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AHC Media

Ventilator Management in the Emergency Department

There was a time when a ventilator in the emergency department (ED) was almost a reportable event. I recall times when I barely got the tube secured before the ICU was there to take the patient. But times have clearly changed. Many of our EDs now hold patients, even those who are critically ill, for hours or even days. Complicated work-ups, consultations, and initial therapy are now done in the ED, with the ED physician responsible for ventilator care. There are even patients who are "too sick for the ICU" who must be stabilized before transfer, even if it is only just down the hall. Therefore, it is prudent that the emergency physician be very familiar with the basic physiology of ventilation and the appropriate settings for newly intubated patients.

— Sandra M. Schneider, MD, Editor

Introduction

Airway management and the treatment of respiratory failure are major components of emergency medicine (EM). There are many techniques and tools with which to manage respiratory failure, and the list of options continues to expand. Mechanical ventilation (MV) is the ultimate tool for the management of acute respiratory failure. EM providers undergo a great deal of training in airway management, but there is often little training in the use of MV.

Many emergency departments (EDs) and hospitals are stretched from a bed and resource standpoint. There is significant overcrowding in EDs throughout the country, and this has a direct effect on patient care. Intubated critically ill patients are spending more time in the ED, and this is associated with a negative impact on their outcomes.[1,2] As evidenced by early therapy for severe sepsis/septic shock and the golden hour in trauma, many life-saving interventions are time sensitive. With intubated patients having longer stays in the ED, it is important for EM providers to become more comfortable with initial and ongoing ventilator management. This paper will discuss the indications for invasive positive pressure ventilation (PPV), respiratory physiology, parameters of the ventilator, ventilator modes, physiologic changes after intubation, ventilator management of specific disease processes, and tips for trouble-shooting common problems.

Indications for Invasive PPV

There are four main indications for intubation: 1) inability to ventilate; 2) inability to oxygenate; 3) airway protection; and 4) anticipated clinical course.[3] It is important to understand how invasive PPV will benefit patients.

Ventilation

Ventilation is the removal of CO_2 from the body. Minute ventilation (V_E) is the volume of air entering or exiting the respiratory system per minute.

V_E = RR x V_T, where RR = respiratory rate and V_T = tidal volume

Ventilation is a dynamic process that relies on matching respiratory drive to

Executive Summary

- There are many methods of delivering positive pressure breaths. In the ED, volume-controlled ventilation is most commonly used. The amount of air delivered is regulated by the volume designated, as opposed to pressure-related ventilation, which delivers air until a set pressure is reached.
- Although ARDS generally develops in patients after they leave the ED, it may be prudent to use lower tidal volumes of 6 mg/kg based on ideal body weight (adjusted by height) in all patients, at least initially.
- PEEP is positive pressure applied at the end of expiration. Its purpose is to keep alveoli open and to recruit or open alveoli that are closed. It is useful when there is a V/Q mismatch or shunt. Suspect a shunt when supplemental oxygen does not reverse hypoxia. The effect of PEEP is not seen for 10-60 minutes.
- Auto-PEEP occurs when incomplete exhalation occurs and is often seen in patients with COPD or asthma. The increase in pressure may lead to hypotension or barotrauma. Acutely it can be managed by disconnecting the patient from the ventilator until a complete exhalation occurs, then readjusting the ventilator.

metabolic demands. Hypoventilation is the result of a respiratory drive that is unable to match intrinsic metabolic demands, which results in an elevated pCO_2 level and acidosis. This may be seen in times of normal or accelerated metabolism and cellular waste production. Inadequate ventilation is due to a relatively low RR, low V_T, or increased dead space ventilation fraction. The alveolar-capillary CO_2 exchange is quite efficient. CO_2 exchanges across the alveolar-capillary exchange interface about 20 times more efficiently than oxygen (O_2).[4] As a result, dysfunctional gas exchange at the level of the alveoli is rarely a primary driver for hypoventilation.

It is critical to determine the acuity of pCO_2 elevation. Patients with chronic respiratory diseases like chronic obstructive pulmonary disease (COPD) or obesity hypoventilation syndrome may have baseline elevated pCO_2 levels. This pCO_2 elevation has occurred over time, and compensatory mechanisms have been able to buffer the acidic CO_2. The most prominent compensatory mechanism is the kidney's excretion of carbonic acid and increased bicarbonate absorption.[5] This process, while powerful, takes 1-5 days to provide significant compensation. This is the key to differentiating the timing of an elevated pCO_2. An elevated pCO_2 with associated acidosis (pH < 7.35) suggests an acute uncompensated hypoventilation. An elevated pCO_2 without acidosis warrants further clinical investigation because the patient may be at his or her baseline state.

While thinking of ventilation in the simple terms of RR and V_T is clinically helpful, it is important to account for the components of V_T.

$V_T = V_A + V_D$, where V_A is alveolar ventilation and V_D is dead space ventilation

Dead space includes the endotracheal (ET) tube, trachea, large airways, and all parts of the airway where gas is not exchanged. These are all fixed volumes and are the first volumes of gas exhaled. Since gas exchange does not occur in this volume, there is not ventilatory contribution. On a ventilator, the typical V_D in an adult is about 150 mL.[4] With this in mind, consider a typical adult patient with shallow breaths (V_T about 250 mL). The patient will only be exchanging about 100 mL of alveolar volume per breath. Even if the RR is 50, the V_E will be 5 liters, which may be too little depending on the clinical situation.

