



The activation of inspiration or a switch from expiration to inspiration is triggered by a trigger variable, also called a triggering mechanism. After successful triggering, inspiration needs to be sustained and limited by at least one of the preset limit variables and terminated by a predefined cycle variable, also known as cycling mechanism.<sup>1-4</sup>

## Trigger variables

A ventilator can be triggered by the machine itself, patient, or manual triggering. There are four common trigger variables: time, flow, pressure, and volume. With time triggering, mechanical inspiration occurs at a regular preset time interval, which is determined by the preset respiratory rate (RR).

A patient's inspiratory effort causes changes in pressure, flow, or volume within the ventilator circuit. When inspiration is initiated by a decrease in the baseline gas flow during exhalation, it is called flow triggering or flowby on some ventilators. If the ventilator is triggered to deliver inspiratory flow by a drop in baseline pressure resulting from a patient's inspiratory effort during exhalation, it is known as pressure triggering. During exhalation, a decrease in volume to a certain extent within the ventilator circuit activates inspiration. It is referred to as volume triggering. <sup>4,5</sup>

Other trigger variables such as flow waveform, chest-wall motion, transthoracic impedance, and diaphragm electrical activity are not available in most ventilators and seldom used. 1,6

Time triggering is also called machine triggering. Flow or pressure triggering are patient triggering and widely used. Flow triggering is claimed to reduce the work of breathing (WOB) compared with pressure triggering. With modern ventilators, these differences have minimal impact on a patient's condition. <sup>1,7,8</sup>

After selecting a trigger variable, the operator has to set up a sensitivity setting that determines the extent of change in flow or pressure, or the amount of a patient's effort that is required to trigger the ventilator. In order to reduce the WOB and avoid patient-ventilator dyssynchrony, it is vital to adjust sensitivity settings to meet the needs of individual patients.<sup>5,9</sup>

# **Limit variables**

Upon triggering a breath, inspiration needs to be sustained and limited by at least one of the following variables: volume, pressure, flow, or time. When the maximum tidal volume  $(V_T)$  that can be delivered to a

Phase variables					
Machine triggering: time					
Patient triggering: flow, pressure, volume					
Volume, pressure, flow, time					
Machine cycling: time, volume					
Patient cycling: flow, pressure					

patient during inspiration is preset, it is called volumecontrolled ventilation. If the maximum pressure rise during inspiration is predetermined, it is referred to as pressure-controlled ventilation. With pressure-support ventilation, inspiration is limited by the preset level of pressure support.

The flow limit involves setting the maximum flow that can be delivered by the ventilator. Setting a flow limit may cause patient-ventilator dyssynchrony if a patient's inspiratory flow demand exceeds the preset flow limit. Most modern ventilators can increase flow rate in response to increasing patient demands. Timelimited ventilation is not commonly used and may be seen in high-frequency ventilation.

A limit variable specifies the relevant parameter that is reached during inspiration. However, it isn't programmed to terminate inspiration. The termination of inspiration and initiation of expiration are controlled by a cycle variable.  $^{4,5,9,10}$ 

## **Cycle variables**

Inspiration can be terminated by a predefined pressure, flow, volume, or time variable. With pressure-cycled ventilation, inspiration is ended once the peak inspiration pressure (PIP) reaches the targeted pressure. In flow-cycled ventilation, the inspiratory flow ceases when its flow rate is dropped to the predetermined percentage of peak inspiratory flow rate.

Flow cycle criteria vary with different kinds of ventilators and are determined by a patient's clinical status. Appropriately set cycling threshold reduces autopositive end-expiratory pressure (auto-PEEP), also called intrinsic PEEP and the WOB.

With volume-cycled ventilation, inspiration is terminated once the preset  $V_{\rm T}$  is delivered. In time-cycled ventilation, the cycling between inspiration and expiration occurs at a regular preset time interval.

Both volume and time cycling are machine cycling.

Phase variables and ventilation modes							
	Volume control ventilation Pressure control ventilation		Pressure support ventilation				
Phase variables	Mandatory breaths	Assisted breaths	Mandatory breaths	Assisted breaths	Assisted breaths		
Trigger	Time	Pressure or flow	Time	Pressure or flow	Pressure or flow		
Limit	Volume	Volume	Pressure	Pressure	Pressure		
Cycle	Time	Time	Time	Time	Usually flow		

Both pressure and flow cycling are patient cycling. Flow cycling is commonly used in pressure support ventilation.  $^{1,2,4,8,9}$ 

# **Breath types**

With MV, there are mandatory and spontaneous breaths. The mandatory breath is triggered, limited, and terminated by the ventilator (time triggered and time-cycled). The spontaneous breath is initiated, controlled, and ended by the patient.

