

Chapter 4

Mechanical Ventilators and Monitors: An Abridged Guide for Engineers



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Who Uses a Ventilator? What Level of Training Is Needed?

Since their invention, ventilators have mainly been used in hospitals to assist patients with different forms of respiratory failure. During the nineteenth and first half of the twentieth centuries, negative-pressure ventilators were large cumbersome devices that were mostly stationary and filled large hospital wards [1]. As positive-pressure ventilators began to take hold in the second half of the twentieth century, these devices have become smaller and more portable, which expanded their footprint from hospitals to the field, and even to patient homes. Modern-day ventilators are no bigger than a purse, can be battery powered, and simply require an oxygen tank for fresh gas flow.

In the hospital and nursing home settings, physicians or mid-level providers (physician assistants and nurse practitioners) typically write the ventilator setting orders, which are then carried out by the respiratory therapist (RT). Because ventilator settings can be changed multiple times a day, this pathway ensures that ventilator settings are not updated by multiple parties, which can lead to confusion for care teams. Bedside nurses are also usually quite familiar with using the ventilator, but are generally encouraged to follow the above pathway, unless there is no RT or physician present.

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RTs are certified medical professionals who create and carry out treatment plans for patients with respiratory issues. They must have a minimum of an associate degree from an accredited respiratory therapy education program, but many go on to earn further credentialing [2]. The responsibility of an RT can drastically vary at different institutions, but in general they work alongside physicians by carrying out physician orders, documenting aspects of respiratory care, administering respiratory therapy, and creating treatment plans for improving a patient's respiratory function. Although most physicians are at some point trained on how to use ventilators, physicians with specified training in critical care are usually titrating the ventilator on a daily basis. Anesthesiologists are also daily users of ventilators, as patients are often on mechanical ventilation during surgery.

In the field, paramedics often are the first to initiate mechanical ventilation for patients in respiratory failure or distress. Paramedic training requires between 1200 and 1800 hours and may last for 6–12 months. They then need to pass certification exams for both skills and knowledge [3].

When used at home, ventilator settings are usually non-titratable as patients that need variable settings are generally kept at nursing home facilities or hospitals. Home caregivers, however, must be able to provide supportive care, such as pulmonary hygiene, and recognize signs of ventilatory dysfunction.

Which Patients Benefit from This Device?

Patients that benefit from mechanical ventilation include the following: Those who (1) require high oxygen concentrations in the lungs, (2) need help clearing carbon dioxide, (3) require respiratory support so their body can concentrate on fighting other processes, (4) no longer have the ability to breathe by themselves, and (5) require help with breathing because they are unconscious [4]. All of these processes describe patients in respiratory failure. Ventilators, in general, are used to support patients in various forms of respiratory failure.

Interestingly, respiratory failure can be caused by problems with the lungs themselves (e.g., pneumonia), but oftentimes has an etiology unrelated to the pulmonary system. For example, common causes of respiratory failure include sepsis (blood-stream infections) or heart failure. During times of physiologic stress, the demand on the respiratory system to bring in oxygen or clear carbon dioxide is increased. Patients can develop respiratory failure simply from this inability to keep up with the increased demand. Respiratory failure is usually split into hypoxemic (problems with low oxygen in the blood), hypercapnic (problems with clearing carbon dioxide in the blood), or mixed (both) etiologies. Ultimately, ventilators assist patients with one or both problems. In most cases, ventilators buy the patient time by providing support until the underlying problem is addressed. A subset of patients may never be able to come off the ventilator. These patients are termed ventilator dependent.

Monitoring Physiologic Parameters

Oxygenation and Pulse Oximetry

Oxygen is an essential element utilized by our cells to produce energy through aerobic cellular respiration. It is brought into the lungs through inhalation, diffuses across the respiratory membrane into the bloodstream, and is attached to hemoglobin molecules as it is transported to various organs and bodily tissues. Because oxygen is vital to cellular function, measuring how much oxygen is in the blood provides important data regarding how well the lungs are functioning [5]. The measurement of quantifying how much hemoglobin in the blood is bound by oxygen is called oxygen saturation. Oxygen saturation is obtained non-invasively through pulse oximetry, which is an electronic device that is usually taped or clipped to the patient's finger. It emits light that travels through the patient's finger to a sensor located on the opposite side which will then measure how much light was not absorbed as it passes through the patient [5]. That measurement is then used to calculate the ratio of oxygenated to deoxygenated hemoglobin and reports the oxygen saturation as a percentage [5]. For a healthy person, a normal oxygen saturation is 95–100%. Pulse oximetry offers a rapid, continuous method of detecting oxygen saturation in patients and is particularly useful in hospital settings to monitor patients undergoing surgery and those whose lung function or breathing may be compromised [6].

Carbon Dioxide and Capnography

Carbon dioxide (CO_2) is a by-product of our cells and is transported through the bloodstream to be eliminated by the body through exhalation. The amount of CO_2 released in one exhaled breath is termed end-tidal CO_2 (ETCO_2). This measurement provides information on how well CO_2 is being transported to and expired from the lungs, but also is indicative of cardiac function and blood flow through the lungs. ETCO_2 can be measured with noninvasive capnography devices and is generally reported in mmHg with normal values between 35 and 45 mmHg [7]. Capnography allows monitoring of the patient's ventilation status in real time and is a valuable tool to inform the provider of any physiological or equipment complications that need to be quickly addressed. There are two main types of capnography devices: mainstream and sidestream. Each has its advantages and disadvantages in how ETCO_2 is measured and in what patient situations it is indicated [7, 8].

Mainstream capnography is designed with the capnograph's CO_2 sensor attached to an airway adapter that is located between the patient's endotracheal tube and the breathing circuit (Fig. 4.1). The airway adapter has an infrared sensor that emits light toward a photodetector on the opposite side of the adapter, which allows for measurement of ETCO_2 [8]. Mainstream capnography is used in intubated patients and