When dealing with a ventilated patient, the two parameters used to modify ventilation will be RR and V_T. If low RR is the only variable driving the acute hypercapnic respiratory failure, this should be evident when examining and monitoring the patient. Hypoventilation secondary to low RR typically stems from the central nervous system (CNS). The CNS may be affected by injury, disease, toxins, or medications (especially inappropriate narcotic and sedative ingestions). Reversing the underlying CNS cause of hypopnea will lead to adequate ventilation. If this is not possible or ineffective, the patient will require PPV with a set minimum RR greater than the patient's intrinsic rate.

There are also a number of conditions that lead to hypoventilation by way of inadequate V_T. One group of conditions is defined by poor respiratory muscle contraction, as seen in neuromuscular disorders like Guillain-Barre syndrome, amyotrophic lateral sclerosis, or myasthenia gravis. Poor chest wall mechanics, as seen in significant trauma or restrictive thoracic anatomy, is another cause of low V_T. Lastly, low volumes of gas reaching alveoli (e.g., obesity, pulmonary edema, or pneumothorax) may contribute to inadequate ventilation. When combined with treatment of the underlying problem, PPV may improve gas exchange by augmenting the patient's V_T.

Failure to ventilate adequately is identified with clinical exam and a blood gas (arterial or venous). Interpreting this information and determining the need for intubation is often more complicated. It is important to take into account the patient's history (including baseline pCO_2 level), symptoms of hypercapnia, and response to therapy. Medications targeted at the

underlying cause (i.e., naloxone) or non-invasive PPV may be appropriate treatment options for patients with hypoventilation. If the patient fails to respond to these interventions or has hypercapnia with severe metabolic or mental status derangements, invasive PPV is most appropriate.

Oxygenation

While ventilation may be clinically simplified into two components, the process of oxygenation encompasses more variables and is more complicated. Oxygenation includes all processes that lead to the delivery of O_2 from the upper airway to the tissues. Hypoxemia may be due to one or more of the following: V/Q mismatch, poor diffusion, low inspired FiO_2, or hypoventilation. (*See previous section.*)

Gas delivery to the alveolus is known as ventilation (V). Transport of gas across the alveolar-capillary membrane into the circulatory system is flow (Q). The ratio of alveolar ventilation (V_A technically but by convention, it is shortened to V) and capillary flow (Q) is an important consideration when treating a patient with hypoxic respiratory failure. In normal lungs, there is an inherent V/Q inequality that is related to gravity. More blood flows to the dependent areas of lung (i.e., there is more Q). This leads to different physiologic zones within the lung related to the gravity-dependent areas. In the most dependent areas, the V/Q ratio is lower. In higher areas, there is relatively more V and less Q, thus the V/Q ratio is higher. This is a normal physiologic situation, but there are certain disease processes that exaggerate this ratio and contribute to respiratory failure. There are a number of factors that may impede V or Q.

Two classic examples of V/Q mismatch are: the lung that is completely obstructed (i.e., main bronchus mucous plug); and the lung that receives no blood flow (i.e., main pulmonary artery embolism). In the first example, the lung segment or alveolus has been completely obstructed so that no air gets to the alveoli. In this case, the V = 0 and Q remains the same. The V/Q = 0, which is known as a pulmonary shunt. In the second example, the lung segment has adequate ventilation but has no blood flow to the alveolar-capillary interface (i.e., during pulmonary embolism). This situation will have $V/Q = \infty$, which is known as dead space. In clinical terms, oxygenation will be poor in the setting of significant V/Q mismatch, regardless of the extreme end of the spectrum on which it lies.[4]

Gas delivery to smaller airways may be disrupted by several mechanisms. It is possible to have ineffective delivery due to bronchial collapse. These conditions may be seen in external mass effect on airways, foreign bodies, or an ET placed into a main stem bronchus. There are also conditions that inhibit alveoli's ability to accommodate gasses. Alveolar collapse (atelectasis) will often cause hypoxia. Atelectasis is related to pressure compression of alveoli, insufficient thoracic negative pressure, surfactant deficiency/dysfunction, or mechanical obstruction of the smaller airways due to mucus. There are also a number of infiltrative processes in which alveolar gas exchange is limited because the alveolar space is occupied by simple fluid, blood, inflammatory cells, or debris.

Oxygen diffusion across the alveolar-capillary membrane is far less efficient than CO_2 diffusion. Therefore, even when gas delivery to alveoli has occurred, there may still be barriers to O_2 diffusion across the alveolar-capillary membrane. Acute processes that alter the interface between alveolus and capillary, like pulmonary edema and pneumonitis, are commonly seen in the ED. In each of these conditions, it is critical to identify the underlying cause and target treatment with appropriate anti-inflammatories, diuretics, or antibiotics. Supplemental O_2 will increase alveolar oxygen concentration and help improve most forms of hypoxemia.

Failure to oxygenate is easy to identify with clinical exam, pulse oximetry, or an arterial blood gas. When supplemental O_2 or non-invasive positive pressure ventilation (NIPPV) is unable to provide adequate oxygenation, invasive PPV is indicated. PPV allows for the best optimization and control of oxygen delivery (DO_2) and mean airway pressure (MAP). There are numerous methods for supplementing O_2 concentration beyond the 21% fraction of inspired oxygen (FiO_2) of room air. Simple non-invasive tools primarily include nasal cannulas, face masks, and face shields. By increasing O_2 concentration in the alveolar gas, a favorable O_2 gradient is created and O_2 diffuses from the alveolus to the alveolar capillary beds. The FiO_2 delivered with supplemental O_2 can reach levels of 100%, but delivering such high concentrations may be detrimental.

