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Overview of Mechanical Ventilation

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Mechanical ventilation can be

- [Noninvasive](#), involving various types of face masks
- Invasive, involving [endotracheal intubation](#)

Selection and use of appropriate techniques require an understanding of respiratory mechanics.

Indications

There are numerous indications for endotracheal intubation and mechanical ventilation (see Table: [Situations Requiring Airway Control](#)), but, in general, mechanical ventilation should be considered when there are clinical or laboratory signs that the patient cannot maintain an airway or adequate oxygenation or ventilation.

Concerning findings include

- Respiratory rate > 30/min
- Inability to maintain arterial oxygen saturation > 90% with fractional inspired oxygen (FiO₂) > 0.60
- PaCO₂ >50 mm Hg with pH < 7.25

The decision to initiate mechanical ventilation should be based on clinical judgment that considers the entire clinical situation and should not be delayed until the patient is in extremis.

Respiratory Mechanics

Normal inspiration generates negative intrapleural pressure, which creates a pressure gradient between the atmosphere and the alveoli, resulting in air inflow. In mechanical ventilation, the pressure gradient results from increased (positive) pressure of the air source.

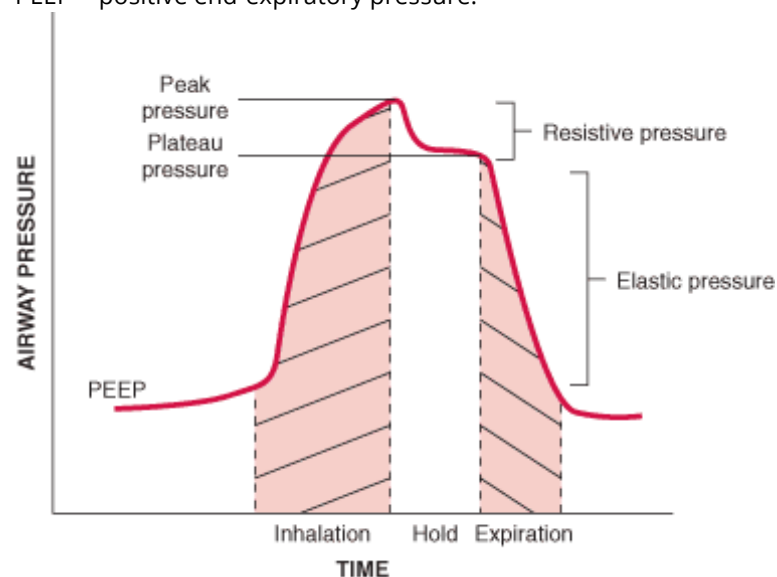
Peak airway pressure is measured at the airway opening (P_{ao}) and is routinely displayed by mechanical ventilators. It represents the total pressure needed to push a volume of gas into the lung and is composed of pressures resulting from inspiratory flow resistance (resistive pressure), the elastic recoil of the lung and chest wall (elastic pressure), and the alveolar pressure present at the beginning of the breath (positive end-expiratory pressure [PEEP]—see Figure: [Components of airway pressure during mechanical ventilation, illustrated by an inspiratory-hold maneuver](#)). Thus

$$\text{Peak airway pressure} = \\ \text{resistive pressure} + \\ \text{elastic pressure} + \text{PEEP}$$

Resistive pressure is the product of circuit resistance and airflow. In the mechanically ventilated patient, resistance to airflow occurs in the ventilator circuit, the endotracheal tube, and, most importantly, the patient's airways. (NOTE: Even when these factors are constant, an increase in airflow increases resistive pressure.)

Components of airway pressure during mechanical ventilation, illustrated by an inspiratory-hold maneuver

PEEP = positive end-expiratory pressure.



Elastic pressure is the product of the elastic recoil of the lungs and chest wall (elastance) and the volume of gas delivered. For a given volume, elastic pressure is increased by increased lung stiffness (as in pulmonary fibrosis) or restricted excursion of the chest wall or diaphragm (eg, in tense ascites or massive obesity). Because elastance is the inverse of compliance, high elastance is the same as low compliance.

