Invasive and Noninvasive Ventilation in the Emergency Department

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The Roman physician Galen (AD 129–217) may have been the first to describe mechanical ventilation using a bellows to inflate the lungs of a deceased animal.\textsuperscript{1} However, it was not until the polio epidemic in the 1950s that mechanical ventilation was widely used. During this period, ventilation was performed by enclosing the thorax in an iron box, called the iron lung. It was designed to apply negative pressure that was transmitted to the intrapleural space, causing room air to enter through the mouth. However, increasing deaths from bulbar poliomyelitis led Engström\textsuperscript{2} to

\textbf{KEYWORDS}

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- Noninvasive ventilation
- Positive pressure ventilation
- Intermittent positive pressure ventilation
- Respiratory insufficiency
- Closed-loop systems
- Dual-control modes

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search for a better way to ventilate these patients. This need led to the invention of the modern positive pressure ventilator. These advances led to a reduction in mortality from bulbar polio from 90% to 20%, and the era of invasive positive pressure ventilation began. Since this era, technological advances and computers have changed the way ventilators operate. Currently, mechanical ventilation is used in many settings, including the Emergency Department (ED), where invasive and noninvasive ventilation (NIV) are essential tools for the treatment of critically ill patients. This article reviews the use of these modalities in the ED. Specifically, this review presents the goals of invasive and noninvasive positive pressure ventilation, the frequent ventilatory modes, and the physiologic principles underlying their use. Different case scenarios help apply these concepts in practice. This article will guide emergency physicians to resolve frequent problems and to prevent complications related to mechanical ventilation.

GOALS OF MECHANICAL VENTILATION AND GAS EXCHANGE PHYSIOLOGY

Goals of Mechanical Ventilation

The main goal of mechanical ventilation is to reduce the work of breathing (WOB) and reverse life-threatening hypoxemia and hypercarbia. It can also support oxygenation and ventilation when patients fail to maintain or protect their airways (eg, coma, sedation, paralysis, poisoning). Thus, it is useful to think of respiration as 2 separate processes: ventilation (alveolar ventilation is indirectly proportional to partial pressure of CO2 in arterial blood [PaCO2]) and oxygenation (assessed by partial pressure of O2 in arterial blood [PaO2] and by pulse oximeter [SpO2]).

Gas Exchange Physiology

When trying to treat hypercarbia, an excess of CO2 in the blood stream, it is useful to think of its determinants. Hypercarbia is caused by increased CO2 production (eg, sepsis, metabolic acidosis), hypoventilation (eg, narcotic overdose with low tidal volume [VT] or low respiratory frequency) or high dead space (eg, pulmonary embolism). Dead space (Vd) is the volume of air that does not participate in CO2 exchange.

Mechanical ventilation is helpful for treating hypoventilation by increasing respiratory frequency and VT. However, mechanical ventilation can increase dead space with alveolar overdistention and excessive VT or positive end-expiratory pressure (PEEP). Positive pressure ventilation can also increase dead space in the case of a pulmonary embolism or any low-output circulatory failure (eg, hemorrhagic shock). Caution is required when using positive pressure ventilation in these situations and appropriate treatment of the underlying cause is essential.

When trying to improve oxygenation, it is useful to think of the 6 different causes of hypoxemia to understand how mechanical ventilation can help: (1) low fractional inspired oxygen (FiO2); (2) hypoventilation; (3) impaired diffusion; (4) ventilation-perfusion (V/Q) mismatching; (5) shunt; and (6) desaturation of pulmonary arterial (mixed venous) blood. Mechanical ventilation can help by solving hypoventilation and by increasing FiO2 and PEEP. Increased FiO2 can solve low FiO2, impaired diffusion, V/Q mismatching, and desaturation of pulmonary arterial blood. Increasing PEEP can help reduce shunting by opening alveoli that are flooded with fluid or are atelectatic (PEEP is discussed further later). Increasing PEEP can also help reduce FiO2 to nontoxic levels (<50%).

POSITIVE PRESSURE VENTILATION

Positive pressure ventilation is divided into invasive mechanical ventilation and NIV.
Invasive Mechanical Ventilation

Ventilator variables
To understand the many different modes of ventilation, it is important to understand the 3 key ventilator phase variables\(^9\) that determine (1) when a breath is delivered (trigger), (2) what limits gas delivery (limit), and (3) what ends gas delivery (cycle). Table 1 describes each mode of ventilation with its respective trigger, limit, and cycle.

Table 1

<table>
<thead>
<tr>
<th>Mode of Ventilation</th>
<th>Trigger</th>
<th>Limit</th>
<th>Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>A/C, SIMV, PSV</td>
<td>Patient</td>
<td>Volume</td>
<td>Volume</td>
</tr>
<tr>
<td>A/C, mandatory</td>
<td>Time</td>
<td>Pressure</td>
<td>Time</td>
</tr>
<tr>
<td>PSV, mandatory</td>
<td>Time</td>
<td>Flow</td>
<td>Time</td>
</tr>
</tbody>
</table>

The trigger variable determines breath initiation and is set by the mode on the ventilator. The patient can initiate the breath (patient triggered) or the ventilator can deliver a breath after an elapsed amount of time (time triggered) (Fig. 1).

Assisted breaths in assist control (A/C) are patient triggered. Other patient-triggered modes include spontaneous breaths in synchronized intermittent mandatory ventilation (SIMV) and pressure-support ventilation (PSV). Examples of time-triggered breaths are controlled breaths in A/C or mandatory breaths in SIMV. With time triggering, the rate of breathing is controlled by the ventilator, which means the patient receives mandatory breaths at a prespecified frequency determined by the clinician. For most modes of ventilation, a preset trigger sensitivity has to be reached before a ventilator delivers flow. Pressure and flow are the most frequently used triggers. With pressure triggering, a set negative pressure must be attained for the ventilator to deliver a breath. This pressure is usually set at \(-2 \text{ cm H}_2\text{O}\). The higher (more negative) the trigger sensitivity, the harder the patient has to work to trigger a breath. With flow triggering, 2 variables must be set: the base flow rate (usually set between 5 and 20 L/min) and the flow sensitivity (usually set between a minimum of 1 L/min and one-half of the base flow). If the trigger sensitivity is set too low (too sensitive), the ventilator can autotrigger because of oscillating water in the ventilator tubing, or by hyperdynamic heartbeats, or when the patient moves.

To control the delivery of gas to the patient during the inspiratory phase, a limit variable is added. This limit variable is usually set as a volume limit (eg, A/C and mandatory breaths in SIMV), a pressure limit (eg, pressure A/C [PAC], PSV, or spontaneous breath in SIMV), a flow limit (eg, A/C or mandatory breaths in SIMV), or a time limit (eg, high-frequency oscillation ventilation [HFOV]). The limit variable does not terminate the breath. For example, a ventilator can be set for pressure ventilation with a limit of \(25 \text{ cm H}_2\text{O}\) and an inspiratory time set at 2 seconds. Such a breath is described as pressure limited and time cycled.

