MECHANICAL VENTILATION

HISTORY

Mechanical ventilatory support is now a major aspect of critical care. The lungs are often central in a patient with multi-organ system failure, and many patients have cardiopulmonary failure secondary to other disorders. The challenge of providing ventilatory support has never been greater, and the equipment with which to do so has never been more varied.

Mechanical ventilation has been around for centuries, as far back as 1530, when Paracelsus used a fire bellows connected to a tube in a patients mouth to ventilate a human.

The first generation of mechanical ventilators were developed in the mid-1800’s. The first mechanical ventilator was the iron lung, a negative pressure ventilator as shown in the pictures below:

Figure 11  iron lung

Dupuis, Ventilators, ©1986, Mosby Year Book.

The iron lung creates a negative pressure in the thorax by creating a vacuum around it. This draws air into the lungs, and can ventilate the patient without the use of an artificial airway.
Iron lungs are still in use today, despite being bulky, difficult to clean and sterilize, and problems with nursing care. The chest cuirass was a type of ventilator utilizing a chest plate that covered the thorax and abdomen.

These are also still used as home ventilators on patients with chronic paralytic disorders, although they do have an unwanted side effect of air entering the stomach when the abdomen is exposed to negative pressure ventilation.

It was during the polio epidemic in Scandinavia in 1952 when positive pressure ventilation came into vogue. At the time, with hundreds of polio patients paralyzed and unable to breathe, medical students and nurses were ventilating patients by hand 24 hours a day to keep them alive, because there were not enough iron lungs or other negative pressure ventilators. Soon afterwards, positive pressure ventilators with cuffed endotracheal and tracheostomy tubes became widely used, mostly because of their reliability and direct access to the airway.

Ventilatory philosophy has also evolved throughout the years. Larger tidal volumes were standard (10-15 mL/kg), with an occasional larger “sigh” breath designed to open up collapsed alveoli. PEEP was introduced to improve FRC, and in the early 1970’s, the desire to improve patient tolerance of mechanical ventilation led to the development of patient-triggered breaths. The mid-1980’s led to a shift in thinking, and mechanical ventilation began to become tailored to individual patient needs, with many
new alternate modes and attention to methods of weaning. Today, the choices for modes and ventilators are endless, and focus has been on the physiology of ventilation and protecting the lung. This workshop will not attempt to describe all modes or ventilators, but will focus on the concepts behind the choices available.

INDICATIONS FOR VENTILATORY SUPPORT

Throughout hospitals across North America, there is no absolute agreement on what constitutes the indications for the beginning of ventilatory support. Table 1 shows a compilation of some of the indices used to determine when to put a patient on a ventilator, but these parameters must be looked at in conjunction with the patient as a whole. For example, one of the criteria is for \( PCO_2 > 55 \), but many patients are chronic \( CO_2 \) retainers and their normal \( CO_2 \) may be much higher than this.

Table 1  Indices for the Initiation of Ventilatory Support

<table>
<thead>
<tr>
<th>MEASUREMENT</th>
<th>UNITS</th>
<th>NORMAL</th>
<th>VENTILATORY SUPPORT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume (( V_T ))</td>
<td>mL/kg</td>
<td>5-8</td>
<td>&lt;5</td>
</tr>
<tr>
<td>Vitai Capacity (VC)</td>
<td>mL/kg</td>
<td>65-75</td>
<td>&lt;10 or &lt;15</td>
</tr>
<tr>
<td>Forced expiratory volume in one second (FEV,)</td>
<td>mL/kg</td>
<td>50-60</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Functional residual capacity (FRC)</td>
<td>% of predicted</td>
<td>80-100</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Respiratory rate (f) or (RR)</td>
<td>breaths/min</td>
<td>12-20</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Maximum inspiratory force (MIF)</td>
<td>cm H( _2 )O</td>
<td>80-100</td>
<td>&lt;20-30</td>
</tr>
<tr>
<td>Minute ventilation (MV) or (( V_E ))</td>
<td>L/min</td>
<td>5-6</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Maximum voluntary ventilation (MVV)</td>
<td>L/min</td>
<td>120-180</td>
<td>&lt;20</td>
</tr>
<tr>
<td>Dead space (( V_D/V_T ))</td>
<td>%</td>
<td>0.25-0.40</td>
<td>&gt;0.60</td>
</tr>
<tr>
<td>( PaCO_2 )</td>
<td>mm Hg</td>
<td>35-45</td>
<td>&gt;50-55</td>
</tr>
<tr>
<td>( PaO_2 ), breathing air</td>
<td>mm Hg</td>
<td>75-100</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Alveolar to arterial PO(_2) gradient ([P(a-a)O_2]), breathing 100% oxygen</td>
<td>mm Hg</td>
<td>25-65</td>
<td>&gt;350-450</td>
</tr>
<tr>
<td>Arterial/alveolar PO(_2) ratio ((PaO_2/PaO_2))</td>
<td>mm Hg</td>
<td>0.75</td>
<td>&lt;0.15</td>
</tr>
<tr>
<td>Arterial PO(_2)/inspired O(_2) ((PaO_2/FiO_2))</td>
<td>mm Hg</td>
<td>350-450</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Intrapulmonary shunt</td>
<td>%</td>
<td>&lt;5</td>
<td>&gt;20-30</td>
</tr>
</tbody>
</table>