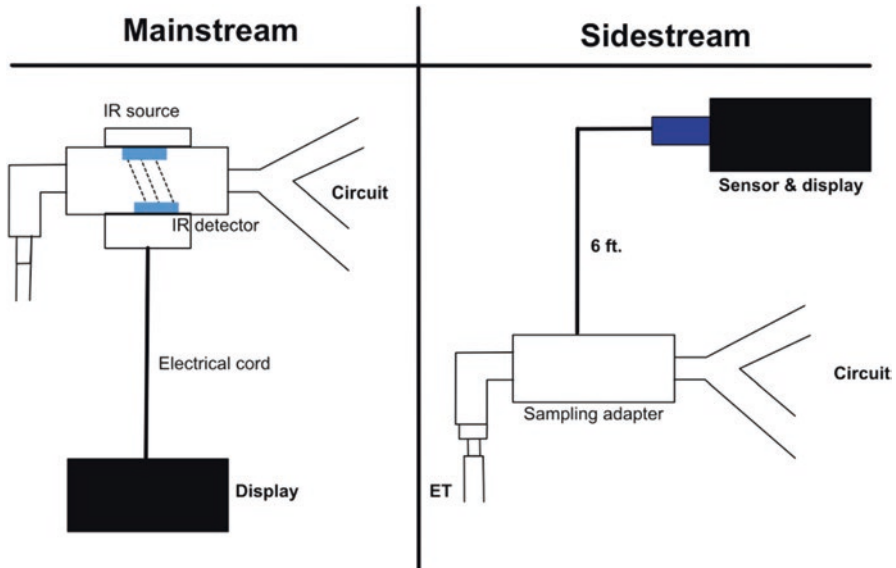


Fig. 4.1 Diagrams illustrating the equipment design of mainstream capnography vs. sidestream capnography

can be used for neonates and children [8]. Many of the advantages of this device type are due to the absence of a sampling tube in the apparatus. Because the ETCO_2 is being measured along the same pathway as the endotracheal tube and breathing circuit, there are no obstructions, pressure drops, or recording delay and there is minimal dispersion of gases [8]. Disadvantages of mainstream capnography include the sensor windows being more easily affected by secretions and the device design being less practical for patients in positions where they are not lying on their back [8].

Sidestream capnography is configured with the CO_2 -sensing device located separately from the patient's endotracheal tube rather than directly along the airway (Fig. 4.1) [8]. CO_2 exhaled from the patient is pumped from the airway through a six- to eight-foot sampling tube to reach the CO_2 -sensing unit. Additionally, any anesthetic gases that are exhaled with the CO_2 can be diverted to a gas scavenger or back to the patient via the breathing circuit [8]. One main advantage of sidestream capnography is that it can be used in patients who are not intubated. The apparatus can be fitted with nasal adapters that allow ETCO_2 measurements to be taken from patients receiving oxygen through nasal cannula [8]. Another advantage is the ability to use the device in patients who are not supine given the sampling tube's length. However, because the sampling tube is separate from the patient's airway, recording delays are expected as the gases must travel farther distances to the sensor and obstructions within the sampling tube can occur [8]. Furthermore, ETCO_2 measurements may be altered due to water vapor pressure changes or pressure drops within the sampling tube [8]. A particular issue that has been noted regarding sidestream capnography within the pediatric population is capnogram alteration due to increased gas dispersion within the sampling tube [8].

Volume Capnography

Volume capnography provides a continuous visual representation of the partial pressure of CO_2 (PCO_2) compared to the volume exhaled by the patient [9]. It offers many advantages for patient care such as real-time monitoring of ventilation quality, ventilation/perfusion (V/Q) ratio, CO_2 production, and early detection of pathological respiratory conditions that may compromise patient safety. An example of a volume capnogram is shown in Fig. 4.2. The volume capnogram is divided into three phases characterized by different expired gas components from distinct locations within the respiratory tract [9]. Phase I is composed of the gas from the end of the previous inspiration that is located within areas that do not undergo gas exchange such as anatomical dead space (e.g., conducting airways) and artificial dead space (e.g., the breathing circuit) [9, 10]. Thus, no CO_2 from the body is exchanged here and the PCO_2 (measured along the y-axis) remains zero. Increases in dead space will cause phase I prolongation [10]. If the PCO_2 is greater than zero during this phase, it suggests that the patient is breathing in previously expired CO_2 or that there is sensor malfunction [10]. Phase II consists of gas that travels to the sensor from the distal airway as well as from the first alveoli that empty upon expiration [9]. The slope is equal to the transition velocity between these two areas of the respiratory tract. Increased airway resistance or V/Q mismatch may extend this phase [10]. Phase III is made up of gas exclusively from the alveoli [10]. The positive slope during this phase represents real-time diffusion of CO_2 into the alveoli and out of the respiratory tract [9].

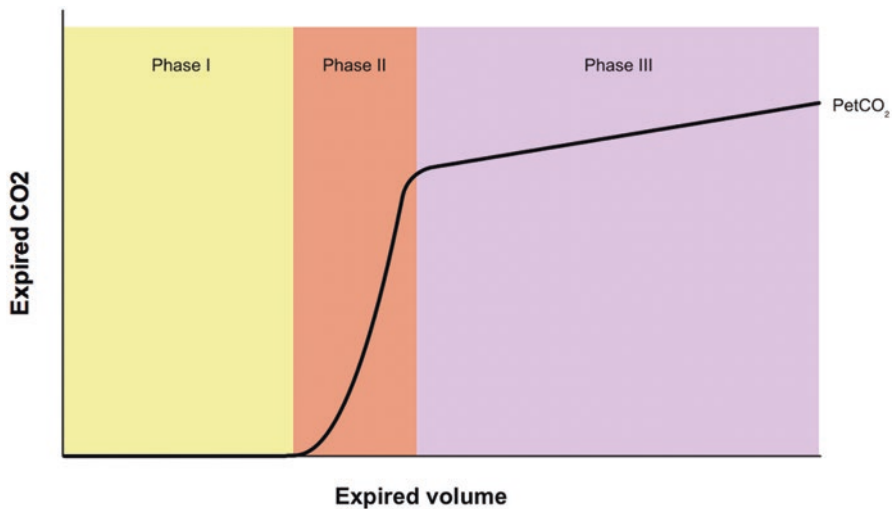


Fig. 4.2 Volume capnogram showing the relationship between expired volume of air and expired PCO_2 (measured in mmHg) during phases I, II, and III

Transcutaneous CO₂ Monitoring

Transcutaneous CO₂ monitoring is another continuous noninvasive method to measure a patient's CO₂ levels and evaluate the quality of ventilation. A transcutaneous monitor is placed on the patient's skin where it measures the partial pressures of oxygen and carbon dioxide, which can be used to estimate the arterial oxygen and carbon dioxide partial pressures [11]. The device works by increasing the skin's temperature at the device's attachment site, leading to increased blood flow to the local area. This increased heat changes the solubility of CO₂ in the blood [11]. It also causes the skin's metabolic rate to increase by 4–5% for each additional degree Celsius gained and thus increases CO₂ production at the site [11]. The sensor, commonly a Severinghaus electrode, measures pH changes to calculate the PCO₂ [12]. An algorithm is then applied that corrects for the additional CO₂ produced locally by the skin as a result of increased temperature and estimates the arterial PO₂ and PCO₂. Transcutaneous CO₂ monitoring can prove to be useful in mechanical ventilation to detect hypoventilation, hypoperfusion, revascularization status, ventilation adequacy, and therapeutic responses to medical interventions [11]. It is especially helpful for monitoring patients who do not have arterial access or cannot have frequent blood draws [11, 12].