In the breath termination, a cycle variable is chosen (volume, flow, or time). Traditional ventilators were classified according to their cycling method. However, new ventilators have microprocessors that allow them to function in many different modes. Volume-cycled ventilation (eg, A/C, SIMV) has a predetermined \(V_T\) set for the patient that is delivered with each inspiration. The amount of pressure necessary to deliver this volume fluctuates based on the resistance and elastance of the patient and ventilator circuit. Flow-cycled ventilation (eg, PSV and spontaneous breaths in SIMV) allows the ventilator to begin the expiratory phase once the flow has decreased to a predetermined value during inspiration. The flow and volume remain unchanged, but the pressure fluctuates from breath to breath based on the resistance and elastance of the respiratory system. Time-cycled ventilation has a predetermined inspiration and expiration time set. The volume and pressure fluctuate. For example, it is used with airway pressure–release ventilation (APRV) and with HFOV (these modes are discussed later).

Inspiratory Flow Patterns

Most ventilators offer at least 3 different types of inspiratory flow patterns for volume-cycled breaths. These flow patterns include a square wave, a sinusoidal wave, and
<table>
<thead>
<tr>
<th>Ventilator Mode</th>
<th>Trigger</th>
<th>Limit</th>
<th>Cycle</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAC</td>
<td>Patient or time</td>
<td>Flow, volume</td>
<td>Time</td>
<td>Reduces WOB Guarantees delivery of set $V_T$ (unless PIP limit exceeded)</td>
<td>Potential adverse hemodynamic effects (with auto-PEEP) May lead to inappropriate hyperventilation and excessive inspiratory pressures</td>
</tr>
<tr>
<td>PAC</td>
<td>Patient or time</td>
<td>Pressure</td>
<td>Time</td>
<td>Allows limitation of PIP</td>
<td>Same as VAC Potential hyperventilation or hypoventilation with changes in airway resistance or elastance</td>
</tr>
<tr>
<td>SIMV</td>
<td>Patient or time</td>
<td>Pressure (patient-triggered breaths) Flow/volume (VC breaths) Pressure (P-SIMV)</td>
<td>Flow (patient-triggered breaths) Volume or time for VC breaths</td>
<td>Less interference with normal cardiovascular function</td>
<td>Increased WOB compared with A/C Asynchrony Poor weaning mode</td>
</tr>
<tr>
<td>PRVC</td>
<td>Patient or time</td>
<td>Dual control: pressure-limited mode using a target $V_T$ for feedback control</td>
<td>Time, flow (patient-triggered breaths)</td>
<td>Minimum MV at the lowest PIP possible; Maintains similar $V_T$ with varying resistance and elastance Intelligent closed-loop ventilation that automatically transitions from controlled breathing to spontaneous when patient is ready</td>
<td>When patients attempt to breathe at a $V_T$ greater than the clinician-set target $V_T$, the burden of inspiratory work is shifted onto the patient Absence of high-level evidence of its superiority on patient-oriented outcomes</td>
</tr>
<tr>
<td>Mode</td>
<td>Type</td>
<td>Description</td>
<td>Time, flow (patient-triggered breaths)</td>
<td>Additional Features</td>
<td></td>
</tr>
<tr>
<td>------</td>
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<td>--------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>ASV</td>
<td>Patient or time</td>
<td>Dual control: pressure-limited mode that uses a target MV for feedback control</td>
<td>Same as PRVC. In addition, the microprocessor calculates the ideal V_T and RR to deliver minimizing WOB and targeting a lung protection strategy. Decreases workload.</td>
<td>When patients attempt to breathe at an MV greater than the clinician-set target MV, the burden of inspiratory work is shifted onto the patient. Absence of high-level evidence of its superiority on patient-oriented outcomes.</td>
<td></td>
</tr>
<tr>
<td>PSV</td>
<td>Patient</td>
<td>Pressure Flow</td>
<td>Patient comfort Improved patient-ventilator synchrony Decreased WOB</td>
<td>Apnea alarm is only backup (newer microprocessor ventilators have a backup mode if patient apneic).</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** A/C, assist control; ASV, adaptive support ventilation; MV, minute ventilation; PAC, pressure assist control; PC, pressure control; PIP, peak inspiratory pressure; PRVC, pressure-regulated volume control; P-SIMV, pressure SIMV (time-triggered breaths are pressure limited); PSV, pressure-support ventilation; RR, respiratory rate; SIMV, synchronized intermittent mandatory ventilation; VAC, volume assist control; VC, volume control; V_T, tidal volume.

a descending ramp wave. In practice, the descending ramp wave is the most popular because it mimics the normal inspiratory pattern with flow increasing rapidly and then decreasing gradually until the end of inspiration.

**Initial Ventilator Settings After Intubation**

According to the clinical setting, ventilator variables are set depending on the mode chosen, the desired $V_T$, inspiratory pressure, inspiratory flow rate, and respiratory rate. Some ventilators also allow the operator to set the inspiratory/expiratory (I/E) ratio directly. For most adults, a normal I/E ratio of 1:2 or 1:3 is used. **Table 2** summarizes the basic initial settings for a multitude of clinical situations that are discussed later.

**Conventional Modes**

Basic modes used to ventilate patients are A/C ventilation, SIMV, and PSV. Refer to **Table 1** for the advantages and disadvantages of each mode.

**A/C ventilation**

With A/C, breath initiation can be patient or time triggered, depending on which occurs first. For patients who are paralyzed, comatose, or deeply sedated, this mode can be used for full ventilatory support. Breaths can be volume limited (volume A/C with
<table>
<thead>
<tr>
<th>Clinical Example</th>
<th>Modes</th>
<th>Respiratory Rate (Breaths/min)</th>
<th>Tidal Volume (mL/kg IBW&lt;sup&gt;a&lt;/sup&gt;)</th>
<th>Flow Rate (L/min)</th>
<th>FiO&lt;sub&gt;2&lt;/sub&gt;</th>
<th>PEEP (cm H&lt;sub&gt;2&lt;/sub&gt;O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal lung mechanics</td>
<td>VAC, PAC, SIMV, PRVC, ASV</td>
<td>10–15</td>
<td>8–10</td>
<td>60</td>
<td>&lt;0.4</td>
<td>3–5</td>
</tr>
<tr>
<td>Severe airflow obstruction</td>
<td>Acute asthma</td>
<td>VAC, PRVC, ASV (100%–110% MV)</td>
<td>8–10</td>
<td>≥60</td>
<td>0.3–0.5</td>
<td>&lt;80% of intrinsic PEEP</td>
</tr>
<tr>
<td>Acute/chronic respiratory failure</td>
<td>COPD exacerbation</td>
<td>VAC, PRVC, ASV (100%–110% MV)</td>
<td>8–10</td>
<td>≥60</td>
<td>0.3–0.5</td>
<td>&lt;80% of intrinsic PEEP</td>
</tr>
<tr>
<td>Acute hypoxemia</td>
<td>Cardiogenic pulmonary edema</td>
<td>VAC, PAC, PSV (spontaneous), PRVC, ASV (100%–110% MV)</td>
<td>8–10 (PS to maintain VT: 8–10 mL/kg)</td>
<td>~60</td>
<td>1.0</td>
<td>10</td>
</tr>
<tr>
<td>Acute hypoxemia</td>
<td>Pneumonia and ARDS</td>
<td>VAC</td>
<td>6–8 and lower to 4–6 subsequently if Ppl &gt;30</td>
<td>~60</td>
<td>1.0</td>
<td>5–24 (ARDS Network PEEP- FiO&lt;sub&gt;2&lt;/sub&gt; chart)</td>
</tr>
<tr>
<td>Neuromuscular weakness and chest wall trauma</td>
<td>PAC, VAC, PRVC, ASV (100%–110% MV)</td>
<td>15–25</td>
<td>8–10</td>
<td>60–80</td>
<td>≤0.5</td>
<td>5–8</td>
</tr>
</tbody>
</table>