VENTILATOR PARAMETERS AND INTER-RELATIONS

Ventilator technology seems to have developed its own language, and discussion of ventilation often sounds like gibberish when using all the abbreviations. To make matters worse, many ventilator parameters and settings are related and sometimes affected by each other. The abbreviations and parameters, along with their formulas and inter-relations, are described below:

\[
\text{MV or } V_E = \text{minute ventilation (total volume in and out of lungs over one minute)}
\]

\[
\text{cycle time = total time in seconds of one complete inspiration and expiration}
\]

\[
V_T = \text{tidal volume (volume moved in and out of lungs during normal breathing)}.
\]

\[
RR \text{ or } f = \text{respiratory rate (breaths per minute)}.
\]

\[
V_T \times RR = MV
\]

\[
60 \text{ seconds} = RR
\]

\[
\text{cycle time}
\]
\[ V = \text{flowrate} \]
\[ T_I = \text{inspiratory time (time of inspiration in seconds)} \]
\[ T_E = \text{expiratory time (time of expiration in seconds)} \]
\[ I:E = \text{inspiratory/expiratory ratio (ratio of time of inspiration vs. expiration)} \]

\[
\frac{\text{cycle time}}{\text{cycle time}} = \frac{T_I}{T_I + T_E} \]

\[
\frac{T_E}{T_I} = x, \text{ then } I:E \text{ is } 1:x\]

\[
MV \times (I + E) = V_{\text{average (Lpm)}}\]

\[
\frac{V_T}{T_I} = V_t\]

**VENTILATORY MODES**

**CONTROLLED VENTILATION**

Controlled ventilation is *time-triggered*; that is, the respiratory rate that is set will control the number of respiratory cycles per minute, and the patient has no control over the ventilation (see fig. 4). This can be very uncomfortable for the patients, and is not often used because they cannot trigger any breaths. It is never appropriate to “lock out” a patient with a respiratory drive using this mode to prevent respiratory alkalosis. Even patients who are paralyzed with neuromuscular blockers or well sedated to prevent undesirable triggering can potentially “wake up” and try to trigger a breath, and may feel suffocated if they cannot do so. This mode is sometimes used on chronic patients with permanent spinal cord injuries who have no ability to trigger the ventilator.

![Figure 4 controlled ventilation](Pilbeam, SP. Mechanical Ventilation 2nd Ed. ©1992 Mosby-Year Book, Inc.)

**ASSIST CONTROL**

Assist/Control (AC) volume control mode is a common mode of ventilation used for patients that cannot support their own ventilation, but still need to be able to trigger breaths (see fig. 5). It is a fully supportive mode, meaning the patient does not have to expend any energy or respiratory muscle function in order to maintain their oxygenation and level of carbon dioxide. A respiratory rate and tidal volume are set and therefore the
ventilating pressures are variable, based on changes in the patient’s resistance and compliance. If the patient triggers the ventilator between these mandatory breaths, they will get a fully supported volume control breath at the set tidal volume. With the tidal volume and the flow set, the peak pressure will be variable.

Figure 5 assist/control ventilation


With this mode, the clinician can maintain control over the minimum minute ventilation, and can completely override the patient’s work of breathing. It is used in patients who are intubated prior to a respiratory evaluation, in patients who require a high minute ventilation, patients with an unstable or absent respiratory drive, patients with respiratory muscle fatigue with need for maximal rest, and those patients in whom it is desirable to minimize oxygen consumption.

Although this mode was designed to prevent the patient from having to do any respiratory work other than initially trigger the ventilator, studies have shown that the patient may do up to 50% of the work of inspiration if the settings are not matched to the patient’s respiratory effort. Therefore the flowrate, flow pattern and tidal volume must be set appropriately.