Arterial Blood Gas vs. Venous Blood Gas

Two additional ways to monitor gases such as O₂ and CO₂ in a patient's bloodstream are through an arterial blood gas (ABG) or a venous blood gas (VBG). While some of the information provided by these two methods may be similar, there are key differences that must be acknowledged, especially when making clinical decisions based on their measurements. An ABG is performed by gaining access to a patient's artery to sample the oxygenated blood. This can be done using needle punctures to take individual samples periodically or by using an indwelling catheter (a tube that remains in the patient's blood vessel) to continuously sample the patient's arterial blood [13]. ABGs provide information including the partial pressures of O₂ and CO₂ (normal PaO₂ >~80 mmHg, PaCO₂ 35–45 mmHg), acidity (physiologic pH 7.35–7.45), oxygen saturation (generally >95%), and concentration of bicarbonate ions (normal range 21–27 mEq/L) in the arterial blood [13]. All values are measured except for the bicarbonate levels, which are obtained through calculation by the Henderson-Hasselbalch formula [13]. PaO₂ and oxygen saturation are useful in determining a patient's oxygen status and whether there is too much or too little oxygen in the blood. PaCO₂ helps to evaluate a patient's ventilation status. The pH of the sample demonstrates the relationship between the PaCO₂ and bicarbonate balance. This is a critical number in determining a patient's overall clinical status since disturbances in pH are not well tolerated physiologically [13]. However, while ABGs provide a wealth of information, they can be technically challenging to obtain and may not be a feasible option in all patients [13].

A VBG is obtained by sampling blood from the veins through intermittent needle punctures or through a patient's indwelling venous catheter. Similar to an ABG, a VBG also measures the partial pressures of O₂ and CO₂ (now PvO₂ and PvCO₂, respectively), oxygen saturation, pH, and bicarbonate concentration [14]. Because blood is being sampled from the veins and has previously released some of its oxygen into the body, a patient's true oxygenation status cannot be determined with a VBG. Additionally, VBG measurements differ from those of ABGs to varying degrees depending on the site collection and the patient's clinical status, and historically have been perceived as less accurate [14]. Therefore, while VBGs can provide helpful information and may be easier to perform, they should be correlated with ABG values when making critical clinical decisions.

Airway Pressures

During normal physiologic inspiration, the contraction of the diaphragm and external intercostal muscles allows the thoracic cavity to expand. Using Boyle's law (pressure is inversely proportional to volume with temperature constant), as the thoracic cavity expands, the volume increase causes a decrease in pressure. This is referred to as negative pressure. The lungs expand in synchrony with the thoracic cavity as they are held to the thoracic cage by the visceral and parietal membranes. The negative pressure within the lungs causes a pressure gradient with the external environment, allowing air from the outside environment to flow into the lungs down the pressure gradient (from high to low pressure). This is called negative-pressure ventilation [15].

For patients on mechanical ventilation, the process is changed. Inspiration on a mechanical ventilator occurs as positive pressure is injected through a breathing tube into the trachea to the lungs. Breathing tubes usually have a balloon, or cuff, that prevents the leakage of the positive airway pressure from going forward. Assuming no blockages in the breathing circuit, the positive pressure from the ventilator needs to overcome the patient's chest wall, airway, and lung resistances in order to generate a breath.

Peak Inspiratory Pressure vs. Plateau Pressure

Peak inspiratory pressure (PIP) is the total pressure generated by the ventilator to overcome airway and alveolar resistance. This is necessary to allow inspiratory flow and designated tidal volume. A simplified equation of *PIP* is

$$\text{Peak airway pressure} = \text{Resistive pressure} + \text{Elastic pressure} \\ + \text{Positive end - expiratory pressure}$$

Resistive pressure is the summation of resistance within the ventilator circuit, the endotracheal tube, and the patient's airways. Elastic pressure is defined as the product of chest wall and lung recoil. Positive end-expiratory pressure (PEEP) refers to the pressure in the lungs at the end of expiration greater than atmospheric pressure.

Peak inspiratory pressure measures the highest pressure applied to the lungs during inhalation whereas plateau pressure, P_{plat} , also known as transpulmonary pressure, is the pressure the alveoli and small airways of the lung are exposed to at peak inspiration when there is no air movement. PIP is the sum of the P_{plat} and the pressure needed to overcome airway resistance. As a result, P_{plat} must be smaller than PIP and is only a direct measurement of the pressure within the airways when there is no airflow [16]. The P_{plat} is measured through the inspiratory pause maneuver as shown in Fig. 4.3.

This maneuver pauses airflow through the lungs, thereby eliminating the pressure contribution from airway resistance, and thus revealing the pressure contribution solely from the alveoli and airways. Peak pressures are considered elevated when there is a 5 mmHg or greater difference between peak pressure and plateau pressure. This occurs when there is a lung pathology causing elevated airway resistance within the respiratory system. Lung pathologies associated with an increased airway resistance include bronchospasms, bronchiectasis, retained secretions, and endotracheal tube tip occlusions. An elevated peak pressure and plateau pressure occur when the difference between peak pressure and plateau pressure is small, suggesting a lung pathology with poor alveolar compliance. Lung pathologies associated with an elevated plateau and peak pressure include pneumothorax, pulmonary edema, acute respiratory distress syndrome, and pneumonia [17].

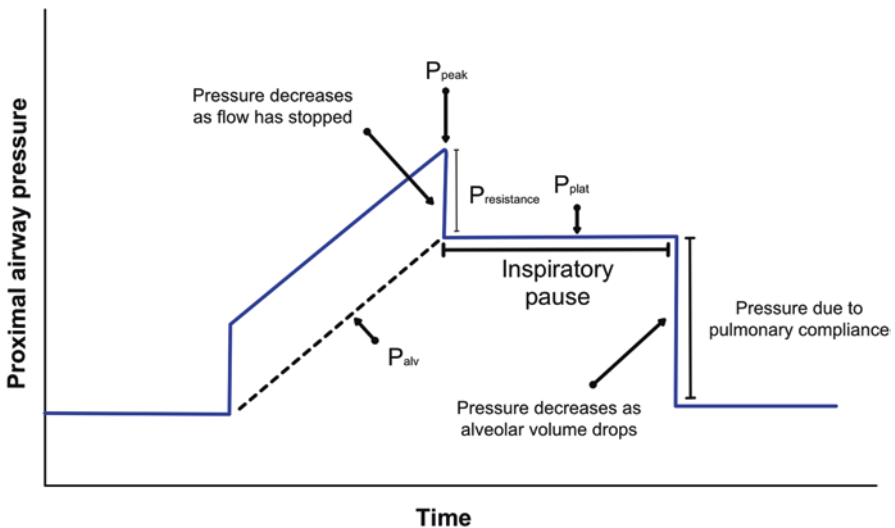


Fig. 4.3 P_{peak} is the maximum pressure applied to the lungs at full expiration whereas P_{plat} is the pressure of the alveoli and small airways during an inspiratory pause maneuver, when there is no flow circulating through the lungs. As indicated in the figure, the primary difference between P_{peak} and P_{plat} is that P_{peak} accounts for the resistance that needs to be overcome to allow airflow into the terminal airways and alveoli

