventilation (without chemical paralysis) ameliorates patient work to a considerable but incomplete extent, resulting in a fall in plasma lactate and improvement in gas exchange [2,3].

The ventilatory muscles continue to perform some work, although the magnitude of this work tends not to be fatiguing when at least 80 percent of the minute ventilation is provided by the machine (show figure 1) [4].
By alleviating patient work, mechanical ventilation decreases the diaphragm’s demand for excessive blood flow and creates an opportunity for reversing the circumstances producing diaphragmatic fatigue and potentially life-threatening hypercapnia\(^\text{[5,6]}\).

**INDICATIONS** — The decision to initiate mechanical ventilation entails a significant commitment of hospital resources and a set of unique and potentially serious complications. The main indication for mechanical ventilation is acute respiratory failure, as reflected by inability to oxygenate the arterial blood adequately and/or loss of the capacity to sustain adequate alveolar ventilation. Clinically, this is usually manifested by the presence of rapid, shallow breathing\(^\text{[5]}\). Physiologic parameters often used to evaluate the need for mechanical ventilation are listed (show table 1).
The objectives of conventional mechanical ventilation are both physiologic and clinical, and are listed (show table 2) [7].

### Abnormalities Suggestive of the Need for Mechanical Ventilation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Loss of ventilatory reserve</td>
<td>&gt;35 breaths/min</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>&lt;5 mL/kg</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>&lt;10 mL/kg</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>Weaker than ~25 cmH2O</td>
</tr>
<tr>
<td>Negative inspiratory force</td>
<td>10 L/min</td>
</tr>
<tr>
<td>Minute ventilation</td>
<td>10 mmHg</td>
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<tr>
<td>Rise in PCO2</td>
<td></td>
</tr>
<tr>
<td>Refractory hypoxemia</td>
<td></td>
</tr>
<tr>
<td>Alveolar–arterial gradient (FiO₂ = 1.0)</td>
<td>&gt;450</td>
</tr>
<tr>
<td>PaO₂/PaCO₂</td>
<td>&lt;0.15</td>
</tr>
<tr>
<td>PaO₂ with supplemental O₂</td>
<td>&lt;55 mmHg</td>
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Ultimately, the decision to initiate mechanical ventilation must take clinical circumstances into account as well as physiologic derangements. In general, it is important to consider mechanical ventilation early in a patient's course of illness, but the method chosen may depend upon the disease process. As an example, a patient with severe COPD and CO₂ retention may have acute on chronic hypercapnia, and should be considered for noninvasive positive pressure ventilation (NPPV) if emergent intubation is not warranted and other contraindications are not present [8]. In contrast, a patient who is unresponsive due to a drug overdose with the same degree of hypercapnia should be intubated and treated with conventional mechanical ventilation. (See "Noninvasive positive pressure ventilation in acute respiratory failure" and see "General approach to drug intoxications in adults").

Common disorders for which mechanical ventilation may be indicated are listed (show table 3).
The general approach to these problems is as follows:

- In acute pulmonary parenchymal diseases, such as severe pneumonia or the acute respiratory distress syndrome (ARDS), conventional mechanical ventilation is usually indicated to treat refractory hypoxemia or excessive work of breathing. (See "Mechanical ventilation in acute respiratory distress syndrome").
- Cardiogenic pulmonary edema produces a similar physiologic derangement of hypoxemia due to alveolar filling, but intubation can often be avoided through the use of NPPV and judicious application of PEEP [9,10]. (See "Noninvasive positive pressure ventilation in acute respiratory failure" and see "Acute decompensated heart failure (cardiogenic pulmonary edema)").
- Exacerbation of underlying obstructive airways disease is a frequent indication for the initiation of NPPV or conventional mechanical ventilation. Refractory hypoxemia, ventilatory failure, or both may serve as an indication for conventional mechanical ventilation. However, NPPV should be considered primarily in the presence of ventilatory failure (ie, hypercapnia); it remains of unproven value in patients with hypoxemic respiratory failure [11,12]. (See "Mechanical ventilation in acute respiratory failure complicating COPD", see "Mechanical ventilation in adults with status asthmaticus", and see "Noninvasive positive pressure ventilation in acute respiratory failure").
- Respiratory failure due to alveolar hypoventilation in the absence of parenchymal lung disease occurs for several reasons, but is a less common indication for mechanical ventilation. Neuromuscular disorders, including myasthenia gravis, Guillain-Barre syndrome, poliomyelitis, and spinal cord trauma are common etiologies. In addition, patients with acute drug or alcohol intoxications may require transient ventilatory support. The presence of primary ventilatory failure is suggested by CO₂ retention in conjunction with a normal or nearly normal alveolar-arterial oxygen gradient. (See "Interpretation of arterial oxygen tension" and see "Respiratory failure from peripheral neuromuscular disease").
• Systemic illnesses leading to respiratory failure include sepsis and shock. Often mechanical ventilation is indicated in these disorders despite the preservation of relatively normal arterial blood pH. If the PaCO2 is higher than expected to compensate for the acidosis (as predicted by the formula: expected PaCO2 = 1.5 x [HCO3-] + 8±2), the patient generally should be promptly intubated and mechanical ventilation initiated. (See "Approach to the adult with metabolic acidosis" and see "Management of severe sepsis and septic shock in adults").

VENTILATOR CYCLING MECHANISMS — Positive pressure ventilation is classified according to the manner in which inspiration is terminated. Common cycling mechanisms include volume-, pressure-, flow-, and time-cycled.

• Volume-cycled, in which inspiration is terminated after delivery of a preset tidal volume, is the most common form of fully supported ventilation (see "Choice of mode" below).
• Pressure-cycled, in which inspiration ceases when a preset maximum pressure is reached. Delivered volume varies with alterations in lung mechanics, and minute ventilation is not assured. Pressure-limited forms of mechanical ventilation, such as pressure-controlled inverse ratio ventilation, have been increasingly used because they may lessen volutrauma in acute lung injury [13]. (See "Alternate modes of mechanical ventilation").
• Flow-cycled, in which inspiration is terminated when a particular flow rate is reached. Pressure support ventilation is an example of flow-cycled mechanical ventilation. Here, a preset airway pressure is applied once the machine is triggered and is cycled off after the inspiratory flow decreases to a predetermined percentage of its peak value (show figure 2).
• Time-cycled, in which inspiration is terminated following a preset inspiratory time. Both the volume of gas delivered and the resultant airway pressure vary from breath to breath as a function of changes in lung mechanics. Home ventilators, such as the Puritan Bennett LP-10, are time-cycled.

Pressure support ventilation Changes in airway pressure, flow, and volume as measured in the distal endotracheal tube during unsupported and pressure-supported spontaneous breaths in intubated patients. The unsupported patient (blue lines) must first generate an initial negative pressure "spike" to open the ventilator demand valves and then must maintain a small amount of negative pressure during inspiration to produce flow through the ventilator circuitry. The addition of increasing levels of pressure support (green lines) provides plateaus of positive pressure that augment the spontaneous tidal volume in accordance with the patient's spontaneous respiratory flow demand and inspiratory time pattern. (Redrawn from Respir Care 1987; 32:447.)
**VOLUME-CYCLED VENTILATION** — With volume-cycled ventilators, the controlled variables of tidal volume and inspiratory flow determine airway pressure and inspiratory time. Variations in airway resistance or lung compliance alter airway pressures but do not affect minute ventilation. Figure 3 shows how variations in airways resistance, lung compliance, inspiratory flow rate, and tidal volume affect airway pressure and inspiratory time (show figure 3).

**Waveforms for volume-cycle ventilator** Pressure, flow, and volume waveforms for a volume-cycle ventilator using a constant flow generator (square wave) at baseline (A), and with increased delivered tidal volume (B), reduced lung compliance (C), and enhanced respiratory flow rate (D). An increase in peak airway pressure occurs in the last three settings. (Adapted from Spearman, CB, Egan, DF, Egan, J, Fundamentals of respiratory therapy, 4th ed, Mosby, St Louis, 1982.)