**Abbreviations:** ARDS, acute respiratory disease syndrome; ASV, adaptive support ventilation; COPD, chronic obstructive pulmonary disease; IBW, ideal body weight; MV, minute ventilation (when using ASV, a percentage of MV is set instead of setting respiratory rate and VT); PRVC, pressure-regulated volume control; PSV, pressure-support ventilation; SIMV, synchronized intermittent mandatory ventilation; VAC, volume assist control.

<sup>a</sup> Female IBW = 45.5 + (0.91 × [height in cm – 152.4]). Male IBW = 50 + (0.91 × [height in cm – 152.4]).

a set $V_T$) or pressure limited (see Fig. 1). To prevent hypoventilation, a backup respiratory rate is set on the ventilator. This backup determines a minimum breathing rate, but the patient has the option of breathing at a faster rate. Thus, every breath is fully supported and has a set $V_T$. Table 2 also presents the different clinical situations where A/C is often used.

**SIMV**

With SIMV, breath initiation can be patient or time triggered (similar to A/C) (Fig. 2). Furthermore, breath termination can be volume cycled (mandatory breaths) or flow cycled (spontaneous breaths).

The ventilator delivers a mandatory breath at a clinician-determined rate and $V_T$. After delivering a mandatory breath, the ventilator then allows the patient to breathe spontaneously without receiving another mandatory breath until the next mandatory breath is due. Thus, the ventilator synchronizes its mandatory breaths with the patient’s spontaneous breaths. When taking a spontaneous breath, patients breathe through the ventilator circuit at a $V_T$ and rate that is determined according to need. Pressure support can be added to spontaneous breaths to decrease the WOB. SIMV has been used in patients with severe respiratory alkalosis, to prevent auto-PEEP (auto-PEEP is discussed later), and to wean patients from the ventilator. This mode is commonly used in the ED after intubation\(^{10}\) because it ensures that a patient

![Fig. 2. SIMV](https://example.com/simv.png)

**Fig. 2.** SIMV. Note that the spontaneous patient-triggered breath is indicated by an arrow. This patient-triggered breath has a different inspiratory pressure determined by the pressure support set by the clinician. The volume generated by this breath is thus different than the other 2 mandatory time-triggered, volume-limited breaths. (*Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 93; with permission.*)
receives a minimal mandatory rate and $V_T$. When patients start recovering from neuromuscular blockade and sedation, SIMV allows patients to breathe spontaneously with pressure support, which is better tolerated in spontaneously breathing patients. However, studies\textsuperscript{11,12} have shown that SIMV prolongs weaning and favors dyssynchrony. Dyssynchrony occurs when there is a mismatch between the patient’s breaths and ventilator-assisted breaths, as well as the inability of the ventilator’s flow delivery to match the patient’s flow demand. This dyssynchrony increases agitation and respiratory distress. Clinicians must closely monitor the $V_T$ of pressure-supported breaths because changing lung elastance and resistance can reduce air entry.

**PSV**

Pressure support is a ventilatory mode that supports a spontaneously breathing patient. The ventilator delivers a predetermined level of positive pressure (determined by the clinician) once the patient triggers a breath. The patient alone determines the respiratory rate, flow rate, inspiratory time, and $V_T$. A constant pressure is maintained until the patient’s inspiratory flow decreases to a specific level (flow cycled) (Fig. 3).

Because patients must be spontaneously breathing, patients receiving PSV need close monitoring because neither their $V_T$ nor their minute ventilation is assured. Clinicians are able to adjust the slope of the pressure and flow curves during inspiration with PSV.\textsuperscript{4} This feature has many different names: rise time, flow acceleration percent, inspiratory rise time, inspiratory rise time percent, and slope adjustment. These terms

![Pressure curve](image)

![Flow curve](image)

*Fig. 3. Pressure-support ventilation. (Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 95; with permission.)*
refer to the time required for the ventilator to rise to the set pressure at the beginning of inspiration. A rise time set too short (with a higher initial flow) can cause a pressure overshoot, and inspiratory flow may end prematurely. A rise time set too long (flow set too low), may not meet a patient’s needs, and asynchrony develops.4

**PEEP**

PEEP is provided to the patient’s airway throughout the respiratory cycle (Fig. 4).

Even though PEEP can be used by itself as an NIV mode for spontaneously breathing patients (in which case it is called continuous positive airway pressure [CPAP]) (Fig. 5), PEEP is often used in addition to other modes of ventilation to improve oxygenation.

PEEP shifts the pressure-volume curve toward normal, increases compliance, recruits alveoli, and increases functional residual capacity (FRC). PEEP is also helpful for left ventricular function because it decreases preload, afterload, and WOB. However, PEEP is detrimental for right ventricular function because it increases right ventricular afterload and might even cause cardiovascular collapse in patients with pulmonary embolism, severe pulmonary hypertension, and right ventricle infarction. Applying low levels of physiologic PEEP (5 cm H₂O) decreases the risk of atelectasis and pneumonia.13 PEEP is indicated in cardiogenic pulmonary edema, acute lung injury (ALI), acute respiratory distress syndrome (ARDS), and to prevent atelectasis in obese patients. The contraindications to applying PEEP are bullous lung disease and preload-dependent shock.

The best level of PEEP to use is called best PEEP. Many different methods exist to try to identify the best level of PEEP to administer to a patient. There are both mechanical and gas exchange approaches. The most pragmatic approach in the ED is to use a gas exchange technique like the one used in the ARDS Network trial (Table 3),14 which used a predetermined PEEP-FiO₂ algorithm designed to provide adequate values for PaO₂ while minimizing FiO₂. The choice between a high and a low PEEP-FiO₂ algorithm is still the subject of ongoing debate15–17 even though a recent systematic review suggested that higher PEEP levels (open-lung ventilation strategy18,19) decreased mortality but increased barotrauma.20 Moreover, no comparative studies have been conducted in the ED, where patients are often hypovolemic and unstable hemodynamically after intubation.21 Because adding extrinsic PEEP can decrease preload, this urges clinicians to be prudent when choosing a high-PEEP strategy immediately after intubation for patients with possible ALI/ARDS. However, a high-PEEP strategy could be useful in hemodynamically stable patients with refractory hypoxemia.