When the mandatory breath is triggered by the patient, it is called an assisted breath. If spontaneous breaths are assisted or supported by the ventilator such as the pressure support mode, they are also classified as assisted breaths.  $^{1,11}$ 

There are volume-controlled or pressure-controlled mandatory breaths. With volume-controlled ventilation, the inspiration of mandatory and assisted breaths is limited by the preset volume. Pressure is variable and affected by both anatomical and artificial airway resistance, chest wall, and lung compliance, as well as different flow patterns. For pressure-controlled ventilation, mandatory, and assisted breaths are limited by the predetermined inspiratory pressure. Volume and flow are variable and influenced by the preset pressure, inspiratory time, respiratory resistance, chest wall, and lung compliance. 11,12

During spontaneous breathing, the patient controls his own tidal volumes and terminates his own inspiration. Both volume and pressure are variable and depend on the patient's inspiratory effort, respiratory resistance and lung compliance. Spontaneous breaths can be supported by a preset pressure-supported or volume-supported ventilation to augment  $V_T$ . Pressure support is commonly prescribed for patients with spontaneous breaths to overcome the resistance created by the ventilator circuit, reduce the WOB, and improve a patient's comfort.  $^{9,13}$ 

With volume-controlled ventilation, the mandatory breath is time triggered, time cycled, and volume limited; the assisted breath is patient triggered, limited, and cycled by the same mechanisms as the mandatory breath. For pressure-controlled ventilation, the mandatory breath is time triggered, time cycled, and pressure limited; the assisted breath is patient triggered, limited, and cycled by the same variables as the mandatory breath. Pressure-support ventilation is patient triggered, pressure limited and usually flow cycled. (See "Phase variables and ventilation modes.")

# VILI and other complications

VILI includes barotrauma, volutrauma, atelectrauma, and biotrauma.

 $\bf Barotrauma$  is evidenced by extra-alveolar air. It is induced by high airway pressure or large  $V_T$  ventilation that damages the alveolar tissue and leads to alveolar rupture. Subcutaneous emphysema, pneumothorax, pneumomediastinum, and pneumoperitoneum are typical forms of barotrauma.  $^{14\text{-}17}$ 

 $\label{eq:VT} \textbf{Volutrauma} \ \text{is caused by large V}_{T} \ \text{ventilation that} \\ \text{leads to alveolar overdistension, which may manifest} \\ \text{itself as pulmonary edema.} ^{14,16,17}$ 

**Atelectrauma** occurs with repeated recruiting and derecruiting unstable alveoli during each breath cycle. Ventilation with low positive end-expiratory pressure (PEEP) predisposes to the development of atelectrauma in patients with heterogeneous damage of lung parenchyma such as acute respiratory distress syndrome (ARDS). <sup>14-16,18</sup>

**Biotrauma** results from the production, activation and release of both local and systemic inflammatory mediators secondary to lung parenchymal damage caused by volutrauma, barotrauma, and atelectrauma. Severe biotrauma may deteriorate to systemic inflammatory response syndrome (SIRS) and contribute to the development of multiple organ dysfunction syn-

drome (MODS).9,14,16,19

A thorough nursing assessment of patients requiring mechanical ventilation is crucial. MV has been associated with complications such as ventilator-associated pneumonia, cardiovascular compromise, gastrointestinal disturbances, and renal impairment. 3,9,19

For example, asymmetric chest movement and decreasing oxygen saturation may indicate pneumothorax. The development of subcutaneous emphysema or pneumothorax suggests barotrauma. Fever, productive cough, decreased breath sounds, and dull-

ness to percussion are signs of pneumonia. Oliguria may indicate decreased renal perfusion. Melena, pallor, and dizziness suggest upper gastrointestinal bleeding.

Critical care nurses assume an increasingly important role in early identification of complications and initiate appropriate measures to minimize adverse effects of MV.

# Improving oxygenation and ventilation

The primary goals of MV are to improve oxygenation and ventilation. The key means of improving oxygenation for ventilated patients are to increase fractional inspired oxygen ( ${\rm FIO}_2$ ) and apply PEEP. Ventilation can be altered by manipulating the parameter setting on the ventilator.

**Administering FIO**<sub>2</sub>. Managing patients with refractory hypoxia often requires administration of high FIO<sub>2</sub> to maintain adequate oxygenation. However, administering FIO<sub>2</sub> greater than 50% for a prolonged period may result in lung tissue injury, known as oxygen toxicity. It may manifest itself as pulmonary fibrosis or atelectasis. In order to reduce oxygen toxicity, FIO<sub>2</sub> should be kept below 50% when possible and the period of administration should be minimized.  $^{20\text{-}24}$ 

**Applying PEEP.** PEEP elevates mean airway pressure, improves gas distribution, and reduces the collapse of alveoli and small airways. It increases functional residual capacity, decreases pulmonary edema and shunting, and improves lung compliance. Ultimately, patient oxygenation is improved that allows clinicians to lower FIO<sub>2</sub>. In addition, PEEP increases alveolar recruitment and minimizes atelectrauma. On the other hand, PEEP raises intrathoracic pressure that reduces venous return



