

## Modern Concepts Artificial Ventilation of the Lungs: Literature Review

*Ojum S., Dr. Nkadam Nwizor Monday*

*Department of Anaesthesia, Rivers State University Teaching Hospital, Port Harcourt, Nigeria*

*Dr. Daniel U, Elem – Ojum*

*Sacred Heart Hospital, Abeokuta*

**Annotation:** This study examines the modern concepts of artificial ventilation of the lungs. Recognizing AV as a life-sustaining intervention rather than a curative therapy, the paper highlights its role in supporting patients with acute or chronic respiratory insufficiency, providing time for recovery or treatment of underlying conditions. The review underscores the significance of optimizing ventilation parameters—such as tidal volume, respiratory rate, minute ventilation, and fraction of inspired oxygen, positive end-expiratory pressure, and airway pressure to ensure adequate gas exchange, minimize ventilator-induced lung injury, and maintain hemodynamic stability. Drawing on contemporary literature, the study explores how different ventilation modes including volume-controlled, pressure-controlled, assist-control, synchronized intermittent mandatory ventilation, pressure support, neurally adjusted ventilator assist, high-frequency oscillatory ventilation, high-frequency percussive ventilation, and airway pressure release ventilation—affect patient outcomes, comfort, and readiness for weaning. The paper argues that effective application of mechanical ventilation requires individualized strategies that account for patient-specific respiratory mechanics, underlying pathology, and tolerance to positive-pressure support. Furthermore, it emphasizes the critical role of continuous monitoring, including arterial blood gas analysis, waveform interpretation, and assessment of lung compliance and airway pressures, in guiding safe and effective ventilation. The study concludes that the impact of mechanical ventilation is intended to make readily available tool that simplify mechanical ventilation discussion the problems arising during prolonged mechanical ventilation, and how they are resolved. One of the recommendations made was that clinicians should tailor ventilator settings and modes based on the patient's underlying condition, lung mechanics, and response to treatment rather than relying on a single standard mode.

**Keywords:** modern, concepts, artificial ventilation, lungs.

### Introduction.

Artificial ventilation of the lungs (mechanical ventilation) is a life-saving procedure, which is often performed in patients with insufficiency of external respiration function, requiring respiratory support. The most important goals of mechanical ventilation are: (1) Ensuring alveolar ventilation lungs (the main criterion of efficiency is the level of PaCO<sub>2</sub>). (2.) Blood oxygenation (the main criterion PaO<sub>2</sub> and SpO<sub>2</sub>). Additional goals of mechanical ventilation include reducing the work of breathing and oxygen consumption by the respiratory muscles, which improves oxygenation of other tissues [1, 2].

These tasks MV must be carried out if possible, without harming the lungs. It is necessary and important to note that mechanical ventilation with positive pressure is only support technology, but does not treat the causes of external respiratory failure in breathing. This method cannot "cure" the disease; it can only help "win time" for the application of other treatment methods that will enhance recovery. (including self-recovery of patient) [3, 4].

Respiratory support in most patients may be necessary during only a short period of time. However, some people cannot be weaned from the ventilator for a long time. Some patients who know that they have a very serious disease of the nervous system or lungs may not even want to use mechanical ventilation at all. This can create additional problems, including ethical ones.

Although the group of patients requiring long-term mechanical ventilation (PMV) is less than 10% of all patients, they consume up to 40% of the resources of intensive care units (ICUs) [5, 6]. Over the past two decades, the prevalence of long-term ventilation has increased dramatically. Soh et al. [7] identified a 190% increase in the incidence of tracheostomy for PMV from 1993 to 2002. Studies of weaning from the ventilator in the early and mid-1990s consistently demonstrated that approximately 10% of ICU patients required respiratory support for at least 14 days [8]. This inevitably leads to a growing number of patients requiring prolonged artificial ventilation. However, it is difficult to accurately determine the number of actual patients on mechanical ventilation, even in the United States. One of the methods of deducting the number of patients using prolonged mechanical ventilation is knowing the patients who had received tracheostomy for ventilation, and this value has increased from 8.3 per 100,000 persons in 1993 to 24.3 per 100,000 in 2002[9]. In 2010 in Boston where a review of patients who were on prolonged MV was done it showed that prolonged MV was done, 7.4 per 100,000 persons was discovered to have been on prolonged artificial ventilation. [10]. If this data are extrapolated for the entire USA population, then approximately 10,966 will require long term mechanical ventilation yearly. [11] This highlights the reality of the need of prolong mechanical ventilation in intensive care units thus the justification for this article.

### **BASIC DEFINITIONS:**

In mechanical ventilation, this process uses positive pressure, which influences breathing mechanism and its parameters, to provide adequate gas exchange support, positive pressure ventilation involve applying ventilatory patterns mimicking normal ones through either masks or artificial airways- tidal volume, Respiratory Rate, minute ventilation, fraction of oxygen in inspired air, pressure in airways. [12]

Ventilation: is the exchange of gases between the lungs and the surrounding medium, another term is the process of delivering of oxygen to the lungs and fro the lungs. This affects delivery of oxygen and removal of carbon dioxide from alveolar space.

Minute Ventilation: MV is a parameter that characterize ventilation, it is the volume of gas that participates in lung ventilation per minute. The PaO<sub>2</sub> is an indicator of this parameter. MV is calculated  $RR * V_t$ , RR is respiratory rate per minute, V<sub>t</sub> is the tidal volume.

Respiratory Rate: This is the number of breaths per minute (bpm). If RR is 15 it means that in every 4 second breathing occurs. This allows us to monitor and evaluate breathing over time. The initial RR should reflect in patient's comfort and is typically 12- 20 bpm. This is adequate and sufficient for MV, normal respiratory metabolic acid –base balance. It is important to note that inadequate respiratory rate could lead to respiratory acidosis, and excessive acidosis can lead to heart attack.

Tidal volume (VT): this tis the volume of gas that is delivered by the ventilator in each breath to the patient. Changing the V<sub>t</sub> leads to changing in minute ventilation ( $V_t * RR$ ), decreasing the V<sub>t</sub> without any change in RR will lead to a decrease in minute ventilation and increase in PaCO<sub>2</sub>, a low tidal volume during mechanical ventilation is a protective mechanism especially for patients with acute respiratory distress syndrome and other lung diseases. Initially the TV is set between 6-8 mL/kg body weight this prevent development of acute ventilator induced lung injury. Therefore, it is important to use a protective lung strategy in ventilation since an increased pressure or volume might lead to unwanted lung injury and lung edema. [4]

Fraction of Inspired Oxygen (FiO<sub>2</sub>): this is the fraction of oxygen in the inhaled air mixture that the patient receives, FiO<sub>2</sub> starts at 100% and gradually reduces depending on the oxygen saturation in blood measured either with pulse oximetry or gas analysis using ABG analyzer. Increasing FiO<sub>2</sub> raises the oxygen content in patients' blood, this can lead to hyperoxia and damage to tissues. Constant increase in FiO<sub>2</sub> will increase oxygen in blood apart from lung injuries it can lead to oxygen toxicity as seen in experimental work done in animals. Authors has agreed on the fact that FiO<sub>2</sub> at 0.4 can be given a prolonged time during ventilation but FiO<sub>2</sub> at 0.8 and above much be avoided is possible. Increase in FiO<sub>2</sub> is less dangerous that hypoxia.

Positive End Expiratory Pressure (PEEP). This is a pressure that is set to remain at the end of expiration, just as  $FiO_2$ , it increases oxygenation and its delivery. According to Henry's law, the solubility of a gas in a liquid is directly proportional at equilibrium to its partial pressure above the liquid. Increasing PEEP will increase the pressure in the system and in the alveolar gas, which will increase the solubility of oxygen and its ability to diffuse through the alveolar-capillary membrane and increase the oxygen content in the blood. PEEP also improves oxygenation as a result of correcting the mismatch between ventilation and perfusion.

Increasing PEEP can contribute to the recruitment of non-ventilated alveoli, that is, the inclusion of non-ventilated alveoli in ventilation, and reduce blood shunting in the lungs. It is necessary to start with a low PEEP of 5 cm H<sub>2</sub>O and titrate as it is for patient's tolerance to the target level of oxygenation. In doing so, close attention must be paid to arterial pressure and patient comfort [14].

Attention should be paid to volumetric curves on the ventilator display, and it is necessary to pay attention to the fact that the curve does not return to zero during exhalation, because if the curve returns to zero (Fig. 1), this indicates incomplete exhalation and the development of auto-PEEP, in this case it is necessary to immediately change the ventilation parameters. Available evidence indicates that high levels of PEEP, compared to low levels, do not reduce in hospital mortality. The data also show that high levels of PEEP did not significantly increase the risk of barotrauma, but rather improved oxygenation in patients on the first, third, and seventh days [15].

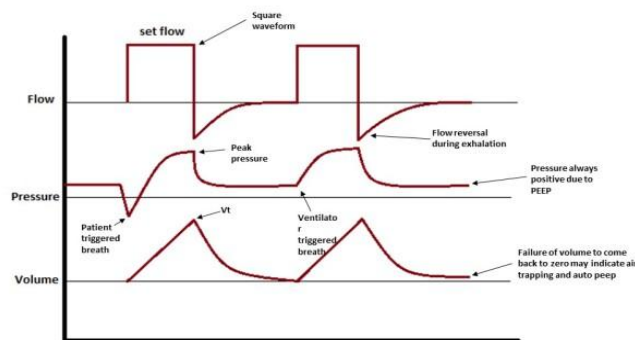


Fig. 1. Assist-controlled (AC) ventilation. Andres Mora Mora Carpio A.L., Mora J.I. Ventilation Assist Control. Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls.

**Flow:** The rate in l/min at which gas mixture is supplied by the ventilator. When the patient is connected to the ventilator in the mode volume control (VCV), inspiratory the flow is usually set to a certain standard value, usually 60 l/min [16]. In many critically ill patients the activity of the respiratory center is increased and this level of flow may be insufficient, to satisfy his needs. As a result, patients will struggle with respiratory impedance, both their own and the device, which leads to an increase in the work of breathing [17]. These violations are usually determined by the jagged shape pressure graph (which means increased patient's work of breathing) [2]. Often, increasing inspiratory flow allows to achieve a more favorable (rounded) shape pressure curve. However, in some patients increasing the flow will lead to a rapid persistent tachypnea [18], with shortening of exhalation time and build-up auto-PEEP. This response to a change in ventilation parameters is due to the Hering-Breuer reflex and differs in different patients [19]. In addition to these four main parameters, is possible to choose a method by which ventilation is carried out.

Ventilator allows you to change the flow; the flow can be constant during inhalation (square waveform) or gradually decreasing (waveform is wavy).

1. A square waveform indicates about faster inhalation and decrease inspiratory time and increase expiratory time. This can be useful for patients with asthma or chronic obstructive pulmonary disease or in cases of increased RR to prevent auto-PEEP and provide enough time to exhale.