![Fig. 4. PEEP. (Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 59; with permission.)](image-url)
Lung Protection Strategies

Since the publication of the ARDS Network trial in 2000, a lung protection ventilation strategy is the norm for all patients with ALI and ARDS. Using such a strategy decreases mortality by 9% (number needed to treat = 11). It is essential that the emergency physician uses initial ventilator settings that protect the lung from further ventilator-induced injury. These settings include a low VT (4–8 mL/kg of ideal body weight [IBW]), aiming for a plateau pressure (Ppl) less than 30 cm H2O and the use of an appropriate PEEP-Fio2 scale (proposed in the ARDS Network trial; see Table 3). Calculating every patient’s IBW is essential to target the appropriate VT, and is easily performed by measuring the height of every patient after intubation and applying the appropriate formula based on the patients’ gender (see Table 2).

The open-lung ventilation strategy combines low VT (4–8 mL/kg) with prevention of atelectrauma using recruitment maneuvers and higher PEEP levels than the ARDS Network trial (for more information on this topic, read the LOVS19 and ExPress18 trials). Modes like APRV and HFOV have been developed to offer alternative strategies to maintain higher average airway pressures to improve recruitment and oxygenation. Because the evidence for these modes is still considered weak and they are mostly used in the intensive care unit (ICU) when a basic lung-protective ventilation strategy is not working, this article only briefly reviews these modes.

APRV

APRV is a time-triggered, pressure-limited, time-cycled mode of ventilation that allows unrestricted spontaneous breathing throughout the ventilatory cycle. With APRV, the ventilator cycles between 2 different levels of pressure (Fig. 6).

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**Table 3**

ARDS PEEP-Fio2 charts. Note that 2 PEEP-Fio2 charts (high and low PEEP) are presented. The decision to choose one chart rather than the other is still a matter of debate (see text). Note that both charts aim for a Pao2 between 55 and 80 mm Hg or a SpO2 between 88% and 95%

<table>
<thead>
<tr>
<th>Lower PEEP/higher Fio2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fio2</td>
</tr>
<tr>
<td>PEEP (cm H2O)</td>
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</table>

<table>
<thead>
<tr>
<th>Higher PEEP/lower Fio2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fio2</td>
</tr>
<tr>
<td>PEEP (cm H2O)</td>
</tr>
</tbody>
</table>

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**Fig. 5.** CPAP. (Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 58; with permission.)
The ventilator maintains a high pressure setting for most of the respiratory cycle (P<sub>High</sub> 20–25 cm H<sub>2</sub>O), which is followed by a periodic release to a low pressure (P<sub>Low</sub> 5–10 cm H<sub>2</sub>O). The purpose of cycling between P<sub>High</sub> and P<sub>Low</sub> is to remove CO<sub>2</sub>. The theoretic advantages of APRV are that it protects the lung (favors alveolar recruitment, decreases overinflation, and enhances gas exchange). However, there is little evidence about its safety and efficacy. To date, low-level evidence suggests that promoting spontaneous breathing with APRV may not be appropriate in patients with severe ALI/ARDS. There is also a fear that APRV might not be protective because of high transpulmonary pressures generated when a patient breathes at P<sub>High</sub>. APRV should not be used with patients with chronic obstructive pulmonary disease (COPD), asthma, or diseases for which permissive hypercapnia is contraindicated.

**HFOV**

HFOV is an emerging ventilatory strategy for adults that has been used successfully in the neonatal and pediatric population. This mode uses high mean airway pressures (25–35 cm H<sub>2</sub>O) to maintain an open lung, and low V<sub>T</sub> (1–4 mL/kg) at a high frequency (3–10 Hz). This allows for adequate ventilation and prevents alveolar overdistension. The application of HFOV has mainly been reported as a rescue ventilatory mode in patients with ARDS in the ICU setting when conventional ventilation has failed. Thus, HFOV is not a ventilatory mode used in the ED. Ongoing studies are evaluating the use of HFOV earlier in the course of ARDS.

**Closed-loop ventilation**

Because of technological advances in computing and in artificial intelligence, intelligent modes with complex closed loops have been developed. Proportional assist ventilation (PAV) and neurally adjusted ventilatory assist (NAVA) are 2 modes that can deliver ventilation proportional to the instantaneous patient effort and seem to improve patient-ventilator interaction. Dual-control modes (eg, pressure-regulated volume control [PRVC] and adaptive support ventilation [ASV]) refer to ventilation modes that allow the clinician to set a volume target and the ventilator delivers pressure-controlled breaths. The common characteristic of these new closed-loop modalities of ventilation is their adaptation to the patient, which explains the improvement in patient-ventilator interaction. Each of these modes changes the level of assistance in response to the patient’s breathing pattern. These modes are promoted as helping the work of a respiratory therapist, who would typically have to remain at the bedside to offer the same level of adaptation to patients.
Nevertheless, and despite their attractiveness, all these new methods of ventilation must show their clinical benefits before being recommended in routine practice.\textsuperscript{28}

**PAV**

PAV delivers an inspiratory pressure proportional to the instantaneous effort of the patient and is amplified according to the patient respiratory mechanics (pulmonary compliance and airway resistance) and the chosen level of assistance (0%–100% assistance for the respiratory muscles).\textsuperscript{28,37–39} Technical improvements made recently (under the name PAV+, available with the Puritan Bennett 840 ventilator, Puritan Bennett, Boulder, CO) allow intermittent and automated measurements of the patient’s compliance and resistance\textsuperscript{40,41} and make this mode easier to use.\textsuperscript{42} Despite its potential, there is currently no evidence that PAV or PAV + changes the clinical course of patients in comparison with pressure support.\textsuperscript{28,43,44}

**Neurally adjusted ventilatory assist (NAVA)**

NAVA is a new mode of ventilation\textsuperscript{30,33} that is available on Servo-i ventilators (MAQUET Critical Care, Solna, Sweden). As for PAV, the level of ventilatory assistance is proportional to the patient’s effort. Its main feature is that the signal used by the ventilator to deliver assistance is not a pressure signal but the diaphragmatic electromyogram signal collected from electrodes placed on an esophageal catheter. Despite these potential benefits, several questions remain concerning the practical use of NAVA. In particular, the ease of placement of the esophageal catheter to record diaphragmatic electromyograms remains uncertain.\textsuperscript{30} Thus, NAVA remains a promising mode of ventilation that needs more evidence of benefit for large-scale use.

**Dual-control Breath-to-Breath, Pressure-limited, Time-cycled Ventilation**

Volume-targeted, pressure-regulated modes of ventilation control $V_T$ through variable levels of pressure and are referred to as dual-control modes. Pressure-regulated, volume control ventilation (PRVC) (AVEA, CareFusion, San Diego, CA, USA; Servo-i and Servo 300, MAQUET Critical Care, Solna, Sweden), AutoFlow (Evita 4, Dräger Medical, Lübeck, Germany), Volume Ventilation Plus (VV+) (Puritan Bennett 840, Puritan Bennett, Boulder, CO, USA), and variable pressure control (VPC) and variable pressure support (VPS) (Venturi Ventilator, Cardiopulmonary Corp., Milford, CT, USA) all possess similar proprietary modes. Breath initiation is patient or time triggered and breath termination is volume cycled, but each breath is pressure limited. All use a pressure-targeted algorithm in which the ventilator determines after each breath whether the pressure applied to the airway was sufficient to deliver the desired $V_T$. For example, if the $V_T$ did not meet the set target, the ventilator will adjust the pressure applied to the airway on the next breath.