End-expiratory pressure in the alveoli is normally the same as atmospheric pressure. However, when the alveoli fail to empty completely because of airway obstruction, airflow limitation, or shortened expiratory time, end-expiratory pressure may be positive relative to the atmosphere. This pressure is called intrinsic PEEP or autoPEEP to differentiate it from externally applied (therapeutic) PEEP, which is created by adjusting the mechanical ventilator or by placing a tight-fitting mask that applies positive pressure throughout the respiratory cycle.

Any elevation in peak airway pressure (eg, > 25 cm H₂O) should prompt measurement of the end-inspiratory pressure (plateau pressure) by an end-inspiratory hold maneuver to determine the relative contributions of resistive and elastic pressures. The maneuver keeps the exhalation valve closed for an additional 0.3 to 0.5 sec after inspiration, delaying exhalation. During this time, airway pressure falls from its peak value as airflow ceases. The resulting end-inspiratory pressure represents the elastic pressure once PEEP is subtracted (assuming the patient is not making active inspiratory or expiratory muscle contractions at the time of measurement). The difference between peak and plateau pressure is the resistive pressure.

Elevated resistive pressure (eg, > 10 cm H₂O) suggests that the endotracheal tube has been kinked or plugged with secretions or that an intraluminal mass or bronchospasm is present.

Increased elastic pressure (eg, > 10 cm H₂O) suggests decreased lung compliance due to

- Edema, fibrosis, or [lobar atelectasis](#)
- Large [pleural effusions](#), [pneumothorax](#) or fibrothorax
- Extrapulmonary restriction as may result from circumferential burns or other chest wall deformity, ascites, pregnancy, or massive obesity
- A tidal volume too large for the amount of lung being ventilated (eg, a normal tidal volume being delivered to a single lung because the endotracheal tube is malpositioned)

Intrinsic PEEP (auto PEEP) can be measured in the passive patient through an end-expiratory hold maneuver. Immediately before a breath, the expiratory port is closed for 2 sec. Flow ceases, eliminating resistive pressure; the resulting pressure

reflects alveolar pressure at the end of expiration (intrinsic PEEP). Although accurate measurement depends on the patient being completely passive on the ventilator, it is unwarranted to use neuromuscular blockade solely for the purpose of measuring intrinsic PEEP. A nonquantitative method of identifying intrinsic PEEP is to inspect the expiratory flow tracing. If expiratory flow continues until the next breath or the patient's chest fails to come to rest before the next breath, intrinsic PEEP is present. The consequences of elevated intrinsic PEEP include increased inspiratory work of breathing and decreased venous return, which may result in decreased cardiac output and hypotension.

The demonstration of intrinsic PEEP should prompt a search for causes of airflow obstruction (eg, airway secretions, decreased elastic recoil, bronchospasm); however, a high minute ventilation (> 20 L/min) alone can result in intrinsic PEEP in a patient with no airflow obstruction. If the cause is airflow limitation, intrinsic PEEP can be reduced by shortening inspiratory time (ie, increasing inspiratory flow) or reducing the respiratory rate, thereby allowing a greater fraction of the respiratory cycle to be spent in exhalation.

Means and Modes of Mechanical Ventilation

Mechanical ventilators are set to deliver a constant volume (volume cycled), a constant pressure (pressure cycled), or a combination of both with each breath. Modes of ventilation that maintain a minimum respiratory rate regardless of whether or not the patient initiates a spontaneous breath are referred to as assist-control (A/C). Because pressures and volumes are directly linked by the pressure-volume curve, any given volume will correspond to a specific pressure, and vice versa, regardless of whether the ventilator is pressure or volume cycled.

Adjustable ventilator settings differ with mode but include respiratory rate, tidal volume, trigger sensitivity, flow rate, waveform, and inspiratory/expiratory (I/E) ratio.

Volume-cycled ventilation

In this mode, which includes volume-control (V/C) and synchronized intermittent mandatory ventilation (SIMV), the ventilator delivers a set tidal volume. The resultant airway pressure is not fixed but varies with the resistance and elastance of the respiratory system and with the flow rate selected.