Auto-PEEP vs. Extrinsic PEEP

In healthy lungs, the end-expiratory pressure is in equilibrium with the environmental pressure. However, in patients with lung pathology where there is an airflow limitation or obstruction, the end-expiratory pressure can be positive. This is referred to as intrinsic PEEP or auto-PEEP. Intrinsic PEEP occurs when the expiratory time is shorter than the time needed to fully deflate the lungs, preventing the lung and chest wall from reaching an elastic equilibrium. This leads to air trapping within the distal alveoli. Intrinsic PEEP can be measured by the expiratory hold maneuver as indicated in Fig. 4.4. This measurement can be performed on the ventilator by pausing the breath in expiration, preventing the delivery of more breaths, and allowing the alveolar pressure to equilibrate with the ventilator circuit. The circuit pressure at the end of the expiratory hold can be measured as the intrinsic PEEP, a rough approximation of the alveolar pressure at the end of expiration [18].

Extrinsic PEEP is the pressure applied by the ventilator throughout the respiratory cycle to maintain alveolar patency and prevent the alveoli from collapsing. Allowing the alveoli to stay open improves oxygenation by increasing the surface area for gas exchange and by decreasing air shunting [19]. Along with improving oxygenation to the distal alveoli, extrinsic PEEP can be used to improve ventilation/perfusion (V/Q) mismatches. A V/Q mismatch occurs (1) when one or more areas of the lung are well oxygenated but have poor blood flow or (2) when the lungs receive adequate blood flow but have poor airflow. The application of a positive pressure through a patient's lungs can open airways that were previously collapsed, improving alveolar oxygenation and thus decreasing V/Q mismatch [20].

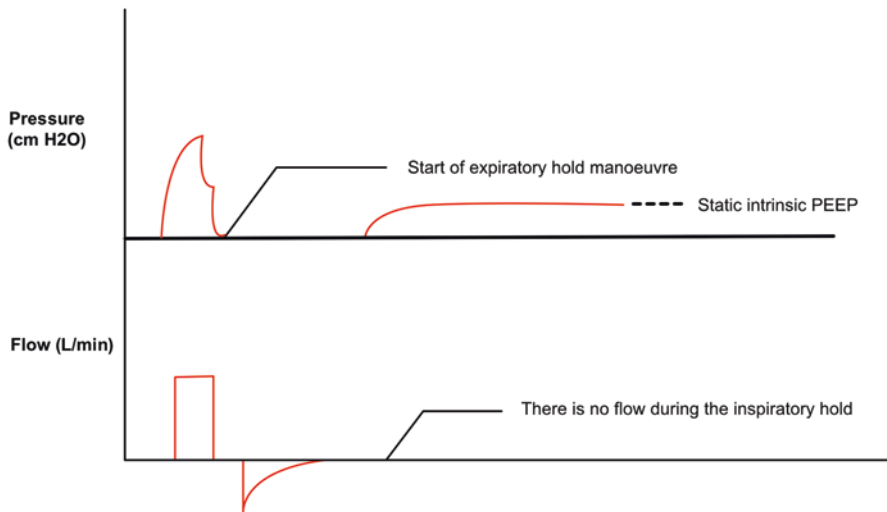


Fig. 4.4 The expiratory hold maneuver pauses the ventilator during expiration, allowing the alveolar pressure to equilibrate with the ventilator circuit. The alveolar pressure at the end of the expiratory hold is known as intrinsic PEEP or auto-PEEP

Extrinsic PEEP decreases the work of breathing for patients that have stiff lungs with low compliance because the positive pressure delivered to alveoli improves oxygenation and compensates for the decreased lung compliance [20]. Patients with stiff lungs will have to increase the number of breaths and total inhaled air volume into their lungs to compensate for their decreased lung compliance. This leads to an overall increase in energy expenditure and lactic acid production that can be counteracted by the ventilator providing an extrinsic PEEP.

A major concern with extrinsic PEEP is that it can decrease blood return to the heart, causing an overall decreased cardiac output. In normal respiratory physiology, the negative pressure created in the airways during inspiration decreases the pressure within the right atrium, generating a suction effect to increase venous return to the heart. Extrinsic PEEP applied from the ventilator generates a more positive pressure in the airways than the typical negative pressure created in normal respiratory physiology. This altered respiratory physiology increases right atrial pressure and decreases venous return to the heart, leading to an overall decrease in cardiac output. This is an important concern for intubated patients who have distributive shock or low blood pressure, as extrinsic PEEP can exacerbate the already decreased cardiac output in these patients [20].

Respiratory Rate

Respiratory rate (RR) is defined as the number of breaths a patient takes in 1 min, known as breaths per minute (bpm). The mechanical ventilator sets the RR to a specific value to allow the same number of breaths to be given to a patient per minute. For example, if the set RR is 20, then the ventilator will deliver 20 bpm or one breath every three seconds [21].

Respiratory Set vs. Actual Rate

The set respiratory rate is the bpm set by the provider on the ventilator. The ventilator will ensure that the set rate is delivered regardless of how many breaths the patient initiates. The actual rate is how many breaths the patient wants to take without intervention. As such, there are multiple combinations of set vs. actual rate. (1) The patient is breathing below the set rate: In these cases, the ventilator will add breaths to the patient's actual rate to ensure that the patient is breathing at the set rate. (2) The patient is not breathing at all: Once again, the ventilator will add breaths to ensure that the patient breathes at the set rate. (3) The patient is breathing above the set rate: Here, what happens largely depends on the ventilator mode. For assist control modes, the ventilator will deliver a fixed tidal volume or drive pressure (depending on the setting of volume control or pressure control) during every inspiration, regardless of whether the breath is initiated by the patient or the ventilator.

During synchronized intermittent mandatory ventilation (SIMV), the ventilator will deliver a fixed tidal volume or drive during every inspiration up until the set rate. Anything above the set rate, the ventilator will deliver pressure support breaths [22].

Ventilator Sensed Rate

Ventilators can deliver flow- vs. time-cycled breaths. Flow-cycled breaths occur when the ventilator senses changes in flow, usually initiated by the patient, and a breath is delivered. Time-cycled breaths occur when enough time has elapsed based on the set rate, leading to a breath delivery.