There are three methods of initiating the inspiratory phase in volume-cycled mechanical ventilators: controlled, assist-control, and intermittent mandatory ventilation (IMV) (show figure 4).
Controlled mechanical ventilation — In controlled ventilation, minute ventilation is completely dependent upon the rate and tidal volume set on the ventilator. Any respiratory efforts made by the patient do not contribute to minute ventilation.

Controlled ventilation is the required ventilatory mode in patients who are making no respiratory effort (e.g., spinal cord injury or drug overdose and those who have been subjected to pharmacologic paralysis). Because oxygen consumption decreases with neuromuscular paralysis [14], the combination of pharmacologic paralysis and controlled ventilation is sometimes used in patients with ARDS for this purpose.

Combined neuromuscular paralysis and controlled mechanical ventilation can also be used to avoid volutrauma in patients with ARDS [15] and to avoid barotrauma in asthmatics who are difficult to ventilate [16,17]. In these settings, hypercapnia is accepted provided that oxygenation is maintained. (See "Permissive hypercapnic ventilation").

Assist-control mechanical ventilation — In the assist-control (A/C) mode, the ventilator senses an inspiratory effort by the patient and responds by delivering a preset tidal volume. Every inspiratory effort that satisfies the ventilator's demand valve trigger threshold initiates delivery of the preset tidal volume (see "Trigger...".)
mode and sensitivity" below). A control mode back-up rate is set on the ventilator to prevent hypoventilation.

Patient work is required to trigger the ventilator, and continues during inspiration [18]. In the presence of auto-PEEP, the effective trigger threshold (and hence patient work) is increased by the amount of auto-PEEP present [19]. (See "Physiologic and pathophysiologic consequences of positive pressure ventilation" section on Auto-PEEP).

**Intermittent mandatory ventilation** — With intermittent mandatory ventilation (IMV), the degree of ventilatory support is determined by the selected IMV rate. At regular intervals, the ventilator delivers a breath based upon a preset tidal volume and rate. In addition, the patient is allowed to breathe spontaneously through the ventilator circuit at a tidal volume and rate determined according to need and capacity.

Most present day ventilators synchronize the intermittent ventilator breaths with inspiratory effort by the patient, a modality termed synchronized IMV or SIMV. However, this modification requires a trigger modality, either a demand valve [20] or flow-by [21], both of which need patient effort to trigger and therefore increase the work of breathing (see "Trigger mode and sensitivity" below).

**Comparison of IMV and A/C** — IMV and A/C are the most frequently used forms of mechanical ventilation (see "Choice of mode" below) [22]. Proposed advantages to IMV compared with A/C include improved synchrony, preservation of respiratory muscle function, lower mean airway pressures, and a decreased tendency to develop auto-PEEP [23]. Alternatively, assist-control ventilation may be better suited to critically ill patients who require full ventilatory support and in whom fluctuations in tidal volume are undesirable.

**Pressure support ventilation** — Pressure support ventilation (PSV) is flow-cycled in that, once triggered by a demand valve, the preset pressure is sustained until the inspiratory flow tapers, usually to 25 percent of its maximal value [24]. PSV tends to be a comfortable ventilatory modality because the patient has greater control over ventilator cycling and flow rates. Close monitoring is required whenever PSV is used alone because neither tidal volume nor minute ventilation is guaranteed.

**Mechanism of benefit** — During PSV, the work of breathing performed by the patient is shifted over to the machine in a manner inversely proportional to the level of pressure support [24], provided the inspiratory flow is sufficient to meet patient demand [25]. In addition, the cycle-per-cycle variability of peak inspiratory flow (which is permitted in pressure-limited modes) may allow better patient comfort [26]. In newer mechanical ventilators at a given level of PSV, increasing the inspiratory rise time, which shortens the time until maximal airway pressure is achieved, can also be used to decrease work of breathing [27].

