

**TITLE:** Mechanical Ventilation 101: Unraveling the Alphabet Soup

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### **Learning Objectives**

1. To understand the three parameters that determine the characteristics of a single ventilator breath
2. To recognize how these are applied in volume-limited and pressure-limited modes of ventilation
3. To appreciate the advantages and limitations of these modes
4. To be able to devise a ventilatory weaning algorithm

### **CLASSIFICATION OF MECHANICAL VENTILATION**

Abbreviations used are summarized in Table 1.

#### **DETERMINANTS OF THE VENTILATOR BREATH PATTERN**

Positive-pressure ventilator modes are defined by inspiratory events or phase variables (Tables 2,3). PEEP is a form of airway pressure therapy which can be applied to any of the ventilator modes and will be discussed separately.

#### **Initiation (Trigger)**

A ventilator breath may be initiated by the machine (machine-triggered, controlled breath) or the patient (patient-triggered, assisted breath).

In a controlled breath, inspiration is triggered at a time set by the ventilator, regardless of the patient's effort and respiratory cycle.

In an assisted breath, inspiration is triggered when the patient generates a negative airway pressure below a preset threshold (usually  $\sim -2$  cmH<sub>2</sub>O), termed the sensitivity. The ventilator rate is dependent on the patient rate, unless efforts are too feeble to reach the sensitivity threshold.

#### **Limit (Target)**

The limit, or target, for each breath is usually used to define the primary ventilator mode, and is either a preset tidal volume or airway pressure.

In the volume limited mode, the ventilator delivers a set flow for a set time until the preset tidal volume is achieved. The peak airway pressure that results is variable, and depends on the peak flow rate, tubing and airway resistance, and lung and chest wall compliance.

In the pressure limited mode, the ventilator delivers a variable (often decelerating) flow to maintain the preset airway pressure limit. The tidal volume that results is variable, and depends on the peak flow rate, tubing and airway resistance and lung and chest wall compliance. In the case of PSV, it also depends on patient effort.

#### **Cycle Off**

The phase variable used by the ventilator to cycle from inspiration to expiration defines the cycle off function, and could be tidal volume, airway pressure, elapsed time or inspiratory flow rate.

The breath may be cycled off to expiration as soon as the preset tidal volume or airway pressure has been achieved (volume-cycled, pressure-cycled), e.g. IMV.

The inspiratory time can be prolonged by the addition of a phase of zero flow, called an inspiratory pause, and the breath cycles off once the predetermined time has elapsed (time-cycled), e.g. PCV.

The breath may cycle off to expiration when the patient's inspiratory flow rate declines to about 25% of the peak flow rate (flow-cycled), e.g. PSV.

## **VENTILATOR MODES (Table 4)**

### **Volume Targeted Modes**

#### ***Volume Controlled Ventilation (VCV)***

This mode is popularly referred to as controlled mechanical ventilation (CMV).

All breaths are initiated at a set time (machine-triggered, controlled breaths) and delivered with a preset flow pattern to achieve a set tidal volume (volume limited). The ventilator cycles off to expiration once the tidal volume has been delivered (volume-cycled), unless an inspiratory pause is added for a predetermined time (time-cycled). Minute ventilation ( $V_E$ ) is determined solely by the ventilator.

The airway pressure alarm limit is usually set to 60 cmH<sub>2</sub>O. If it is exceeded, which could occur with coughing, bucking, bronchospasm or stiff lungs, an alarm sounds and the ventilator cycles to expiration without delivering the entire preset tidal volume.

VCV is limited to patients who are anesthetized, heavily sedated, paralyzed or who have severe neuromuscular disorders. VCV completely eliminates the patient's work of breathing, an advantage in ARDS, when lung stiffness induces work of breathing that exceeds the patient's cardiopulmonary reserve.

#### ***Volume Assisted Ventilation (VAV)***

This mode is popularly referred to as assisted mechanical ventilation (AMV).

All breaths are initiated by patient inspiratory effort above threshold (patient-triggered, assisted breaths). In all other respects a VAV breath is identical to a VCV breath, i.e. it is volume limited and volume- or time-cycled. When a breath is triggered, the full preset tidal volume is delivered, i.e. the patient cannot breathe spontaneously between machine breaths.  $V_E$  is determined by the product of the machine delivered tidal volume and the patient's respiratory rate. Patient work of breathing is greater than VCV, but is still largely provided by the ventilator.

