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# Mechanical Ventilation and Its Utilization

[1]Arieza Putri Revina Octavia, [2]Dr. Dadang Prasetyo Jatmiko

[1]PhD Candidate at Harvard University
[2]Registered Financial Consultant at International Association of Registered Financial Consultants (IARFC)

***Abstract:*** **Knowing how mechanical ventilator functions, its normal working modes and the intricacy related with its utilization is a fundamental however basic expertise though escalated couldn't care less unit (ICU) clinicians. After mechanical ventilation was brought into clinical practice, there was a whirlwind of enthusiasm for creating more up to date methods of ventilation that would profit patients with respiratory disappointment. This sort of approach depended on an observation that mechanical ventilation is a kind of treatment for patients with expiratory disappointment. Be that as it may, there is nothing restorative about mechanical ventilation. Truth be told, the most noteworthy revelation about mechanical ventilation since it was first presented is the way that it harms the lungs and in a roundabout way harms different organs also. Mechanical ventilation is a system that restricts the typical physiology of ventilation by making positive weight rather than negative weight to ventilate the lungs and not amazing that it is dangerous. The present pattern of utilizing lower tidal volumes amid mechanical ventilation is a stage in the correct course in light of the fact that "a lesser is better" methodology is the special case that bode well with a system that is so non physiological. Since mechanical ventilation is a bolster measure and not a treatment methodology, nothing that is finished with a ventilator will favorably affect the result of the essential sickness. Then again, mechanical ventilation can negatively affect results by making antagonistic impacts. This implies the best method of mechanical ventilation is the one with least antagonistic impacts.**

***Keywords:*** **mechanical ventilation, positive pressure, lower tidal volume, adverse effect.**

## 1. INTRODUCTION

The description of positive pressure ventilation was first proposed by Vesalius 400 years ago, but the application of the concept in patient management began in 1955, when polio epidemics occurred almost all over the world. At that time, an assisted ventilation which might act as a negative pressure ventilator tank known as iron lung was required. In Sweden, all medical education centers were closed and all students worked for 8 hours a day as human ventilator which pumps lung in patients with ventilation disorders. Similarly, Boston, USA, Emerson Company successfully created a prototype of a positive pressure pulmonary inflation tool which was then used at Massachusetts General Hospital and delivered satisfactory results in a short period of time. Since then, a new era of positive mechanical pressure ventilation and medical science and intensive care began.

## 2. CONVENTIONAL MECHANICAL VENTILATION

The first positive pressure ventilator was found aiming to develop the lungs to achieve the preset pressure. This type of pressure-cycled ventilation is less favorable because the volume of inflation varies according to changes in mechanical properties in the lungs. In contrast, volume-cycled ventilation may develop the lungs into an initial predetermined volume and deliver a constant alveolar volume despite changes in the mechanical properties of the lungs, thus volume-cycled ventilation serves as a standard method of positive pressure mechanical ventilation.

**Inflation Pressure:**

The lungs are developed at constant flow rates and this results in a steady increase in lung volume. The pressure on the proximal airway shows a sudden initial increase followed by a gradual increase in subsequent lung inflation, but the pressure on the alveoli ($P_{ALV}$) shows a gradual increase during lung inflation.

Initially, the sudden increase in proximal airway pressure is a reflection of the airway resistance. An increase in airway resistance causes an increase in initial proximal airway pressure, while alveolar pressure at the end of lung development remains unchanged. As airway resistance increases, higher inflationary pressure is also required to distribute the volume of inflation, but the alveoli is not exposed to higher inflationary pressure. This is not a problem when lung compliance (distensibility) decreases. Under these conditions, there is an increase in proximal and alveolar airway pressures. Hence, as lung compliance decreases, higher inflation pressure is required to distribute the volume of inflation to the alveoli. Increased alveolar pressure in the affected lung may cause pressure-induced lung injury.

**Cardiac Performance:**

The effect of positive pressure ventilation on cardiac performance is quite complex and results in preload and afterload changes on both sides of the heart. To illustrate these changes, a review of the effect of intrathoracic pressure on transmural pressure is important because it determines ventricular filling (preload) and resistance to ventricular emptying (afterload).

**Mechanism of Thoracic Pump during Normal Spontaneous Breathing and Positive Pressure Ventilation:**

In the last few decades, it has been known that decreased cardiac output is the major complication of positive pressure ventilation. This phenomenon can be understood by comparing intrapleural (intrathoracic) pressure changes during normal spontaneous breathing to positive pressure ventilation.