2. The wavy waveform indicates a decrease in flow, since the delivered volume increases. This is usually more comfortable for the patient and provides better distribution and equalization of volume in patients with heterogeneous lungs, such as in ARDS. The speed at which the flow moves, so also can be controlled by setting the inspiratory and expiratory time. This indicator can be adapted for patient comfort or prevention of auto-PEEP. After the patient breaths in, the expiratory valve of the ventilator opens and air is allowed to escape until the pressure in the system reaches PEEP. (fig. 1)

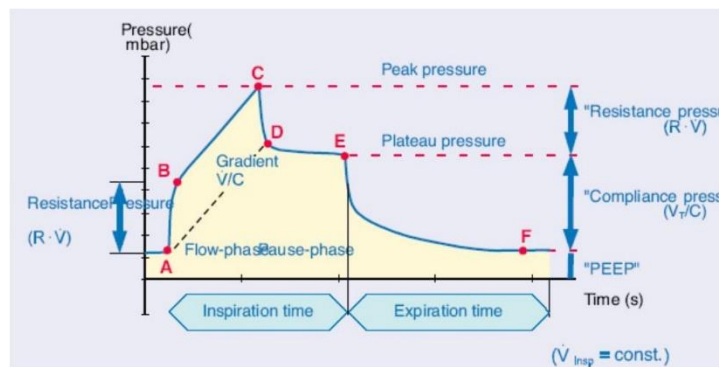
**AIRWAY PRESSURE.**

It is necessary to evaluate two important pressures in the system during mechanical ventilation:

1. Peak pressure – the pressure reached during inspiration, when air is inhaled into the lungs, is a measure of resistance of the respiratory tract, as well as compliance and includes resistance in the bronchus and bronchial tree.
2. Plateau pressure – static pressure, reached at the end of a full breath. In order to measure plateau pressure, we must we need to maintain an inspiratory pause, to allow the pressure to equalize in breathing circuit (Fig.2). Pressure plateau reflects alveolar pressure and lung compliance. Normal plateau pressure should be below 30 cm H2O; higher pressure can cause barotrauma.

If an increase in peak pressure is observed, the first step that needs to be taken, maintain an inspiratory pause and check the plateau pressure. Increased peak pressure and normal plateau pressure indicates high resistance in airways in normal compliance.

Compliance (C) – increase in lung volume in response to increased pressure in the respiratory ways. Normally, C of the lungs and chest wall reaches 100 ml/cm H2O during spontaneous breathing, and decreases to 50 ml/cm H2O during expiratory cycle of mechanical ventilation.



The level of pressure at “B” is affected by the resistance and the flow  $R = \frac{\Delta p}{\dot{V}}$

Fig. 2. Normal Pressure-Time Curve Drager —Curves and Loops in Mechanical Ventilation, Drager.

This means that in a normal lung, a tidal volume of 500 ml will be provided by a positive alveolar pressure of 10 cm H2O. However, in intensive care, we rarely work with normal lungs, and compliance can be higher or much lower. Any disease that destroys the lung parenchyma, such as emphysema, will increase compliance; any disease that increases lung stiffness (ARDS, pneumonia, pulmonary edema, pulmonary fibrosis) reduces lung compliance.

**EFFECT OF MECHANICAL VENTILATION WITH POSITIVE PRESSURE ON PHYSIOLOGY**

Positive pressure produced by the ventilator, is transmitted to the upper respiratory tract and finally into the alveoli, and then into the chest cavity, creating positive pressure (or, at least, less negative pressure) in the pleural cavity (Fig. 3). This increases pressure in the right atrium (RA) and reduces

venous return, causing decrease in preload, which leads to decrease in cardiac output. As a result, the mean arterial may decrease pressure (MAP) if there is no compensatory response that increases systemic vascular resistance (SVR). This is very important point, especially in patients who may not be able to increase their SVR, for example, patients with distributive shock (septic, neurogenic or anaphylactic shock). On the other hand, ventilation with positive pressure can significantly reduce the work of breathing. This allows reduction in blood flow to the respiratory muscles and redistributes it to more critical organs. Reducing the work of breathing muscles also reduces CO<sub>2</sub> production and lactate in these muscles helps reduce acidosis.

Effects of ventilation with positive pressure on venous return may be useful in patients with cardiogenic edema of the lungs. In these patients with volume overload, a decrease in venous return directly reduces the severity of pulmonary edema, reducing the cardiac output of the right ventricle. At the same time, a decrease in return may reduce overstretching of the left ventricle, placing it at a more advantageous point Frank-Starling curve and possibly improve improving cardiac output.

## REVIEW OF LITERATURE INDICATIONS FOR CONTINUED USE ARTIFICIAL LUNG VENTILATION.

The most common indication for mechanical ventilation is hypercapnic (ventilatory) and/ or hypoxemic (parenchymal) acute respiratory failure, which is diagnosed by clinical signs, arterial blood gas analysis, capnography and pulse oximetry.

## CONTRAINDICATIONS

There are no absolute contraindications for artificial lung ventilation, as this method is often life-saving of the patient, and all patients who need lung ventilation.

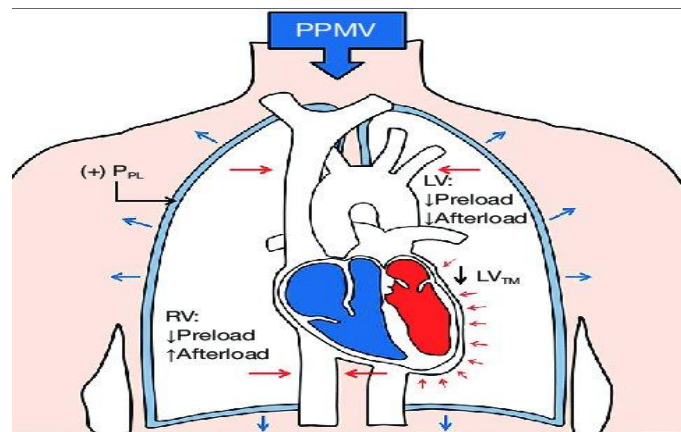


Fig. 3. Hemodynamic effects of artificial ventilation

The pressure in the right heart and intrathoracic large veins depends on the pleural pressure (PPL), which changes during the entire respiratory cycle. During mechanical ventilation with positive pressure (PPMV), the pressure in the right ventricle (RV) is inversely proportional to the intrathoracic pressure, and since PPL becomes more positive, the end-diastolic pressure of the RV decreases, causing a detectable decrease in cardiac output. On the other hand, PPMV reduces the end-diastolic pressure and afterload for the left ventricle (LV). For all vascular structures within the chest cavity, the transmural pressure (PTM) is influenced by changes in PPL and respiratory efforts ( $PTM = \text{intravascular or intraventricular systolic pressure} - PPL$ ). Blue arrow – expansion of the chest walls directed outward PPMV, Red arrows – effect of positive PPL on the intrathoracic vascular network and left ventricle. LVTM, left ventricular transmural pressure to offer the possibility of transfer to mechanical ventilation, if there are indications. The only absolute contraindication indication for artificial lung ventilation is tension pneumothorax, but after decompression of the pleural cavity, this contraindication is excluded.

A contraindication may also be the refusal of a patient who is in a clear state of mind to use mechanical ventilation, but this issue has not been legally developed in Ukraine.

## MONITORING

All patients on mechanical ventilation require special monitoring of important life functions: heart rate, breathing rate, blood pressure and oxygen saturation. Other tests that can be done include chest x-ray chest, capnography and measurement of partial pressure of oxygen and carbon dioxide in arterial blood (PaO<sub>2</sub> and PaCO<sub>2</sub>). ABG should be obtained 30 minutes after intubation and mechanical ventilation. Members of the medical team providing mechanical ventilation (including ICU doctors, nurses, respiratory doctors) should use this information to assess the patient's status and, if necessary, make changes to the ventilatory parameters [4]. Monitoring also includes control of pressure in the airways and compliance of the respiratory system.

## MODERN MECHANICAL VENTILATION MODES

Modern mechanical ventilators were developed in the 1950s to support patients with respiratory failure accuracy [20]. Although newer machines provide significantly more clinical opportunities, the variety of available modes can be challenging to understand and apply them in clinical settings. Many of these modes are considered nontraditional compared to the modes that which have been used for many years. There is insufficient evidence to support the superiority of one mode compared to compared to another. Regardless of trademarks newer models can be divided into 2 main categories: volume-controlled ventilation by volume (VCV) and pressure-controlled pressure (PCV) [21 -23]. Dual control modes management has properties of both pressure and by volume. Some dual modes controls can be further categorized as feedback ventilation modes, which provide variable assist -controlled breathing depending on the degree of the patient's respiratory effort [24, 25].

After intubating the patient and connecting to a ventilator, it is necessary to select the ventilation mode to be used. Several principles must be followed to make this optimal for the particular patient in a consistent manner. Regardless of the selected mode of mechanical ventilation, to ensure safe and effective use of artificial ventilation in patients, the American College of Emergency Physicians (ACEP) [14] recommends the following: the strategy of artificial ventilation should be individualized, taking into account the underlying disease process of the patient. The initial setting of ventilator parameters may vary depending on the cause of intubation and the scope of what we are aiming to achieve. Nevertheless, there is a choice of some basic setting parameters for most cases.

Consider lung-protective strategies that include limited tidal volume, maintaining lung tissue recruitment, limiting airway pressure, and reducing oxygen toxicity. Lung compliance and plateau pressure should be assessed to help reduce the incidence of barotrauma and lung injury. Continuous quantitative waveform capnography is recommended, and blood gas measurements after intubation can be obtained to ensure adequate ventilator parameters (e.g., respiratory rate, tidal volume, FiO<sub>2</sub>). Patients should continue to receive adequate doses of analgesia and sedation to maintain comfort during mechanical ventilation. Unless contraindicated, elevate the head of the bed at least 30 degrees to reduce the risk of ventilator-associated pneumonia.

Since prolonged periods of hyperoxia can lead to iatrogenic injury, titrate FiO<sub>2</sub> to maintain adequate oxygen saturation. There are several different ventilation modes that differ minimally from each other. In this review, we will focus on the ventilation modes commonly used and their clinical application. Controlled ventilation can be provided with volume or pressure control. Therefore, there is volume controlled or pressure-controlled ventilation.

**Volume-controlled ventilation (VCV)** mode in which ventilation provides a specific tidal volume and, accordingly, minute ventilation. Although the tidal volume remains constant with each breath, the inspiratory pressure in the airways may vary depending on the resistance of the airways and the compliance of the lung and chest wall [22]. For example, if airway resistance increases or lung compliance decreases, inspiratory pressure will increase even though the delivered tidal volume remains constant. During VCV, an increase in inspiratory pressure, since tidal volume is delivered, ends with peak inspiratory pressure when the entire tidal volume is delivered by the ventilator. In this mode, the waveform of pressure increase is often described as an "shark fin" (Fig. 4). Volume-

controlled ventilation is a mode that is usually combined with assist-controlled ventilation, or synchronized intermittent mandatory ventilation.

**Assist-Control (AC)** Assist-Control is one of the most common modes of mechanical ventilation in the intensive care unit, especially in a recently intubated patient. This mode provides good comfort and easy control of some of the most important physiological parameters. Ventilation in AC mode is a periodic volume regulated ventilation method. Assisted control (volume regulation) is the preferred method used in most intensive care units in the United States because it is convenient. Four ventilator setting parameters can be easily selected (respiratory rate, tidal volume, FiO<sub>2</sub> and PEEP).

**Fixed tidal volume (V<sub>t</sub>)** which the ventilator provides in the ventilation in intervals or when the patient starts breathing. V<sub>t</sub>, provided by the ventilator during AC, will always be the same regardless of compliance, peak pressure or plateau pressure in the lungs.