V/C ventilation is the simplest and most effective means of providing full mechanical ventilation. In this mode, each inspiratory effort beyond the set sensitivity threshold triggers delivery of the fixed tidal volume. If the patient does not trigger the ventilator frequently enough, the ventilator initiates a breath, ensuring the desired minimum respiratory rate.

SIMV also delivers breaths at a set rate and volume that is synchronized to the patient's efforts. In contrast to V/C, patient efforts above the set respiratory rate are unassisted, although the intake valve opens to allow the breath. This mode remains popular, despite the fact that it neither provides full ventilator support as does V/C, facilitates liberating the patient from mechanical ventilation, nor improves patient comfort.

Pressure-cycled ventilation

This form of mechanical ventilation includes pressure control ventilation (PCV), pressure support ventilation (PSV), and several noninvasive modalities applied via a tight-fitting face mask. In all of these modalities, the ventilator delivers a set inspiratory pressure. Hence, tidal volume varies depending on the resistance and elastance of the respiratory system. In this mode, changes in respiratory system mechanics can result in unrecognized changes in minute ventilation. Because it limits the distending pressure of the lungs, this mode can theoretically benefit patients with [acute respiratory distress syndrome](#) (ARDS); however, no clear clinical advantage over V/C has been shown, and, if the volume delivered by PCV is the same as that delivered by V/C, the distending pressures will be the same.

Pressure control ventilation is a pressure-cycled form of A/C. Each inspiratory effort beyond the set sensitivity threshold delivers full pressure support maintained for a fixed inspiratory time. A minimum respiratory rate is maintained.

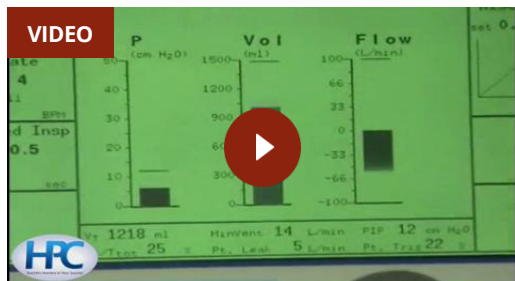
In **pressure support ventilation**, a minimum rate is not set; all breaths are triggered by the patient. The ventilator assists the patient by delivering a pressure that continues at a constant level until the patient's inspiratory flow falls below a preset algorithm. Thus, a longer or deeper inspiratory effort by the patient results in a larger tidal volume. This mode is commonly used to liberate patients from mechanical ventilation by letting them assume more of the work of breathing. However, no studies indicate that this approach is more successful.

Noninvasive positive pressure ventilation (NIPPV)

NIPPV is the delivery of positive pressure ventilation via a tight-fitting mask that covers the nose or both the nose and mouth. Helmets that deliver NIPPV are being studied as an alternative for patients who cannot tolerate the standard tight-fitting face masks. Because of its use in spontaneously breathing patients, it is primarily applied as a form of PSV or to deliver end-

expiratory pressure, although volume control can be used.

Noninvasive Positive Pressure Ventilation (NIPPV) Using Bilevel Positive Airway Pressure



NIPPV can be given as

- Continuous positive airway pressure (CPAP)
- Bilevel positive airway pressure (BiPAP)

In **CPAP**, constant pressure is maintained throughout the respiratory cycle with no additional inspiratory support.

With **BiPAP**, the physician sets both the expiratory positive airway pressure (EPAP) and the inspiratory positive airway pressure (IPAP), with respirations triggered by the patient.

In either mode, because the airway is unprotected, aspiration is possible, so patients must have adequate mentation and airway protective reflexes and no imminent indication for surgery or transport off the floor for prolonged procedures. Obtunded patients and patients with copious secretions are not good candidates. NIPPV also should be avoided in patients who are hemodynamically unstable and in those with evidence of impaired gastric emptying, as occurs with ileus, bowel obstruction, or pregnancy. In such circumstances, swallowing large quantities of air may result in vomiting and life-threatening aspiration. Also, IPAP must be set below esophageal opening pressure (20 cm H₂O) to avoid gastric insufflation.

Indications for conversion to [endotracheal intubation](#) and conventional mechanical ventilation include the development of shock or frequent arrhythmias, myocardial ischemia, and transport to a cardiac catheterization laboratory or surgical suite where control of the airway and full ventilatory support are desired.