During spontaneous ventilation, a decrease in intrathoracic pressure causes air to flow into the lungs and blood to the large thoracic vessels and heart. Thus, the right ventricular preload will increase as blood returns to the right heart increases as well as the right cardiac stretch and volume increase that the stroke volume of the right-sided heart also increases. The right ventricle receives less blood during spontaneous passive exhalation for at that time, the intrathoracic pressure is less negative. The volume of the left-sided heart tends to follow the state of the right-sided heart volume.

**Transmural Pressure:**

The mechanical effect of the lungs on the transmission of intrathoracic pressure is illustrated in Figure 2. The left figure shows what happens when the normal lungs are developed with a volume of 700 ml derived from a positive pressure source. In these circumstances, an increase in alveolar pressure is entirely transmitted to the pulmonary capillaries and does not cause a change in the transmural pressure ($P_{tm}$) along the capillary capillaries. However, when the same lung development develops in the non-compliance lung (right figure), an increase in alveolar pressure is not entirely transmitted to the capillaries and an increase in transmural pressure occurs which may lead to suppression of capillaries. Therefore, in circumstances where there is a decrease in lung compliance (pulmonary edema, pneumonia), a lung development with positive pressure may cause suppression of the heart and intrathoracic blood vessels. This emphasis can bring about the advantages or disadvantages as illustrated below.

**Preload:**

Lung development with positive pressure can cause ventricular filling to decrease through several mechanisms. First, the intrathoracic positive pressure decreases the venous pressure gradient to the thorax, although positive pressure lung inflation may result in increased intraabdominal pressure and tends to maintain venous flow to the thorax. Second, positive pressure urges the outer surface of the heart to reduce the distensibility of the heart so that it can reduce ventricular filling during the diastole phase. In general, suppression of the pulmonary vessels may increase pulmonary vascular resistance and this may interfere with right ventricular output stroke. In these circumstances, the right ventricle dilates and presses the septum toward the left ventricle, causing a reduction in the size of the ventricular chamber and the filling of the left ventricle. This phenomenon is known as ventricular interdependence which is a mechanism of the left heart failure due to the right heart failure.

**Afterload:**

Disorders of ventricular filling during the diastolic phase due to heart suppression resulting from intrathoracic positive pressure also occur at the time of ventricular emptying during the systolic phase. This may be likened to the pressure of the hand pressing the ventricle during the systolic phase and this is termed as ventricular afterload. Thus, ventricular afterload or ventricular emptying impedance is a function of the peak pressure of the transmural wall during the systolic phase. Transmission of incomplete intrathoracic pressure into the ventricular chamber will reduce the transmural pressure through the ventricle during the systolic phase, which in turn reduce the ventricular afterload.[1,3,6]

**Cardiac Output:**

Positive pressure lung inflation tends to reduce ventricular filling during the diastolic phase, but this also causes an increase in ventricular emptying during the systolic phase. Overall, the effect of positive pressure ventilation on cardiac output depends on the more prominent effect between preload or afterload. When normal intravascular volume and intrathoracic pressure are not too large, the effects of decreased afterload become more prominent and positive pressure ventilation increases cardiac stroke output. Increased stroke volume increases systolic blood pressure during lung development, a phenomenon known as reverse pulsus paradoxus. In these circumstances, it can explain the beneficial effects of chest compression to increase cardiac output at the cardiac arrest.[1,3]

The opposite occurs in hypovolemia. When intravascular volume is reduced, the more prominent effect of intrathoracic positive pressure is the decrease in ventricular preload. In these circumstances, positive pressure ventilation lowers cardiac stroke output. Therefore, it emphasizes the importance of efforts to avoid hypovolemia in the management of patients with dependent ventilator.

## 3. THE EFFECT OF MECHANICAL VENTILATION ON ORGAN SYSTEMS

- Intracranial Pressure and Cerebral Perfusion:

The amount of blood flowing to the brain is determined by the cerebral perfusion pressure (CPP). CPP is the result of a reduction of the mean systemic arterial blood pressure (MABP) with intracranial pressure (ICP). Cerebral perfusion pressure may potentially decrease because positive pressure ventilation with or without positive end-expiratory pressure (PEEP) can decrease cardiac output and MABP. For example, if MABP decreases from 100 to 70 mmHg and ICP 15 mmHg, CPP will decrease from 85 mmHg (100 – 15 = 85) to 55 mmHg (70 – 15 = 55).[3]

Positive pressure ventilation can increase central venous pressure (CVP) so that the venous return from the head will decrease causing an increase in ICP and decrease CPP. This can be known clinically in the presence of increased distention of the jugular vein. Therefore, decreased cerebral perfusion may cause cerebral hypoxemia and increased ICP may increase cerebral edema.