In order for the ventilator to recognize a patient's breath, it must be able to sense changes in pressure within the patient's respiratory system. The ventilator has sensors within its tubing that detect the increase in negative pressure within the thoracic cavity that occurs with inspiration. If the change in pressure surpasses the ventilator's trigger-sensitivity threshold, the ventilator will deliver a breath at the fixed tidal volume. However, if the patient fails to make a new inspiratory effort, the ventilator is programmed to initiate a breath in a time-dependent manner, known as time-triggered breaths. Time-triggered breaths will always follow the set rate of bpm established by the ventilator unless the patient attempts to inspire [23].

Humidity (Heat and Moisture Exchangers vs. Heated Humidifier vs. Heated-Wire Circuits)

When a person breathes normally, the upper respiratory tract acts to filter, warm, and humidify the inspired air before it reaches the alveolar air sacs in the lungs. However, mechanical ventilation bypasses much of the anatomy that carries out these important bodily functions and places the burden of accomplishing them on the lower respiratory tract [24]. This can lead to lower respiratory tract damage that can negatively affect the patient's ventilation and cause complications. To avoid this, mechanical ventilators are equipped with devices that can optimize the humidity of the ventilated air. Humidifiers can be divided into two general categories: passive and active.

Passive humidifiers use a heat and moisture exchanger (HME) or artificial nose which contains a condenser that captures the water vapor and heat from the patient's exhaled air and adds it to the air that will be subsequently inhaled [24]. The device is located between the patient and the Y-piece, where the inspiratory and expiratory limbs separate to connect to the ventilator [24]. HMEs can further be characterized by their design elements as hygroscopic, hydrophobic, combined hydrophobic hygroscopic, and filtered [24, 25]. Hygroscopic HMEs contain hygroscopic salts such as lithium chloride or calcium chloride that have a high capacity to absorb moisture from exhaled air and release it back into inhaled air [24]. Hydrophobic

HMEs have condensers that repel water and maintain the temperature gradient, allowing water vapor droplets to accumulate on the filter's surface to humidify the next inhaled breath [24]. Combined hydrophobic hygroscopic HMEs contain both a water-repelling and hygroscopic salt element. Additionally, pleated filters with dense fibers or electrostatic filters can be applied to each of these HME types to create barriers to viral and bacterial pathogens [24]. Passive humidifiers are advantageous because they remove condensation that may accumulate in other problematic portions of the breathing circuit. However, they are associated with increased airway resistance and dead space, are more prone to occlusion, and are contraindicated in certain patient populations [24, 25].

Active humidifiers function by utilizing a heated water reservoir that warms the air. The device is located in the inspiratory limb of the ventilator circuit between the patient and ventilator [24]. Heated humidifiers (HH) can be further designated as bubble, passover, inline vaporizer, and counterflow [24]. Bubble humidifiers push gas into a tube that opens at the bottom of the water reservoir. As the gas forms bubbles that rise to the top of the reservoir, the amount of water vapor the gas contains increases. Additional ways to increase water vapor content using a bubble humidifier include slowing the flow rate, using a longer water column, and having a diffuser that creates smaller bubbles [24]. For passover humidifiers, air travels over the heated water reservoir which supplies it with increased water vapor. Passover humidifiers can further be modified with wicks or membranes which increase the gas-water interface and thus the water vapor content of the gas being inhaled by the patient [24]. Inline vaporizers consist of a plastic capsule in the circuit's inspiratory limb that adds water vapor to the gas and heats it via a disk heater. Because the capsule is located closer to the patient, this system does not use heated wires or additional temperature monitoring [24]. Lastly, counterflow humidifiers work by using externally heated water that travels through a humidifier and down a surface while gas travels across that surface in the opposite direction. Both inline vaporizers and counterflow humidifiers are newer technologies that are still being researched [24]. Compared to passive humidifiers, active humidifiers can be used more broadly and can attain a greater breadth of humidity and temperature. However, active humidifiers have a greater risk of contamination due to possible condensation of water vapor within the breathing circuit [24].

Heated humidifiers can be designed with water traps or heated wires to mitigate the risk of water vapor condensing along the inspiratory limb as a result of temperature differences along its length [24]. Specifically in regard to heated-wire circuits, these are located within the inspiratory limb of the breathing circuit and help regulate the temperature at the Y-piece [24]. Temperature probes are located at the humidifier and at the Y-piece and provide feedback to the system to achieve the desired temperature [24]. Heated-wire circuits can be divided into single-heated-wire (SHW) and double-heated-wire (DHW) circuits. While both have a heated wire within the inspiratory limb, DHW circuits contain a second wire in the expiratory limb to decrease condensation of water vapor within that portion as well [24]. The decrease in condensation with the use of heated-wire circuits makes these versions of active heated humidifiers advantageous compared to those without heated wires [24, 26].

Tidal Volume

Tidal volume (V_t) is defined as the amount of air that is moved into or out of the lungs during normal inhalation or exhalation and is typically about 500 mL in a healthy adult [27]. V_t is an important value to monitor and set in mechanical ventilation particularly in volume control modes. Too small of a V_t may cause inadequate oxygenation/ventilation and too large of a V_t may cause barotrauma to the lungs. Therefore, the set V_t must be titrated to the patient's physiological status and oxygen needs [27]. For a patient with healthy lungs, providers commonly start with a V_t of 6–8 mL/kg of predicted body weight [28]. For patients whose lung function is compromised such as in acute respiratory distress syndrome, the V_t may be set lower at 4–6 mL/kg [28]. Generally, V_t is not set above 10 mL/kg as research has shown increases in morbidity with similar or greater values [28].

Set V_t may be the same as or different than actual inhaled V_t depending on the ventilator setting being used. In continuous mandatory ventilation (CMV), the ventilator will provide breaths at a set V_t and RR despite the patient's own breathing efforts. CMV is commonly used in patients who are paralyzed and therefore the set V_t should match the actual inhaled V_t [29]. However, there are other volume control modes that allow for spontaneous breaths by the patient in addition to those initiated by the ventilator. In assist-control ventilation (ACV), the ventilator also has a set V_t and RR [29]. Unlike CMV, ACV also responds to the patient's spontaneous breaths by providing the set V_t when triggered and resetting the time it will deliver the next set breath. Because the ventilator is still providing the set V_t to each mechanically or patient-induced breath, set V_t should equal actual inhaled V_t [29]. Intermittent mandatory ventilation (IMV) is another mode that allows for spontaneous breathing by the patient while it continues to deliver set V_t at the set rate. For patients with this setting, there is no assistance by the ventilator during these spontaneous breaths [29]. This can lead to a discrepancy between the set V_t and actual inhaled V_t depending on the patient's spontaneous breathing against the airway circuit resistance. Finally, synchronous intermittent mandatory ventilation (SIMV) combines preset breaths with the patient's spontaneous breathing and synchronizes the timing of the mechanical breaths to avoid stacked breathing (delivering a mechanical breath while the patient has already initiated a spontaneous breath) [29]. If the patient's spontaneous breathing does not match the ventilator's settings, it is possible for set V_t to differ from actual inhaled V_t [29].