NIPPV can be used in the outpatient setting. For example, CPAP is often used for patients with [obstructive sleep apnea](#), whereas BiPAP can be used for those with concomitant obesity-hypoventilation syndrome or for chronic ventilation in patients with neuromuscular or chest wall diseases.

Ventilator settings

Ventilator settings are tailored to the underlying condition, but the basic principles are as follows.

Tidal volume and **respiratory rate** set the minute ventilation. Too high a volume risks overinflation; too low a volume allows for [atelectasis](#). Too high a rate risks hyperventilation and [respiratory alkalosis](#) along with inadequate expiratory time and autoPEEP; too low a rate risks inadequate minute ventilation and [respiratory acidosis](#). A low tidal volume of 6 to 8 mL/kg ideal body weight (IBW—see [Initial Ventilator Management in ARDS](#)) was initially recommended for patients with ARDS; however, such low tidal volume is usually also appropriate in certain patients who have normal lung mechanics ([1](#), [2](#)), such as those who are on mechanical ventilation during surgery ([3](#), [4](#)). Other patients (eg, those with trauma, obtundation, severe acidosis) may be started at slightly higher tidal volume (eg, 8 to 10 mL/kg). IBW rather than actual body weight is used to determine the appropriate tidal volume for patients who have lung disease and who are receiving mechanical ventilation:

IBW (kg) Males:

$$50 + 2.3(\text{height in inches} - 60)$$

or $50 + 0.91(\text{height in cm} - 152.4)$

IBW (kg) Females:

$$45.5 + 2.3(\text{height in inches} - 60)$$

or $45.5 + 0.91(\text{height in cm} - 152.4)$

Sensitivity adjusts the level of negative pressure required to trigger the ventilator. A typical setting is -2 cm H₂O. Too high a setting (eg, more negative than -2 cm H₂O) causes weak patients to be unable to trigger a breath. Too low a setting (eg, less negative than -2 cm H₂O) may lead to overventilation by causing the machine to auto-cycle. Patients with high levels of **autoPEEP** may have difficulty inhaling deeply enough to achieve a sufficiently negative intra-airway pressure.

The **I:E ratio** is the ratio of time spent in inhalation versus that spent in exhalation. The I:E ratio can be adjusted in some modes of ventilation. A normal setting for patients with normal mechanics is 1:3. Patients with asthma or COPD exacerbations should have ratios of 1:4 or even more to limit the degree of autoPEEP.

The **inspiratory flow rate** can be adjusted in some modes of ventilation (ie, either the flow rate or the I:E ratio can be adjusted, not both). The inspiratory flow should generally be set at about 60 L/min but can be increased up to 120 L/min for patients with airflow limitation to facilitate having more time in exhalation, thereby limiting autoPEEP.

Fi₂ is initially set at 1.0 and is subsequently decreased to the lowest level necessary to maintain adequate oxygenation.

PEEP can be applied in any ventilator mode. PEEP increases end-expired lung volume and reduces airspace closure at the end of expiration. Most patients undergoing mechanical ventilation may benefit from the application of PEEP at 5 cm H₂O to limit the atelectasis that frequently accompanies endotracheal intubation, sedation, paralysis, and/or supine positioning. Higher levels of PEEP improve oxygenation in disorders such as cardiogenic **pulmonary edema** and **ARDS**. PEEP permits use of lower levels of Fi₂ while preserving adequate arterial oxygenation. This effect may be important in limiting the lung injury that may result from prolonged exposure to a high Fi₂ (≥ 0.6). PEEP increases intrathoracic pressure and thus may impede venous return, provoking hypotension in a hypovolemic patient, and may overdistend portions of the lung, thereby causing ventilator-associated lung injury (VALI). By contrast, if PEEP is too low, it may result in cyclic airspace opening and closing, which in turn may also cause VALI from the resultant repetitive shear forces. It is important to keep in mind that the pressure-volume curve varies for different regions of the lung. This variation means that, for a given PEEP, the increase in volume will be lower for dependent regions compared to nondependent regions of the lung.