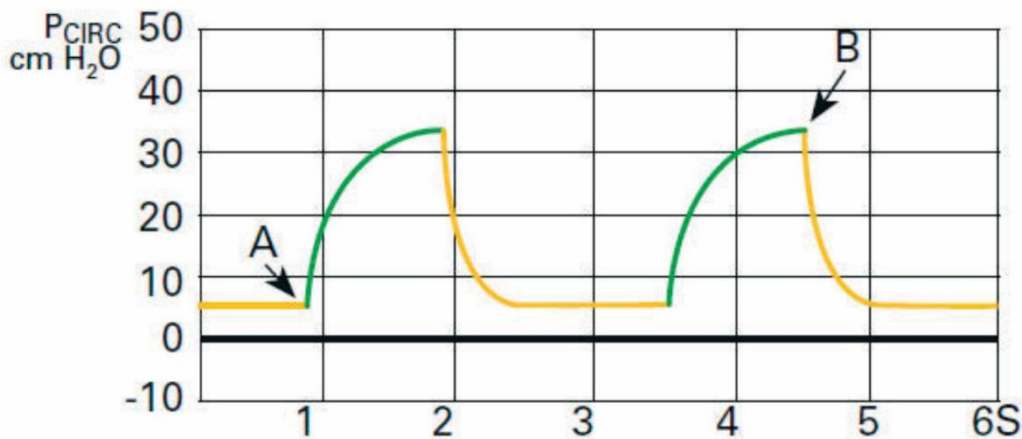


Fig. 4. Pressure/time curve for volume-controlled ventilation. Point A represents the beginning of the ventilator-initiated breath. The shape of the pressure wave increase, ending with the peak inspiratory pressure (point B), is noted. The horizontal axis represents time in seconds. Abbreviation: PCIRC, pressure in the circuit. (taken from Gallagher JJ. *Alternative Modes of Mechanical Ventilation*. AACN Advanced Critical Care Volume 29, Number 4, pp. 396-404, 2018)

With AC, the RR indicator can affect the patient's breathing, meaning that if the patient takes a breath, the ventilator will detect a decrease in pressure and provide the specified number of breaths, even if the patient breathes less often. For example, if the patient breathes at a rate of 20 bpm, and the ventilator RR is set at 15 bpm, the ventilator will follow the patient and provide 20 bpm (each time the patient initiates a breathing effort). When using the AC method, the ventilator is programmed to respond to changes in systemic pressure when the patient begins breathing. When the diaphragm contracts, the intrathoracic pressure becomes more negative. Negative pressure is transmitted to the airways and then to the ventilator hoses, where sensors detect a change in pressure and change the tidal volume. The threshold level of negative pressure at which breathing should be triggered is called trigger sensitivity and is usually adjusted by a respiratory physician. This makes this method of controlled or assisted ventilation very convenient for the patient, since each of his or her breathing efforts will be reinforced by the ventilator, and if the patient's breathing is suppressed, the ventilator will provide a minimum minute volume of ventilation. As with increasing V<sub>t</sub>, increases RR, we will increase minute ventilation and this will reduce pCO<sub>2</sub> in the patient's blood. However, it should be remembered that by increasing RR, will also increase dead space, so increasing RR may not be as effective to increase minute volume as increase in V<sub>t</sub>. AC mode is an excellent method to guarantee good ventilation. It is often used in cases of metabolic or respiratory acidosis.

AC is a mode primarily used for ventilation in the "Ventilation with Lower Tidal Volumes as Compared with Traditional Tidal Volumes for Acute Lung Injury and the Acute Respiratory Distress Syndrome" study, which used the ARDSNET protocol. In principle, this is the only proven regimen that provides a survival benefit in patients with ARDS and should be the method of choice in patients

with this pathology. It is important to understand that when using the method AC tidal volume can be selected, and the pressure in the airways will depend on lung compliance.

In the case of good compliance, there will be a low plateau pressure, while in the case of a stiff lung, the same tidal volume will produce a much higher pressure (that is, this can be expected in patients with pulmonary edema, ARDS, pneumonia or pulmonary fibrosis). It is important to understand this in order to prevent ventilator- induced lung injury or barotrauma.

Each breath can be initiated by time (respiratory rate trigger) (if the patient's respiratory rate is lower than the set rate on the ventilator, the ventilator will provide breaths within the set time interval), or patient-triggered if the patient provides additional ventilation on their own.

Further adjust the parameters is necessary, taking into account the analysis of arterial blood gases, blood oxygen saturation on a pulse oximeter, capnography, to determine whether further correction of ventilator parameters is necessary. The advantage of the AC method is increased comfort, easier correction of respiratory acidosis/alkalosis, and low patient work of breathing. Some disadvantages include the inability to directly control airway pressure, which can cause barotrauma, and hyperventilation often occurs with breath stacking (unintentionally high tidal volume due to incomplete exhalation between adjacent forced breath cycles), auto-PEEP, and respiratory alkalosis.

Thus, Advantages of the AC method:

- Increases patient comfort and in results in providing the necessary ventilation;
- The doctor can regulate PaCO<sub>2</sub> levels to correct respiratory acidosis/alkalosis;
- Provides low work of breathing for the patient.

Disadvantages of the AC method:

- The system is regulated by volume, and there is a risk of barotrauma, especially with stiff lungs; usually regulating the plateau pressure should help prevent this complication.

If the patient has pronounced tachypnea or if there is not enough time to perform a full exhalation, the patient may develop auto-PEEP. In this case, not all of the gas volume will be exhaled with each breath, which increases the air volume in the patient's lung, plateau volume, and intrathoracic pressure. This can lead to hypotension due to decreased venous return. It is necessary to disconnect the patient from the ventilator to ensure sufficient time for a full exhalation and then correct the ventilation parameters. Since the patient may breathe frequently, hyperventilation can lead to respiratory alkalosis. This can be prevented by providing good sedation.

**SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION (SIMV)** SIMV is another commonly used mode of ventilation, although its use is less reliable in providing tidal volume, but in some patients demonstrates better results compared to AC. “Synchronized” means that the ventilator adapts its breaths to the patient's attempts at spontaneous breathing. “Intermittent” means that not all breaths are necessarily supported by the device, and “mandatory” ventilation means that, as with AC, the ventilator will provide a mandatory set minute volume of breathing regardless of the patient's breathing efforts. Obligatory breaths of the respirator can be regulated by time if the patient's RR respiratory rate becomes less than the RR set on the ventilator (as with AC). The difference from the AC mode is that in the SIMV mode the ventilator will only provide those breaths, the frequency of which is set on the respirator, any breath initiated by the patient above this frequency will not necessarily receive full support by pressure or volume. This means that the tidal volume of each breath taken by the patient above the set breathing rate will depend solely on pulmonary compliance and patient effort. This mode was proposed as a method of “training” the diaphragm to maintain muscle tone and promote faster weaning the patient from the respirator. Nevertheless, many multiple studies have been unable to show any advantages of the mode SIMV compared to other modes artificial ventilation of the lungs. In addition, SIMV requires higher patient work of breathing than AC, which contributes to fatigue of the respiratory muscles. With this mode, regulation by volume or pressure is possible, the advantages of using these modes have not been identified.

## PRESSURE SUPPORT VENTILATION (PSV)

PSV – Pressure-controlled mode ventilation. With PCV, the gas flow is supplied according to the intended inspiratory pressure limit, not the tidal volume. The inspiratory pressure limit is supported by inspiratory time until exhalation. Pressure is controlled or limited from breath to breath, but breathing volume may vary depending on airway resistance and compliance of the lung and chest wall. For example, in as much as airway resistance increases or compliance of the lung decreases, inspiratory pressure may be reached more quickly, limiting the supply tidal volume. On the contrary, improvement of lung compliance may limit increasing tidal volume. Like VCV, PCV can be combined with ventilation assisted control and synchronized intermittent mandatory ventilation; PCV, however, can also be used with spontaneous breathing option, such as pressure support. PCV breathing produces a pressure waveform more square shape than produced by VCV.

This form waves follow from inspiratory pressure, which rises rapidly to a predetermined pressure limit and maintained for duration of the specified inspiratory time until exhalation (Fig. 5).

Since there is no reserved level minute volume, this method should not be used in patients with impaired consciousness, shock, or circulatory arrest. Tidal volumes will depend exclusively on the patient's efforts and pulmonary compliance. PSV is often used for weaning from a ventilator, as this method only facilitates the patient's breathing efforts, but does not guarantee any minute volume or respiratory rate.

Volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) are not different breathing modes, but are different control variables within the mode. Most studies comparing the effects of VCV and PSV were not controlled or prospective, and offer little new to our understanding of when and how to use each control variable. Any benefit associated with PSV relative to respiratory variables and gas exchange is likely due to the associated decelerating flow waveform available during VCV on many ventilators.

Furthermore, useful features of both VCV and PCV can be combined into so-called dual control modes, which are volume regulated, pressure-limited, and time limited. PSV mode does not offer an advantage over the VCV mode in the absence of patient spontaneous breathing, especially when flow deceleration is available during VCV. PSV may offer a lower work of breathing and improved comfort for patients with increased and variable respiratory needs [26].

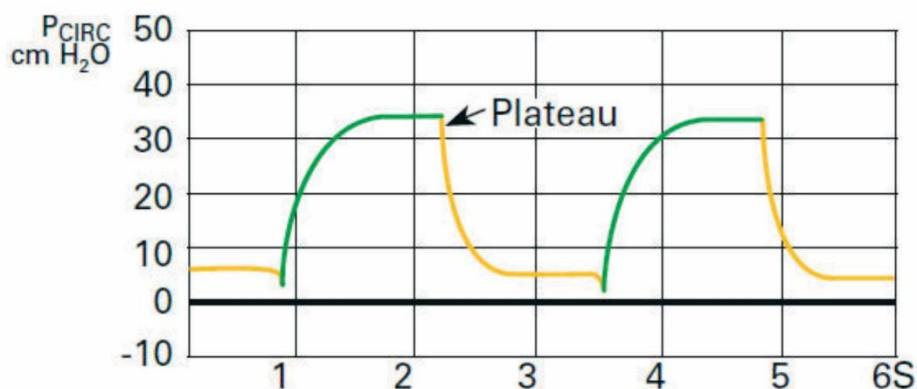


Fig. 5. Pressure/time curve during pressure-controlled ventilation. An early increase in pressure is noted, which reaches a plateau and is maintained throughout the inspiratory time until the start of exhalation. The horizontal axis represents time in seconds. Abbreviation: PCIRC, pressure in the circuit. (taken from Gallagher JJ. *Alternative Modes of Mechanical Ventilation*. AACN Advanced Critical Care Volume 29, Number 4, pp. 396-404, 2018)

The biggest disadvantage of PSV is unreliable minute ventilation, which can cause CO<sub>2</sub> retention and acidosis, as well as higher work of breathing, which can lead to fatigue of the respiratory muscles. To eliminate this shortcoming, a new algorithm has been proposed for PSV, called volume support ventilation (VSV). VSV is a method similar to PSV, but with this method, the tidal volume is

controlled by a feedback mechanism, because the pressure support provided to the patient will be constantly adjusted by tidal volume. With this method, in cases of decreased tidal volume, the ventilator will increase pressure support, and if the tidal volume increases, then the pressure support will decrease to maintain tidal volume close to the desired to ensure adequate minute ventilation. There is some evidence that the use of VSV can reduce the time of assisted ventilation, the full time of weaning from the ventilator and reduce the need for sedatives.

Currently available data are insufficient to confirm or refute the benefits of pressure-controlled or volume-controlled ventilation in patients with ARDS. More extensive studies including a larger number of patients in whom PSV and VCV will be compared may provide more reliable evidence on which more robust conclusions can be based [27].

### **NEURALLY ADJUSTED VENTILATOR ASSIST (NAVA)**

Neuro-regulated ventilator support uses electromyography (EMG) signal from the diaphragm, which controls the flow and cycles of respiratory support [28, 29]. In this mode, sensors in the diaphragm determine the timing and intensity of the patient's respiratory effort, which are synchronized with the ventilation thus preventing barotrauma and providing the necessary support by the ventilator. The extent of ventilator support increases or decreases and is proportional to the input signal. In the case of NAVA the applied pressure is proportional to the integral of the electrical activity of the diaphragm. Thus, as the patient's effort increases and the electrical activity of the diaphragm is greater, the level of assistance is greater.