VAV mandates spontaneous ventilation and is contraindicated with heavy sedation, muscle relaxation or in patients too weak to trigger breath initiation. VAV is dangerous in central hyperventilation syndromes. Assisted patient tachypnea generates an extremely high  $V_E$ , acute respiratory alkalosis, hypokalemia and arrhythmias.

#### ***Volume Assisted-Controlled Ventilation (VACV)***

This mode is popularly referred to as assist-control ventilation (ACV).

VACV is a combination of VAV and VCV. A minimum controlled ventilator rate is set, but patient-triggered breaths are allowed if the spontaneous rate exceeds the controlled rate. VACV can thereby prevent hypoventilation, but like VAV, it will exacerbate hyperventilation.

Ventilator weaning is not possible by weaning the controlled rate. For example, a patient with a spontaneous rate of 16 breaths/min has the controlled rate set at 12 breaths/min, with  $V_T$  1.0 L. The actual  $V_E$  the patient will receive is 16 L/min, while the guaranteed  $V_E$  is 12 L/min. If the controlled rate is decreased to 8 breaths/min and the patient's spontaneous rate does not change,  $V_E$  remains 16 L/min. If the patient stops breathing, the ventilator would still provide 8 controlled breaths/min, and  $V_E$  would be 8 L/min.

### ***Intermittent Mandatory Ventilation (IMV)***

IMV is essentially VCV imposed upon spontaneous ventilation.

At an operator set frequency (the IMV rate) the ventilator provides a positive pressure breath to the patient regardless of the phase of the patient's cycle. It is identical to a VCV breath (machine-triggered, volume-limited, volume- or time-cycled). However, an additional circuit provides a continuous gas flow which allows the patient to breathe spontaneously between machine breaths. Total  $V_E$  is the sum of machine  $V_E$  and patient  $V_E$ .

Synchronous IMV (SIMV) is a modification of IMV in which the patient is allowed to trigger the IMV breath in a manner analogous to VAV. However, if the patient does not breathe within an allotted "window" (i.e. 6 sec if the IMV rate is set at 10 breath/min), the ventilator delivers a VCV breath. Thus, SIMV may be thought of as VACV superimposed upon spontaneous ventilation.

### ***Advantages and Limitations of IMV***

The benefits of IMV are directly related to the relative preponderance of spontaneous versus IMV ventilation. The physiologic impact of high IMV rates is almost identical to VCV or VACV.

- Avoidance of heavy sedation, paralysis or hyperventilation to eliminate the ventilatory drive.
- No exacerbation of central hyperventilation syndromes - the IMV rate is fixed, and tachypnea is not augmented by large machine-delivered tidal volumes.
- Improved intrapulmonary matching of ventilation and perfusion.
- Increased venous return and enhanced cardiac output and renal perfusion.
- Decreased potential for barotrauma because of decreased mean airway pressure.
- In patients with COPD and  $CO_2$  retention, IMV allows the adjustment of  $V_E$  to preserve hypercarbia and facilitate ventilatory weaning.

### ***Ventilatory Weaning with IMV***

IMV is an extremely useful mode for weaning ventilation in postoperative patients. By decreasing the IMV rate in stepwise fashion over time (e.g. 10, 8, 6, 4 breaths/min), there is a gradual increase in patient work of breathing versus ventilator work of breathing.

Inadequate recovery from the residual effects of opioid anesthesia or sedation is revealed by lack of appropriate "overbreathing" and the development of respiratory acidosis. Also, a sudden decline in  $PaO_2$  at low IMV rates exposes the patient's spontaneous ventilation to be shallow, with decreased FRC and poor ventilation-perfusion matching. These findings suggest that tracheal extubation should be delayed until the patient has recovered more fully.

The original IMV systems provided fresh gas in a continuous flow mode, which minimized patient work to open the exhalation valve. SIMV uses a demand flow system to actuate the patient-triggered IMV breath, which imposes increased patient work. Significant patient work is also performed in overcoming the resistance of the ventilator tubing circuit. Moreover, in IMV patient support is an "all or none" phenomenon: the ventilator provides 100% of the work during the IMV breath and zero work during the spontaneous breath. Thus, patients with limited ventilator reserve or poor lung compliance may become anxious, tachypneic and fail weaning at low IMV rates.

In postoperative patients, these limitations may be overcome by adding 5 - 10 cmH<sub>2</sub>O pressure support, but in difficult weaning conversion to PSV mode may offer substantial advantages.