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and cardiac output (CO), and lowers blood pressure. Moreover, excessive PEEP may cause alveolar overdistension and produce VILI. 14,25,26 Low PEEP recruits inadequate recruitable alveoli and does not prevent recruited unstable alveoli from collapse. In addition, it necessitates an increase in FIO2 to maintain adequate oxygenation. When applying PEEP, a patient's lung condition, the degree of hypoxia and hemodynamic status should be considered. Adequate oxygenation is often achieved by the combined administration of FIO2 and PEEP. At times it is very difficult to achieve optimal

oxygenation, and consequently, suboptimal oxygenation may be accepted.  $^{15,19,22}$ 

Furthermore, oxygenation can be improved by inversion of inspiratory to expiratory (I:E) ratio, prone positioning, and lung recruitment maneuvers (RM). However, ventilation with inverse I:E ratio is very uncomfortable and often requires sedation and paralysis. The prone position is cumbersome and may lead to inadvertent extubation and the development of pressure ulcers. Performing RM may reduce venous return and CO, induce hypotension, and has a potential risk of barotrauma. <sup>16,27-29</sup>

**Improving ventilation.** The partial pressure of arterial carbon dioxide ( $PaCO_2$ ) and pH level in arterial blood gases (ABGs) are significantly affected by minute ventilation. Hypoventilation elevates  $PaCO_2$  and may lead to respiratory acidosis. Hyperventilation lowers the level of  $PaCO_2$  and may cause respiratory alkalosis.  $^{22,30}$ 

With volume-controlled ventilation, the settings of  $\rm V_T$  and RR determine minute ventilation. For pressure-controlled ventilation, the preset inspiratory pressure, RR, inspiratory time, respiratory resistance, and lung compliance primarily affect minute ventilation. Pressure support augments spontaneous  $\rm V_T$  and subsequently, minute ventilation. As a result, it is often prescribed to lower the level of  $\rm PaCO_2$  for spontaneously breathing patients.  $^{9,18}$  The level of pressure support is determined by the patient's clinical condition, minute ventilation,  $\rm PaCO_2$  and arterial pH.  $^{22,31,32}$ 

Ventilation with large tidal volumes can produce volutrauma. High RR will shorten expiratory time, increase auto-PEEP, and compromise CO. Elevating inspiratory

Strategies for improving ventilation					
Modes of ventilation	Strategies for improving ventilation (lowering PaCO <sub>2</sub> )				
Volume control	Increasing V <sub>T</sub> , RR or both				
Pressure control	Increasing inspiratory pressure, RR or prolonging inspiratory time, decreasing airway resistance (bronchodilators and airway suctioning)				
Pressure support	Increasing pressure support level				

pressure increases the risk of barotrauma. In order to reduce VILI and other complications, controlled hypoventilation and permissive hypercapnia are allowed in some critical settings, such as severe asthma, ALI, or ARDS. 33-35

## The nurse's role

Critical care nurses can identify subtle changes in a patient's clinical status and initiate appropriate nursing interventions rapidly and effectively. The main components of nursing care for mechanically ventilated patients include the following:

- 1. Performing frequent assessments including level of consciousness and vital signs.
- 2. Verifying prescribed ventilator settings and appropriate alarm limits. Nurses should also properly secure the endotracheal tube and respond to and troubleshoot ventilator alarms, adhere to infection control guidelines, and identify complications or mechanical problems associated with MV, such as an air leak or kink in the ventilator circuit.
- 3. Ensuring emergency equipment, such as manual resuscitation bags and oropharyngeal and nasopharyngeal airways, are immediately available.
- 4. Assessing the adequacy of cardiac output. MV compromises hemodynamic status and predisposes patients to hypotension and renal dysfunction. Maintaining adequate perfusion is paramount.
- 5. Evaluating the adequacy of oxygenation. Oxygen saturation and partial pressure of arterial oxygen ( $PaO_2$ ) are key indicators of oxygenation.
- 6. Assessing the adequacy of ventilation. It is essential to monitor the patient's  $PaO_2$ ,  $PaCO_2$ , and acid-base balance.
- 7. Monitoring the patient-ventilator interaction. Using

accessory muscles for breathing suggests increasing WOB. Common causes include increasing airway resistance as a result of bronchospasm, excessive sputum, or small ETT size.  $^{2,9,10,36}$ 

When the flow delivered by a ventilator does not meet patient needs, flow dyssynchrony occurs. The inspiratory flow rate is adjusted to decrease the WOB and alleviate patient discomfort. If the termination of the inspiratory flow by a ventilator does not synchronize with the end of a patient's neural inspiration, it results in cycle dyssynchrony. With pressure-support ventilation, optimizing flow cycle threshold will ameliorate cycle dyssynchrony.<sup>2,10,32,36</sup>

- Educating patients and their families (with the patient's consent) about the patient's illness, the need for respiratory support and the application of MV.
- Involving patients in the decision making regarding medical treatment and nursing care, and encouraging them to actively participate in the ventilator weaning process.

MV can cause numerous complications. A sound knowledge of MV and a patient's clinical status enables clinicians to fine-tune ventilator settings to maximize the benefits of ventilatory support while minimizing VILI. Critical care nurses play a crucial role in improving the effectiveness of MV, preventing harm, and optimizing patient outcomes.

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