The greatest clinical risk associated with cerebral perfusion is in patients with high ICP with increased cerebral edema. Patients with closed head injury, cerebral or post-neurosurgical tumors fall into this category. If the patient has a normal intracranial hemodynamic condition, positive pressure ventilation will not increase intracranial pressure (ICP). In patients with abnormal cerebral function, changes that occur in perfusion and cerebral pressure will greatly affect hemodynamic conditions. If there is an increase in ICP, there will be hyperventilation to decrease ICP by reducing $PaCO_2$ to 25 – 30 mmHg. Alkalosis caused by low $PaCO_2$ can cause vasoconstriction of blood vessels. Theoretically, it may decrease ICP and increase cerebral perfusion, but lasts only 24 to 36 hours. Therefore, controversy about the benefit and usefulness of the theory still exists and differs from its application in various institutions.

Some patients with head injury or cerebral dysfunction require PEEP to treat refractory hypoxemia caused by increased shunting of functional residual capacity (FRC). PEEP may increase ICP, but PEEP is needed to maintain oxygenation so it is lifesaving. Therefore, in these patients, it is necessary to monitor ICP.

- **Renal Function:**

The effect of positive pressure ventilation on renal function has been known since 4 decades ago. This change occurs in 3 areas, namely:

- Renal response to hemodynamic changes arising from the increased intrathoracic pressure:

Reduced cardiac output due to alveolar positive pressure tends to decrease renal blood flow (RBF) and glomerular filtration rate (GFR) decreasing urine production. The decreased urine production is not solely because of a decrease in cardiac output as the return of cardiac output to an adequate value is not always accompanied by an increase in the urine production in parallel. When the kidneys are not affected by neural and humoral factors, urine production remains constant at arterial pressures with a fairly wide range. When the glomerular capillary pressure decreases below 75 mmHg, the glomerular flow rate decreases and the urine flow decreases. In severe hypotension, the urine flow can be stopped.[3]

When using positive pressure ventilation, arterial blood pressure is usually compensated. Decrease in pressure is not a factor causing a significant decrease in urine production during mechanical ventilation. Redistribution of blood in the kidney affects changes in renal function itself. The flow to the outer cortex decreases, while the flow to the inner cortex and the juxtaglomerular nephrons is increased so that less urine, creatinine and sodium are excreted. This occurs because

the juxtaglomerular nephrons near the renal medulla are more efficient to absorb sodium than those in the outer cortex so that more sodium are absorbed followed by increased water absorption. Redistribution of blood is a response to sympathetic stimulation such as increased catecholamines, vasopressin, and angiotensin. Possible explanations of this effect are related to changes in renal venous pressure due to inferior vena cava vasoconstriction, changes in blood pressure of the inferior vena cava or congestive heart failure.

- Humoral responses include changes in antidiuretic hormone (ADH), atrial natriuretic peptide (ANP) and renin-angiotensinaldosterone (RAA)

Urine production during positive pressure ventilation will decrease. This is due to changes in perfusion and endocrine function. Increased release of antidiuretic hormone (ADH) from the posterior pituitary may decrease urine production. As the name implies, ADH inhibits water excretion. The higher the ADH is released into the circulation, the less urine formation resulting in the greater the volume of fluid in the body.[3,6]

The main determining factor of ADH release is plasma osmolality. Other factors include blood pressure, nausea, vomitus, and various drugs such as narcotics and nonsteroidal anti-inflammatory drugs. Changes in blood pressure caused by positive pressure ventilation may increase ADH release by the following mechanism, ie, the volume receptor present in the left atrium sends nerve impulses through the vagal path to the hypothalamus. This neural activity can stimulate increased or decreased ADH production and secretion. Baroreceptors present in the carotid body and along the aortic arch make sense of pressure changes and may increase or decrease ADH levels. At the time of giving positive pressure ventilation, these receptors are exposed to changes in intrathoracic pressure, volume and blood pressure. It is known that negative pressure ventilation inhibits the release of ADH and causes a diuretic effect, conversely positive pressure ventilation increases ADH release resulting in oliguria.