## Pressure Targeted Modes

### *Pressure Support Ventilation (PSV)*

PSV is a unique, patient-triggered, pressure-limited, flow-cycled mode which allows close tracking of the patient's ventilatory effort and very precise decremental withdrawal of ventilatory support of the work of breathing. It provides the greatest patient-ventilator interaction of any of the conventionally used ventilatory modes.

Once initiated, there is a rapid flow of fresh gas until the airway pressure limit (above baseline PEEP) is achieved. Thereafter, the inspiratory flow adjusts via microprocessor circuitry to keep the airway pressure constant. The patient can continue to actively inhale and increase the delivered tidal volume above that provided by the ventilator alone. When the patient's inspiratory flow rate declines to 25% of the peak flow rate, the ventilator cycles off into expiration.

The actual tidal volume delivered for a given level of PSV depends on patient effort, airway resistance and chest wall and lung compliance, and must be measured with each breath.

### *Advantages and Limitations of PSV*

- PSV provides support with every breath that the patient takes, with a variable flow pattern. Many patients feel more comfortable on PSV than IMV.
- It can be used to support spontaneous ventilation during IMV (see above). However, it cannot be used as an adjunct to VCV or VACV, in which unassisted spontaneous breathing is not allowed.
- At a high level of PSV, the ventilator work is almost 100%, patient work almost zero. PSV can be slowly decreased in decrements of 1-2 cmH<sub>2</sub>O, so that patient work of breathing is very gradually increased over a time continuum (no "all or none").
- Peak airway pressure is lower than with volume limited modes, because part of the tidal volume is generated by the patient's spontaneous effort.
- The PSV breath is patient-triggered, and cannot be used in heavily sedated or paralyzed patients. In central hyperventilation syndromes PSV exacerbates high V<sub>E</sub> and acute respiratory alkalosis, just like VACV.
- PSV is a weaning mode, and is not appropriate for patients with active lung pathology and poor lung compliance (e.g. ARDS)

### *Ventilatory Weaning with PSV*

The initial level of PSV depends on airway resistance, lung compliance, patient fatigue and muscle reserve, and must be individualized to each patient. To generate a tidal volume of ~ 10 mL/kg and provide about 95% of the work of breathing, PSV of between 20 and 30 cmH<sub>2</sub>O is required. Usually a tidal volume of ~ 7 mL/kg is sufficient (PSV 15-20 cmH<sub>2</sub>O).

PSV is most successful in patients with rapid, shallow breathing due to low FRC or muscle fatigue. The level of PSV is "ramped up" at the bedside until the desired tidal volume is achieved, which should be matched by a slowing of the ventilatory rate to < 20 breaths/min and stable or decreased V<sub>E</sub>. The IMV rate can then be rapidly weaned to zero, or left at 2-4 breaths/min to provide "sighs".

Unchanged ventilatory rate and increased V<sub>E</sub> indicate a central hyperventilation syndrome, a relative contraindication to PSV.

Once the patient is stabilized, ventilatory weaning is performed by gradual decrease in the level of PSV (decrements of 1-2 cmH<sub>2</sub>O, q8 to q 24 hr). The rate of weaning must be individualized to the patient.

Too rapid PSV weaning results in shallow, rapid breathing and respiratory muscle fatigue. Too slow PSV weaning prolongs muscle atrophy and discoordination.

PSV should not be weaned below 8 cmH<sub>2</sub>O, because this level is required to overcome the resistance of the endotracheal tube; instead, patients should be assessed for tracheal extubation.

### ***Pressure Controlled Ventilation (PCV) and Inverse Ratio Ventilation (IRV)***

PCV is a machine-triggered, pressure-limited and time-cycled mode intended for patients with acute respiratory failure, severe hypoxemia and poor lung compliance.

Immediately after initiation of the breath, fresh gas flow rapidly achieves the preset pressure limit above baseline PEEP. For example, PCV of 25 cmH<sub>2</sub>O added to 10 cmH<sub>2</sub>O PEEP achieves a peak airway pressure of 35 cmH<sub>2</sub>O. Thereafter, flow decelerates to zero with an inspiratory pause that sustains airway pressure at the limit for the defined inspiratory time, when it cycles off. This allows precise definition of inspiratory and expiratory ratios.

Prolongation of inspiratory time such that the I:E ratio is greater than 1:2 is known as inverse ratio ventilation (IRV).