A similar but more complex dual-control mode, adaptive support ventilation (Hamilton G5, Hamilton C2, Galileo, Raphael, Hamilton Medical, Bonaduz, Switzerland) provides automatic ventilation in which minute volume (MV) is controlled through a $V_T$–respiratory rate ($V_T$–RR) combination based on respiratory mechanics. Clinicians must set a MV depending on the IBW (0.1 L/kg IBW per minute). The setting is expressed as a percentage of the target (% MV). The % MV can be adjusted later according to the target $P_{a\mathrm{CO}_2}$ in passive patients and according to the clinical condition in active patients.\textsuperscript{30} In patients unable to trigger a breath (passive or paralyzed patients), the ventilator generates a pressure-controlled breath, automatically adjusting inspiratory pressure and timing to achieve the target $V_T$ and respiratory rate. Based on the Otis equation\textsuperscript{45} and on the patient’s own expiratory flow-volume curve,\textsuperscript{46–48} the target $V_T$–RR combination minimizes WOB for a clinician-set MV.\textsuperscript{45,46} ASV
automatically determines the \( V_T \) and respiratory rate that minimizes auto-PEEP\(^{30} \) and maintains the peak airway pressure at less than the target level.\(^{45} \)

Even if these dual modes are promising, clinical studies have not shown a clear clinical benefit in comparison with other modes of ventilation.\(^{48,49} \) Certain investigators have suggested that ASV could be appropriate for patients with ARDS, but others have found inappropriate tidal volumes for this population.\(^{49} \) Other investigators have raised the possibility that patients’ WOB increase when patients attempt to breathe at a \( V_T \) or MV greater than the clinician-set target \( V_T \) or MV with different dual-control modes.\(^{50} \)

**SmartCare**

The SmartCare system (Evita XL; Dräger, Lübeck, Germany) uses pressure support as primary mode\(^{30} \) and integrates an algorithm that (1) maintains the patient in a respiratory comfort zone by adapting the level of pressure support; (2) gradually decreases the level of the pressure support in case of stability; and (3) implements automated spontaneous breathing trials performed with minimal levels of pressure support. The first multicenter study comparing this system of automated weaning with usual weaning was published recently\(^{51} \) showing shorter weaning periods, duration of mechanical ventilation, and duration of ICU stay.

**NIV**

Since the introduction of CPAP to treat obstructive sleep apnea in the 1980s\(^{52,53} \) and the discovery that masks were a convenient conduit to assist ventilation, noninvasive positive pressure ventilation has become an important modality to treat acute respiratory failure and to prevent the need for invasive mechanical ventilation.\(^{3} \) NIV is postulated to improve physiologic effects of various diseases by reducing the WOB and improving oxygenation and alveolar ventilation.\(^{54} \) In the acute care setting, NIV has been proven to reduce the need for intubation\(^{55,56} \) and its related complications, reduce mortality,\(^{57} \) and shorten the hospital stay for certain patients requiring mechanical ventilation.\(^{58,59} \) It also provides flexibility in initiating and removing mechanical ventilation, reduces sedation requirements, and preserves airway defenses, speech, and swallowing mechanisms. However, NIV can cause gastric distension, pressure sores, facial pain, nose dryness, eye irritation, discomfort, claustrophobia, and poor sleep.

In this article, NIV includes both noninvasive bilevel positive pressure ventilation (NPPV) and noninvasive CPAP. NIV can be delivered via a variety of portable NIV ventilators and adult acute care ventilators.\(^{60} \)

Most portable NIV ventilators are microprocessor controlled and use a blower to regulate gas flow into the patient’s circuit to maintain the preset pressure.\(^{60} \) They have a single-circuit gas delivery system that uses an intentional leak port for patient exhalation instead of a true exhalation valve. This system allows the continuous flow of gas to help maintain pressure levels and flush exhaled gases from the circuit. NIV ventilators are often pressure-limited, flow-triggered and time-triggered, flow-cycled and time-cycled ventilators. They deliver an inspiratory positive airway pressure (IPAP) and an expiratory positive airway pressure (EPAP) (Fig. 7).

The IPAP level set on most NIV ventilators is equivalent to the total inspiratory pressure (pressure-support + PEEP) found in the PSV mode used on acute care ventilators. Most portable NIV ventilators can offer the following modes: CPAP, assist mode (similar to PSV), and an A/C mode (similar to pressure A/C). NIV ventilators have the ability to compensate for air leaks\(^{61} \) that make breaths easier to trigger and terminate\(^{60} \) compared with ICU ventilators. One of the limitations of portable NIV ventilators is
that oxygen delivery can be difficult. Because most NIV ventilators do not have an integrated oxygen blender, supplemental oxygen must be blended into the system. In the past, Respironics BiPAP Vision (Philips, Amsterdam, Netherlands) was the only NIV ventilator with this feature. However the newer high-performance NIV ventilators now include oxygen blenders.

**Indications and contraindications**

Recently, Keenan and colleagues produced clinical practice guidelines about the use of NIV in acute care settings based on a comprehensive review of the literature. These investigators recommend that NPPV be used for patients with severe exacerbation of COPD (pH <7.35 and relative hypercarbia) (grade 1A recommendation) and that either NPPV or CPAP be used for patients with respiratory failure caused by cardiogenic pulmonary edema in the absence of shock or acute coronary syndrome requiring urgent coronary revascularization (grade 1A recommendation). They also suggest that NPPV be used for immunosuppressed patients who have acute respiratory failure (grade 2B recommendation). No recommendation was made about the use of NPPV for acute asthma exacerbations, because of inconclusive studies. They recommend not to use CPAP for patients who have ALI (grade 1C recommendation), but no recommendation about the use of NPPV for ALI was made. The recommendation not to use CPAP for patients with ALI comes from an RCT comparing the use of CPAP plus usual therapy versus usual therapy alone. In a subgroup of patients with ALI, the addition of CPAP did not affect endotracheal intubation or hospital mortality, but was associated with more adverse events (including 4 cardiac arrests). Other contraindications to NIV include cardiorespiratory arrest, severe encephalopathy, severe upper gastrointestinal bleeding, hemodynamic instability or unstable cardiac arrhythmia, facial or neurologic surgery, trauma or deformity, upper airway obstruction, inability to cooperate or protect the airway, inability to clear secretions, or high risk for aspiration.

**Interfaces (nasal, oronasal, full face, helmet)**

To prevent skin pressure lesions, discomfort, and mask leaks, the choice of interface is important. Keenan and colleagues suggest the use of an oronasal mask rather than a nasal mask for patients in acute respiratory failure (grade 2C recommendation). No other recommendation was made regarding other interfaces currently available (helmet interface or full face). Modifications to the interfaces are available to avoid pressure sores to the nasal bridge, such as forehead spacers, masks with ultrathin silicon seals, or heat-sensitive gels that minimize skin trauma. A heated humidifier should be used to prevent drying of the airway during prolonged NIV. In addition, further research is needed.
needed to determine the optimal period of rest to allow oral intake, speech, and pressure relief on skin surfaces.  