The idea of oxygen toxicity or the production of dangerous free radicals at high levels of FiO_2 has been classically taught, but its true clinical impact is heavily debated. There is some evidence that in specific populations, hyperoxia may be associated with increased mortality.[6] Yet, hyperoxia has been used as a potential therapy for severe traumatic brain injury and has not been shown to cause increased free radical production or pulmonary damage related to a pro-inflammatory state.[7,8] Despite maximizing FiO_2, there will be situations in which hypoxia prevails. These are cases in which V/Q = 0, and PPV is required to improve the shunt physiology before the hypoxia will be corrected.

There are several factors that contribute to the mean airway pressure (MAP), including inspiratory time and inspiratory pressure, but the most clinically significant factor is positive end expiratory pressure (PEEP). MAP augmentation is a critical tool in the treatment of hypoxia related to intrapulmonary shunting. PEEP is a continuous pressure that remains after completion of the exhaled phase. By keeping some positive airway pressure during exhalation, there is less alveolar collapse. The underlying pulmonary pathology, level of PEEP, and pulmonary

compliance all factor into how well alveoli respond to PEEP. In general, the amount of alveolar collapse will decrease as PEEP is increased. With time and increasing levels of PEEP, collapsed alveoli may open up. This is alveolar recruitment. Using PEEP is a way to try to expose more alveoli to an elevated MAP with each respiratory phase. The alveoli open and then stay open because of PEEP. Alveolar recruitment improves pulmonary compliance and improves shunt physiology by increasing the functional reserve capacity. As more alveoli open and are able to accommodate air flow, there is an increase in V_A that will help correct V/Q mismatching.

Once a patient is connected to invasive PPV and V_A is optimized, it is important to account for the extrapulmonary components of oxygen delivery.

$DO_2 = CO \times [(1.34 \times Hgb \times SaO_2) + (0.003 \times PaO_2)]$, where CO = cardiac output, Hgb = hemoglobin

This equation highlights the efficiency of hemoglobin's oxygen transport and the importance of attaining adequate O_2 saturations. There are only a few situations (e.g., severe anemia or carbon monoxide poisoning) in which focusing on the small contribution of dissolved oxygen (pO_2) will yield significant clinical effects. The techniques for optimizing CO and appropriate hemoglobin levels fall outside this discussion on MV, but their consideration is paramount in critically ill patients who show evidence of inadequate DO_2.

Airway Protection

This indication for MV is the most subjective. There are many different etiologies to consider when dealing with upper airway compromise. For patients with upper airway obstruction, intubation or other airway bypass means are often necessary. Many etiologies, like angioedema and neck hematoma, tend to worsen over time without definitive treatment. Early intubation while treating the underlying reason necessitating airway protection is the best treatment plan. It is easier to identify obstructive airway tissues than it is to assess a patient's ability to protect his or her airway. There is a wide range of diseases that could compromise a patient's ability to protect his or her airway from occlusion or aspiration. Differentiating patients based on the etiology is helpful, especially if the underlying problem is immediately reversible. However, many patients have metabolic or neurologic dysfunction that cannot be reversed easily. The provider must rely on subjective features, including alertness, swallow/speech function, and secretion quality, to make a determination regarding airway protection. There have been attempts to assign airway protection prognostication to more objective measures like the Glasgow Coma Scale (GCS) and the presence of a gag or cough. A depressed GCS of 8 or less has value in determining which patients require intubation, but it alone is not an adequate decision tool.[9,10] In controlled experiments, 37% of healthy subjects did not have a gag reflex.[11] In every case, the provider must carefully consider the GCS and individual clinical situation when determining if intubation is needed for airway protection.

Anticipated Clinical Course

There are situations in which the EM provider has to evaluate and treat the patient while trying to predict downstream events. Many times, careful foresight and a controlled intubation can prevent a crash airway emergency. Consider a patient with blunt trauma who arrives in the ED with a borderline hemodynamic status and waning mental status. One could argue that immediate intubation is unnecessary because the patient is protecting his or her airway, oxygenating well, and has a GCS greater than 8. However, there is the high likelihood of serious injury and a clinical trajectory of decreasing mental and hemodynamic status. This patient will need prompt clinical and radiographic evaluation and possibly emergent operative intervention. Transporting the patient away from the ED for imaging, the operating room, or to another facility may put him at risk of aspiration or other airway failure. Pre-emptively securing a patient's airway in the ED is far safer than having to emergently manage an airway while in the halls of the hospital or in the back of an ambulance.

In addition to patients with multisystem trauma, there are medical patients who will benefit from early intubation. For example, fatigue is highly likely in an elderly patient with pneumonia, mild hypoxia on supplemental O_2, tachypnea, and using some accessory muscles. Even with antibiotics and oxygen, the clinical picture is not likely to improve over the next several hours. Intubating the patient early in the clinical course is preferred to letting the patient become exhausted and use up his or her physiologic reserve. There are also patients with severe non-pulmonary illness who benefit from early intubation. Consider a patient with bacteremia and associated septic shock. This patient has increased metabolic demands and physiologic strain. The pulmonary function may be normal, but in states of critical illness, the respiratory system may account for up to 24% of metabolic demands.[12] Intubating these patients will offload physiologic strain and allow for more appropriate allocation of metabolic resources.

Ventilator Parameters and Modes

Current ventilators have a variety of modes. Many clinicians tout the benefits of one mode over another, but most of this is based in anecdotes and personal preference. There is not one mode of ventilation that is best for all patients, but there are some modes that may be more appropriate than others in certain clinical situations.[13] The modes typically seen in the ED include Assist Control (AC), Synchronized Intermittent Mandatory Ventilation (SIMV), Pressure Support Ventilation (PSV), and Airway Pressure Release Ventilation (APRV). There are additional modes, but those are used less

frequently. The variety of modes, numerous acronyms, and different terminology may lead to confusion, but breaking MV down to its basic parameters may aid in the understanding of each mode.