Exhaled V_t is another parameter that is important to monitor as it can provide information on both mechanical and spontaneous ventilation. Generally, the exhaled V_t should be approximately the same as the set V_t . If the exhaled V_t is lower than the set V_t , there may be air leakage within the ventilatory circuit [29]. Common causes of air leakage occur around the endotracheal (ET) tube if the inflatable cuff does not create a leakproof seal or if a cuffless ET tube is too small to prevent air leakage around it [29]. Lower exhaled V_t compared to set V_t can also suggest insufficient exhalation time which could be due to the patient's lung health or due to mismatched timing of the ventilator-assisted breathing and the patient's spontaneous breathing rates [29].

Patient-Ventilator Synchrony Monitoring

Patient-Ventilator Synchrony/Dyssynchrony Introduction

Patient-ventilator synchrony refers to the mechanical ventilator functioning in synchrony with the patient's own respiratory drive. The respiratory system is dynamic and is constantly being influenced by the body's own mechanical, chemical, behavioral, and reflex mechanisms to create subtle changes in breath-to-breath adjustments. This multitude of factors affecting each patient's breathing pattern can present a major challenge for the ventilator to respond appropriately to the patient's expiratory and inspiratory signals [30].

Respiratory drive is dependent on both voluntary and autonomic control. These control centers act in synchrony to determine whether inspiration or expiration should be inhibited or stimulated, sending outgoing neuronal information to the phrenic and intercostal nerves to adjust the various aspects of ventilation. The ventilator must respond to these adjustments to maintain patient-ventilator synchrony. Patient-ventilator dyssynchrony (PVD) occurs when the ventilator is unable to recognize or adjust to a patient's breathing pattern [30].

Ineffective Triggering

Ineffective triggering is a form of PVD where the ventilator fails to trigger a breath when the patient attempts inhalation. Ventilators respond by initiating a breath when the flow or pressure within a patient's respiratory drive changes. A pressure-triggered breath occurs when the inspiratory effort by the patient creates a large enough negative pressure within the airways that it surpasses the pressure threshold on the ventilator. With a flow-triggered breath, the patient's inspiration must draw a continuous flow greater than the flow threshold on the ventilator. In either case, if the flow or pressure generated by a patient's inspiratory effort is unable to surpass the ventilator's threshold value, then the ventilator will not initiate a breath when the patient attempts inhalation, leading to ineffective triggering. Ineffective triggering occurs in patients with severe respiratory weakness and can even exacerbate their weakness by forcing them to increase their inspiratory efforts in an attempt to produce a triggered breath. Ways to prevent ineffective triggering include lowering the flow or pressure threshold on the ventilator and addressing the underlying cause of the patient's respiratory weakness [30].

Double Triggering and Reverse Triggering

As respiratory drive increases, the duration of a patient's inspiration, also known as neural inspiratory time (neural T_i), can outlast the ventilator's programmed inflation time (ventilator T_i), causing the ventilator to trigger an additional breath in response

to the patient's continued inhalation, known as double triggering. Double triggering occurs when the patient's diaphragm continues to contract as the ventilator begins the expiratory phase. When the diaphragm continues to contract, the pressure sensor on the ventilator will recognize the increased negative pressure in the patient's proximal airways and immediately initiate a new breath. As a result, the ventilator will deliver two breaths without exhalation in between, thereby essentially doubling the patient's fixed V_t . The elevated V_t can lead to overdistention of the alveoli and small airways and cause clinical manifestations such as pneumothorax, pneumomediastinum, and other pathologies associated with volutrauma or barotrauma. Figure 4.5 represents double triggering with neural T_i being longer than ventilator T_i , forcing the ventilator to respond to the lengthened neural T_i time by initiating a new breath. In order to correct double triggering, the ventilator can be turned off to allow the episode causing the double triggering to resolve. The ventilator's flow or volume settings should be adjusted to meet the patient's respiratory demands in order to correct the double triggering when the cause of double triggering does not resolve on its own [30, 31].

Reverse triggering, also known as entrainment, is another type of patient-ventilator dyssynchrony that occurs when a patient's respiratory center is activated by the ventilator passively inflating the lungs. Reverse triggering that occurs during pressure-controlled ventilation shows detectable flow changes during the inspiratory phases and continued patient effort during expiratory flow. During volume-controlled ventilation, reverse triggering is identified by pressure changes during the inspiratory phases or continued patient effort during the expiratory flow waveform. Reverse triggering originates in the patient's diaphragm as it contracts in response to the ventilator triggering a breath. If the diaphragm continues to contract during the ventilator exhalation, the patient will be inhaling while the ventilator is in the expiratory phase, forcing the ventilator to initiate another breath, known as

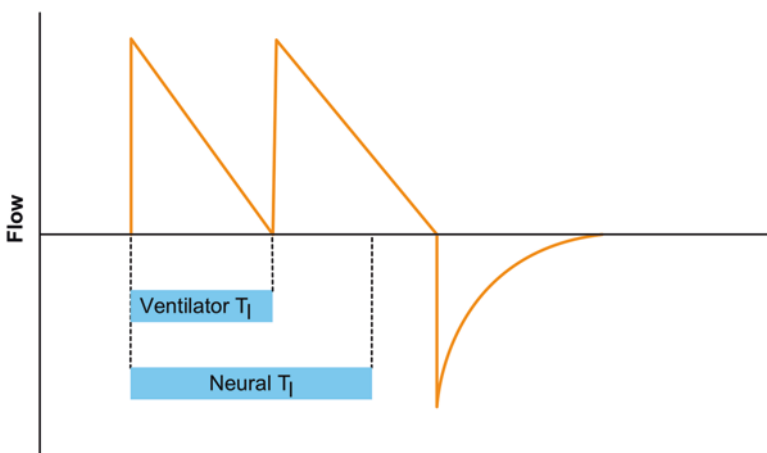


Fig. 4.5 The patient's inspiration time, neural T_i , is greater than the ventilator's inspiration time, ventilator T_i , prompting the ventilator to initiate a new breath. This phenomenon is known as double triggering

breath stacking. Similar to double triggering, breath stacking can cause excessive regional lung stress as the tidal volume can dramatically increase due to excessive inspiratory volume delivery [30, 31].