**Initiation and monitoring of NIV**  
Several factors are vital to the success of NIV: careful patient selection; properly timed initiation; comfortable, well-fitting interface; coaching and encouragement; and careful monitoring. Initial ventilator pressure settings should be started at low to maximize patient compliance and comfort and then can be increased to alleviate respiratory distress. Typical starting pressures are an IPAP of 10 to 12 cm H2O and an EPAP of 4 to 5 cm H2O, maintaining a gradient of at least 5 cm H2O. IPAP can be increased to 20 cm H2O, and EPAP to 10 or 12 cm H2O. However, these higher pressures are limited by air leaks and by the amount of pressure needed to keep a tight fit of the mask on the patient’s face. Most importantly, monitoring the VT is essential in determining whether adequate ventilation is generated by these settings.

Once NIV is initiated, patients should be closely monitored in the ED, a critical care unit, or a step-down unit. Improving signs of respiratory distress (abdominal paradox, accessory muscle use, and tachypnea) and improving pH within the first 2 hours of NIV are indications of success. However, if these signs of distress persist, adjustments must be made. Leaks should be sought and corrected, patient-ventilator synchrony should be optimized, and pressures may have to be increased. Improvement in mental status, gas exchange, respiratory rate, and heart rate is expected in the first 1 or 2 hours. If this does not occur, clinicians must consider immediate intubation because delays are associated with worse outcomes.

**Sedation during NPPV**  
Patient agitation has been considered a relative contraindication for the use of NPPV because of the fear of using sedation. A cross-sectional Web-based survey performed on American and European physicians concluded that most physicians infrequently use sedation and analgesic therapy for patients receiving NPPV. If used, sedation was usually administered as an intermittent intravenous bolus, outside of a protocol. A benzodiazepine alone was the most preferred (33%), followed by an opioid alone (29%). Short-acting narcotics like remifentanil and newer agents like dexmedetomidine have been reported to help agitated patients cooperate with mask ventilation without inducing respiratory depression. Although sedation can help reduce anxiety and respiratory rate (notably helping reduce auto-PEEP with patients who have COPD), it must be administered with caution in a monitored setting. Sedation levels, breathing amplitude, respiratory rates, and blood gases must be monitored closely.

**Boussignac CPAP system and other portable CPAP systems**  
The Boussignac CPAP system includes a valve capable of delivering a PEEP level ranging from 2.5 to 10 cm H2O without using a generator. The valve is a small plastic tube attached to a face mask that creates continuous positive pressure. It has been proven to be equivalent to NPPV for the treatment of acute pulmonary edema in the ED and is used to treat acute pulmonary edema in certain prehospital settings. Other portable CPAP systems also exist, like the WhisperFlow Fixed CPAP Generator (Philips Respironics, Andover, MA, USA), and PORTO2VENT CPAP device (Emergent Respiratory, LLC, Anaheim, CA, USA), which are mainly used in prehospital settings because they offer a portable CPAP system offering a low consumption of oxygen.
High-flow nasal oxygen

High-flow nasal (HFN) oxygen delivery is also a potential alternative to CPAP because it delivers air and oxygen via a humidified circuit at greater flow rates than those traditionally used with a nasal interface (up to 40 L/min). Similar to CPAP, this therapy may provide augmented airway pressures that can improve oxygenation. However, more research is needed to define the ideal population for this modality.

Case Scenarios

Normal lung mechanics: poisonings

Following ingestion of benzodiazepines and antidepressants, a 30-year-old woman without any past medical history is intubated for airway protection. This patient has normal lung mechanics and gas exchange, but remains apneic after intubation. The following modes could all be used to guarantee adequate minute ventilation: volume or pressure A/C, SIMV and dual-control modes (PRVC, ASV). An extrinsic PEEP set at 5 cm H₂O is often used to offset the gradual loss of functional residual volume (FRC) in the supine mechanically ventilated patient. Adequate minute ventilation must be ensured to maintain normocarbia. Table 2 presents the initial ventilator settings (including V₁ and respiratory rate) that are suggested for this patient.

Severe airflow obstruction: acute asthma

A 16-year-old boy known for severe asthma is brought to the ED with a progressively worsening asthma exacerbation. At arrival, the patient is unresponsive and presents obvious signs of respiratory failure with abdominal paradox. The patient is intubated using ketamine and a neuromuscular blocker. After intubation, the peak inspiratory pressure (PIP) is measured at 50 cm H₂O and the Ppl is measured at 25 cm H₂O. This situation is an example of an airway resistance problem with normal compliance. In asthma, bronchospasm, mucosal edema, and secretions contribute to an increase in airway resistance. Increased airway resistance causes an increase in the PIP. PIP is the sum of the pressure required to force gas through the resistance of the airways and the pressure of gas in the alveoli. The Ppl reflects the effect of the elastic recoil on the gas volume inside the alveoli. If the peak pressure during a mechanical breath is 50 cm H₂O and the Ppl is 25 cm H₂O, the pressure lost in the airway because of airway resistance is 25 cm H₂O (Fig. 8). Normally this should measure less than 10 cm H₂O with a proper-sized endotracheal tube.

Another problem with airflow obstruction is auto-PEEP. Gas can be trapped in the lungs during mechanical ventilation if not enough time is allowed for exhalation. When

![Fig. 8. Effect of an increase in airway resistance on the peak pressure. (Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 159; with permission.)](image-url)
air trapping occurs, the increased alveolar pressure reduces venous return and cardiac output. Because the cause of auto-PEEP in the asthmatic is edema in the bronchi, applying extrinsic PEEP to these patients remains controversial.62

Intrinsic or auto-PEEP is related to hyperinflation caused by air trapping and is most often found in patients with acute asthma, COPD, or in patients with normal lungs when insufficient time is allowed to exhale. The simplest way of detecting auto-PEEP is to examine the expiratory flow rate waveform (Fig. 9), because flow never returns to zero at the moment of taking the next breath.

Auto-PEEP can also be measured by performing the end-expiratory airway occlusion method, which consists of occluding the inspiratory and expiratory valves at the end of expiration for 3 to 5 seconds. Pressure then equalizes and any positive pressure remaining in the circuit is auto-PEEP. This maneuver must be performed in patients without any spontaneous inspiratory effort because this interferes with the measurement.