Parameters

Parameters are features and settings on the ventilator that determine how a breath is initiated, how large a breath will be, and how long a breath will last. Parameters are independent of the mode of ventilation. One mode may have several possible combinations of the same parameters. Regardless of the mode, there will always be these four parameters. The selection and combination of the parameters helps define the mode and determines how the patient and ventilator will interact.

Control

The term control has multiple meanings when talking about MV. A ventilator breath can be referred to as controlled or assisted. Breaths that are initiated by the ventilator are termed controlled, and those that are initiated by the patient are termed assisted. Control, as a parameter of the ventilator, is what the ventilator delivers to the patient. Volume control or pressure control are most frequently used. For volume control, a specific V_T is set. The ventilator then modulates pressure delivery in order to attain the set volume. As pulmonary compliance worsens, the ventilator will use a higher pressure to achieve the set volume. Volume control is most commonly used when patients are initiated on MV because it allows for close control of V_E. In pressure control (sometimes called PCV), a maximum pressure is set. Volume is delivered until the desired pressure is reached. With pressure control, V_T will vary with each breath. As pulmonary compliance worsens, the resultant V_T will be smaller. Since there may be dramatic breath by breath variation in the V_T during pressure control, it may be more difficult to ensure a consistent V_E.

Trigger

Trigger is the parameter that determines what initiates inhalation. In most cases, this will be time, pressure, or flow. There are some applications in which esophageal pressure or neurologic impulses will be used as a trigger, but these are rarely seen in the ED. When time is used as the trigger, the RR is selected and each minute is divided into equal blocks based on that rate. The ventilator will then ensure that a controlled breath is given at least once in every time period. In some modes, the ventilator will not deliver a time-triggered breath if the patient initiates his or her own breath in that specified time block. For example, if the RR is set for 10 breaths per minute, there will be a time trigger every six seconds. If the patient does not initiate an assisted breath, then the ventilator will ensure that a controlled breath occurs every six seconds.

In addition to time, pressure or flow may be a trigger to initiate a breath. Pressure or flow triggering relies on the patient taking a breath. When the patient inhales, there will be a decrease in the pressure within the ventilator tubing. This attempted breath will also create negative flow. If the pressure decrease or negative flow meets the set threshold, then an assisted breath will be delivered. Pressure and flow triggering allows the ventilator to support or augment breaths in those patients with an intrinsic drive but insufficient strength. Some breaths within a set mode may be time-triggered and others may be pressure- or flow-triggered.

Cycle

Cycle is what ends the inspiratory phase of the ventilator. This is different than starting exhalation. Exhalation is a passive process on a ventilator, unless using a high-frequency mode like high-frequency oscillatory ventilation. The cycle may be volume, pressure, flow, or time. Once the inspiratory phase has reached the set endpoint, the ventilator will stop delivering a positive pressure breath. The patient may continue inhaling beyond this point, but the ventilator will no longer be assisting. As soon as the inhalation stops, it gives way to the exhalation phase of breathing.

Limit

Limits are the safety mechanisms on the ventilator. A limit may be set to a specific pressure, volume, flow, or time. Limits are determined for the control, the trigger, and the cycle. If a limit is reached, then an alarm goes off. This alarm is a warning to the provider that there is a problem with the control, the trigger, or the cycle. The purpose of having a limit is that if the ventilator goes beyond the limit, then there is a higher likelihood of injury to the respiratory system.

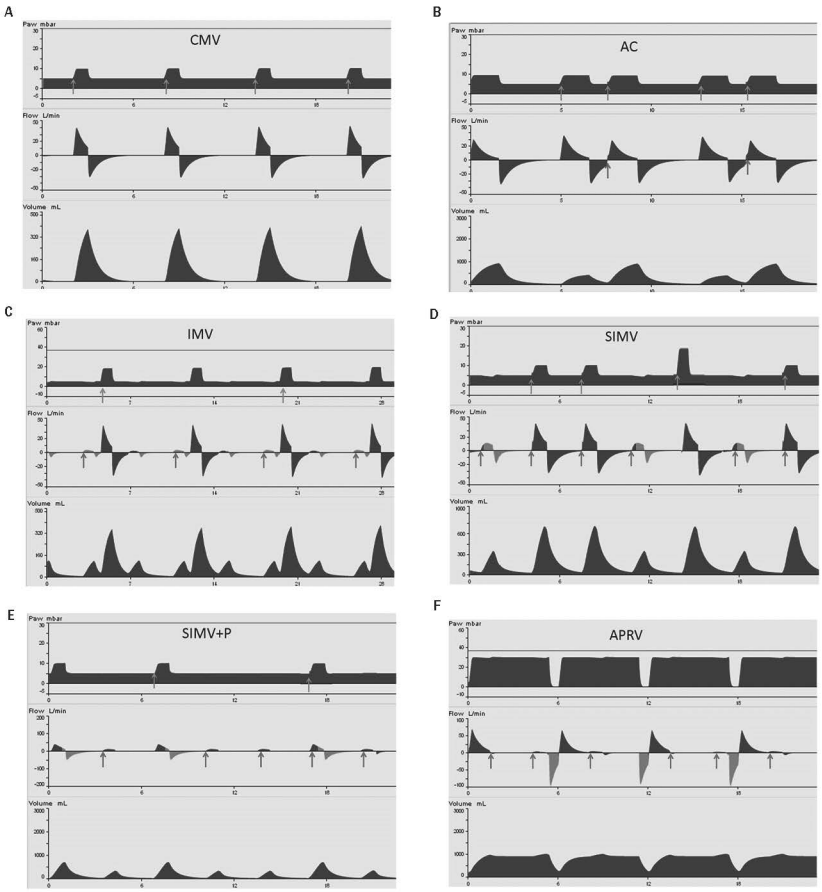
Modes

The mode of ventilation refers to the manner in which the ventilator provides inspiratory support to the patient. Each mode uses some combination of the above parameters to ensure that gas is delivered to the patient. The mode will determine how the patient and ventilator interact. Some modes disregard a patient's respiratory drive and focus only on delivering controlled breaths. Other modes may not deliver controlled breaths and only assist when the patient attempts a breath. Mode names may vary depending on the ventilator brand. Focusing on the underlying parameters and the nature of the patient-ventilator interaction will guide the appropriate mode choice for each clinical situation. Figure 1 shows the interaction between the ventilator and the patient for the different modes below.