Flow Dyssynchrony and Auto-Triggering

An increased respiratory drive oftentimes necessitates an increased flow from the ventilator. Flow dyssynchrony occurs when the ventilator is unable to meet the patient's increased flow demand. For example, this occurs when the flow rate set by the ventilator is too low to meet the patient's own inspiratory demand. During volume ventilation while the flow is fixed, flow dyssynchrony can be identified on pressure-time waveforms as a "scooped" appearance on the pressure wave during inhalation, as demonstrated in Fig. 4.6. Improving flow dyssynchrony necessitates a reduction in a patient's inspiratory demand or an increase in flow through the ventilator. Increasing ventilator flow delivery can be accomplished by directly increasing the flow or by adjusting the inspiratory flow patterns [30].

Inappropriate ventilator sensitivity levels can cause auto-triggering, a process by which the ventilator misinterprets the initiation of a patient's breath and triggers a breath spontaneously. Signals that can induce auto-triggering include condensation in the circuit, ET cuff leaks, circuit leaks, cardiogenic oscillations, increased cardiac output, or elevated ventricular filling pressure. The management of auto-triggering is to minimize both ET and circuit leaks, remove condensation from the circuit, and decrease the sensitivity of the trigger threshold [30].

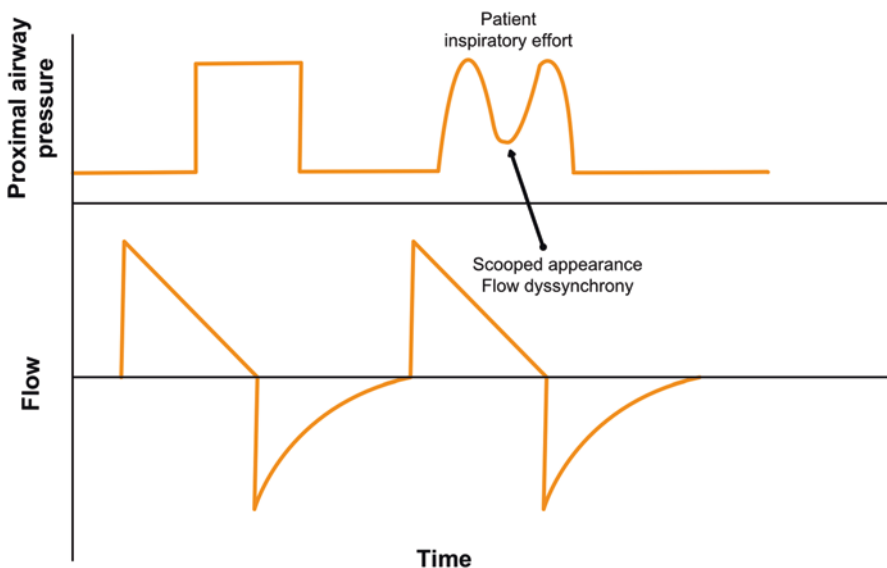


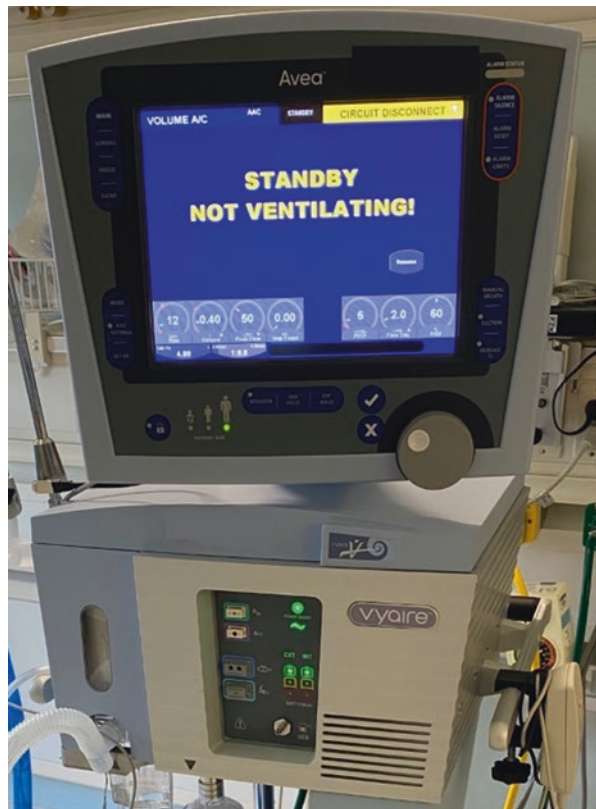
Fig. 4.6 The scooped appearance in the figure showcases flow dyssynchrony as the flow rate set by the ventilator is insufficient to meet the patient's elevated inspiration flow rate

Setup and Form Factor of Contemporary Ventilators

Modern mechanical ventilators come in an array of shapes and sizes and have gone through many generations of modifications. In the ICU, the ventilator is usually positioned at the head of the patient’s bed to be closer to the patient’s airway. It can be either to the left or the right of the patient, but is commonly positioned closer to the entrance of the room for ease of access. A standard intensive care unit (ICU) ventilator typically has wheels so that it can be moved from room to room. However, once it is set up for a patient, it is usually not moved around the room. It also requires an AC plug for power and wall gas hookups, unless it is a portable transport ventilator.

The current generation of ICU ventilators (fourth generation) are mainly distinguished from older ventilators by their plethora of features and ventilation modes available to the user. With the advent of microprocessor chips, ventilators have become extremely advanced in their ability to control oxygen percentage, pressures/volumes, flows, and respiratory cycle times [1]. As seen in Fig. 4.7, modern ventilators have a computer screen with dials and buttons that allow the user to make

Fig. 4.7 Avea™ CVS Ventilation System by Vyair Medical with touch screen



setting changes to the ventilator. Many modern-day ventilators are even touch screen with high-resolution monitors. Each setting on the ventilator is dialed in through the buttons, screen, and dials, allowing the medical professional intricate control.

Since ventilators can deliver anywhere from 21% oxygen (room air) to 100% oxygen, each ventilator also requires gas connections of air and oxygen. In the United States, room air is indicated with the color yellow, and oxygen with the color green. Specialty-specific ventilators may have connections for other forms of gas (e.g., anesthesia ventilators usually have a nitrous oxide line which is indicated with the color blue). Gas connections can come directly from the hospital's central supply, which directly hook into the wall (Fig. 4.8), or can come in the form of canisters. Special pegs and keys on the cannisters and wall hookups are used to ensure that the different gases are not interchanged, which would cause the ventilator to deliver the wrong gas. This could cause significant harm to the patient by delivering hypoxic gas mixtures.

Modern-day ventilators are usually circle-system circuits. They have an inspiratory and expiratory limb controlled by one-way valves that regulate the direction of airflow. One end of the circuit is hooked up to the ventilator (Fig. 4.9) and the other end converges at the "Y" connector, which is then hooked to the patient's

Fig. 4.8 Oxygen (green) and Air (yellow) wall hookup. BeaconMedæ's™



Fig. 4.9 Inspiratory and expiratory limbs of the ventilator circuit with accordion tubing



Fig. 4.10 “Y” connector that hooks up to the endotracheal tube



endotracheal tube or noninvasive ventilator mask (Fig. 4.10). The circuit tubing is usually configured in an accordion shape, which allows it to stretch and reach the patient, but also works to trap water droplets in the warm and humidified air.