Assist/Control can cause respiratory alkalosis due to hyperventilation, air trapping (if the respiratory rate is set too high and the patient does not have time to exhale), and excessive intrapulmonary pressures (if the tidal volume is set too high).

PRESSURE SUPPORT

Pressure Support (PS) is a mode in which the clinician can set a positive inspiratory pressure throughout most of the inspiratory phase (see fig. 6). Pressure support is a spontaneous mode; the patient must trigger a breath to get the pressure support. It can be used in conjunction with SIMV and CPAP. When the patient triggers a breath, the ventilator responds with flow to meet the patient’s inspiratory flow demands and to maintain the inspiratory pressure at a preset level. As long as the patient’s inspiratory effort is maintained, the preselected airway pressure stays constant, with a variable flow rate of gas from the ventilator. When the patient’s inspiratory flowrate slows down to a certain level, the pressure support shuts off to allow the patient to exhale. All ventilators are a little different in the level of flow at which the pressure support shuts off, but with most it is at approximately 25% of the patient’s peak inspiratory flow. Tidal volume will
vary in this mode, determined by patient effort, compliance, resistance, and the set level of pressure support.

![Airway Pressure Diagram](image)

**Figure 62 pressure support ventilation**


Pressure support ventilation is used to decrease the patient’s work of breathing. It can be set low, just to overcome the resistance of the ventilator circuit and endotracheal tube, or high enough to provide a large tidal volume. In order to take a breath, a patient must overcome the elastic resistance of the lung, and the resistance of the airways, artificial airway, and ventilator circuit. The inspiratory pressure can be set to overcome this work and make it easy for the patient to get a breath. Pressure support without SIMV should only be used on patients who have an intact respiratory drive and some stability of ventilation. It is often used as a weaning mode to condition and strengthen the muscles of ventilation – the lower the pressure support level, the harder the patient must work.

Initial settings of pressure support should be set in order to receive the desired tidal volume. A pressure support set too high will result in hyperinflation and possible cardiorespiratory compromise. Once set, the pressure support level should be turned down as tolerated by the patient, watching for signs of respiratory distress such as an increased respiratory rate, decreased tidal volume, and accessory muscle use. The patient is often extubated when the pressure support reaches 5 – 7 cm H$^2$O, as this is the approximate level needed to overcome the resistance of most artificial airways. If the diameter of the endotracheal or tracheostomy tube is excessively small, this level may need to be higher.

**IMV/SIMV**

**IMV** stands for Intermittent Mandatory Ventilation. This mode was developed in the 1960’s out of a necessity to allow infants with hyaline membrane disease to breathe intermittently between respiratory cycles, because the sensing mechanisms at that time were not responsive enough to allow the tiny infants to trigger the ventilator, and they would grow progressively hypoxic and hypercapnic. IMV provided a set respiratory rate, but for the first time allowed the infants to breathe spontaneously from a continuous flow in between mandatory breaths (see fig. 7). IMV systems were then adapted to adult
ventilators, allowing adults to breathe independently of mandatory breaths. With an inspiratory flowrate, the ventilator would deliver flow to meet the patient’s demand.

Sometime later, **SIMV**, or **Synchronized Intermittent Mandatory Ventilation** was developed to synchronize the patient’s spontaneous breaths with the ventilator’s mandatory breaths (see fig. 7). This prevented the ventilator from delivering a mandatory breath when the patient was exhaling, thereby preventing “stacked” breaths and resultant barotrauma.

![Figure 7 IMV vs. SIMV](image)

**Figure 7 IMV vs. SIMV**

IMV/SIMV can be used with PEEP and pressure support. If used with both of these adjuncts, the patient will exhale down to the set PEEP level. On mandatory breaths, the patient will get the set tidal volume. On spontaneous breaths, the patient will get pressure-supported breaths.

Proponents of IMV/SIMV modes claim that they avoid respiratory alkalosis when compared with assist/control modes, they decrease the requirement for sedation and paralysis, they decrease mean airway pressure, improve V/Q matching, prevent respiratory muscle atrophy, and expedite weaning. Others argue that IMV modes can increase the risk of carbon dioxide retention, increase the work of breathing, cause respiratory muscle fatigue, and that weaning is best accomplished using other methods. IMV/SIMV modes certainly have a place in ventilatory therapy, but with all the choices available today, all ventilatory modes should be tailored to meet the needs of individual patients.