Positive pressure ventilation and PEEP decrease atrial filling pressure with mechanical compression in the atria by decreasing right atrial stretch due to decreased venous return. A decrease in atrial stretch causes a decrease in the production of other hormones, ie atrial natriuretic peptide (ANP). ANP serves to maintain fluid and electrolyte balance. Decreased levels of ANP play a role in water and sodium retention during positive pressure ventilation.[3,6]

The stimulation of sympathetic nervous system leads to an increase in plasma renin activity (PRA) and is one of the factors affecting water and sodium retention during positive pressure ventilation and PEEP. The increase in PRA activates the RAA cascade resulting in sodium and water retention. Synthesized prostaglandins in the kidneys tend to compensate for these effects, but they are not sufficient to cope with those effects thoroughly.

- The Effects on the kidney due to abnormal pH, $PaCO_2$ and $PaO_2$

Changes in $PaO_2$ and $PCO_2$ are the effects of ventilation on the kidneys. The decrease in $PaO_2$ in patients with respiratory failure indicates a decline in urine production and renal function. $PaO_2$ below 40 mmHg (severe hypoxemia) leads to decreased renal function. Similarly, $PaCO_2$ above 65 mmHg may also decrease renal function.

- The Effects of Mechanical Ventilation on Hepar and Gastrointestinal Function

Patients receiving positive pressure ventilation and PEEP show hepatic dysfunction characterized by an increase in serum bilirubin greater than 2.5 mg/100 ml without prior history of hepatitis disease. This is due to decreased cardiac output, downward diaphragm movement opposite to the hepar, decreased portal venous flow or increased splanchnic resistance resulting in ischemia in hepatic tissue as well as other factors that interfere with hepatic function.

Positive pressure ventilation increases splanchnic resistance, decreases splanchnic vein flow and plays a role in triggering gastric mucosal ischemia. Ischemia is what ultimately often increases the incidence of gastrointestinal bleeding and gastric ulcer that occur frequently in critically ill patients. This occurs due to increased permeability of the gastric mucosal barrier. Therefore, these patients are given antacids or cimetidine to prevent gastrointestinal bleeding due to acute stress ulceration. These drugs increase the gastric pH associated with an increased risk of nosocomial pneumonia in ventilated patients. In these circumstances, oral sucralfate which can overcome gastrointestinal bleeding without altering pH can be administered.

Patients who receive positive pressure ventilation are also at risk for severe gastric distension by swallowing air leaking around the endotracheal tube or when this positive pressure ventilation is administered through the mask. The installation of a nasogastric tube can dispose of inflow air and decompress the gastric.

## 4. MECHANICAL VENTILATION INDICATION

The act of intubation and initiation of mechanical ventilation are a complicated thing to decide upon. Before doing so, there are some rules that must be well understood, among others:

- Indications of intubation and mechanical ventilation should be considered well. There is a tendency to delay intubation and mechanical ventilation as much as possible in the hope that it is not necessary. However, planned intubation is less dangerous than emergency intubation. In addition, delaying intubation can cause harm to patients who may otherwise be avoided. If the patient's condition is considered severe enough and requires immediate intubation and mechanical ventilation, do not delay to perform the action.

- Intubation is not an act performed by an incompetent person. Nurses tend to apologize for having done intubation when they are on duty of night watch, as if the act is something they cannot do. On the contrary, intubation should be done with a strong principle and no one is blamed for taking an airway control on an unstable patient.

- The act of initiating mechanical ventilation is not a "death gate." The notion that once we use a ventilator will forever depend on the ventilator is incorrect. It should not affect our decision to initiate mechanical ventilation. The use of ventilator does not cause a person to become dependent, except in patients with severe cardiopulmonary disease and neuromuscular disorders.

**Mechanical Ventilation Settings:**

The parameters to be set vary widely depending on the mode of ventilation used. Some of these parameters include:

- Respiratory rate

The respiratory rate used in the mandatory ventilator range widely. This depends on the target value of minute ventilation that varies in each individual and certain clinical conditions. In general, the respiratory rate ranges from 4 to 20 times per minute and in most stable patients, it ranges from 8 to 12 times per minute. In adult patients with acute respiratory distress syndrome, the use of low tidal volume should be balanced by an increase in respiratory rates up to 35 times per minute to maintain adequate minute ventilation.

- Tidal volume

In some cases, tidal volume should be lower especially in acute respiratory distress syndrome. When adjusting tidal volume in certain modes, the approximate roughly ranges from 5 to 8 ml/kg of ideal body weight. In patients with normal intubated lung for some reasons, tidal volume is used up to 12 ml/kg of ideal body weight. The tidal volume should be adjusted so as to maintain plateau pressure below 35 $cmH_2O$. Plateau pressure is determined by a breath-holding maneuver during inspiratory called alveolar pressure of end-inspiratory in patients who are relaxed.