### ***Advantages and Limitations of PCV and IRV***

- Limiting peak airway pressure may decrease the risk of barotrauma. However, mean airway pressure is increased, and excessive alveolar distension (volutrauma) may play an even more important role.
- Precise control of inspiratory flow rate, inspiratory time and mean airway pressure allows IRV, which enhances alveolar inflation, recruitment and oxygenation, and decreases required FIO<sub>2</sub>.
- PCV and IRV evoke patient discomfort and heavy sedation is required. Coughing or "bucking" disrupt precise delivery of prolonged inspiratory time, resulting in desaturation. In severe cases, muscle relaxation may be necessary.
- Increased mean airway pressure impairs cardiac filling, although transmission of high airway pressures to the vasculature may be attenuated by lung stiffness.
- Inverse I:E ratio may result in inadequate expiratory time, air trapping, breath "stacking", and the development of auto-PEEP (intrinsic PEEP). This in turn results in hypercarbia and decreased effective PCV. It is much more likely to occur with high ventilatory rates. Paradoxically, hypercarbia may respond favorably to a slower ventilator rate, as long as the inspiratory time is not changed.
- Transport of patients dependent on high mean airway pressure requires the use of an AMBU fitted with a PEEP valve, and a ventilator capable of PCV in the OR or the radiology suite. Sudden release of mean airway pressure with standard AMBUs or anesthesia ventilators may be disastrous.

### ***Management of Hypoxemia with PCV***

The primary treatment goal is a PaO<sub>2</sub> > 60 mmHg (SpO<sub>2</sub> > 90%) at the lowest possible FIO<sub>2</sub>. A useful index is the PaO<sub>2</sub>: FIO<sub>2</sub> ratio (P to F ratio). Normal, > 300; acute lung injury, 300-200; ARDS <200.

PCV is initiated by selecting a pressure limit that keeps the peak airway pressure ≤ 40 cmH<sub>2</sub>O. Thereafter, mean airway pressure (normally < 10 cmH<sub>2</sub>O) is adjusted by progressively increasing inspiratory time (normally < 0.5 sec) which inverts the I:E ratio (normally < 1:2). In severe ARDS, this may require mean airway pressure > 25 cmH<sub>2</sub>O, inspiratory time > 2.0 sec and I:E ratio > 3:1.

During these manipulations ventilator rate must be kept constant. For example, if  $f$  is 10 breaths/min, and inspiratory time is 2.0 sec, I:E is 1:2. If  $f$  is doubled to 20 breaths/min, I:E is 1:1 even though inspiratory time has not been changed.

Lower tidal volumes (5 mL/kg) are acceptable, but must be closely monitored. A sudden decline in tidal volume and oxygen saturation may result from inadequate sedation or muscle relaxation, or from mucus plugging.

Although auto-PEEP may improve oxygenation by increasing mean airway pressure, it decreases effective PCV and results in lower tidal volumes and hypercarbia. It may be managed by decreasing extrinsic PEEP by the amount of auto-PEEP, by decreasing the ventilator rate to increase expiratory time, and/or by accepting low tidal volumes and hypercarbia (permissive hypercapnia).

### ***Pressure Assist-Control Ventilation (PACV)***

PACV facilitates lightening of sedation and onset of spontaneous breathing in patients who still have residual lung stiffness. Successful transition to PSV usually requires weaning of mean airway pressure  $\leq 15$  cmH<sub>2</sub>O and inspiratory time  $< 0.8$  sec.

### **POSITIVE END-EXPIRATORY PRESSURE (PEEP)**

PEEP can be applied to any of the modes of ventilation already described; when applied during spontaneous ventilation it is referred to CPAP.

The primary function of PEEP is to increase FRC by preventing collapse of patent alveoli ( $< 10$  cmH<sub>2</sub>O) and recruiting collapsed alveoli ( $> 10$  cmH<sub>2</sub>O). In pulmonary edema, PEEP redistributes (but does not decrease) EVLW, improves FRC, lung compliance and oxygen diffusion.

PEEP of 5 cmH<sub>2</sub>O is routinely used to maintain FRC during postoperative mechanical ventilation, to substitute for "physiologic" PEEP provided by glottic closure in the non-intubated patient. Moderate levels (6 - 10 cmH<sub>2</sub>O) are used to reverse atelectasis during postoperative mechanical ventilation. High levels ( $>10$  cmH<sub>2</sub>O) augment airway pressure therapy for the treatment of ARDS.