This problem can be prevented by an optimal treatment of the underlying cause of obstruction, a lower respiratory frequency, and allowing the patient to exhale. Therefore, asthmatic patients require a shorter inspiratory time, higher inspiratory flow, and a longer time for expiration. In patients with normal airway resistance, the normal I/E ratio is 1:2 or 1:3. In patients with obstructive airway disease, the goal is to have an I/E ratio closer to 1:4 or 1:5. This ratio is achieved by decreasing the respiratory rate (8–10 breaths per minute), decreasing the VT (4–8 mL/kg) and increasing the flow rate (>60 mL/min). Typical settings for a patient with severe airflow obstruction are found in Table 2. Volume-limited or pressure-limited modes may be used, but

Fig. 9. Auto-PEEP. (Adapted from Pilbeam SP, Cairo JM. Mechanical ventilation: physiologic and clinical applications. 4th edition. St Louis (MO): Mosby Elsevier; 2006. p. 373; with permission.)
a volume-limited mode may be preferable to ensure a minimal $V_T$. It may be acceptable to allow permissive hypercapnia (pH > 7.10–7.20) except if the patient has severe pulmonary hypertension, ventricular dysfunction, or increased intracranial pressure. Plateau pressure has to be maintained at less than 30 cm H$_2$O. A dual-control mode like PRVC or ASV may also be an option if the physician wants to limit the pressure delivered with each breath. Deep sedation and paralysis are often required to gain rapid control of the patient’s breathing pattern and to allow a prolonged I/E ratio.

**Acute/chronic respiratory failure: COPD exacerbation**

The same principles as for asthmatic patients apply to patients with COPD except that the application of extrinsic PEEP is indicated for patients with COPD. For patients with COPD, the cause of air trapping is more related to dynamic expiratory airway collapse than the fixed severe distal airway edema that is found in asthmatic patients. NPPV is also the initial intervention strategy to favor in acute respiratory failure caused by COPD because this often responds well.

If intubation is necessary, a common mistake (as discussed for acute asthma) is to set a respiratory rate too high, causing auto-PEEP. If this mistake is not recognized, hypotension and cardiovascular collapse will occur. If this happens, an important step is to simply disconnect the patient from the ventilator and allow the patient to exhale. Then, resetting a lower respiratory rate, minimizing inspiratory time, and applying extrinsic PEEP help prevent the formation of auto-PEEP.

Extrinsic PEEP can also be used to prevent the formation of auto-PEEP in patients with COPD, which is especially important when treating patients with COPD who are having difficulty in triggering the ventilator. Auto-PEEP imposes extra work on patients because patients must generate enough inspiratory pressure to overcome auto-PEEP (see Fig. 9). Applying extrinsic PEEP (up to 80% of measured auto-PEEP) to the ventilator circuit can balance expiratory pressure throughout the ventilator circuitry to reduce the triggering load. A more pragmatic approach is simply to titrate extrinsic PEEP until every inspiratory effort made by a patient triggers the ventilator.

**Acute hypoxemia: cardiogenic pulmonary edema**

A 75-year-old man presents with rapidly progressive shortness of breath. He is known to have congestive heart failure. He is tachypneic with a saturation of 89% on a non-rebreather mask, and has a normal blood pressure, ECG, and mental status. NIV using CPAP in addition to other therapies (nitroglycerine and furosemide) is attempted first. However, increasing the CPAP level and changing the mode to bilevel positive pressure ventilation with an IPAP of 15 cm H$_2$O and EPAP of 5 cm H$_2$O could not decrease the signs of respiratory failure. After an hour of treatment, the patient’s mental status is decreased. Considering that the patient is failing NIV therapy, the clinician decides to intubate. Initial settings for patients with acute pulmonary edema are found in Table 2.

**Acute hypoxemia: pneumonia and ARDS**

A 60-year-old man with severe pneumonia is intubated on arrival to the ED because of hypoxemic respiratory failure. His postintubation chest radiograph shows diffuse bilateral infiltrates. The patient is not known for any cardiac problems and a bedside ultrasound reveals normal left ventricular contractility. He is diagnosed with ARDS with a $\text{PaO}_2/\text{FiO}_2$ less than or equal to 200.

Known to have decreased compliance, patients with ARDS benefit from a lung-protective ventilation strategy. This strategy is used to prevent pulmonary barotraumas
and ventilator-induced lung injury (VILI) by minimizing airway pressures. The goals of ventilation in ALI/ARDS are found in Table 2. As previously stated, the low Vₜ (6–8 mL/kg IBW and 4–6 mL/kg IBW if Ppl > 30) and low Ppl (<30 cm H₂O) are desired. PEEP is set according to FiO₂ requirement as per ARDS Network guidelines (see Table 3) aiming for arterial saturation to be more than 88%. Once the patient is stabilized, the FiO₂ can be titrated down to maintain a PaO₂ > 60 mm Hg.

The management of refractory hypoxemia in patients with ARDS includes decreasing oxygen consumption (antipyretics, sedatives, analgesics, and paralytics⁷⁴–⁷⁷), improving oxygen delivery, and manipulating mechanical ventilatory support. These interventions include rescue therapies like recruitment maneuvers,⁷⁸ HFOV, prone ventilation,⁷⁹,⁸⁰ and inhaled nitric oxide.⁸¹,⁸²

**Neuromuscular weakness and chest wall trauma**

A 40-year-old woman is brought to the ED after falling off a horse. She has a saturation of 85% with 100% FiO₂ and is unable to move her arms or legs. She is intubated to correct her hypoxia. She is also found to have multiple rib fractures and lung contusions.

When initiating mechanical ventilation in patients with polytrauma, the physician must take into account the implications of potential hypovolemia, poor lung compliance, and, in the patient with traumatic brain injury (TBI), increased intracranial pressure. Determining a safe PEEP level in TBI is difficult without intracranial pressure monitoring. Even so, low levels of PEEP seem to be safe. PEEP can also exacerbate hypotension. In cases of TBI, hypercapnia and acidosis should be avoided. A PCO₂ between 35 and 40 mm Hg is recommended.⁸³ However, patients with significant thoracic trauma can have poor pulmonary compliance, increasing the risk of barotrauma.

The patient in the present case scenario has neuromuscular weakness (quadriplegia), restriction of her chest wall (rib fractures), and decreased lung compliance (pulmonary contusions). This patient remains a challenge to ventilate because she may require larger Vₜ and respiratory rates to minimize the air hunger and atelectasis that she may experience with her neuromuscular weakness. The same principles apply with neuromuscular diseases such as Guillain-Barré syndrome or myasthenia gravis. However, with the reduced compliance, Ppl must be monitored to avoid inducing VILI or barotrauma. Initial settings for patients with neuromuscular disorders are shown in Table 2.

**Complications**

Although mechanical ventilation is lifesaving, it is associated with several complications that can be related to the direct effects of the ventilator, the endotracheal tube, the toxicity of the oxygen, as well as systemic complications.⁵

VILI encompasses barotrauma, volutrauma, atelectrauma, biotrauma, and oxygen toxicity. Barotrauma (eg, pneumothorax, pneumomediastinum, pneumopericardium) occurs when the transalveolar pressure increases to a degree that disrupts the structural integrity of the alveolus. Volutrauma (eg, ALI) occurs when the lung parenchyma is damaged by overdistending the alveoli. To prevent these complications, the physician should try to keep Ppl at less than 30 cm H₂O and Vₜ between 4 and 8 mL/kg IBW. Atelectrauma and biotrauma are caused by cyclic opening and closing of lung units. Application of a PEEP to keep the lung open may attenuate these problems.