The EMG sensor consists of multiple electrodes mounted on a trans esophageal catheter, which is placed in the esophagus at the level of the diaphragm. The trigger start of the ventilator breath, thus, actually occurs simultaneously with the onset of excitation of the diaphragmatic nerve, and the cycles of breathing is closely related to the cessation of contraction of the respiratory muscle. The flow delivery is stimulated by the intensity of the EMG signal (electrical activity of the diaphragm [EADi]), and the clinician establishes this intensity in ml/millivolt. Like PAV, NAVA depends solely on the patient's effort, which affects the timing, intensity, and duration of the respiratory cycle. Thus, as with PAV mode, clinicians should choose appropriate sensory indicators specifically for patients with unreliable respiratory drives. Also as with PAV mode, clinicians should set the PEEP and FiO<sub>2</sub> levels. Small clinical studies have demonstrated improved triggering mechanism, and synchronization of the patient's breathing cycle with NAVA compared to conventional modes [30–32]. However, there is no consensus on what level of support to start with and what level to maintain thereafter. As with PAV mode, some physicians try to start with high levels of support and wean from the ventilator at low levels, while others indicate that maintaining a constant level of support along with regular SBT makes more sense.

Also as with PAV mode, there is insufficient evidence that NAVA mode demonstrates improved outcomes (e.g., reduced duration of mechanical ventilation, need for sedatives). Another problem with NAVA mode is the cost of EMG sensors.

**High-frequency oscillatory ventilation** (High-frequency oscillatory ventilation (HFOV) – ventilation with a very high respiratory rate (120–900 breaths per minute [bpm] in adults) and with very small with minute volumes (usually less than anatomical dead space and often less than 1 ml/kg at the alveolar level). To ensure gas exchange and transport of gases in the lungs up and down the trachea-bronchial tree under these seemingly non physiological conditions, mechanisms such as Taylor dispersion, coaxial flows, and extensive diffusion are involved [33]. HFOV provides a substantial mean airway pressure, but exerts very little pressure or volume fluctuations in the alveoli, so this mode is sometimes called continuous positive airway pressure (CPAP) with gas oscillation. The alleged benefits for HFOV are twofold.

First, very little periodic pressure oscillation in the alveoli minimizes cyclic over distension and derecruitment of alveoli [34]. Second, high mean airway pressure may also prevent alveolar derecruitment. Interestingly, the mean pressure used during HFOV is often reported to exceed 30–35

cmH<sub>2</sub>O, i.e. the pressure used during conventional ventilation [35–37]. This may explain the expansion of alveolar membranes that may occur when a slowly applied constant pressure is applied, rather than cyclic short periodic pressure breaths during conventional ventilation [38]. In adults, common initial HFOV settings are: frequency 300 bpm and mean pressure 5 cmH<sub>2</sub>O higher compared to previous conventional ventilation [37, 39]. HFOV ventilation to a large extent controlled by mean pressure and FiO<sub>2</sub>, CO<sub>2</sub> clearance is largely controlled “power setting” that controls the amplitude pressure fluctuations. Lower frequency increased CO<sub>2</sub> clearance, to a large extent, because more slow frequencies allow for larger changes in volume. The greatest clinical experience with high-frequency ventilation methods is in newborns and in the pediatric group of patients [40, 41]. The results of these studies suggest that high-frequency ventilation appears to improve long-term disease outcomes in these patients. Recently, new devices have become available for use of high-frequency ventilation methods in adults that are able to adequately maintain gas exchange in the lungs. In 2010, the McMaster University Evidence Based Medicine Group updated a meta-analysis of HFOV use in ARDS [37]. They analyzed eight clinical trials of HFOV in patients with ARDS. This population included some pediatric patients who met the criteria for ARDS.

In this analysis, six of the eight studies used HFOV within 48 hours of intubation, and in five of the eight studies, the ARDS Network low tidal volume ventilation strategy was used as a control group. Four hundred and nineteen patients were included in these studies. The concluding meta-analysis showed that HFOV provided a significant reduction in mortality with a risk ratio of 0.77 and a 95% confidence interval of 0.61 to 0.98. This suggests the appropriateness of using HFOV in severe respiratory failure associated with ARDS. This conclusion, however, was questioned in a recent publication of two large randomized controlled trials of HFOV in moderate ARDS, in which this mode did not offer benefit and in one HFOV was associated with harm. These results determine the indications for HFOV use: (1) in patients in whom conventional protective ventilation methods have failed and (2) study by clinicians of HFOV technology.

A variant of high-frequency ventilation is high-frequency percussive ventilation (HFPV) – a mode in which high frequency pulsed pressure oscillations are applied to a normal ventilation pattern. This technique is thought to provide two things: first, high-frequency pulsations can increase gas exchange in the airways, and thus gas exchange between the alveoli and the blood.

Second, high-frequency pulse can increase secretion clearance. Indeed, it was this latter application that stimulated its popularity in patients with burns of the respiratory tract, where improved pulmonary toilet is required. One of the few randomized studies of this technique was conducted in 2010 [42], where 62 patients with military burn trauma to the respiratory tract were randomized to HFPV or conventional protective to develop a strategy for mechanical ventilation. Approximately one-third of patients had significant inhalation injuries.

Although gas exchange in the lungs was satisfactory in the HFPV group, the final results in terms of survival rate, ventilator free days, and hospital length of stay did not differ statistically. HFPV, therefore, remains an attractive theoretical additional technique in patients, especially with severe respiratory tract injuries, but new evidence is needed to improve outcomes with its use. For reader in low income countries, this mode is only of theoretical interest, due to the lack of appropriate equipment.

### **AIRWAY PRESSURE RELEASE VENTILATION (APRV)**

APRV, also known as Bi-level– new mechanical ventilation strategy aimed at preventing VILI. It is cyclical in time, pressure-controlled form of respiratory support [43–45]. As one might assume from the name, in APRV mode the ventilator will provide constant, high pressure in the airways, providing oxygenation, and ventilation is provided by due to periodic reduction of this pressure. This means that the ventilator provides constant high pressure (P high) for a specified period of time (T high), and then then reduces this pressure, usually to zero (P low) in a much shorter period of time (T low). The respiratory rate in mode APRV often ranges from 8 to 12 breaths/min. Using this mode, T high can vary from 4 to 6 seconds. Patients can breathe spontaneously and independently of the supplied

machine breathing. This spontaneous breathing supports alveolar ventilation and gas movement to the larger airways for exhalation during the short expiratory phase. Although spontaneous breathing theoretically can occur both during inhalation and during exhalation, the short T low often does not allow the patient to attempt spontaneous breathing during exhalation, especially if T low is less than 1 s [46]. Often T low is set empirically between 0.2 and 0.8 s. Since the exhalation time is so short, P low is often set at 0 cm H<sub>2</sub>O. If a longer T low is set (for example, T low > 1.0 seconds), P low can prevent alveolar collapse during a longer expiratory time.

Airway pressure release ventilation is often used to recruit, support, and stabilize alveolar units in conditions of widespread alveolar collapse, such as in ARDS. Prolonged inspiratory time is key to the recruiting effort, increasing mean airway pressure above or below the desired plateau pressure. The short release period allows for exhalation to remove carbon dioxide from the lungs, theoretically preventing alveolar derecruitment between breaths. The idea of this mode is that during T high (occupying 80-95% of the respiratory cycle time), constant alveolar recruitment occurs, which improves oxygenation, since the time during which high pressure is maintained is much longer than with any other type of ventilation ("open lung" strategy). This reduces the intensity of constant stretching-contraction of the lung compared to other ventilation modes, preventing ventilator-induced lung injury. Minute ventilation will depend on T low and the patient's tidal volumes during T high.

APRV is actually a modification of pressure-controlled SIMV, which allows the patient's spontaneous breathing (with or without pressure support) to combine both during inflation and during deflation. APRV differs from conventional pressure controlled SIMV by the choice of inspiratory and expiratory time (I: E), specifically, with conventional pressure-controlled SIMV, the "physiological" inspiratory time I: E ratio is less than 1:1. Spontaneous breathing thus occurs during the expiratory phase. In contrast, APRV provides a longer inspiratory time, producing so-called inverse ratio ventilation (IRV with I:E ratios up to 4:1 or 5:1). Spontaneous breathing, therefore, in this case occurs during this long inflation period. The alleged advantages of this approach are similar to those in the second mode with a long inspiratory time (IRV) [47, 48].

APRV mode has recently gained popularity as an alternative for patients with difficulties in providing oxygenation (for example, in severe ARDS), in whom all other modes have not achieved their goals.

#### **Indications for APRV:**

1. ARDS, in which it is difficult to provide oxygenation with AC;
2. Postoperative atelectasis.

#### **Advantages of APRV:**

APRV is a good method for protective lung ventilation. The ability to set P high means that the operator controls the plateau pressure, which can be set significantly lower than 30 cm of water. If pressure is above this, is a high risk of barotrauma of the lungs.

Since the patient makes his own breathing efforts, the mode provides the best distribution of oxygen which in turn leads to secondarily improved V/Q matching.

Constant high pressure means good lung recruitment (open lung strategy open lung), so APRV can improve oxygenation in patients with ARDS, in which it is difficult to provide oxygenation with AC mode.

APRV can reduce the need for sedatives and neuromuscular relaxants, as the patient feels more comfort than with other mechanical ventilation modes.

#### **Disadvantages and contraindications:**

Given that spontaneous breathing is important element of APRV, this method is not ideal for patients requiring deep sedation. There is no data on the use of APRV in patients with neuromuscular disorders transmission or obstructive pulmonary disease and its use should be avoided in this group patients.

Theoretically, constant high intrathoracic pressure can cause high pressure in the pulmonary artery and worsen intracardiac shunts in patients with pathology Eisenmenger.

Several randomized, controlled studies to evaluate APRV has been done, in the first of these, showed it was beneficial on the outcomes in the group APRV, but these results were difficult to interpret, because the management strategy required 3 days of muscle paralysis, and this seemed to significantly worsen gas exchange [49]. In a later study, the authors compared APRV with a more common SIMV strategy and found no significant differences in outcomes [50].

In a recent study, the authors compared APRV with the ARDS Network using low tidal volume in 64 patients with trauma-induced ARDS [51]. This study also found no significant differences in any disease outcomes. Specifically, the number of days in the ICU, length of stay, and mortality were all comparable regardless of ventilation mode. Finally, an interesting analysis of a very large database of mechanical ventilation use was published [52]. This study included 234 subjects who were on APRV. A control group of patients were selected who had propensity to benefit from assist-control. In the control group, mortality, without ventilator, or length of stay did not differ from the study group. Taken together, these studies suggested that APRV, although a physiologically interesting mode, has not yet been shown to improve disease outcomes in patients with severe ARDS.

Regarding this mode, it can be concluded that the APRV mode must be used in patients in whom AC mode cannot be effective and safe to provide safe plateau pressure and blood oxygenation, that is, in patients with severe ARDS. Choosing APRV as a ventilation method, rather than a more common mechanical ventilation method, such as AC, should be used with strong clinical justification.

#### **AUTOMATIC MECHANICAL VENTILATION MODES:**

New modes aimed at improving patient-ventilator interaction, providing feedback between the volume of the ventilator relative to the intended pressure. As noted earlier, the intended pressure with variable flow characteristically often synchronizes with the patient's breathing pattern. The disadvantage is in planning the pressure however, the tidal volume cannot be guaranteed. This may be especially important if the patient's respiratory drive is variable, or mechanics external respiration is unstable and, therefore, minute ventilation or target tidal volume (e.g. 6 - 8 ml/kg) cannot be reliably achieved.

Over the past 2 decades, several technical innovations have attempted to describe the pressure and target flow functions, creating feedback algorithms that allow some volume control with pressure planning. The most common approach is to use a weighted volume value to control the pressure level of the subsequent intended inspiratory pressure [53, 54]. It is important to remember that no algorithm can replace attempts to correct patient parameters at the bedside [55].

**Adaptive support ventilation (ASV)** is an assisted, pressure-controlled, time-cycled ventilation mode in which the breathing pattern is automatically determined – breathing is an assisted, pressure-controlled, time-cycled ventilation mode in which the breathing pattern is automatically determine tidal volume and frequency of breathing, based on the mechanics of the respiratory system to minimize ventilator work [56].