**Clinical use** — PSV can be added during full or partial support with SIMV to overcome endotracheal tube and ventilator circuitry resistance encountered during spontaneous breaths. The resistance of the endotracheal tube varies as a function of tube diameter and inspiratory flow rate [28]. Pressure support levels in excess of 10 cmH2O may be needed to overcome the resistance of an endotracheal tube, particularly with small (<7 mm) endotracheal tubes [29] or in patients with COPD.
At higher levels of pressure support (>10 cmH2O), tidal volume is augmented and respiratory rate slows.

Attempting to provide full ventilatory support to patients with PSV often requires relatively high levels of pressure, since low pressures increase the risk of alveolar collapse [31]. In one report of eight patients with acute respiratory failure, PSV levels of 27±5 cmH2O were required to attain a stable breathing pattern [32]. The use of lower levels of PSV, in conjunction with adequate PEEP, has been reported in selected patients with acute lung injury and an intact respiratory drive. Although patients who successfully tolerate this approach are usually able to be ventilated at PSV levels below 20 cmH2O, these patients are less severely ill than those who cannot tolerate this modality [33].

Lower levels of pressure support may also be appropriate for some patients with obstructive airway disease. As an example, in a study of patients 36 patients, most of whom had COPD, physiologic variables improved progressively with increasing levels of pressure support, but patient comfort was greatest in the middle ranges of PSV, around 10 to 15 cm H2O [34]. The application of PEEP did not affect patient comfort, and resulted in a higher tidal volume and lower respiratory rate at each level of PSV studied [34].

When PSV is used during weaning from mechanical ventilation, contraction of the sternocleidomastoid muscle and increases in airway occlusion pressure (Pao) suggest that pressure support has decreased to a potentially fatiguing level [35,36].

**Limitations** — PSV may be poorly tolerated in patients with high airway resistance and may not provide a sufficient minute ventilation because of the preset high initial flow and terminal inspiratory flow algorithms that are standard on most critical care ventilators [7,37]. Central apnea or hyperpnea may complicate PSV during sleep, resulting in increased sleep fragmentation when compared to assist-control ventilation [38].

In addition, the application of PSV to patients with COPD does not decrease auto-PEEP. The continued presence of auto-PEEP can increase patient work and worsen respiratory muscle fatigue, making PSV unsuitable for full ventilatory support in these patients [39]. PSV can be used to provide noninvasive respiratory support via a tight-fitting nasal or face mask. The benefits of noninvasive and invasive PSV on diaphragmatic function, respiratory mechanics, and gas exchange are similar; however, noninvasive ventilation may be better tolerated and may decrease the risk of nosocomial infection [40,41]. (See "Noninvasive positive pressure ventilation in acute respiratory failure").

Finally, as with A/C and SIMV, ventilator asynchrony can occur during PSV employed for full ventilatory support [42]. Thus, PSV is poorly suited to provide full ventilatory support to patients with acute respiratory failure, and is more appropriate during weaning from mechanical ventilation. (See "Methods of discontinuing mechanical ventilation").

**CHOICE OF MODE** — The initial selection of ventilator mode is often based on the physician's familiarity with various modes, the clinical setting (surgical versus medical ICU), geography, and institutional guidelines, if available. A point prevalence survey of 1638 patients in 412 ICUs in North America, South America, Spain, and
Portugal found that assist-control was the most frequently utilized ventilator modality, employed in 47 percent of patients at the time of the survey. SIMV, usually in combination with pressure support, was used in an additional 31 percent [22]. A 28-day international survey by the same investigators noted that over 60 percent of patients with COPD or ARDS complicated by respiratory failure were treated with assist-control during the first week of hospitalization [43].

During both A/C ventilation and SIMV, the diaphragm and other respiratory muscles continue to perform some work, which may prevent atrophy. In contrast, prolonged ventilation with CMV (controlled mechanical ventilation) appears to result in ventilator-induced diaphragmatic dysfunction [44,45]. Unfortunately, the optimal balance of diaphragmatic rest that achieves recovery and avoids atrophy is unknown. When a patient is fully supported and the A/C mode is changed gradually over to decreasing amounts of mechanical support in the SIMV mode, the amount of inspiratory work per liter of ventilation increases progressively. At levels of SIMV supporting less than 80 percent of the assisted minute ventilation, a potentially fatiguing inspiratory load is created [4].