Continuous Mandatory Ventilation (CMV) and Assist Control (AC)

The oldest and simplest mode of mechanical ventilation is CMV. It may be volume- or pressure-controlled but will always be time-triggered. The provider sets the desired volume or pressure and then sets the desired rate. A controlled breath will

Figure 1: Waveforms of Different Ventilator Modes



Waveforms of different ventilator modes A) CMV: Note the arrows in the Paw represent controlled breaths. B) AC: In this diagram, the first and third arrows in the Paw are controlled breaths, the second and fourth are the assisted breaths. In the Flow section, the arrows are the patient's attempts to breathe. C) IMV: In the Paw section, the arrows represent controlled breaths and the arrows in the Flow section are the patient attempts to breathe. D) SIMV: In this diagram, the first, second, and fourth arrows in the Paw are assisted breaths, the third is a controlled breath. The arrows in the Flow section are the patient attempts to breathe. E) SIMV+P: In this diagram, the first arrow in the Paw is an assisted breath, the second is a controlled breath. The arrows in the Flow section are the patient attempts to breathe. F) APRV: The arrows in the Flow section are the patient attempts to breathe. (Images adapted from Dräger Evita V500 Product Demonstrator manual simulation available at www.Draeger.com.)

be delivered at the specified rate. It is an appropriate mode for patients who have no intrinsic respiratory drive or are paralyzed. The downside of CMV is when the patient has a RR greater than the set rate and attempts a spontaneous breath between controlled breaths. These spontaneous patient breaths occur against a closed circuit. For the patient, this is analogous to inhaling through a tube with a cork on the end. This creates discomfort and anxiety and may make weaning from MV difficult.

Because of the poor patient-ventilator interaction in CMV, it has largely been replaced by AC. If a patient is paralyzed or has an intrinsic RR less than that set on the ventilator, then CMV and AC are the same. The ventilator will deliver a volume- or pressure-controlled breath at the set time. The major difference between CMV and AC is how the ventilator responds when a patient initiates a spontaneous breath. Rather than the spontaneous breath being against a closed circuit as in CMV, in AC the breath will be assisted. The ventilator will still ensure that a breath occurs during each time cycle, using controlled breaths if the patient does not initiate a breath. In AC, the ventilator will also give an assisted breath whenever the patient attempts a spontaneous breath. AC is a well-tolerated and comfortable mode of ventilation that is able to dramatically decrease a patient's work of breathing. This feature makes AC a valuable mode in the treatment of respiratory failure related to shock or sepsis. However, since the patient receives the set V_T with each spontaneous effort, the V_E may be excessive. Patients with an inappropriately high RR will be at risk for hyperventilation.[14,15]

Intermittent Mandatory Ventilation (IMV) and Synchronized Intermittent Mandatory Ventilation (SIMV)

IMV is a volume- or pressure-controlled mode of ventilation that uses a time trigger. There is a set rate of volume- or pressure-controlled breaths. At first glance, it appears to be the same as CMV. In patients with no respiratory drive or one with an intrinsic rate under that set on the ventilator, IMV is essentially the same as CMV. However, there is a major difference between CMV and IMV when a patient has a spontaneous breath. In IMV, those spontaneous breaths are against an open circuit. It is as if the patient is breathing through an open-ended tube rather than a tube that is occluded. The patient is able to breathe whenever he wants, but there are still time-triggered controlled breaths. This may lead to breath stacking when controlled breaths are delivered on top of a patient's spontaneous breaths. The resultant V_T may be quite large and put the patient at risk for barotrauma or volutrauma.

SIMV was developed to avoid these air-stacking situations and create a better patient-ventilator interaction. SIMV is a modification of IMV that is time- and patient- (pressure or flow) triggered. For a set RR, the ventilator will divide a minute into equal blocks or segments. The ventilator will ensure that one controlled breath is delivered during each of those blocks. If the patient initiates a spontaneous breath during the block, the ventilator will deliver an assisted breath. That breath will satisfy the requirement for the specific block of time, and the ventilator will wait for the next segment. If the patient does not take a spontaneous breath during the time segment, a time-triggered controlled breath will be delivered. The ventilator will wait until the end of the time segment before delivering a controlled breath, thereby giving the patient an opportunity to trigger a breath on his or her own. For example, if the ventilator's rate is set at 10, each minute will be divided into 10 six-second blocks. The patient may take spontaneous breaths in all or none of those blocks but will still get at least 10 full breaths per minute. If the patient initiates a breath two seconds into the block, then the ventilator will deliver an assisted breath, which will satisfy the breath requirement for that block. Like IMV, the patient is also able to take extra breaths. If the patient initiates two or more breaths during a block, only the first breath will be a full assisted breath. The subsequent breaths in the same time block will not be assisted by the ventilator.