Adaptive support ventilation (ASV) is a ventilation mode that adapts continuously to support patients' independent attempts. In this ventilation mode, the clinician enters the patient's gender and height during the initial ventilator setup, and these parameters determine the ideal minute ventilation. Another set of parameters includes the percentage of desired minute ventilation support (e.g. 100%), the maximum pressure limit, FiO<sub>2</sub> and PEEP [57–59].

The machine determines the optimal breathing volume and frequency to achieve the ideal minute target ventilation to optimize the work of breathing. In a patient who is breathing spontaneously (active), the ventilator will perform the regulated volume and pressure to support function. In a patient who is not breathing spontaneously (passive), the ventilator will provide the volume-intended function and regulated pressure.

ASV calculates the minimum work after the first measurement of the mechanics of the respiratory system using several test breaths. Expiratory time constants ( $RC_e$   $\frac{1}{4}$  resistance compliance) are then measured to ensure an inspiratory time of at least one  $RC_e$  and an expiratory time of at least three  $RC_e$ . This data is then inserted into the following algorithm to calculate the frequency associated with the minimum work for a given alveolar ventilation: where –  $RC$  is the respiratory time constant,  $V_A$  and  $V_D$  – alveolar ventilation and dead space ventilation  $A D$  respectively, and a constant that depends on the shape of the flow wave. Boundary rules exist to prevent excessive lung stretching. Under normal conditions, the minute volume is set within 100% of the normal minute volume based on the predicted body weight of 0.1 l/kg/min. Thus, in a 70-kilogram patient, Minute ventilation would be set at 7.0 l/min. ASV regulates inspiratory pressure to achieve a respiratory structure ( $V_T$  and frequency) that minimizes the work of breathing, based on Otis' equation. It is important, however, that the range of  $V_T$  and pressure should be selected by the clinician. Breathing alternates between regulated pressure and pressure support breathing, based on the presence or absence of spontaneous breathing efforts by the patient.

Conceptually, As ASV can provide synchronized ventilation and PSV. During mandatory breathing, ASV controls inspiratory time and inspiratory: expiratory ratio (I: E), based on the measurement of the expiratory time constant. At lower compliance, I: E is lower, while at high resistance, a more significant I: E ratio is selected to avoid air immobilization. Ideal body weight can also be used to calculate the desired minute ventilation based on metabolic requirements and predicted dead space. Clinicians should also set the PEEP and  $FiO_2$  levels.

One caveat should be made associated with this mode – the combination of tidal volume and frequency used to achieve the intended minute ventilation may be accompanied by a large tidal volume that will be above the target [60, 61]. Low tidal volume ventilation – this problem can be corrected by adjusting the pressure limit to achieve sufficient tidal volume, although doing so, the machine adapts to a higher frequency in order to achieve the intended minute ventilation.

ASV allows automatic adaptation breathing pressure and breathing rate with minimal work from the patient to ensure that the target minute ventilation is achieved. Conceptually, this minimal work of the ventilator, can use minimal effort to straighten the lungs, which may reduce the risk of lung injury.

ASV as a pure control mode was evaluated by several methods. The initial model demonstrated [62] that the ASV algorithm responded appropriately to abrupt changes in external breathing mechanics. Several early clinical studies compared the ASV mode with the traditional clinical physician-selected mode and found that ASV tends to choose a lower tidal volume and a higher frequency (and thus a lower inspiratory pressure) compared to the modes that clinicians choose [62, 63]. The results of two other studies suggest that ASV also adapts appropriately to changes in patient position and even to the transition to single lung ventilation [64, 65]. Another study suggested that the I: E ASV algorithm provided less Minute ventilation in patients with chronic obstructive pulmonary disease (COPD) [64]. In longer-term clinical studies, ASV has shown that the algorithm provided appropriate support in anesthetized patients [63], as well as in patients with respiratory failure [66]. More recent ASV assessments have focused on the ability of this mode to provide adequate protective small tidal volumes. Indeed, when respiratory system compliance is low, the ASV algorithm provides a protective tidal volume structure similar to that recommended by the ARDS Network [56]. Importantly, separate studies [67] find that ASV required less clinical physician/ventilator intervention, fewer arterial blood gas analyses, fewer ventilator errors, and a lower incidence of postoperative atelectasis on radiographs.

But there is a problem arise, when respiratory system compliance decreases to a lesser extent (e.g., patients with more moderate forms of ARDS). Under these conditions, the ASV algorithm tends to deliver often excessive tidal volumes up to 10 ml/kg [68]. The clinical significance of this phenomenon is unknown, but this is potential problem should be considered by the clinicians wishing to use this mode.

A further improvement of the mode ASV is the intellivent ASV mode. The iASV mode is more likely to provide a suboptimal range for maximum pressure. As in previous studies, the iASV mode provided

more manipulations and required less clinical physician interaction and variability in volumes and pressures in the airways [69]. Fot et al. [70] evaluated iASV compared ventilator weaning and SIMV plus PSV ventilation after coronary artery bypass surgery. They found that iASV and ventilator weaning had similar outcomes and reduced ventilator weaning compared to traditional methods. The authors noted a decrease in clinical physician interaction with the ventilator, but found no other benefits. Later, Arnal et al. [71] compared iASV either with PSV or with volume-controlled continuous mandatory ventilation in 60 subjects. They concluded that iASV reduces the number of manual ventilator parameter changes without a difference in the number of arterial blood gas studies or sedation use. Using a Likert scale, staff rated iASV as a method that is easier to use compared to conventional ventilation modes [71].

### **SMARTCARE PS MODE**

In SmartCare PS mode, the level of pressure support changes, based on the desired breathing rate, which is determined by the ETCO value. SmartCare PS regulates pressure support to maintain a “normal” range of ventilation in the patient ventilation. The normal ventilation range is usually defined as VT 300 ml, frequency breathing between 12 and 30 breaths/min, and ETCO less than 55 mm Hg. Range acceptable values for these variables may be changed by the clinician for a given patient, for example, in COPD or non-neurological injuries. In chronic processes, the acceptable level of ETCO is higher, while in acute ranges, ETCO is lower. Thus, decision-making is based on individual patient characteristics. SmartCare PS is designed to wean from the ventilator and, therefore, is not used clinically for respiratory support in adults with acute respiratory failure [72].

**Proportional assist-control ventilation (PAV)** is a new approach to assisted ventilation [73]. The PAV mode is an intermittent controlled “test breath” in which the resistance and compliance of the external respiratory system are determined. In this mode, measured flow and volume can be used to calculate both resistive and elastic work. The clinician must set the desired proportion of total work to be performed by the ventilator.

The ventilator then measures the flow and volume requirement with each breath and adds both pressure and flow to provide the selected proportion of the work of breathing. PAV can be compared to power steering in a car, an analogy that has a lot of rules. Like PAV, power steering reduces the work to turn the wheels, but does not automatically drive the car – the driver must control the final characteristics of the car, as the patient must ultimately control the intensity of breathing and the choice of breathing pattern. In PAV mode, the more effort the patient makes, the more pressure, flow, and volume are delivered. Unlike assist-volume, when flow and volume are not triggered by effort and when, in fact, the pressure exerted can be “dumped” by effort. PAV also contrasts with pressure assist, when flow and volume are triggered by effort, but not pressure. Because PAV requires sensors in the ventilator circuit to measure patient effort, this mode is susceptible to the same sensor performance and built-in PEEPi problems that affect breathing in other modes [74]. As with other assistive modes, the clinician The physician should set PEEP and FiO<sub>2</sub> [75,76]. These studies also demonstrated the effectiveness of safety mechanisms to prevent excessive pressure (“runaway”), and also emphasize the importance of having adequate alarm systems, because PAV provides minimal support with little effort and no support if effort ceases. Thus, PAV should be used with caution in patients with unreliable respiratory drives (e.g., neurological disorders, variable sedation/opioid use). Clinical studies have compared PAV with other modes of assisted ventilation, and this mode has been found to create favorable conditions for little stress on the muscle and patient comfort [77, 78]. Whether the use of PAV mode improves significant disease outcomes (e.g., sedation needs, shorter mechanical ventilation needs) will be determined in future studies.

**Flow-controlled exhalation–flow controlled expiration (FLEX).** This mode of mechanical ventilation has only been tested in experiment [79]. FLEX slows expiratory peak flow rate and maintains reduced flow throughout exhalation, thus prolonging the non-zero flow phase (and, in turn, the total expiratory flow time) and increases the average pressure in the airways. This is expected to

reduce airway collapse and edema formation, especially in damaged lungs. FLEX is expected to reduce lung injury.

**'Expiratory ventilation assistance'-'expiratory ventilation assistance'(EVA)** The Enk working group originally developed a new ventilation mode 'expiratory ventilation assistance' (EVA) as an emergency ventilation system designed to restore ventilation through a tracheostomy. A detailed description of the EVA operating principle can be found in the articles by Hamaekers et al [80, 81]. As a further development of the EVA mode, a prototype of automated control of mechanical ventilation was developed. This mode produces fully controlled exhalation, linearizing the expiration flow in accordance with the controlled exhalation flow. In the study by Schmidt et al. [82] positioned the EVA mode as an automated ventilator. Compared to the usual VCV protocol, when using expiratory control, the EVA mode increases lung tissue aeration and improves oxygenation, without changing the minimum and maximum pressure in the airways. These study results give reason to assume that the EVA mode may represent a new approach to protective lung ventilation. However, clinical studies are needed to confirm the feasibility and effectiveness of using this mode in practice.

Proposed automatic modes closed-loop control is thought to offer many advantages over traditional physician control regarding lung ventilation. The sheer number of physiological variables available to the clinician is daunting. Along with the dozens of clinical decisions that must be made daily, the potential for error is significant.

Despite our best efforts, the ability of the human mind to deal with this volume of information is limited [83]. Long night hours only exacerbate these problems. Therefore, automatic ventilation control modes offer additional benefits, which include reducing labor costs, providing more adequate treatment in the absence of experts, reducing ventilator weaning time, and making recommendations based on factual data [84].

## REASONS FOR INSUFFICIENT VENTILATOR PERFORMANCE

A good understanding of the concept under discussion describing ventilator-related complications and problem solving should become second nature to the attending anesthesiologist. The most common types of corrections that should be made when choosing ventilation parameters should ensure normal oxygenation and ventilation.

**Hypoxia:** As discussed earlier, to improve oxygenation, an increase in FiO<sub>2</sub> and PEEP (T high and P high for APRV) is used. To correct hypoxemia, either of these parameters (or both) is increased. Particular attention should be paid to the possible negative effects of raising PEEP, which can cause barotrauma and hypotension. Raising FiO<sub>2</sub> also adds its problems, as high FiO<sub>2</sub>, can cause oxidative damage to the alveoli. Another important aspect of oxygen content should determine the oxygenation threshold. In general, there is little benefit from maintaining an oxygen saturation threshold above 92-94% except in cases of carbon monoxide poisoning. A sudden decrease in oxygen saturation should raise suspicion of dystopia of the intubation tube, pulmonary embolism, pneumothorax, pulmonary edema, atelectasis, or obstructive syndrome.

To improve oxygenation against the background of PEEP a recruitment maneuver is used, which includes a periodic increase in airway pressure during mechanical ventilation in order to open ('recruit into breathing') collapsed acini and increases the number of alveoli involved in periodic ventilation. Recruitment maneuvers are often used to treat patients who have acute respiratory distress syndrome (ARDS), but the effect of this treatment on disease outcomes has not been well established. This conclusion is based on the results of 5 RCTs, which included an "open lung strategy," in which the intervention group differed from the control group only by the use of a recruitment maneuver (which included higher PEEP, or different ventilation modes with higher plateau pressure). A ventilation strategy that included recruitment in patients with ARDS reduced ICU mortality without increasing the risk of barotrauma, but did not affect 28-day and in-hospital mortality (low level of evidence) [85]. Another mode that is used to reducing hypoxemia – ventilation in prone position, which can improve

the mechanics of external respiration and gas exchange in the lungs, and, accordingly, the results of treatment in patients with ARDS [86].