### ***Adverse Effects and Limitations of PEEP***

- PEEP directly increases peak, mean and baseline airway pressures and exacerbates the circulatory and barotrauma effects of mechanical ventilation.
- PEEP is directed to the more compliant (apical) lung zones, or, in the case of nonuniform lung disease, to areas of normal lung. Alveolar overdistension can increase intrapulmonary dead space, air trapping, carbon dioxide retention and hypercapnia. Patients with emphysema or acute bronchospasm are at highest risk.
- In spontaneously breathing patients PEEP may provide an intolerable increase in the work of breathing.

## **ADVERSE EFFECTS OF MECHANICAL VENTILATION**

### **VENTILATION-PERFUSION ( $V_A/Q$ ) MISMATCH**

Positive pressure ventilation invariably results in  $V_A/Q$  mismatch, increased AaDO<sub>2</sub> and higher required FiO<sub>2</sub>.

Gravity augments perfusion to dependent lung zones (Zones 2, 3), but gas delivered by positive pressure ventilation follows the path of least resistance, so that ventilation is greatest in the

non-dependent zones (Zone 1). During spontaneous breathing the diaphragm actively ventilates dependent lung zones, but with paralysis it is passively pushed cephalad by the abdominal viscera into the dependent lung zone, further impeding ventilation to that area.

### **CIRCULATORY IMPAIRMENT**

Increased intrathoracic pressure decreases venous return, cardiac output and RBF. Measured intravascular pressure (CVP, PAOP) and cardiac preload appear to increase, but the effective transmural pressure declines because of increased intrapleural pressure.

Circulatory effects are directly proportional to the elevation of intrathoracic pressure, and are exacerbated by hypovolemia. Fortunately, the adverse circulatory effects of mechanical ventilation are less in stiff, noncompliant lungs, which attenuate the transmission of intraalveolar pressure to the intravascular space.

PEEP > 10 cmH<sub>2</sub>O may compress the pulmonary capillary bed, increase RV afterload and pressure, shift the intraventricular septum and impede LV filling.

RBF declines because of the decrease in cardiac output, but also because of mechanoreceptor and baroreceptor responses resulting in activation of the sympathetic, renin-angiotensin-aldosterone system, and antidiuretic hormone. ANP release is impeded by decreased atrial transmural pressure. The net effect is renal vasoconstriction, decreased GFR and urine flow, and salt and water retention.

Airway pressure therapy impairs venous return from the head and may increase ICP and neurologic injury. Impairment of hepatic vein outflow may result in passive hepatic congestion.

### **Therapeutic Strategies**

- Choose a mode of ventilation that provides the lowest mean airway pressure while still sustaining adequate levels of oxygenation, e.g. wean high IMV rates if tolerated.
- Enhance cardiac and renal function by the use of preload augmentation, inotropic agents or vasodilator therapy (e.g. dopamine).
- In head injury, simple elevation of the head of the bed attenuates the effect of PEEP on ICP while allowing its beneficial effect on lung function.

### **BAROTRAUMA**

#### **Pathogenesis**

Barotrauma, physical damage induced by pressure applied in the airways and alveoli, includes epithelial injury, increased microvascular permeability, and inflammatory changes indistinguishable from ARDS.

The sequence of injury is rupture of the fragile alveolar epithelium, pulmonary interstitial emphysema (perivascular linear shadows on chest X-ray), pneumomediastinum, subcutaneous emphysema, and pneumoperitoneum (which may mimic visceral rupture). Pneumothorax occurs when the tougher visceral pleura finally tears, and tension may develop immediately.

Barotrauma is directly related to the severity of lung disease, and the magnitude of airway pressure (usually > 40 cmH<sub>2</sub>O). Lung disease predisposing to barotrauma includes ARDS, bullous emphysema, necrotizing pneumonia, neoplasm and radiation pneumonitis. Excessive airway pressure is induced by excessive tidal volume, high gas flow, bronchospasm, mucus plugging, coughing, bucking, high levels of PEEP and/or endobronchial intubation.

### **Prevention and Management**

No one method of ventilation has been proven superior in preventing barotrauma. Modes using high airway pressure therapy (PCV-IRV, high PEEP) have the greatest potential for barotrauma, PSV and IMV less (depending on the ratio of ventilator and patient effort), and CPAP least.

Barotrauma may be decreased by permissive hypercapnia, i.e. the use of low tidal volumes (5-7 mL/kg) and the acceptance of elevated PaCO<sub>2</sub> (60-110 mmHg) as long as pH is > 7.25. Excess CO<sub>2</sub> can be washed out by intratracheal oxygen insufflation, and increases in pulmonary artery pressure respond to inhaled nitric oxide.