Since the additional breaths in a time period are unsupported, it is common to combine SIMV with Pressure Support Ventilation (PSV). This new "mode" is called SIMV+P, but it is actually two ventilator modes working together. SIMV+P only differs from SIMV when taking into account the patient's extra spontaneous breaths in a time segment. In SIMV, the patient's extra breaths are unsupported, but in SIMV+P, these extra breaths are given a set amount of pressure support. The addition of PSV allows the patient's subsequent spontaneous breaths in a time period to contribute more to the minute ventilation. One of the main benefits of SIMV+P is thought to be its ease in weaning, although there is little evidence to support this.[13] SIMV+P is especially advantageous in patients who have inappropriate tachypnea. Patients are less likely to hyperventilate than if they were on AC.

Pressure Support Ventilation (PSV)

PSV is a pressure-controlled mode of ventilation with flow or pressure triggering. Often, providers think of PSV as an invasive form of BiPap. With PSV, there are two pressures that may be adjusted. The first is the inspiratory pressure, which is delivered when the patient generates enough pressure or flow to trigger the ventilator. The second pressure is PEEP. As the expiratory phase ends, the ventilator will keep a set level of PEEP, thereby preventing alveolar pressures from returning to 0 cm H_2O. Each breath the patient takes will get the same inspiratory pressure and PEEP. The rate is up to the patient. The resultant V_T will vary depending on the patient's pulmonary compliance, chest wall compliance, and respiratory effort.

A key difference between PSV and controlled modes, such as AC or SIMV, is that PSV is only pressure- or flow-triggered. There are no breaths unless the patient has respiratory effort, thus there is no way to guarantee a minimum V_E. An apneic or very weak patient who is unable to mount a satisfactory inspiratory force will not trigger a breath. It is for this reason that PSV is not an acceptable mode for patients who are paralyzed, have profound diaphragm weakness, or are prone to apnea. PSV is, however, a comfortable and well-tolerated mode for patients with a good respiratory drive. There is less patient-ventilator dyssynchrony than with other modes of ventilation, and the patient is able to control his or her V_E. PSV is most commonly used in patients intubated for upper airway protection and those who are weaning toward extubation.

There are several considerations regarding the use of PSV in the ED. First, it will rarely be an appropriate mode immediately post-intubation. Most induction agents blunt respiratory drive, and paralytics are often used. Therefore, the patient will not have the drive or ability to trigger breaths. In addition, variations in the patient's RR and V_T make it difficult to ensure a consistent V_E. Instead, a mode that is volume- or pressure-controlled with a time trigger, such as AC or SIMV+P, will be most appropriate. However, when critically ill patients are spending hours to days in the ED, switching to PSV outside the peri-intubation period may be appropriate.

Airway Pressure Release Ventilation (APRV)

APRV (sometimes referred to as bi-level) is a mode that focuses on elevating the mean airway pressures for the purpose alveolar recruitment. It is typically used in patients with poor oxygenation and poor pulmonary compliance. It is a pressure-controlled and time-triggered mode of ventilation. Since it is pressure-controlled, there is no specific V_T to set. Instead, the pressures (P_{high} and P_{low}) and times (T_{high} and T_{low}) are set and modified to attain adequate oxygenation and ventilation. The majority of oxygenation occurs at P_{high}, when the alveoli are subject to a sustained pressure. This pressure may force open alveoli, and the continuous nature of it tends to keep the alveoli open, thereby leading to alveolar recruitment. The recruited alveoli increase V_A. With increased V_A, gas exchange should improve. This concept is most helpful with oxygenation. CO_2 removal primarily occurs during the patient's unassisted spontaneous respirations at P_{high} and during pressure drops (releases) from P_{high} to P_{low}. The pressure gradient from P_{high} to P_{low} is a major factor for determining amount of CO_2 removed.

APRV is excellent for oxygenation, but adequate ventilation may be difficult. Since a good deal of CO_2

Table 1: Suggestions for Initial Ventilator Settings Based on Different Clinical Pictures

	Airway protection	Hypoxia	COPD or Asthma	Increased metabolic demands
Key concerns or considerations	Apnea	Lung protection	Auto-PEEP	Matching respiratory alkalosis
Mode	PSV, AC, SIMV+P	AC, SIMV+P	AC, SIMV+P	AC, SIMV+P
V_T (mL/kg of IBW)	6	6	6	6
RR	10-14	12-20	6-8	18-24
PEEP (cm H_20)	5	8	0-5	5
FiO_2	0.4	0.8-1.0	0.4-1.0	0.4
I:E	1:2	1:2	1:3-1:5	1:2
Inspiratory flow (L/min)	60	60	100	60
Each setting should take into account the underlying pathology and key physiologic consideration.				

removal depends on the patient's spontaneous breaths, this mode should be avoided in heavily sedated or paralyzed patients.

Initial Ventilator Settings

Whenever a patient is placed on MV, it is important to have an idea of why the patient needs the ventilator. This knowledge will help determine what the initial ventilator settings should be. (*See Table 1.*) First, choose the mode. A volume-controlled mode with time trigger, such as AC or SIMV+P, is usually the first choice for most clinicians since it will ensure a minimum V_E and is appropriate for apneic patients. PSV is a potential option but only in spontaneously breathing patients without risk of apnea.

Second, set (in the case of a volume-controlled mode) or target (in the case of a pressure-controlled mode) a V_T of 6 mL/kg predicted body weight. Low tidal volumes of 6 mL/kg of ideal body weight (IBW) have been shown to decrease mortality and ICU length of stay in patients with acute respiratory distress syndrome (ARDS).[16] Even when the clinical scenario may not meet all criteria for ARDS, there is emerging evidence that patients without ARDS have better outcomes when using low V_T.[17] It is also important to use IBW to calculate V_T, which is based on a patient's height. Approximately 10-15 minutes after initiating a V_T of 6 mL/kg IBW, it is important to assess the plateau pressure (P_{plat}). This is done by performing an inspiratory hold on the ventilator. Most modern ventilators have a button/knob for this feature. If P_{plat} is greater than 30 cm H_2O, it is important to lower the V_T to achieve a P_{plat} less than 30 cm H_2O because this lessens the risk of pressure-related injury and yields better patient outcomes.