Increased plateau pressure does not necessarily increase the risk of barotrauma. The risk is determined by the transalveolar pressure which results from a reduction between alveolar pressure and pleural pressure. In patients with chest wall edema, abdominal or ascites distension, chest wall compliance decreases. This causes increased pleural pressure during lung development. Increased transalveolar pressure is rare in patients who have normal lung compliance.

- Inspiratory pressure

In pressure-controlled ventilation (PCV) and pressure-supported ventilation, the inspiratory pressure is adjusted so that plateau pressure is less than or equal to 35 $cmH_2O$. The tidal volume should also be maintained in the predetermined range.

- Fraction of inspired oxygen ($FiO_2$)

In most cases, $FiO_2$ should be 100% when the patient is intubated and connected to the ventilator for the first time. When placement of the endotracheal tube has been established and the patient has been stabilized, $FiO_2$ should be lowered to the lowest concentration that can still sustain oxygen saturation of hemoglobin, because high oxygen concentration can cause pulmonary toxicity. The main purpose of ventilation is to maintain a saturation value of 90% or more. Sometimes the value may change, for example in circumstances requiring a protection against the lungs from too large tidal volume, oxygen pressure and concentration. In this situation, the oxygen saturation target can be lowered to 85% when the factors contributing to oxygen delivery are being optimized.

- Positive end-expiratory pressure (PEEP)

As the name implies, PEEP serves to maintain positive airway pressure at some levels during the expiratory phase. PEEP is distinguished from the continuous positive airway pressure (CPAP) based on when it is used. PEEP is only used in the expiratory phase, while CPAP takes place during the respiratory cycle.[7]

The use of PEEP during mechanical ventilation has potential benefits. In acute hypoxaemia respiratory failure, PEEP increases the mean alveolar pressure, increases the area of atelectasis re-expansion and can push fluid from the alveolar space to the interstitial thus allowing previously covered or submerged alveoli to participate in gas exchange. In cardiopulmonary edema, PEEP can reduce left ventricular preload and afterload, thus improving cardiac performance.[7]

In hypercapneal respiratory failure caused by airway obstruction, patients often experience lack of time for expiration resulting in dynamic hyperinflation. This leads to auto-PEEP, ie alveolar expiratory pressure is higher than atmospheric pressure. When auto-PEEP is obtained, it requires trigger ventilator in the form of a higher negative pressure of the airway than trigger sensitivity or auto-PEEP. If the patient is not able to achieve it, the inspiratory effort becomes futile and can improve the work of breathing. Administration of PEEP can overcome this because it can reduce auto-PEEP from the total negative pressure required to trigger the ventilator. In general, PEEP is increased gradually until the patient's breathing effort can constantly trigger the ventilator up to 85% of the predicted auto-PEEP.

- Trigger sensitivity

The trigger sensitivity is the negative pressure that the patient must initiate to initiate a respiratory aid by the ventilator. This pressure should be low enough to reduce the work of breathing, but should also be high enough to avoid excessive sensitivity to the patient's breathing effort. This pressure ranges from -1 to -2 $cmH_2O$. This trigger ventilator arises when the patient's breathing flow decreases 1 to 3 l/min.

- Flow rate

This is often overlooked in volume-targeted mode. This flow rate is particularly important for patient comfort as it affects the work of breathing, dynamic hyperinflation and auto-PEEP. In most ventilators, the flow rate is regulated directly. In other ventilators, eg Siemen 900 cc, the flow rate is determined indirectly from the respiratory rate and I:E ratio.

**Examples are as follows:**

| | |
|---|---|
| Respiratory rate | = 10 |
| Respiratory cycle time | = 6 seconds |
| I:E ratio | = 1:2 |
| Inspiratory time | = 2 seconds |
| Expiratory time | = 4 seconds |
| Tidal volume | = 500 ml |
| Flow rate | = volume/inspiratory time |
| | = 500 ml every 2 seconds |

- Comparison of inspiratory time to expiratory time

In line with the inspiratory flow rate, the respiratory therapist regulates the I:E ratio without the physician's request. However, clinicians are required to understand this change that may affect the mechanics of the respiratory system and patient comfort. I:E ratio commonly used is 1: 2. In acute hypoxaemia respiratory failure, this ratio may increase with the elongation of inspiratory time, mean airway pressure or fluid-filled alveoli that can improve oxygenation. In severe hypoxemia, I:E ratio is sometimes reversed to 2:1, so that caution must be maintained to overcome adverse effects on hemodynamics and lung integrity.