Oxygen toxicity is explained by production of free radicals causing a spectrum of lung injury, ranging from mild tracheobronchitis to diffuse alveolar damage. To prevent this, the physician may accept PaO₂ as low as 50 mm Hg.
Complications related to the endotracheal tube include vocal cord or airway edema, mucosal ulceration, airway granulomas, tracheal stenosis, tracheoesophageal fistula, and vocal cord paralysis. Ventilator-associated pneumonia (VAP) represents another major problem.

**Troubleshooting**

In urgent situations, when a patient develops severe respiratory distress or hemodynamic instability while receiving mechanical ventilation, the physician should immediately know what to do. **Fig. 10** presents an algorithm of the various steps to troubleshoot a patient who has decompensated.

The first step is to disconnect the patient from the ventilator and provide manual bagging ventilation with 100% $\text{FiO}_2$. This step removes a large number of potential ventilator-related problems and enables the clinician to concentrate on the patient.

![Troubleshooting Algorithm](image)

Fig. 10. Troubleshooting algorithm.
If the patient gets better with this simple maneuver, the physician should try to change the ventilator settings to match patients’ effort to the required support. If the patient continues to deteriorate, this may be a sign of tension pneumothorax, displaced or obstructed endotracheal tube, intrinsic PEEP, failure in oxygen supply, or dysrhythmia. A useful mnemonic for the causes of unexpected acute decompensation of mechanically ventilated patients is DOPE: dislodgement of the endotracheal tube, obstruction of the endotracheal tube, pneumothorax, and equipment failure.

Consulting any alarms that were triggered can also be helpful in determining the cause of distress. Alarms warn of possible danger related to the patient-ventilator system. Low-pressure alarms are used to detect ventilator disconnection and leaks in the system. High-pressure alarms ring when the patient coughs, when there is high resistance, or when there is a drop in compliance. Apnea alarms are used to monitor mandatory and/or spontaneous breaths. The low gas source alarms notify the operator that the available high-pressure gas source is no longer functional. It cannot be silenced if gas is critical to ventilator operation.

**Important bedside respiratory mechanics**

In addition to the DOPE mnemonic and assessing the alarms, it is also essential to understand basic respiratory system mechanics to decide what to do with patients who are difficult to ventilate. An easy way to remember these basic concepts is to represent the respiratory system by a block attached to a wall with a spring, acted on by a unidirectional force (Fig. 11).

The spring represents the elastance of the system (lungs and chest wall), the friction offered by moving the block represents the resistive element (mainly airway resistance), the distance the block is moved represents the volume of lung inflation, the velocity of moving the block represents the flow of air, and the unidirectional force represents the sum of the pressure generated by the mechanical ventilator and by the diaphragm’s effort to inflate the lungs.

At the bedside, clinicians can easily measure these pressures to guide their assessment of any patient who is difficult to ventilate. PIP represents the pressure needed to overcome both the resistive and elastic elements. The difference between PIP and Ppl is the pressure required to overcome airway resistance. Ppl is the pressure generated by the elastance of the lung filled with a certain VT. Patients must first be put in a volume-preset mode to control the volume administered. Patients must not generate any spontaneous breathing (ie, deeply sedated) that would falsify these measures.

Assuming inflation onset with a constant flow, an initial change in pressure is recorded, which precedes alveolar filling, and corresponds with the resistive pressure (PIP–Ppl) related to gas flow in the airways. At the end of inspiration, a step-off in
resistive pressure occurs during an applied airway occlusion (Fig. 12). When the flow of gas falls to zero, the remaining pressure is called the Ppl and represents the static summation of elastic recoil forces corresponding with the applied VT.

In the clinical setting, peak airway pressures that differ by more than 10 cm H2O from the Ppl should prompt the clinician to look for a cause of increased resistance in the airways (kinked endotracheal tube, inspissated secretions, right mainstem intubation, bronchospasm, patient biting the endotracheal tube). If Ppl is increased (>30 cm H2O), clinicians should look for causes of increased elastance (tension pneumothorax, ARDS, pulmonary edema, obesity, and abdominal compartment syndrome). Auto-PEEP can also be a cause of increased Ppl.

If signs of respiratory distress persist without any abnormal change in the PIP or Ppl, the clinician must then consider a pulmonary embolism. In situations of respiratory distress, clinicians should also ensure that the triggers are not too sensitive (triggering frequent breaths) or not sensitive enough (failing to trigger). Adequate sedation and analgesia are essential in managing a patient who is difficult to ventilate.

As previously stated, the main goal of mechanical ventilation is to reduce the WOB. In acute respiratory failure, the inability of the respiratory pump, mainly the diaphragm and extrinsic respiratory muscles, to meet metabolic demands is a result of either increased ventilatory load at more than the critical level or inability of these respiratory muscles to generate sufficient force to overcome the elastic and resistive forces previously presented. Measuring the WOB is still difficult at bedside, but careful clinical examination is paramount in determining whether sufficient ventilatory support is offered. This determination consists in assessing the breathing pattern, the use of extrinsic respiratory muscles, and the presence of paradoxic motion of the thorax and abdomen. If there are signs of persistent increased WOB, physicians must attempt to correct the underlying cause and adapt the ventilatory support to target the resistive or elastic components of respiratory failure.

**SUMMARY**

To reduce WOB and reverse life-threatening hypoxemia and hypercarbia, emergency physicians need to understand basic lung mechanics, the patient’s underlying disease, and the different modes of invasive ventilation and NIV. Following the initiation of ventilatory support, the emergency physician must then evaluate the interaction between the patient and the ventilator to see whether hypoxemia and/or hypercarbia are resolving.
Basic modes used to ventilate patients are A/C, SIMV, and PSV. In cases of ALI/ARDS, it is recommended to ventilate patients in volume A/C, to set low Vt (4–8 mL/kg IBW), and to maintain low Ppl (<30 cm H2O). For patients with acutely decompensated COPD (pH<7.35 and relative hypercarbia), NIV should be started early. Starting NIV early helps to avoid unnecessary intubation. If invasive mechanical ventilation is needed, it is essential to decrease the set respiratory rate because air trapping occurs (auto-PEEP) with high respiratory rates. Increasing inspiratory flow to decrease inspiratory time and prolong expiratory time also helps to avoid auto-PEEP formation. Extrinsic PEEP is also essential in patients with COPD to prevent distal airway collapse, thus preventing more air trapping. NIV should also be started early for patients with acute cardiogenic pulmonary edema, but only in the absence of shock or acute coronary syndrome. NIV is also recommended for immunosuppressed patients who have acute respiratory failure.

Although mechanical ventilation is lifesaving, it is associated with several complications. Therefore, avoiding mechanical ventilation when NIV is indicated and having a systematic approach to initiating ventilation, making adjustments, and troubleshooting is essential. Emergency physicians must also understand how airway resistance and elastance affect the ventilated patient. This understanding helps them perform basic bedside measurements (inspiratory occlusion pressure and expiratory occlusion pressure) to identify the cause of a patient difficult to ventilate.

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REFERENCES