There are clinical situations in which a low V_T may not be appropriate as an initial setting. Patients with profound metabolic acidosis, or those with sensitivity to elevated pCO_2 (i.e., increased intracranial pressures) may not have adequate ventilation with low V_T, despite maximizing RR. In these cases, a higher V_T may be appropriate, but the amount of time at a higher V_T should be limited.

The third step in setting up the ventilator is to select the RR. The goal is to match or exceed the pre-intubation V_E. For clinical conditions in which the patient has a high metabolic demand and V_E, such as septic shock, salicylate toxicity, or diabetic ketoacidosis (DKA), it is important to ensure that the post-intubation V_E is high. For example, a patient with DKA may have a pH of 6.9 while generating his or her own V_E of 30 L/min. In this case, the patient is barely compensating for the severe metabolic acidosis despite an extremely high V_E. Choosing a V_T and RR that yields a lower V_E than the pre-intubation value of 30 L/min may lead to cardiac arrest since the lower V_E will allow CO_2 to rise, which will cause the pH to fall below 6.9. After setting the RR, it is important to frequently reassess the patient's pH and ventilator parameters. In a case such as this, end tidal CO_2 measurement before, during, and after intubation may be particularly useful. Monitoring of real-time trends in ventilation may allow for quicker intervention than conventional titration by blood gases.[18] With regard to the severity of lung injury, if P_{plat} is elevated, a V_T of 4-5 mL/kg may be needed. In this situation, it is often necessary to increase the RR to maintain an appropriate V_E.

The next setting to consider is the breakdown of each breath into an inspiratory and expiratory phase. The most common inspiratory to expiratory ratio (I:E) is 1:2. This means that twice as much time is allotted to the expiratory phase of the breath. It is necessary to consider the patient's underlying lung pathology and monitor the ventilator outputs for evidence of auto-PEEP. Patients with a prolonged expiratory phase due to

Table 2: Tables from ARDSnet Protocol Summary Showing Different Combinations of PEEP and FiO_2 [20]

Higher PEEP and Lower FiO_2													
FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18-24
Lower PEEP and Higher FiO_2														
FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	5	8	10	12	14	14	16	16	18	20	22	22	22	24
The goal oxygenation is PaO_2 55-80 mmHg or SpO_2 88-95%.														

bronchospasm (e.g., COPD) who are prone to air trapping will often need a longer I:E, occasionally as high as 1:8.

The last major settings are FiO_2 and PEEP. As mentioned, each of these relates to oxygenation, and the choice of settings will rely heavily on the clinical situation. An FiO_2 up to 100% may be used initially, but this amount of FiO_2 is usually not needed when PEEP and FiO_2 are titrated to maintain an O_2 saturation of > 89%. If a patient is intubated for airway protection and pre-intubation oxygenation was not an issue, then an FiO_2 of 40%-60% with PEEP of 5 should be adequate. For those patients with significant hypoxia, an initial FiO_2 of 80-100% may be more appropriate. A higher PEEP of 8-12 cm H_2O would also be reasonable in an effort to raise the MAP and improve oxygenation. The ARDS network has developed a set of tables (*see Table 2*) to assist providers with initial choice and titration of PEEP and FiO_2. These tables help guide settings that optimize recruitment while minimizing the harmful effects of excessive PEEP.[19] It is important to know that there is no outcome difference between the different tables.[20] It is best to pick one strategy and continue it throughout the clinical course.

When initiating APRV, P_{high} is set at approximately 5 cm H_2O above the plateau pressure with a maximum P_{high} of 35 cm H_2O. P_{low} is set to 0 cm H_2O to provide the largest pressure gradient and optimize ventilation. To optimize oxygenation, it is important to maximize the time with high airway pressure (T_{high}) and minimize time with low airway pressure (T_{low}). The net effect of this strategy will raise the MAP and improve oxygenation. The more time spent at T_{low} may lead to alveolar collapse or derecruitment. Derecruitment may be further minimized by setting a T_{low} that is short enough so that P_{high} restarts before the expiratory/release flow rate has gone below 50%. A common starting point is to set T_{high} for 5.4 seconds and T_{low} for 0.6 seconds. On these settings, there will be one release every six seconds or 10 releases per minute.

Physiologic Changes Due to PPV

The process of induction, paralysis, and initiation of PPV has dramatic effects on a patient's physiology. Most patients who are emergently intubated in the ED are critically ill and have poor physiologic reserve. Failure to account for and anticipate the physiologic changes that occur post-intubation may lead to poor patient outcomes. Most commonly used induction medications cause vasodilation leading to hypotension. Some medicines, such as propofol, may also contribute directly to hypotension by inducing myocardial depression.

Once PPV is initiated, thoracic physiology is changed. Each breath is delivered by positive pressure from the ventilator rather than the negative pressure derived from diaphragm contraction and thoracic expansion. In addition, there is the contribution of PEEP that creates a baseline positive pressure environment even at times of exhalation. The increase in intrathoracic pressure during and between each breath may cause a significant change in cardiovascular function. Under normal circumstances, when a person inhales, venous return to the right atrium (RA) is assisted by the negative intrathoracic pressure. The negative pressure gradient minimizes impedance on venous return and will provide added gradient that helps draw venous blood into the RA. When on PPV, the positive intrathoracic pressure may dramatically decrease venous return and cardiac preload by negating the normal physiologic advantages. There will be loss of favorable pressure gradient from abdomen to thorax, which will also impede venous return to the RA.