## **HYPERCAPNIA**

To change the content of CO<sub>2</sub> in the blood, it is necessary to change alveolar ventilation. To do this, you need to increase breathing tidal volume or respiratory rate (T low and P low with APRV). Increasing the respiratory rate or tidal volume, as well as increasing T low, will increase ventilation and decrease CO<sub>2</sub>. But it is necessary to take into account that by increasing the respiratory rate, we simultaneously we increase the volume of dead space and this measure may not be as effective as increasing the tidal volume. When increasing the volume or frequency, special attention should be paid to the volume flow loop in order to prevent the development of auto-PEEP.

Another important circumstance is the increase pressure. As discussed above, two pressures are important in the system: peak and plateau. Possible reasons: (1) check the patency of the intubation tube, (2) obstruction by sputum-aspiration of sputum, (3) bronchospasm – the solution is to use bronchiolitis agents.

Increased peak pressure and pressure plateau: compliance issues.

Possible causes of these disorders include:

- Intubation of the main bronchus: diagnosis auscultation of the patient lungs, unilateral absent of breath sounds and their absence in the contralateral lung (atelectasis lung).
- Pneumothorax: diagnosis by auscultation - breathing unilateral and hyper resonant breathing in the contralateral lung. Both complications are reliable diagnosed using X-ray radiography.
- Atelectasis: (may occur both with endo bronchial intubation and obstruction of the afferent bronchus). Initial management consists of restoring bronchial patency, if necessary, using fiber optic bronchoscopy and then recruiting maneuvers.
- ARDS: Use of low respiratory volumes, depending on the severity ARDS ventilation with high PEEP, ventilation in the prone position.

## **Dynamic hyperinflation or auto-PEEP:**

This is a process in which part of the inhaled air is not completely exhaled at the end of the respiratory cycle. The accumulation of remained exhaled air will increase the pressure in the airways and cause barotrauma and hypotension. The patient will be difficult to ventilate. To prevent and resolve auto-PEEP, it is necessary to provide enough time for air to exhale. The goal in management is to reduce the ratio of inspiration to expiration, this can be achieved by reducing the respiratory rate and/or tidal volume (a higher volume will require a longer time to exhale), and increase the inspiration flow (if air is introduced quickly, the expiratory time will be longer at any given respiratory rate).

The same effect can be achieved by using a square wave shape for the inspiratory flow, this means that we can force the ventilator to deliver full flow from the beginning to the end of inflation. Other methods that can be implemented should guarantee an appropriate sedative effect in order to apply hyperventilation and the use of bronchiolitis agents and steroids to reduce airway obstruction. If auto PEEP causes severe hypotension, briefly disconnecting the patient from the ventilator may be a life-saving measure.

## **DESYNCHRONIZATION OF PATIENT'S BREATHING WITH THE VENTILATOR**

Another common problem that occurs in patients on mechanical ventilation is desynchronization of the patient's breathing with the ventilator, usually referred to as “patient fighting the with the ventilator”. Important reasons include: hypoxia, auto-PEEP, hypercapnia, pain and discomfort. Clinical signs desynchronization - tachypnea, shortness of breath, sweating and tachycardia. After exclusion such important causes as pneumothorax or atelectasis, it is necessary to achieve comfort in the patient, provide adequate sedative effect, and analgesia. It is necessary to consider changing the ventilation

mode, since some patients may respond better to different ventilation modes. Convenient interactive support requires that the clinical doctor optimized all three phases of respiratory cycle: trigger start of breathing, delivery flow and circulation. Graphically, desynchronization manifests itself with excessive peaks of negative pressure in the airways, preceding the trigger start of breathing or the absence of any flow delivery in response to observed effort, as well as a significant decrease in pressure in the airways during inhalation. Cycle that there is desynchronization is manifested by prolonged respiratory effort of the patient and sometimes a double trigger, if the cycle is too early. Desynchronization of the cycle can also be manifested by increased pressure in the airways during expiratory muscle activity, if the cycle is too long.

Common strategies to optimize synchronization during trigger breathing, flow delivery, and circulation include several options. Optimal trigger breathing involves the sensitivity of the trigger mechanism of assisted breathing, which is as sensitive and sensitive as possible without auto cycling [113].

## NEGATIVE CONSEQUENCES (COMPLICATIONS) OF MECHANICAL VENTILATION

How does the patient feel while is he on a ventilator? The ventilator itself does not cause pain. Some people don't like the feeling of having an intubation tube. They cannot speak because the tube passes between the vocal folds into the trachea. They also cannot eat orally. A person may feel uncomfortable, because air is blown into their lungs. Sometimes a person will try to exhale when the ventilator will try to exhale. People who are on mechanical ventilation need to be given sedatives and/or painkillers, so that they feel more comfortable. But these drugs can cause drowsiness and cause depression of breathing center. Sometimes medications are used that temporarily prevent muscle movement (neuromuscular block agents) to allow a person synchronize breathing with the ventilator. These drugs are usually used when a person has a very severe injury lung damage; discontinuation of relaxants should be done as soon as possible, as long-term their use is associated with a number of negative consequences and, above all, with dystrophy respiratory muscles [87].

- **Respiratory tract and lung infections:** Tracheal intubation the tube and tracheostomy contribute to microbial invasion into the lower respiratory ways. This can cause complications such as ventilator-associated pneumonia (VAP). For the prevention and treatment of VAP, the patient's position with the head end raised, thorough toileting of the respiratory tract and rational antibiotic therapy are usage.
- **Pneumothorax.** Mechanical ventilation increases the risk of lung damage with air escaping into the pleural cavity. If this complication occurs, air must be immediately removed from this space. To decompress the pleural cavity, drainage is usually necessary and the drainage tube should remain for some time to ensure that the failure has stopped.
- **Ventilator-induced lungs Injury (VILI).** VILI is probably the most important problem facing clinicians providing long-term mechanical ventilation. Lung damage associated with mechanical assisted breathing is often called ventilator-induced lung injury or VILI [88 – 90]. Today, the physician must strike a balance between providing adequate gas exchange in the lungs and preventing VILI associated with high positive airway pressure and high oxygen concentration in the gas mixture. On the one hand, patients with respiratory failure need adequate oxygenation and maintenance of acid base balance; on the other hand, the lungs are fragile structures, easily injured by excessive stress, repeated collapse and opening of alveoli and high oxygen concentration. This problem is exacerbated by the fact that lung damage is usually heterogeneous, so gas exchange can be improved in one area (for example, at a higher peak pressure), but cause damage in another [91].

The main cause of VILI is damage alveoli caused by over distension at the end inhalation (over distension) and collapse at the end exhalation, long periods of periodic oxygenation higher than the normal physiological levels and cyclic recurring atelectasis that occurs during periods of ventilation with positive pressure. In general, the risk of VILI increases when the increase in plato pressure by more than 30 cmH<sub>2</sub>O when tidal volume, is more than 8mL/kg (ideal body weight). It is important to

note that VILI is associated with systemic inflammatory response with the release of cytokines and translocation of bacteria from the lungs. [92]

VILI is a component that causes multiorgan dysfunction, which has high mortality rate. The frequency of VILI is 24% in patients on mechanical ventilation not associated with ARDS [93,94]. For prophylaxis and treatment of VILI, is important to ventilate patient with a lung protective method which comprises of low tidal volume and PEEP, plato pressure not more than 30 cmH<sub>2</sub>O. [95]

**NON INVASIVE VENTILATION AND ITS INDICATION:** The terms non-invasive ventilation (NIV) of the lungs refers to all ventilation without inserting tubes in the patient trachea. [96]. The main indication for NIV includes prevention of complications associated with intubation of trachea [97]. Reduction of patient's discomfort and preservation of patients protective reflexes of the airways. One of the advantages of NIV is its usage in ventilating patient with chronic obstructive airway disease [ COPD] [98], acute cardiogenic edema and patient s with immunodeficiency [99]. A study multicenter of patient in the intensive care unit in France, a study that continued for 15 years show that the use of NIV increased within that period up to 42% in 2011, NIV as a method of choice reduced the mortality rate on the 60<sup>th</sup> day and frequency of infection compared to invasive mechanical ventilation. [100]. Increasing role of NIV in the intensive care units in the years has brought about production of devices for its usage and also new ventilation settings. The important factor is the removal of gas leakages at the patient-ventilator interface, and also reduction of dead space.

#### **NIV REGIMES:**

**CONTINUOUS POSITIVE AIRWAY PRESSURE [CPAP].** This means that there is continuous positive airway pressure in the airways, the simplest mode of NIV. In this mode the ventilator provides a controlled positive pressure in the airways all through the respiration cycles, both expiratory and inspiratory. CPAP does not directly influence respiration but increases the volume of alveoli and surface areas of gases exchange. [101], increases lung compliance and reduces work load in breathing muscles. [102] PEEP reduces venous return to the heart thereby reducing the pre and afterload without interfering with myocardial contractility. [101] In general cardiac output increases without changes myocardial oxygen demand [103]. CPAP relives alveoli from collapse, lowers the works of respiratory muscles and improves lung compliance [104]. It also reduces respiratory distress, hemodynamic stability [105], oxygenation, reduces hypercapnia and hypoxemia in intubation [106].

**BIPHASIC POSITIVE AIRWAYS PRESSURE [BiPAP],** The BiPAP mode uses a lower pressure as the ventilator makes an effort to inhale and provides positive pressure breathing support in the airways during exhalation [107]. The addition of inspiratory positive assistance reduces the work of breathing during inhalation [108]. BiPAP is more effective in reducing the work of breathing and reducing oxygen consumption [104] compared to CPAP. There are more advantages of BiPAP which includes, reduction in RR, HR, there is also improvement in oxygen concentration in the arterial blood (increases PaO<sub>2</sub> in blood and reduces PaCO<sub>2</sub> within 30 minutes) [109]. BiPAP also reduces the need for endotracheal intubation and its associated complication [110]. BiPAP reduces the risk of nosocomial infection when compared to invasive mechanical ventilation [111]. This mode can be used in the process of weaning from respirator. Non-invasive ventilation of the lungs is used as an initial auxiliary breathing in patients with COPD and ARDS, but the benefit of using this method is unclear, and in patients in whom gas exchange does not improve, intubation may be delayed, which may lead to adverse outcomes. Is therefore important and mandatory to have a clear indication for selecting patients who will benefit from this technique [112].

#### **Conclusion**

In Conclusion, the impact of mechanical ventilation is intended to make readily available tool that simplify mechanical ventilation discussion the problems arising during prolonged mechanical ventilation, and how they are resolved. Highlighting the commonly used modes of mechanical ventilation in clinical practice. Mechanical ventilation remains a vital supportive intervention in managing respiratory failure by ensuring adequate oxygenation and ventilation while reducing the

work of breathing. However, it does not treat the underlying disease but serves as a bridge to recovery or further medical intervention. This is intended to make readily available a tool that simplify mechanical ventilation discussion the problems arising during prolonged mechanical ventilation, and how they are resolved. The most frequently used modes of mechanical ventilation are also discussed. Therefore, the effectiveness of mechanical ventilation largely depends on the clinician's understanding of respiratory physiology and the principles underlying each ventilatory mode. Proper selection and adjustment of ventilator parameters are essential to ensure patient safety, comfort, and optimal outcomes. Additionally, adherence to lung-protective strategies and careful monitoring can help minimize complications such as ventilator-induced lung injury.

### Recommendations

1. Clinicians should tailor ventilator settings and modes based on the patient's underlying condition, lung mechanics, and response to treatment rather than relying on a single standard mode.
2. Consistent use of low tidal volumes, controlled airway pressures, and careful FiO<sub>2</sub> titration should be prioritized to reduce the risk of ventilator-induced lung injury and improve patient outcomes.
3. Continuous monitoring (ABG analysis, capnography, and ventilator parameters) alongside structured weaning strategies should be implemented to reduce the duration of mechanical ventilation and associated ICU resource burden.