**PRESSURE CONTROL**

Pressure Control (PC), unlike assist/control, is a mode in which a peak pressure is set throughout inspiration, as well as a respiratory rate (see fig. 8). With pressures set instead of a volume, the volume the patient receives will vary based on changes in their compliance and resistance. When the patient triggers a ventilator while on this mode, they receive a fully supported pressure control breath.
Pressure control is often used to improve patient oxygenation and protect the lung against barotrauma. With a pressure held constant, the length of the inspiration can be manipulated in order to recruit collapsed alveoli and improve oxygenation. The lung can be protected against high ventilating pressures if the patient’s compliance is worsening.

Pressure control is a mode that should only be used with careful ventilator and patient monitoring. If the patient’s compliance improves or resistance decreases, the tidal volume can suddenly increase and increase the risk of pneumothorax. Conversely, if the compliance decreases or the resistance increases, there is a risk of hypoventilation and resulting acidosis.

MANDATORY MINUTE VENTILATION

Mandatory minute ventilation (MMV), also called minimum minute ventilation, or augmented minute ventilation, was developed during the late-1970’s as a weaning mode. The clinician sets a minimum minute volume that the patient must meet. The ventilator will monitor the patient’s spontaneous minute volume, and if it falls below the set minute volume, the ventilator will deliver set tidal volumes at a set respiratory rate until the minute volume increases above the set minimum. This mode is used for weaning but precautions must be taken – the patient should be monitored closely to ensure they are getting effective tidal volumes (greater than deadspace) and to see how much spontaneous ventilation they are doing on their own.

AIRWAY PRESSURE RELEASE VENTILATION

Airway pressure release ventilation (APRV) is a new mode developed in 1987. Using this mode, the patient breathes at a high level of CPAP, and occasionally the pressure is released down to baseline to allow the lungs to deflate passively and clear PCO₂. The length of pressure release is less than 2 seconds. This mode was designed to improve oxygenation in the non-compliant lung (see fig. 9).
There is a common mistake made by those unfamiliar with ventilation to confuse the terms CPAP and PEEP. These terms are often used interchangeably, but in fact mean different things. The terms are differentiated below.

1. **CPAP** Continuous Positive Airway Pressure  CPAP is a technique that holds a continuous positive pressure in the lungs during spontaneous ventilation. This means that the pressure is held constant in the lungs at all times, and the patient takes breaths without any assistance.

2. **PEEP** Positive End Expiratory Pressure  PEEP refers to holding a pressure in the lungs during the exhalation phase during assisted ventilation. This means that the patient receives an assisted breath from the ventilator, and exhales down to the set PEEP level.
CPAP is used as a mode unto itself. It can be used with nasal CPAP systems to provide noninvasive ventilation, such as with nocturnal nasal CPAP systems for obstructive sleep apnea. It can also be used for patients who can effectively maintain their tidal volume and respiratory rate on a ventilator, but have difficulty with oxygenation. It is sometimes used as a weaning trial mode for patients on ventilators who need to slowly build up respiratory muscle strength. CPAP can be used on almost every ventilator available.

PEEP is usually set with any assisted or controlled ventilatory mode. Preventing the lungs from exhaling down to atmospheric pressures has several advantages. The lungs will be less vulnerable to collapse and atelectasis, oxygenation and ventilation/perfusion matching will improve, and the compliance of the lung may increase. It will help to recruit previously collapsed alveoli, and may help to redistribute extravascular lung water (see fig. 12).

![PEEP may help to redistribute extravascular lung water and prevent mild ongoing pulmonary hemorrhage](image)

Figure 12 redistribution of lung water
Shapiro et al, Clinical Application of Respiratory Care, 4th Ed., ©1991 Mosby Year Book

PEEP valves can also be attached to manual ventilation devices to provide a constant positive expiratory pressure in the lungs while using a bag/mask resuscitator.

Positive expiratory pressure can dramatically improve a patients oxygenation and ventilatory status, but can have serious detrimental effects. PEEP and CPAP must be used cautiously, trying to achieve the best PaO₂ with the least adverse effects. There is much controversy over what constitutes the “best PEEP”. One approach is to titrate the PEEP to achieve adequate PaO₂ (usually >60 mm Hg, or SaO₂ >0.90), with a non-toxic level of FiO₂ (<0.50) without causing a significant reduction in cardiac output. Other methods include titrating PEEP to optimum pulmonary compliance, or to a shunt <15%.

**ADVERSE EFFECTS OF MECHANICAL VENTILATION**

There are many adverse effects to positive pressure ventilation, adversely affecting many body systems. However, most intensive care units are well aware of these
and take steps to avoid them, and the beneficial effects of ventilation usually outweigh the bad, because in most cases the alternative to no ventilation is death.