When a patient receives more than 50 percent of minute ventilation from the machine in the SIMV mode, the hemodynamic benefit of negative pressure from spontaneous breaths through the ventilator circuitry is lost. Thus, higher levels of cardiac output, mean blood pressure, and wedge pressure during SIMV compared with A/C are seen only when the machine provides the patient with less than half the minute ventilation [46]. However, increases in oxygen consumption above values seen in the A/C mode also occur at these low levels of SIMV support. Thus, the hemodynamic benefits gained at low SIMV back-up rates appear to be offset by the increase in work of breathing.

In view of these observations, patients receiving essentially full ventilatory support by SIMV should have at least 80 percent of their minute ventilation supplied by machine-supported breaths. At this level of support, there is no particular advantage of one modality over the other, and the choice may be more a function of clinician familiarity than patient benefit. An exception to this general rule was noted in an ACCP consensus statement that cautioned against the initial use of A/C in awake patients with obstructive airways disease, since this can lead to progressive hyperinflation [7].

**VENTILATOR SETTINGS** — Ventilatory support requires consideration of trigger mode and sensitivity, respiratory rate, tidal volume, flow rate, flow pattern, and the fraction of inspired oxygen (FiO2).

**Trigger mode and sensitivity** — Full ventilatory support via either A/C or SIMV requires a demand valve trigger that senses a negative airway pressure deflection generated by the patient during breath initiation. This method is often referred to as "pressure triggering." Demand valve sensitivity should allow the patient to trigger the ventilator easily and avoid a prolonged period between the initial effort and the machine breath. A trigger sensitivity of -1 to -3 cmH2O is most often used. If, however, the trigger sensitivity set on the machine is too sensitive, the machine may "auto-cycle" due to patient movement or the subtle pressure deflections caused by water moving within the ventilator tubing.
Auto-PEEP increases the work needed to trigger the ventilator, since the patient must generate enough negative pressure in the ventilator tubing to overcome not only the trigger sensitivity set on the ventilator, but also the auto-PEEP. Patients can become dyspneic and "locked out" from obtaining sufficient ventilator breaths to meet their needs if they are unable to generate this additional negative pressure (show figure 5) [47].

The application of small amounts of PEEP on the ventilator can offset this effect by decreasing the inspiratory pressure the patient must generate to trigger a machine breath, without worsening dynamic hyperinflation (show figure 6) [19,48].
Prolongation of the expiratory time, by either decreasing the tidal volume or increasing the inspiratory flow rate, will reduce auto-PEEP and may be sufficient to keep the patient from being locked out.

**Flow-by triggering** involves the use of a continuous gas (baseline) flow circulating past the patient. A predetermined deviation from this flow (trigger sensitivity) created by the onset of patient inspiration results in machine triggering (show figure 7).
Flow-by triggering requires less work of breathing when used in patients receiving continuous positive airway pressure (CPAP) [49]. Flow-by triggered CPAP decreases inspiratory work in COPD patients; a possible mechanism is that the base flow creates a modest PEEP effect [50].

Flow-by triggering decreases the inspiratory effort during spontaneous SIMV breaths but not machine-triggered breaths [21]. As a result, the use of flow-by triggered SIMV for purposes of full ventilatory support probably confers no advantage over demand valve triggering. Similarly, flow-by triggering offers no advantage over demand valve triggering when using pressure support ventilation [51].