In extreme cases heavy sedation and muscle relaxation may be required to provide control of airway pressures, and it may be justified to accept the increased risk of oxygen toxicity from a higher FIO<sub>2</sub> in order to limit further increases in airway pressure. Prophylactic thoracostomy tubes may induce more injury than they prevent.

**TABLE 1 ABBREVIATIONS**

AaDO <sub>2</sub>	alveolar-arterial oxygen gradient	IRV	inverse ratio ventilation
ACV	assist-control ventilation	f	frequency (ventilator or respiratory rate)
AMV	assisted mechanical ventilation	SIMV	synchronous intermittent mandatory ventilation
ANP	atrial natriuretic peptide	PACV	pressure assisted-controlled ventilation
APRV	airway pressure release ventilation	PAOP	pulmonary artery occlusion pressure
ARDS	adult respiratory distress syndrome	PEEP	positive end-expiratory pressure
CMV	controlled mechanical ventilation	PSV	pressure support ventilation
COPD	chronic obstructive pulmonary disease	PCV	pressure controlled ventilation
CPAP	continuous positive airway pressure	RBF	renal blood flow
CVP	central venous pressure	V <sub>A</sub> /Q	ventilation-perfusion ratio
EVLW	extravascular lung water	VAV	volume assisted ventilation
GFR	glomerular filtration rate	VACV	volume assisted-controlled ventilation
ICP	intracranial pressure	VCV	volume controlled ventilation
I:E ratio	inspiratory:expiratory ratio	V <sub>E</sub>	minute ventilation
IMV	intermittent mandatory ventilation	V <sub>T</sub>	tidal volume

**Table 2. Application of Breath Functions (Phase Variables)**

<b>Function</b>	<b>Mode</b>	<b>Primary parameter</b>	<b>Comment</b>
initiation	control	predetermined time interval	machine-triggered
	assist	threshold negative airway pressure	patient-triggered
limit	volume	delivery of preset tidal volume	airway pressure variable
	pressure	attainment of preset airway pressure	tidal volume variable
cycle off	volume	preset volume achieved	
	pressure	preset pressure achieved	
	time	predetermined time interval passed	inspiratory pause (zero flow)
	flow	decline to preset minimum flow rate	patient inspiratory flow pattern

The primary parameter is that which is set by the operator. It determines how each of the three functions (initiation, limit, cycle off) is expressed by the mode used.

**Table 3. Breath Types Defined by Machine vs. Patient Control**

<b>Breath Type</b>	<b>Phase Variable</b>		
	<b>Initiation (Trigger)</b>	<b>Limit (Target)</b>	<b>Cycle-off</b>
mandatory	machine	machine	machine
assisted	patient	machine	machine
supported	patient	machine	patient
spontaneous	patient	patient	patient

A *mandatory breath* is triggered, limited and cycled-off by the machine, which does all the ventilatory work.

An *assisted breath* is triggered by the patient, and limited and cycled-off by the machine. The patient does the work of initiating the breath only, while the ventilator does the rest.

A *supported breath* is triggered by the patient, limited by the ventilator, and cycled-off by the patient. The patient does the work of initiating the breath, and then interacts with the ventilator to perform a variable amount of the remaining work.

A *spontaneous breath* is triggered, limited and cycled-off by the patient, who does all the ventilatory work.

(Modified from the American Association for Respiratory Care Consensus Statement on the Essentials of Ventilators, 1992: Resp Care 37:1000, 1992)

**Table 4. Modes of Mechanical Ventilation**

<b>Ventilatory Mode</b>	<b>Breath Type</b>	<b>Initiation</b>	<b>Cycle Off</b>
<b><i>Volume Targeted</i></b>			
volume controlled ventilation (VCV)	mandatory	time	volume, time
volume assisted ventilation (VAV)	assisted	pressure	volume, time
volume assist-control ventilation (VACV)	assist/mandatory	pressure/time	volume, time
intermittent mandatory ventilation (IMV)	spont/mandatory	time	volume, time
synchronized IMV (SIMV)	spont/mandatory	pressure/time	volume, time
<b><i>Pressure Targeted</i></b>			
pressure support ventilation (PSV)	supported	pressure	flow
pressure control ventilation (PCV)	mandatory	time	time
pressure assist control ventilation (PACV)	assist/mandatory	pressure/time	time

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