CLINICAL CALCULATOR:

[Ideal Body Weight](#)



Ventilator settings references

1. [Serpa Neto A, Cardoso SO, Manetta JA, et al](#): Association between use of lung-protective ventilation with lower tidal volumes and clinical outcomes among patients without acute respiratory distress syndrome: A meta-analysis. *JAMA* 308:1651–1659, 2012. doi: 10.1001/jama.2012.13730.
2. [Gajic O, Dara SI, Mendez JL, et al](#): Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. *Crit Care Med* 32:1817–1824, 2004.
3. [Futier E, Constantin JM, Paugam-Burtz C, et al](#): A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. *N Engl J Med* 369(5):428–437, 2013. doi: 10.1056/NEJMoa1301082.
4. [Yang M, Ahn HJ, Kim K, et al](#): Does a protective ventilation strategy reduce the risk of pulmonary complications after lung cancer surgery? A randomized controlled trial. *Chest* 139:530–537, 2011. doi: 10.1378/chest.09-2293.

Patient positioning

Mechanical ventilation is typically done with the patient in the semiupright position. However, in patients with ARDS, prone

positioning may result in better oxygenation primarily by creating more uniform ventilation. Uniform ventilation reduces the amount of lung that has no ventilation (ie, the amount of shunt), which is generally greatest in the dorsal and caudal lung regions, while having minimal effects on perfusion distribution.

Although many investigators advocate a trial of prone positioning in patients with ARDS who require high levels of PEEP (eg, > 12 cm H₂O) and F_{IO}₂ (eg, > 0.6), until recently trials have not shown any improvement in mortality with this strategy (however, these trials have typically been underpowered). A recent, large, multicenter, prospective trial assessed patients who had moderately severe ARDS (PaO₂:F_{IO}₂ < 150 mm Hg on an F_{IO}₂ ≥ 0.6, PEEP > 5 cm H₂O) and who were on a tidal volume of about 6 mL/kg. These patients were randomized to undergo ≥ 16 h of prone positioning or be left in the supine position during ventilation. The study, which included a total of 466 patients, identified lower 28- and 90-day mortality in the prone-positioning group without a significant incidence of associated complications (1).

Prone positioning is contraindicated in patients with spinal instability or increased intracranial pressure. This position also requires careful attention by the ICU staff to avoid complications, such as dislodgement of the endotracheal tube or intravascular catheters.

Patient positioning reference

1. [Guérin C, Reignier J, Richard JC, et al](#): Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med* 368(23):2159–2168, 2013. doi: 10.1056/NEJMoa1214103.

Sedation and comfort

Although many patients tolerate mechanical ventilation via endotracheal tube without sedatives, some require IV administration of sedatives (eg, [propofol](#), [lorazepam](#), [midazolam](#)) and analgesics (eg, [morphine](#), [fentanyl](#)) to minimize stress and anxiety. These drugs can also reduce energy expenditure to some extent, thereby reducing carbon dioxide production and oxygen consumption. Doses should be titrated to the desired effect, guided by standard sedation/analgesia scoring systems. Patients undergoing mechanical ventilation for ARDS typically require higher levels of sedation and analgesia. The use of [propofol](#) for longer than 24 to 48 h requires periodic monitoring of serum triglyceride levels. There is evidence that continuously administered IV sedation prolongs the duration of mechanical ventilation. Thus, the goal is to achieve adequate but not excessive sedation, which can be accomplished by using continuous sedation with daily interruption or by using intermittent infusions.

Neuromuscular blocking agents are not used routinely in patients undergoing mechanical ventilation because of the risk of prolonged neuromuscular weakness and the need for continuous heavy sedation; however, one study did show reduced mortality at 90 days in patients with early, severe ARDS who received 48 h of neuromuscular blockade (1). Exceptions who may benefit from neuromuscular blockade include patients who fail to tolerate some of the more sophisticated and complicated modes of mechanical ventilation and to prevent shivering when cooling is used after cardiac arrest.

Sedation and comfort reference

1. [Papazian L, Forel JM, Gacouin A, et al](#): Neuromuscular blockers in early acute respiratory distress syndrome. *N Engl J Med* 363:1107–1116, 2010. doi: 10.1056/NEJMoa1005372.