Transport ventilators (Fig. 4.11) are smaller battery-operated ventilators that can appear in ambulances, helicopters, and aircrafts to transport patients between hospital settings. Transport ventilators are also used in the hospital to transport patients between different areas in the hospital, such as between the ICU and operating room. Many mishaps can occur when patients are transported with ventilators [32]. Thus transport ventilators are built for their ease of maneuverability, compact size and form factor, and simplistic operating functions. Transport ventilators usually use air and oxygen cannisters as their source of fresh gas flow. Studies have shown that these ventilators are effective and safe for short periods of time, despite their simplistic design [33].

Fig. 4.11 LTV™1200 by Vyair Medical transport ventilator with oxygen cannister



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References

1. Kacmarek RM. The mechanical ventilator: past, present, and future. *Respir Care*. 2011;56(8):1170–80.
2. Your Complete Guide to Becoming a Respiratory Therapist | University of Cincinnati. University of Cincinnati. Published May 2019. Accessed 3 July 2021.
3. What's the Difference Between an EMT and a Paramedic? UCLA CPC. Published September 18, 2014. Accessed 3 July 2021.
4. American Thoracic Society Patient Educations Information Series: Mechanical Ventilation. *Am J Respir Crit Care Med*. 196, P3–4, 201. <https://www.thoracic.org/patients/patient-resources/resources/mechanical-ventilation.pdf>. Accessed July 2021.
5. Hafen BB, Sharma S. Oxygen Saturation. *Nih.gov*. Published May 7, 2021. Accessed 3 July 2021.
6. Pulse Oximetry. Yale Medicine. Published March 2, 2021. Accessed 3 July 2021.

7. Richardson M, Moulton K, Rabb D, et al. Introduction. [Nih.gov](#). Published March 2016. Accessed 3 July 2021.
8. Bhavani Shankar Kodali MD. Types of Capnographs - Capnography. *Capnography*. Published 2021. Accessed 3 July 2021.
9. Kreit JW. Volume capnography in the intensive care unit: potential clinical applications. *Ann Am Thorac Soc*. 2019;16(4):409–20. <https://doi.org/10.1513/annalsats.201807-502cme>.
10. Munir K, Himmelstoss M. Volumetric capnography. Bonaduz: Hamilton Medical; 2016.
11. Transcutaneous CO2 Monitoring, October 2018. American Association of Sleep Technologists.
12. Restrepo RD, Hirst KR, Wittnebel L, Wettstein R. AARC Clinical Practice Guideline: Transcutaneous Monitoring of Carbon Dioxide and Oxygen: 2012. *Respir Care*. 2012;57(11):1955–62. <https://doi.org/10.4187/respcare.02011>.
13. Theodore AC. Arterial Blood Gases. UpToDate. Published 2021. Accessed 3 July 2021.
14. Theodore AC. Venous Blood Gases and Alternatives to Arterial Blood Gases. UpToDate. Published 2021. Accessed 3 July 2021.
15. Mechanics of Breathing | Boundless Anatomy and Physiology. [Lumenlearning.com](#). Published 2021. Accessed 3 July 2021.
16. Jade H. Peak airway pressure in mechanical ventilation definition & interpretation. Health Jade. Published December 18, 2019. Accessed 4 July 2021.
17. Cassone M, Cocciolone A, Melnychuk E. Your First Shift in the Unit: Demystifying Ventilator Alarms. [Emra.org](#). Published December 16, 2019. Accessed 4 July 2021.
18. Yartsev A. Intrinsic PEEP and the expiratory hold manoeuvre | Deranged Physiology. [Derangedphysiology.com](#). Published 2015. Accessed 4 July 2021.
19. Merck Manuals. Noninvasive positive pressure ventilation (NIPPV) using bilevel positive airway pressure. Merck Manuals Professional Edition. Published 2021. Accessed 4 July 2021.
20. Mora AL, Mora JI. Positive End-Expiratory Pressure. [Nih.gov](#). Published August 29, 2020. Accessed 4 July 2021.
21. Mora AL, Mora JI. Ventilation Assist Control. [Nih.gov](#). Published April 28, 2021. Accessed 4 July 2021.
22. Modes of Mechanical Ventilation. [Openanesthesia.org](#). Published 2021. Accessed 4 July 2021.
23. Mancebo J. Chapter 6. Assist-control ventilation. In: Principles and practice of mechanical ventilation, 3e. New York: AccessMedicine, McGraw Hill Medical; 2021. [Mhmedical.com](#). Accessed 4 July 2021.
24. Al Ashry HS, Modrykamien AM. Humidification during mechanical ventilation in the adult patient. *Biomed Res Int*. 2014;2014:1–12. <https://doi.org/10.1155/2014/715434>.
25. Cerpa F, Cáceres D, Romero-Dapueto C, et al. Humidification on ventilated patients: heated humidifications or heat and moisture exchangers? *Open Respir Med J*. 2015;9(1):104–11. <https://doi.org/10.2174/1874306401509010104>.
26. Restrepo RD, Walsh BK. Humidification during invasive and noninvasive mechanical ventilation: 2012. *Respiratory Care*. 2012;57(5):782–8. <https://doi.org/10.4187/respcare.01766>.
27. Hallett S, Toro F, Ashurst JV. Physiology, tidal volume. [Nih.gov](#). Published May 9, 2021. Accessed 4 July 2021.
28. Hyzy RC, Mcsparron JI. UpToDate. Published 2021. Accessed 4 July 2021.
29. Staff RT. Ventilation modes and monitoring. RT: For decision makers in respiratory care. Published February 7, 2020. Accessed 4 July 2021.
30. Mellott KG, Grap MJ, Munro CL, Sessler CN, Wetzel PA. Patient-ventilator dyssynchrony: clinical significance and implications for practice. *Crit Care Nurse*. 2009;29(6):41–55. <https://doi.org/10.4037/ccn2009612>.
31. Poor H. Patient-ventilator dyssynchrony. Basics of Mechanical Ventilation. Published online 2018:75–93. https://doi.org/10.1007/978-3-319-89981-7_7.
32. Waydhas C. Intra-hospital transport of critically ill patients. *Crit Care*. 1999;3(5):R83–9. <https://doi.org/10.1186/cc362>. Epub 1999 Sep 24. PMID: 11094486; PMCID: PMC137237
33. Hurst JM, Davis K Jr, Branson RD, Johannigman JA. Comparison of blood gases during transport using two methods of ventilatory support. *J Trauma*. 1989;29(12):1637–40.