Respiratory rate — A respiratory rate set between 10 and 20 breaths/minute is generally sufficient for clinically stable patients in acute respiratory failure. If SIMV is used, the rate should be adjusted to achieve at least 80 percent of the patient's minute ventilation. During A/C ventilation, the rate should be set to about four breaths per minute lower than the patient's spontaneous rate. This allows assessment of a patient's PCO2 and minute ventilation and prevents a significant decrease in minute ventilation if apnea occurs [52].
In normal volunteers, increases in inspiratory time decrease respiratory rate [53]. Since inspiratory time is equal to tidal volume divided by inspiratory flow, an increase in tidal volume or a reduction in inspiratory flow may be useful in attempts to decrease respiratory rate. In addition, medications are commonly employed to relieve anxiety and help blunt respiratory drive, thereby improving synchrony with the ventilator. Intermittent intravenous bolus sedation is preferred to continuous intravenous sedation whenever possible, as the latter has been associated with a prolongation in the duration of mechanical ventilation [54]. (See "Use of sedative medications in critically ill patients").

Conversely, patients with acute lung injury ventilated using low tidal volumes may require an increase in respiratory rate to maintain appropriate minute ventilation; this in turn may cause progressive auto-PEEP. In a study of 14 patients receiving low tidal volume ventilation, increasing the respiratory rate led to increased auto-PEEP unless the inspiratory flow rate was also increased [55]. Thus, to increase minute ventilation in this setting, both respiratory rate and inspiratory flow need to be increased simultaneously.

**Tidal volume** — Historically, the use of supraphysiologic tidal volumes (10 to 15 mL/kg) was found to prevent atelectasis-induced hypoxemia in anesthetized mechanically ventilated patients with relatively normal lungs [56] and obviated the need for periodic hyperinflation "sigh" breaths [57]. However, inflation of the lung at the high end of the pressure-volume curve increases the risk of adverse side effects [58]. Consequently, a maximum tidal volume of 10 mL/kg should be employed for mechanically ventilated patients who do not have significant lung disease, as in the postoperative setting or following drug overdose.

Lower tidal volumes less than 10 mL/kg should be used in patients with lung disease, such as ARDS, obstructive airways disease, fibrotic lung disease, or after lung resection. In these settings, large tidal volumes can lead directly to barotrauma or induce and perpetuate lung injury, a phenomenon called volutrauma or ventilator-associated lung injury (VALI) [59,60]. (See "Physiologic and pathophysiologic consequences of positive pressure ventilation", and see "Inflammatory mechanisms of lung injury during mechanical ventilation").

The ARDS Network trial demonstrated decreased mortality (31 versus 40 percent) in patients with acute lung injury (PaO2/FiO2 ratio <300) who were treated with a lower tidal volume strategy (6 mL/kg) compared to the conventional ventilation group (12 mL/kg) [61]. As a result, an initial tidal volume of 6 mL/kg ideal body weight has become common in patients with acute lung injury, although some controversy still remains regarding this approach. (See "Mechanical ventilation in acute respiratory distress syndrome").

**Flow rate and I:E ratio** — The peak flow rate determines the maximum inspiratory flow delivered by the ventilator during inspiration. Flow rates of 60 L/min are often sufficient; however, higher rates of inspiratory flow are frequently necessary to produce adequate gas exchange, especially in patients with obstructive airways disease. Insufficient inspiratory flow rates produce dyspnea, spuriously low peak inspiratory pressures, and scalloping of the inspiratory airway pressure tracing caused by the patient's inspiratory effort [56].
For a given tidal volume and respiratory rate, increasing the inspiratory flow rate shortens inspiratory time and decreases the inspiratory to expiratory (I:E) ratio. Generally, an I:E ratio of 1:2 or less is required, and a ratio of 1:3 or less is preferable in spontaneously breathing patients. In the setting of dynamic hyperinflation, prolonging the expiratory time results in lower mean airway pressure and less dynamic hyperinflation and reduced auto-PEEP. The degree of dynamic hyperinflation is more predictive of the likelihood of barotrauma than is peak inspiratory pressure [62]. (See “Positive end-expiratory pressure (PEEP”).)

**Flow pattern** — Microprocessor-controlled mechanical ventilators can deliver several inspiratory flow patterns (show figure 8):

- Constant (square wave)
- Decelerating (ramp wave)
- Sinusoidal

![Ventilator flow and pressure waveforms](image)

*Ventilator flow and pressure waveforms* Airway pressure (Paw) and flow rate shown for constant, decelerating, and sinusoidal inspiratory flow waveforms. Inspiratory time and tidal volume were held constant. Peak inspiratory airway pressures are similar with all waveforms, but mean airway pressure is highest with the decelerating inspiratory flow wave. (Redrawn from Banner, HJ, Lanpontang, S, in Current Respiratory Care, Kacmarek, RM, Stoller, JK (Eds), BC Decker, Philadelphia, 1968).