## 5. NEW STRATEGIES FOR MANAGEMENT OF MECHANICAL VENTILATION

In the early use of positive pressure mechanical ventilation, it is recommended to provide a large inflation volume to avoid the occurrence of alveolar collapse. In normal spontaneous breathing, the required tidal volume is 5 – 7 ml/kg (ideal

body weight), while the standard inflation volume during volume-cycled ventilation is 2 times the tidal volume value, namely 10 – 15 ml/kg. This volume incompatibility is even magnified by the addition of mechanical sigh 1.5 to 2 times larger than standard inflation volume (15 – 30 ml/kg) and is distributed 6 to 12 times per hour.[1]

The use of large inflation volume in conventional mechanical ventilation can cause damage to the lungs and can cause injury to other organs through the mechanism of inflammatory cytokine release. The discovery of ventilator-induced lung injury changes the way of ventilation administration drastically.

**Ventilator-Induced Lung Injury:**

In pulmonary diseases most of which require mechanical ventilation such as acute respiratory distress syndrome (ARDS) and pneumonia, pathological changes occur uniformly throughout the lungs. Even if lung condition problems such as ARDS that are on chest X-rays appear uniformly homogeneous throughout the lungs. The inflation volume tends to spread in the normal area of the lung because the inflation volume can only be distributed in area of the lungs that have normal function. This tendency arises especially if the administered inflation volume is too large.

Hyperinflation in the normal lung area during mechanical ventilation may cause stress fracture of alveolar capillary interface. This arises because of the excessive alveolar pressure (barotrauma) or excessive alveolar volume (volutrauma). Alveolar rupture causes adverse effects such as accumulation of alveolar gas in the pulmonary parenchyma that may cause interstitial pulmonary emphysema in mediastinum leading to pneumomediastinum and in the pleural cavity causing pneumothorax. Another consequence is inflammatory lung injury that is difficult to distinguish from ARDS, in addition to the possibility of multi-organ injury due to the release of inflammatory mediators into the bloodstream known as biotrauma.

**Lung-Protective Ventilation:**

The risk of lung injury caused by large inflation volume has led to clinical studies evaluating lower tidal volume in positive pressure ventilation. The largest study was conducted on 800 patients with ARDS (acute respiratory distress syndrome) and compared ventilation with tidal volume of 6 ml/kg with 12 ml/kg using predicted weight (body weight with normal lung volume). Ventilation with low tidal volume is associated with an absolute decrease of the mortality rate, ie 9% at end-inspiratory plateau pressure below 30 mmHg.

Low volume ventilation or current lung protective ventilation has been recommended for all patients with ARDS, but there is also evidence that ventilator-induced lung injury also occurs in other cases. Therefore, lung protective lung with low tidal volume is considered as a useful strategy for all patients with acute respiratory failure. This procedure is designed to achieve and maintain a 6 ml/kg tidal volume (using predicted weight).

**Ventilation Mode:**

Ventilation mode is a concise term to describe how the ventilator works in certain situations. This term is found by physicians, therapists, or ventilator manufacturers who develop different types of ventilation. Mode is a special setting of control variables and stages. In other words, we can describe the mode with pressure waveforms, flow and volume obtained from the type of ventilation mode applied to the patients.

**Table 1: Procedures for Lung-Protective Ventilation**

| |
|---|
| • Select the assist-control mode and $FiO_2$ 100%<br>• Set the initial tidal volume ($V_T$) 8 ml/kg using predicted body weight (PBW)<br>Male : PBW = 50+[2.3X(body heigh in inch – 60)]<br>Female : PBW = 45.5+[23X(body heigh in inch – 60)]<br>• Select the respiratory rate (RR) to reach the pre ventilator minute ventilation (MV), but do not exceed RR = 35x/min<br>• Add PEEP 5 – 7 $cmH_2O$ |
| • Reduce $V_T$ by 1 ml/kg every 2 hours until $V_T$ 6 ml/kg<br>• Adjust $FiO_2$ and PEEP to maintain $PaO_2$> 55 mmHg or $SaO_2$> 88% |
| • When $V_T$ decreases to 6 ml/kg, measure: |

- Plateau pressure
- $PCO_2$ and arterial pH
- If Ppl > 30 $cmH_2O$ or pH < 7.30, follow the recommended low volume ventilation procedure on ARDS

Historically, the trigger mechanism is often referred to as the term 'mode'. Control mode (time trigger), assist mode (pressure trigger) and assist/control mode (time and pressure trigger) are the most common mode used to trigger the ventilator during inspiratory. Thereafter, other ventilation modes such as IMV (intermittent mandatory ventilation), SIMV (synchronize intermittent mandatory ventilation), PEEP (positive end-expiratory pressure), CPAP (continuous positive airway pressure), pressure control, PS (pressure support), and APRV (airway pressure release ventilation) are developed.