**CARDIOVASCULAR EFFECTS**

When a patient breathes spontaneously, the negative pressure created in the thorax not only draws air into the lungs, but also draws blood into the major thoracic vessels, pulling venous blood into the heart. This is called the “thoracic pump”. Based on Starling’s law of the heart, an increased preload to the heart tends to stretch the ventricles and the force of contraction is greater, resulting in a bigger stroke volume, and thus a greater cardiac output.

During positive pressure ventilation, the thoracic blood vessels are compressed and central venous pressure increases. Less blood returns to the heart because of the increased pressure gradient and the lack of negative intrathoracic pressure. This causes a drop in preload and therefore a drop in cardiac output. High levels of PEEP can cause an increase in right ventricular pressure and a shift in the interventricular septum to the left, causing left ventricular filling pressures to decrease, which will reduce cardiac output. A falling cardiac output could have negative effects on myocardial perfusion through the coronary arteries, which in turn could cause myocardial ischemia.

**INTRACRANIAL EFFECTS**

Blood flow into the brain is determined by the cerebral perfusion pressure (CPP). Cerebral perfusion pressure is calculated by subtracting the intracranial pressure (ICP) from the mean systemic arterial pressure (MAP).

Mechanical ventilation can increase central venous pressure by compressing the heart and increasing intrathoracic pressure. This high central venous pressure can decrease the venous return to the heart from the jugular veins, thereby causing an increased ICP. Other hemodynamic effects from mechanical ventilation can cause a decrease in the MAP, and therefore a drop in the CPP.

Normal patients can easily handle a decreased cerebral perfusion pressure or an increased intracranial pressure through normal autoregulation, which automatically controls blood flow into the brain. However, many patients with severe head injuries or cerebral edema can develop life-threatening complications due to an increase in their ICP from mechanical ventilation or high PEEP.

**RENAL EFFECTS**

Renal performance can be compromised during positive pressure ventilation because as cardiac output decreases, renal blood flow decreases, glomerular flow rates drop, and urine output slows. Mechanical ventilation can cause other renal effects, including an increase in the levels of antidiuretic hormone (ADH), and reduced levels of atrial natriuretic peptide (ANP) (a naturally produced diuretic). Increased ADH causes an inhibition of urine secretion, and decreased ANP can cause water and sodium retention.
BAROTRAUMA

This term describes the physical damage occurring to the lungs as a result of pressure. In fact, lung damage is more likely to result from alveolar overdistention (volutrauma). The incidence of barotrauma in patients on IPPV and PEEP can be as high as 15%. Some pre-existing conditions shown in table 18 are known to predispose a patient to barotrauma.

Table 2 Conditions Predisposing a Patient to Barotrauma

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>High peak airway pressures with low end-expiratory pressures</td>
</tr>
<tr>
<td>Bullous lung disease (e.g. emphysema and history of tuberculosis)</td>
</tr>
<tr>
<td>High levels of PEEP with high tidal volumes</td>
</tr>
<tr>
<td>Aspiration of gastric acid</td>
</tr>
<tr>
<td>Necrotizing pneumonias</td>
</tr>
<tr>
<td>ARDS</td>
</tr>
</tbody>
</table>

An excessive level of positive airway pressure can cause subcutaneous emphysema (a condition where air leaking from ruptured alveoli can track into the mediastinum or elsewhere in the thorax). Subcutaneous emphysema can also result from high positive airway pressures used with a tracheostomy tube when air is forced into the subcutaneous layer of the skin. Subcutaneous emphysema will feel like crackling air bubbles when the skin is palpated. It is a condition that does not usually cause problems in itself unless the excess air builds to a level that begins to compress structures underneath. However, it is a sign of an air leak or pneumothorax, conditions that can be fatal.

Pneumothorax is defined as the presence of air in the pleural space. It is caused by alveolar rupture and can lead to a buildup of pressure in the pleural space, compressing and collapsing the lung and mediastinum underneath.

An unrelieved buildup of pressure is called a tension pneumothorax, and can rapidly progress to respiratory and cardiac compromise. Pneumothorax needs immediate medical attention.

Figure 134 pneumothorax

Barotrauma can also take the form of pneumomediastinum, pneumoperitoneum, and damage at the cellular level.

**OTHER EFFECTS**

Studies have been done suggesting that further detrimental effects of positive pressure ventilation can include liver malfunction, gastric mucosal ischemia, and increased fibrinolysis.