### REFERENCES

1. Laghi F., Tobin M.J. Indications for mechanical ventilation. In: Tobin M.J., editor. Principles and practice of mechanical ventilation, 3rd ed. New-York, NY: McGraw- Hill; 2012. pp. 129–162.
2. Tobin M.J. Mechanical ventilation. *N. Engl. J. Med.* 1994; 330:1056–1061.
3. Al-Hegelan M., MacIntyre N.R. Novel Modes of Mechanical Ventilation. *Semin Respir Crit Care Med* 2013; 34(04): 499-507.
4. Tobin M., Manthous C. Mechanical Ventilation. *Am. J. Respir. Crit. Care Med.* 2017;196(2): P3-P4.
5. Engoren M., Arslanian-Engoren C., Fenn-Buderer N. Hospital and long-term outcome after tracheostomy for respiratory failure. *Chest* 2004; 125:220–7.
6. Carson S.S. Outcomes of prolonged mechanical ventilation. *Curr. Opin. Crit. Care* 2006; 12:405–11.
7. Cox C., Carson S.S, Holmes G., et al. Increase in tracheostomy for prolonged mechanical ventilation in North Carolina, 1993-2002. *CritCareMed* 2004; 32:2219–26.
8. Esteban A., Frutos F., Tobin M.J., et al. A comparison off our methods of weaning patients from mechanical ventilation. Spanish Lung Failure Collaborative Group. *N. Engl. J. Med.* 1995; 332:345–50.
9. Carson S.S. Outcomes of prolonged mechanical ventilation. *Curr. Opin. Crit. Care* 2006; 12:405–11.
10. Divo M.J., Murray S., Cortopassi F., et al. Prolonged mechanical ventilationin Massachusetts: the prevalence survey. *Respir Care* 2010; 55:1693–8.
11. King A.C. Long-term home mechanical ventilationin the United States. *Respir Care* 2012; 57:921–30.
12. Al-Hegelan M., Mac-Intyre N.R. Novel Modes of Mechanical Ventilation. *Semin Respir Crit Care Med* 2013; 34(04): 499-507.
13. Slutsky A.S. Mechanical ventilation. American College of Chest Physicians' Consensus Conference. *Chest* 1993; 104(6):1833–1859. Erratum: *Chest* 1994;106(2):656.

14. Moraò Carpio A.L., Mora J.I. Ventilation Assist Control. Stat Pearls [Internet]. TreasureIs land (FL): Stat Pearls Publishing; 2018-Oct 27.
15. Santa Cruz R., Rojas J.I., Nervi R., Heredia R., Ciapponi A. High versus low positive end-expiratory pressure (PEEP) levels for mechanically ventilated adult patients with acute lung injury and acute respiratory distress syndrome. *Cochrane Database Syst Rev.* 2013 Jun6;(6): CD009098.
16. Tobin MJ. Advances in mechanical ventilation. *N. Engl. J. Med.* 2001; 344:1986–1996.
17. Tobin M.J., Jubran A., Laghi F. Fighting the ventilator. In: Tobin M.J, editor. Principles and practice of mechanical ventilation, 3rd ed. New-York, NY: McGraw- Hill; 2012. pp. 1121–1136.
18. Puddy A., Younes M. Effect of inspiratory flow rate on respiratory output in normal subjects. *Am. Rev. Respir. Dis.* 1992; 146:787–789.
19. Laghi F., Segal J., Choe W.K., Tobin M.J. Effect of imposed inflation time on respiratory frequency and hyperinflation in patients with chronic obstructive pulmonary disease. *Am. J. Respir. Crit. Care Med.* 2001; 163:1365–1370.
20. Slutsky A.S. History of mechanical ventilation. From Vesalius to ventilator-induced lung injury. *Am. J. Respir. Crit. Care Med.* 2015;191(10):1106-1115.
21. Goligher E.C., Douflé G., Fan E. Update in mechanical ventilation, sedation and outcomes 2014. *Am. J. Respir. Crit. Care Med.* 2015;191(12):1367-1373.
22. Rittayamai N., Katsios C.M., Beloncle F., Friedrich J.O., Mancebo J., Brochard L. Pressure-controlled vs volume controlled ventilation in acute respiratory failure: a physiology-based narrative and systematic review. *Chest.* 2015;148(2):340-355.
23. Chatburn R.L. Understanding mechanical ventilators. *Expert. Rev. Respir. Med.* 2010;4(6):809-819.
24. Branson R.D., Chatburn R.L. Controversies in the critical care setting. Should adaptive pressure control modes be utilized for virtually all patients receiving mechanical ventilation? *Respir. Care.* 2007;52(4):478-485.
25. Lellouche F., Brochard L. Advanced closed loops during mechanical ventilation (PAV, NAVA, ASV, Smart Care). *Best Pract. Res. Clin. Anaesthesiol.* 2009;23(1):81-93.
26. Campbell R.S., Davis B.R. Pressure-controlled versus volume-controlled ventilation: does it matter? *Respir. Care.* 2002 Apr;47(4):416-24.
27. Chacko B., Peter J.V., Tharyan P., John G., Jeyaseelan L. Pressure-controlled versus volume-controlled ventilation for acute respiratory failure due to acute lung injury (ALI) or acute respiratory distress syndrome (ARDS). *Cochrane Database Syst Rev.* 2015 Jan 14;1:CD008807.
28. Sinderby C. Neurally adjusted ventilatory assist (NAVA). *Minerva Anesthesiol* 2002;68(5):378–380.
29. Sinderby C., Navalesi P., Beck J., et al. Neural control of mechanical ventilation in respiratory failure. *Nat. Med.* 1999;5(12):1433–1436.
30. Coisel Y., Chanques G., Jung B., et al. Neurally adjusted ventilator assist in critically ill postoperative patients: a crossover randomized study. *Anesthesiology* 2010;113(4): 925–935.
31. Terzi N., Pelieu I., Guittet L., et al. Neurally adjusted ventilator assisting patients recovering spontaneous breathing after acute respiratory distress syndrome: physiological evaluation. *Crit. Care Med.* 2010; 38(9): 1830–1837.
32. Bengtsson J.A., Edberg K.E. Neurally adjusted ventilator assist in children: an observational study. *Pediatr. Crit. Care Med.* 2010;11. (2):253–257.

33. Chang H.K. Mechanisms of gas transport during ventilation by high-frequency oscillation. *J. Appl. Physiol.* 1984;56(3):553–563.
34. Froese A.B. High-frequency oscillatory ventilation for adult respiratory distress syndrome: let's get it right this time! *Crit. Care Med.* 1997;25(6):906–908.
35. Fessler H.E., Hager D.N., Brower R.G. Feasibility of very high-frequency ventilation in adults with acute respiratory distress syndrome. *Crit. Care. Med.* 2008;36(4):1043–1048.
36. Derdak S. High-frequency oscillatory ventilation for acute respiratory distress syndrome in adult patients. *Crit Care Med* 2003;31(4, Suppl): S317–S323.
37. Sud S., Sud M., Friedrich J.O., et al. High frequency oscillation in patients with acute lung injury and acute respiratory distress syndrome (ARDS): systematic review and meta-analysis. *BMJ*2010;340:c2327.
38. Young D., Lamb S.E., Shah S., et al; OSCAR Study Group. High frequency oscillation for acute respiratory distress syndrome. *N. Engl. J. Med.* 2013;368(9):806–813.
39. Derdak S. High-frequency oscillatory ventilation for acute respiratory distress syndrome in adult patients. *Crit. Care Med.* 2003;31(4, Suppl): S317–S323.
40. Ferguson ND, Cook DJ, Guyatt GH, et al; OSCILLATE Trial Investigators; Canadian Critical Care Trials Group *N Engl J Med*2013;368(9):795–805.
41. Plataki M, Hubmayr RD. The physical basis of ventilator induced lung injury. *Expert Rev Respir Med* 2010;4(3):373–385.
42. Chung K.K., Wolf S.E., Renz E.M., et al. High-frequency percussive ventilation and low tidal volume ventilation in burns: a randomized controlled trial. *Crit. Care Med.* 2010;38(10):1970–1977.
43. Habashi N.M. Other approaches to open-lung ventilation: airway pressure release ventilation. *Crit. Care Med.* 2005;33(3, Suppl): S228–S240.
44. Putensen C., Zech S., Wrigge H., et al. Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. *Am. J. Respir. Crit. Care Med.* 2001;164(1):43–49.
45. Varpula T., Valta P., Niemi R., Takkunen O., Hynynen M., Pettilä V.V. Airway pressure release ventilation as a primary ventilator mode in acute respiratory distress syndrome. *Acta Anaesthesiol Scand* 2004;48(6):722–731.
46. Singer B.D., Corbridge T.C. Pressure modes of invasive mechanical ventilation. *South. Med. J.* 2011;104(10):701-709.
47. Cole A.G.H., Weller S.F., Sykes M.K. Inverse ratio ventilation compared with PEEP in adult respiratory failure. *Intensive Care Med* 1984;10(5):227–232.
48. Tharratt R.S., Allen R.P., Albertson T.E. Pressure controlled inverse ratio ventilation in severe adult respiratory failure. *Chest* 1988;94(4):755–762.
49. Putensen C., Zech S., Wrigge H., et al. Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. *Am. J. Respir. Crit. Care Med.* 2001;164(1):43–49.
50. Varpula T., Valta P., Niemi R., Takkunen O., Hynynen M., Pettilä V.V. Airway pressure release ventilation as a primary ventilator mode in acute respiratory distress syndrome. *Acta Anaesthesiol Scand* 2004;48(6):722–731.

51. Maxwell R.A., Green J.M., Waldrop J., et al. A randomized prospective trial of airway pressure release ventilation and low tidal volume ventilation in adult trauma patients with acute respiratory failure. *J. Trauma* 2010;69(3):501–510.
52. Gonzalez M., Arroliga A., Frutos-Vivar F., et al. Airway Pressure release ventilation versus assist-control ventilation: a comparative propensity score and international cohort study. *Int. Care Med.* 2010;36(5): 817–827.
53. Branson R.D. Dual control modes, closed loop ventilation, handguns, and tequila. *Respir Care* 2001;46(3):232–233.
54. Branson R.D., Davis K. Jr. Dual control modes: combining volume and pressure breaths. *Respir. Care Clin. N. Am.* 2001;7(3):397–408.
55. Tobin M.J., Jubran A., Laghi F. Fighting the ventilator. In: Tobin M.J., editor. *Principles and practice of mechanical ventilation*, 3rd ed. New-York, NY: McGraw- Hill; 2012. pp. 1121–1136.
56. Veelo D.P., Dongelmans D.A., Binnekade J.M., Paulus F., Schultz M.J. Adaptive support ventilation: a translational study evaluating the size of delivered tidal volumes. *Int. J. Artif. Organs* 2010;33(5):302–309.
57. Sulemanji D., Marchese A., Garbarini P., Wysocki M., Kacmarek R.M. Adaptive support ventilation: an appropriate mechanical ventilation strategy for acute respiratory distress syndrome? *Anesthesiology.* 2009;111(4):863-870.
58. Agarwal R., Srinivasan A., Aggarwal A.N., Gupta D. Adaptive support ventilation for complete ventilator support in acute respiratory distress syndrome: a pilot, randomized controlled trial. *Respirology.* 2013;18(7): 1108-1115.
59. Arnal J., Wysocki M., Novotni D., et al. Safety and efficacy of a fully closed-loop control ventilation (IntelliVent-ASV®) in sedated ICU patients with acute respiratory failure: a prospective randomized crossover study. *Intensive Care Med.* 2012;38(5):781-787.
60. Turner D.A., Rehder K.J., Cheifetz I.M. Nontraditional modes of mechanical ventilation: progress or distraction? *Expert. Rev. Respir. Med.* 2012;6(3):277-284.
61. Dongelmans D.A., Paulus F., Veelo D.P., Binnekade J.M., Vroom M.B., Schultz M.J. Adaptive support ventilation may deliver unwanted respiratory rate-tidal volume combinations in patients with acute lung injury ventilated according to an open lung concept. *Anesthesiology.* 2011;114(5):1138-1143.
62. Laubscher T.P., Heinrichs W., Weiler N., Hartmann G., Brunner J.X. An adaptive lung ventilation controller. *IEEE Trans Biomed. Eng.* 1994;41(1):51–59.
63. Weiler N., Eberle B., Latorre F., von Paczynski S., Heinrichs W. Adaptive lung ventilation (AVL). Evaluation of new closed loop regulated respiration algorithm for operation in the hyperextended lateral position [in German]. *Anesthetist* 1996;45(10):950–956.
64. Belliato M., Palo A., Pasero D., Iotti G.A., Mojoli F., Braschi A. Evaluation of the adaptive support ventilation (ASV) mode in paralyzed patients. *Int. J. Artif. Organs.* 2004;27(8):709– 716.
65. Tassaux D., Dalmas E., Gratadour P., Jolliet P. Patient ventilator interactions during partial ventilatory support: a Preliminary mandatory ventilation plus inspiratory pressure support. *Crit Care Med* 2002;30(4):801–807.
66. Linton D.M., Potgieter P.D., Davis S., Fourie A.T., Brunner J.X., Laubscher T.P. Automatic weaning from mechanical ventilation using an adaptive lung ventilation controller. *Chest* 1994;106(6):1843–1850.
67. Esquinas A.M., Cravo J., De-Santo L.S. Adaptive support ventilation weaning protocols in cardiac surgical patients: complex speculations with little practical impact. *J. Crit. Care* 2017; 37:250.