- Full and Partial Ventilatory Support

Full Ventilatory Support (FVS) and Partial Ventilatory Support (PVS) are terms to describe the degree of mechanical ventilation provided. FVS consists of two components: the ventilator provides all the energy needed to maintain effective alveolar ventilation and this FVS only occurs when the ventilator breathing rate is 8 or more and the tidal volume is between 8 – 12 ml/kg of ideal body weight, since this ventilation setting can causing $PaCO_2$ less than 45 mmHg. In PVS, ventilator breathing rate and tidal volume are given less than FVS, so that the patients participate in the work of breathing (WOB) to maintain effective alveolar ventilation.

FVS is generally provided by assist-control mode as well as volume ventilation or pressure ventilation. The mode should be set in such a way that the patient receives adequate alveolar ventilation without taking into account whether the patient can breathe spontaneously or not. In PVS, any ventilation mode can be used, but patients can actively participate in maintaining adequate $PaCO_2$.

In acute respiratory failure, the initial purpose of ventilation is immediate respiratory aid to provide rest periods for the respiratory muscles. After several hours to several days, it is expected that the patient's condition has stabilized and begins to recover. If ventilation mode is maintained, there will be weakness of the muscles or atrophy so some clinicians do not recommend the use of FVS and prefer PVS from the beginning. However, FVS is still needed to avoid atrophy of the respiratory muscles.

- **Controlled Mechanical Ventilation:**

Control mode is a time trigger. All breathing, either in the form of volume or pressure breathing, are all mandatory. Patients cannot trigger their own breathing. In some ventilators, the difference between control and assist/control is only in the sensitivity setting. Controlled ventilation (time-triggered inspiratory) can only be applied to patients who do not have their own breathing effort or when the ventilation is administered, the patient must be controlled throughoutly. However it is not advisable to maintain this ventilation mode without having the patient have his own breathing effort. Controlled ventilation is suitable for patients who are unconscious due to drug effects, cerebral function disorders, spinal and phrenic nerve injuries and patients with motor neuron palsy leading to loss of voluntary respiratory effort.

- **Assist-Control Ventilation:**

Assist-control ventilation is a ventilation with time trigger setting or patients with minimum respiratory rate, sensitivity and respiratory type. Patients may trigger their breathing at a faster rate but a preset volume or fixed pressure is applied to each breath.

When there is a patient breathing effort, an assist-control mode may be used. With this mode, each breath (time or patient trigger) is regulated breathing. The trigger of the patient arises because the ventilator is sensitive to pressure or flow changes as the patient attempts to breathe. When there is a mild negative pressure (-1 $cmH_2O$) or a decrease of flow (2 – 3 l/min under the expiratory bias flow), the inspiratory cycle begins. The minimum respiratory rate should be adjusted to the ventilator to ensure expiratory volume. If desired, patient may be given additional breath.

Previously, assist-control ventilation was assumed to resemble a work of breathing, but it is now known that patients can perform inspiratory work of 33 – 50% or more. This occurs especially when there is active inspiratory and the gas flow does not correspond the inspiratory flow required by the patient. Clinically. If the pressure does not increase smoothly and quickly to reach the top, the flow is inadequate An overview of the concave-shaped pressure curve suggests active inspiratory. The flow should be increased until the patient's needs are met and the curve shows a slightly convex-shaped.

Another problem with assist-control ventilation is sensitivity. When the machine is too sensitive to the patient's breathing effort, the machine can be easily triggered (auto triggering) without flowing volume or pressure. This can be corrected by making the machine less sensitive to the patient's breathing effort. Conversely, if the inspiratory effort shows a pressure of -3 $cmH_2O$ to the readings in the manometer, the machine is less sensitive to the patient's breathing effort, therefore, the sensitivity should be increased. Without the use of muscle relaxants or respiratory depressants, it is difficult to avoid the occurrence of respiratory alkalosis. $PCO_2$ can reach the limit of apnea (32 mmHg) in some patients.