Complications and safeguards

Complications of mechanical ventilation can be divided into those resulting from

- Endotracheal intubation
- Mechanical ventilation itself
- Prolonged immobility and inability to eat normally

The presence of an endotracheal tube causes risk of sinusitis (which is rarely of clinical importance), ventilator-associated pneumonia (see [Hospital-Acquired Pneumonia](#)), tracheal stenosis, vocal cord injury, and, very rarely, tracheal-esophageal or tracheal-vascular fistula. Purulent tracheal aspirate in a febrile patient who has an elevated WBC count > 48 h after ventilation has begun suggests ventilator-associated pneumonia.

Complications of ongoing mechanical ventilation itself include [pneumothorax](#), oxygen toxicity, hypotension, and VALI.

Oxygen toxicity refers to inflammatory changes, alveolar infiltration, and, eventually, pulmonary fibrosis that can develop

following prolonged exposure to high F_{iO_2} (eg, >0.6). Toxicity is both concentration- and time-dependent. An F_{iO_2} > 0.6 should be avoided unless necessary for survival. An F_{iO_2} < 0.6 is well tolerated for long periods.

Ventilator-associated lung injury (VALI), sometimes termed ventilator-induced lung injury, is alveolar injury related to mechanical ventilation. Possible mechanisms include alveolar overdistention (ie, volutrauma) and the shear forces created by repetitive opening and collapse of alveoli (ie, atelectrauma), leading to release of inflammatory mediators resulting in increased alveolar permeability, fluid accumulation, and loss of surfactant.

If acute hypotension develops in a mechanically ventilated patient, particularly when it is accompanied by tachycardia and/or a sudden increase in peak inspiratory pressure, [tension pneumothorax](#) must always be considered; patients with such findings should immediately have a chest examination and a chest x-ray (or immediate treatment if examination is confirmatory). More commonly, however, hypotension is a result of sympathetic lysis caused by sedatives or opioids used to facilitate intubation and ventilation. Hypotension can also be caused by decreased venous return due to high intrathoracic pressure in patients receiving high levels of PEEP or in those with high levels of intrinsic PEEP due to asthma or COPD. If there are no physical findings suggesting tension pneumothorax, and if ventilation-related causes of hypotension are a possible etiology, pending a portable chest x-ray, the patient may be disconnected from the ventilator and gently bagged manually at 2 to 3 breaths/min with 100% oxygen while fluids are infused (eg, 500 to 1000 mL of 0.9% saline in adults, 20 mL/kg in children). An immediate improvement suggests a ventilation-related cause, and ventilator settings should be adjusted accordingly.

Relative immobility increases the risk of venous thromboembolic disease, skin breakdown, and atelectasis.

Most hospitals have standardized protocols to reduce complications. Elevating the head of the bed to > 30° decreases risk of ventilator-associated pneumonia, and routine turning of the patient every 2 h decreases the risk of skin breakdown (see [prevention of pressure ulcers](#)). All patients receiving mechanical ventilation should receive deep venous thrombosis prophylaxis, either [heparin](#) 5000 units sc bid to tid or low molecular weight [heparin](#) or, if [heparin](#) is contraindicated, sequential compression devices. To prevent GI bleeding, patients should receive an H₂ blocker (eg, [famotidine](#) 20 mg enterally or IV bid) or [sucralfate](#) (1 g enterally qid). Proton pump inhibitors should be reserved for patients with a preexisting indication or active bleeding. Routine nutritional evaluations are mandatory, and enteral tube feedings should be initiated if ongoing mechanical ventilation is anticipated.

The most effective way to reduce complications of mechanical ventilation is to limit its duration. Daily “sedation vacations” and spontaneous breathing trials help determine the earliest point at which the patient may be liberated from mechanical support.

Drugs Mentioned In This Article

Drug Name	Select Trade
famotidine	PEPCID
sucralfate	CARAFATE
lorazepam	ATIVAN
midazolam	No US brand name
fentanyl	ACTIQ, DURAGESIC, SUBLIMAZE
morphine	DURAMORPH PF, MS CONTIN
propofol	DIPRIVAN
heparin	PANHEPRIN