68. Dongelmans D.A., Paulus F., Veelo D.P., Binnekade J.M., Vroom M.B., Schultz M.J. Adaptive support ventilation may deliver unwanted respiratory rate-tidal volume combinations in patients with acute lung injury ventilated according to an open lung concept. *Anesthesiology* 2016; 82:657–68.
69. Bialais E., Wittebole X., Vignaux L., et al. Closed-loop ventilation mode (IntelliVent-ASV) in intensive care unit: a randomized trial. *Minerva Anestesiologica* 2016; 82:657–68.
70. Fot E.V., Izotova N.N., Yudina A.S., et al. Automated weaning from mechanical ventilation after off-pump coronary artery bypass grafting. *Front Med. (Lausanne)* 2017; 4:31.
71. Arnal J.M., Garnero A., Novotni D., et al. Closed loop ventilation mode in intensive care unit: a randomized controlled clinical trial comparing the numbers of manual ventilator setting changes. *Minerva Anestesiologica* 2018; 84(1):58–67.
72. Dojat M., Harf A., Touchard D., et al. Evaluation of a knowledge-based system providing ventilatory management and decision for extubation. *Am. J. Respir. Crit. Care Med.* 1996; 153:997–1004.
73. Younes M. Proportional assist ventilation, a new approach to ventilator support. *Theory. Am. Rev. Respir. Dis.* 1992; 145(1): 114–120.
74. Grasso S., Ranieri V.M. Proportional assist ventilation. *Respir. Care Clin. N. Am.* 2001; 7(3):465–473.
75. Sassoon C.Sh. Triggering of the ventilator in patient ventilator interactions. *Respir. Care* 2011; 56(1):39–51.
76. Dreher M., Kabitz H.J., Burgardt V., Waltersbacher S., Windisch W. Proportional assist ventilation improves exercise capacity in patients with obesity. *Respiration* 2010; 80(2):106–111.
77. Moderno E.V., Yamaguti W.P., Schettino G.P., et al. Effects of proportional assisted ventilation on exercise performance in idiopathic pulmonary fibrosis patients. *Respir Med* 2010; 104(1):134–141.
78. Grasso S., Ranieri V.M. Proportional assist ventilation. *Respir Care Clin. N. Am.* 2001; 7(3):465–473.
79. Mitrouska J., Xirouchaki N., Patakas D., Siafakas N., Georgopoulos D. Effects of chemical feedback on respiratory motor and ventilator output during different modes of assisted mechanical ventilation. *Eur Respir J* 1999; 13(4):873–882.
80. Goebel U., Haberstroh J., Foerster K., Dassow C., Priebe H.-J., Guttman J., Schumann S. Flow-controlled expiration: a novel ventilation mode to attenuate experimental porcine lung injury. *British Journal of Anesthesia* 113 (3): 474–83 (2014).
81. Hamaekers A.E., Gotz T., Borg P.A., et al. Achieving an adequate minute volume through a 2 mm trans tracheal catheter in simulated upper airway obstruction using a modified industrial ejector. *Br. J. Anaesth.* 2010; 104: 382–386.
82. Hamaekers A.E., Borg P.A., Gotz T., et al. Ventilation through a small-bore catheter: optimizing expiratory ventilation assistance. *Br. J. Anaesth.* 2011; 106:403–409.
83. Schmidt J., Wenzel C., Mahn M., Spassov S., Schmitz H.C., Borgmann S., Lin Z., Haberstroh J., Meckel S., Eiden S., Wirth S., Buerkle H., Schumann S. Improved lung recruitment and oxygenation during mandatory ventilation with a new expiratory ventilation assistance device A controlled interventional trial in healthy pigs. *Eur. J. Anaesthesiol.* 2018; 35:736–744.
84. Miller G. The magical numbers even plus or minus two: some limits on our capacity for processing information. *Psychol. Rev.* 1956; 63:81–97.
85. Branson R.D. Modes to facilitate ventilator weaning. *Respir Care* 2012; 57:1635–48.

86. Hodgson C., Goligher E.C., Young M.E., Keating J.L., Holland A.E., Romero L., Bradley S.J., Tuxen D. Recruitment maneuvers for adults with acute respiratory distress syndrome receiving mechanical ventilation. *Cochrane Database Syst Rev.* 2016 Nov 17;11:CD006667.
87. Bloomfield R., Noble D.W., Sudlow A. Prone position for acute respiratory failure in adults. *Cochrane Database Syst. Rev.* 2015 Nov13;(11):CD008095.
88. Tobin M., Manthous C. Mechanical Ventilation. *Am. J. Respir. Crit. Care Med.* 2017 Jul. 15;196(2): P3-P4.
89. Kolobow T., Moretti M.P., Fumagalli R., et al. Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation. An experimental study. *Am. Rev. Respir. Dis.* 1987;135(2):312–315.
90. Dreyfuss D., Saumon G. Ventilator-induced lung injury: lessons from experimental studies. *Am. J. Respir. Crit. Care Med.* 1998;157(1):294–323.
91. Tremblay L., Valenza F., Ribeiro S.P., Li J., Slutsky A.S. Injurious ventilatory strategies increase cytokines and cfos m-RNA expression in an isolated rat lung model. *J Clin Invest* 1997;99(5):944–952.
92. International consensus conferences in intensive care medicine: ventilator-associated lung injury in ARDS. *Am. J. Respir. Crit. Care Med.* 1999;160(6):2118–2124.
93. Nahum A., Hoy J., Schmitz L., Moody J., Shapiro R., Marini J.J. Effect of mechanical ventilation strategy on dissemination of intratracheally instilled *Escherichia coli* in dogs. *Crit. Care Med.* 1997;25(10):1733–1743.
94. Rouby J.J., Brochard L. Tidal recruitment and over inflation in acute respiratory distress syndrome: yinandyang. *Am. J. Respir. Crit. Care Med.* 2007;175(2): 104–106.
95. Villar J., Kacmarek R.M., Perez-Mendez L., Aguirre-Jaime A. A high positive end-expiratory pressure, low tidal volume ventilator strategy improves outcome in persistent acute respiratory distress syndrome: a randomized, controlled trial. *Crit. Care Med.* 2006;34(5):1311–1318.
96. Slutsky A.S., Ranieri V.M. Ventilator-induced lung injury. *N. Engl. J. Med.* 2013; 369:2126–2136.
97. Bello G., De Pascale G., Antonelli M. Noninvasive Ventilation, *Clinics in Chest Medicine*, 2016, V 37, Issue 4, P. 711-721.
98. Pingleton S.K. Complications of acute respiratory failure. *Am. Rev. Respir. Dis.* 1988; 137:1463–93.
99. Nava S., Hill N. Non-invasive ventilation in acute respiratory failure. *Lancet* 2009; 374:250–9.
100. Keenan S.P., Sinuff T., Burns K.E., et al, Canadian Critical Care Trials Group/Canadian Critical Care Society Noninvasive Ventilation Guidelines Group. Clinical practice guidelines for the use of noninvasive positive-pressure ventilation and noninvasive continuous positive airway pressure in the acute care setting. *CMAJ* 2011;183: E195–214.
101. Schnell D., Timsit J.F., Darmon M., et al. Noninvasive mechanical ventilation in acute respiratory failure: trends in use and outcomes. *Intensive. Care Med.* 2014; 40:582–91.
102. Chadda K., Annane D., Hart N., Gajdos P., Raphael J.C., Lofaso F. Cardiac and respiratory effects of continuous positive airway pressure and noninvasive ventilation in acute cardiac pulmonary edema. *Crit Care Med.* 2002;30(11):2457-2461.
103. Cross A.M. Review of the role of non-invasive ventilation in the emergency department. *J. Accid. Emerg. Med.* 2000; 17:79-85.

- 104.Masip J., Betbese A.J., Paez J., Vecilla F., Canizares R., Padro J. Non-invasive pressure support ventilation versus conventional oxygen therapy in acute cardiogenic pulmonary oedema: a randomized trial. *Lancet*. 2000; 356:2126-2132.
- 105.Nava S., Carbone G., Dibattista N., Bellone A., Baiardi P., Cosentini R. Noninvasive ventilation in cardiogenic pulmonary edema. *Am. J. Respir. Crit. Care Med*. 2003; 168:1432-1437.
- 106.Kaminski J., Kaplan P.D. The role of noninvasive positive pressure ventilation in the emergency department. *Top. Emerg. Med*. 1999;21(4):68-73.
- 107.Chadda K., Annane D., Hart N., Gajdos P., Raphael J.C., Lofaso F. Cardiac and respiratory effects of continuous positive airway pressure and noninvasive ventilation in acute cardiac pulmonary edema. *Crit. Care Med*. 2002;30(11):2457-2461.
- 108.Masip J., Betbese A.J., Paez J., Vecilla F., Canizares R., Padro J. Non-invasive pressure support ventilation versus conventional oxygen therapy in acute cardiogenic pulmonary oedema: a randomized trial. *Lancet*. 2000; 356:2126-2132.
- 109.Cross A.M. Review of the role of non-invasive ventilation in the emergency department. *J. Accid. Emerg. Med*. 2000; 17:79-85.
- 110.Mehta S., Jay G.D., Woolard R.H., Hipona R.E., Connolly E.M., Cimini D.M. Randomized, prospective trial of bilevel versus continuous positive airway pressure in acute pulmonary edema. *Crit. Care Med*. 1997;25(4):620-627.
- 111.Kaminski J., Kaplan P.D. The role of noninvasive positive pressure ventilation in the emergency department. *Top. Emerg. Med*. 1999;21(4):68-73.
- 112.Brochard L. Noninvasive ventilation for acute respiratory failure. *JAMA*. 2002;288(8):932-934.
- 113.Chawla R., Mansuriya J., Modi N., Pandey A., Juneja D., Chawla A., Kansal S. Acute respiratory distress syndrome: Predictors of noninvasive ventilation failure and intensive care unit mortality in clinical practice. *J. Crit. Care*. 2016 Feb;31(1):26-30.
- 114.Sassoon C.Sh. Triggering of the ventilator in patient ventilator interactions. *Respir Care* 2011;56(1):39–51. Glumcher F., Dubrov S., Diomin E.