The ramp wave pattern may distribute ventilation more evenly than other types of wave forms, particularly when airways obstruction is present [63]. Peak inspiratory pressure, dead space, and pCO2 decrease in patients with obstructive airways disease ventilated with a ramp wave form; oxygenation remains unchanged [64].

The effects on mean airway pressure and therefore the risk of hemodynamic sequelae and barotrauma are unpredictable and must be empirically determined. Microprocessor-controlled ventilators have the ability to apply an inspiratory hold, which inhibits exhalation until there has been a preset pause (end-inspiratory pause). This intentionally results in elevations of mean airway pressure, but can lead to improvements in the distribution of gas through collateral ventilation (Pendelluft flow), which may be of value in conditions such as ARDS.
Fraction of inspired oxygen — Attempts should always be made to utilize the lowest possible fraction of inspired oxygen (FiO2) that maintains the arterial oxygen saturation above 90 percent; this is usually achieved at an arterial PO2 above 60 mmHg. An FiO2 below 0.5 (50 percent oxygen) is preferable to minimize oxygen toxicity. The 1993 ACCP consensus conference acknowledged that an arterial saturation slightly below 90 percent is acceptable in ARDS patients when the tradeoff would be a higher plateau pressure and greater risk of volutrauma [7]. (See "Oxygen toxicity").

PATIENT-VENTILATOR INTERACTION — Ideally, the ventilator should be set to cycle in synchrony with the patient's intrinsic respiratory rhythm in order to minimize dyspnea and promote unloading of the respiratory muscles. Despite this, dyspnea is felt to be common in mechanically ventilated patients, although it is difficult to study quantitatively [65]. Adjusting the inspiratory flow rate and tidal volume to match the patient's needs is one way that clinicians may be able to decrease dyspnea and improve patient comfort [66]. Although these interventions may improve patient-ventilator interaction, increased inspiratory flow rates may cause profound elevations in peak airway pressure [67].

A second consideration relevant to patients receiving mechanical ventilation is that, in general, inspiratory time (tidal volume divided by inspiratory flow) is inversely proportional to respiratory rate. Thus, attempts to increase minute ventilation solely by raising tidal volume can prove futile unless inspiratory flow is also increased to maintain a constant inspiratory time [55,68]. If this is not done, the inspiratory time will rise, and the respiratory rate will fall.

Ineffective triggering (resulting in a "locked out" patient) appears to be relatively common, and may occur in up to one-third of a patient's inspiratory efforts [68]. The time at which a patient commences an expiratory effort in relation to the end of mechanical inspiration determines the success of the ensuing effort in triggering the ventilator. The earlier that a patient begins exhalation, the greater the probability that triggering the subsequent breath will be ineffective. (See "Positive end-expiratory pressure (PEEP)").

Sedation — Sedation is generally required in order to perform mechanical ventilation safely in intubated patients in the ICU. Mechanical ventilation can cause a significant amount of anxiety in a patient regardless of the etiology of respiratory failure [69]. Rapid diagnosis and treatment of anxiety should be made, whether it is directly or indirectly caused by the ventilator. (See "Acute toxic-metabolic encephalopathy in the intensive care unit").

Propofol and midazolam are widely used to sedate patients with acute respiratory failure during mechanical ventilation. Both are rapid acting and do not suppress adrenal function; however, propofol has been shown to result in earlier extubation and earlier ICU discharge, when compared with midazolam [70,71]. Daily interruption of sedation is recommended in all mechanically ventilated patients as this approach had been associated with a decreased length of stay in the ICU by 3.4 days and decreased days on the ventilator by 2.4 days, without any increase in associated complications [72,73]. (See "Use of sedative medications in critically ill patients").