- **Intermittent Mandatory Ventilation:**

Problems related to lung emptying that are not entirely in assist-control ventilation have led to the development of a ventilation mode known as intermittent mandatory ventilation (IMV) introduced for the first time in 1971. At that time, this mode was used to provide ventilation in neonates with respiratory distress syndrome typically characterized by respiratory frequency above 40 times/min. IMV is designed to provide partial ventilation assistance. This mode combines an assist-control venting period with a patient's spontaneous breathing period. This spontaneous breathing period may help to prevent pulmonary hyperinflation and auto PEEP in patients with rapid breathing. In addition, the purpose of using this ventilation is to prevent atrophy of the respiratory muscles due to long-term mechanical ventilation. Disadvantage of this IMV is the increase in work of breathing and decreased cardiac output.

- **Pressure-Controlled Ventilation:**

Pressure-controlled ventilation (PCV) uses constant pressure to develop the lungs. Such ventilation is less preferred because the lung development volume is not the same, but it is still used because the risk of lung injury induced by the ventilator is lower in this mode. Ventilation with PCV as a whole is regulated by the ventilator without patient participation (similar to assist-control ventilation).

- **Pressure-Support Ventilation:**

Breathing with reinforced pressure allowing the patient to determine the volume of inflation and the duration of the respiratory cycle is called pressure-support ventilation (PSV). This method is used to strengthen spontaneous breathing, not to provide overall breathing assistance. In addition, PSV can overcome respiratory resistance through the ventilator circuit aiming to reduce work of breathing during the weaning of the ventilator. The purpose of PSV is not to strengthen the tidal volume, but to provide sufficient pressure to overcome the resistance generated by the endotracheal tube and the ventilator circuit. Inflation pressure between 5 to 10 $cmH_2O$ is good enough for this purpose. PSV is quite popular as one of the non-invasive mechanical ventilation methods. For non-invasive ventilation, PSV is provided through a face mask or special nasal mask with pressure 20 $cmH_2O$.

- **Positive End-Expiratory Pressure (PEEP):**

In patients with ventilator-dependent, at the end of breathing, there is generally a distal air space collapse resulting in frequent atelectasis that may interfere with gas exchange and exacerbate respiratory failure. Attempt to overcome the atelectasis is decreasing lung compliance with the consequences of common lung abnormalities in ventilator-dependent patients, eg ARDS and pneumonia. To anticipate the tendency for alveoli collapse at the end of breathing, a positive end-expiratory pressure (PEEP) is made. This pressure acts as a stent to keep the small airway open at the end of expiration. This PEEP has become a standard measure in the management of patients with ventilator-dependent.

PEEP is not recommended in patients with localized lung disease such as pneumonia because the suppressed pressure can be distributed to normal lung areas and this can lead to excessive distention causing alveoli rupture.

- **Continuous Positive Airway Pressure (CPAP):**

Spontaneous breathing with positive pressure maintained during the respiratory cycle is called continuous positive airway pressure (CPAP). In this ventilation mode, the patient does not need to produce negative pressure to receive the inhaled gas. This is made possible by a special inhalation valve that opens when the air pressure is above atmospheric pressure. CPAP should be distinguished by spontaneous PEEP. In spontaneous PEEP, negative airway pressure is required for inhalation. Spontaneous PEEP has been replaced by CPAP because it can decrease work of breathing.

The clinical use of CPAP is in non-intubated patients. CPAP can be supplied through a special face mask equipped with a pressure regulating valve. CPAP face mask has been proven successful in delaying intubation in patients with acute respiratory failure, but this mask should be fitted properly and strongly and cannot be removed when the patient is eating, so that it can only be used temporarily. Special nasal mask is more tolerable by patients especially in patients with obstructive sleep apnea, as well as in patients with acute exacerbation of chronic obstructive pulmonary disease.

## 6. MECHANICAL VENTILATION COMPLICATIONS

There are several complications of mechanical ventilation, among others: 1). Risks associated with endotracheal intubation, including difficult intubation, blockage of endotracheal tubes by secretions. 2). Long-term endotracheal intubation can cause damage to the larynx, especially the vocal cords and trachea. Generally after 14 days of tracheostomy, however, some institutions currently perform percutaneus tracheostomy early. 3). Ventilation gas can cause the effects of airway drain and secretion retention and disrupt coughing process so that it can cause lung infection. 4). Problems associated with the administration of sedation and anesthesia that have the effects of cardiac depression, delayed gastric emptying, decreased mobility and prolonged the recovery process. 5). Hemodynamic disorders primarily in the use of IPPV and PEEP can reduce venous return, cardiac output and blood pressure thus reducing blood flow to the gastrointestinal tract and kidneys. 6). Barotrauma and volutrauma

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