PEEP will also reduce left ventricular (LV) afterload. This effect may be related to the decrease in venous return to the heart that causes a reduced preload that is translated into a decreased mean arterial pressure. The PEEP-induced decrease in LV afterload may also be due to the reduction in the LV end systolic transmural pressure. This occurs when intrathoracic pressure is elevated and arterial pressure is constant. The LV needs less force to eject blood into the aorta. Regardless of the mechanism, the reduction in LV afterload does not correspond to an increase in cardiac output, unless the patient has congestive heart failure and is hypervolemic.[21]

Using PEEP to increase MAP is one of the primary methods for treating hypoxia, but there are also negative effects associated with

elevated PEEP that must be considered. The effects of PEEP are dose-dependent. In cases of poor pulmonary compliance, high levels of PEEP may dramatically decrease venous return to the RA and also dramatically increase pulmonary vascular resistance. High PEEP will also cause an increase in right heart afterload. In some cases, usually PEEP greater than 20 cm H_2O, cor pulmonale may occur.[22]

It is clear that the initiation of PPV dramatically affects cardiovascular physiology. Many common processes requiring intubation (e.g., trauma, severe sepsis, or other shock states) are associated with hypovolemia and inadequate preload. Initiating PPV may be enough to lead to cardiovascular collapse. Hence, EM providers must take care to evaluate fluid and preload status prior to intubation. More importantly, it is necessary to reevaluate the patient's hemodynamics in the first 10-15 minutes following intubation.

Specific Clinical Situations and Ventilator Considerations

Airway Protection. Patients who require intubation for airway protection are usually suffering from a neurologic insult. Whether the insult is related to a drug-induced encephalopathy or a direct damage to the brain, the patient who is unable to protect his or her airway will need a definitive airway. The ET is placed in an effort to protect the patient from aspiration and upper airway occlusion. Assuming the patient has metabolized the induction medications and paralytics and still has an adequate respiratory drive, PSV may be an appropriate mode of ventilation. PSV would allow the patient to breathe comfortably with a protected airway, while controlling his or her own VE.

Severe Hypoxemia and ARDS. Based on the new Berlin Criteria, ARDS is characterized as acute (less than 1 week onset), non-cardiogenic respiratory failure with bilateral opacities on chest radiograph, and PaO_2/FiO_2 ratio < 300 mmHg. There are separate classifications for mild (PaO_2/FiO_2 ratio < 300 mmHg), moderate (PaO_2/FiO_2 ratio < 200 mmHg), and severe (PaO_2/FiO_2 ratio < 100 mmHg) ARDS. Although the definition has changed, the clinical implication is the same. ARDS is a condition of hypoxia that may stem from a variety of illnesses, and is characterized by alveolar edema, endothelial damage, and neutrophil deposition.[23]

Patients may not initially present to the ED with florid ARDS, but it does not take long for ARDS to develop. As critically ill patients spend more time in the ED, EM providers must become familiar with ventilation management in ARDS.[24] While many ARDS management options may have little utility in the ED, there is an ARDS management strategy that is easily applied upon initial presentation. The use of low V_T is the cornerstone of ARDS management. In the original ARMA trial, V_T of 12 mL/kg IBW was compared to V_T of 6 mL/kg IBW. The low V_T group was associated with a dramatic decrease in mortality (31% vs. 40% at 28 days).[16] With this initial result, as well as several follow-up studies and analyses, the estimate for the number needed to treat is only about 10 to attain a mortality benefit.[25] The benefits of lower V_T ventilation in ARDS extend beyond mortality and include decreased time on the ventilator and decreased ICU length of stay.

The evidence for using low V_T is quite strong, but there are a number of real-world implications that must be taken into consideration. First of all, low V_T is based on IBW. Providers tend to do a poor job of estimating a patient's height while he lies in the stretcher.[26] For this reason, providers should take a brief moment to measure a patient's height before calculating the goal V_T.

Another issue to consider is ventilation. When using a low V_T, V_E becomes more reliant on a higher RR. There are times when the RR is limited by obstructive issues or auto-PEEP. Other times, the metabolic demands are so high that even with a high RR, the patient's V_E may still be inadequate. Despite this, there should still be an emphasis of maintaining a low V_T. In fact, if this clinical situation occurs, it is tolerable to allow the pCO_2 to rise greater than 45 mmHg or allow the pH to drift below 7.35. This is termed permissive hypercapnia.[27] Even in patients with a significant respiratory acidosis (pH < 7.2), it is recommended to continue low V_T ventilation. Treatment of the associated respiratory acidosis is controversial, but it is reasonable to add a buffer infusion like sodium bicarbonate or THAM to keep pH between 7.15 and 7.2028. There is even some suggestion that permissive hypercapnia may itself carry some mortality benefit outside the strict adherence to low V_T.[29–31]

Despite the benefits seen with ventilator strategies that rely on permissive hypercapnia, it is important to keep in mind the select patient populations that may be hurt by elevated pCO_2 levels. This includes patients with increased intracranial pressures or other significant neurologic issues, since the elevated pCO_2 will cause an increase cerebral blood flow. Patients with significant arrhythmias, heart failure (especially right ventricle), or pulmonary hypertension may not tolerate hypercapnia well either.

As mentioned above, ARDS may not be apparent in the ED, and it is difficult to determine which patients with acute respiratory failure will progress to that severity of illness. Despite that, the dramatic benefits seen with low V_T make it reasonable to use this ventilation strategy even if ARDS criteria are not met. In summary, patients intubated for hypoxic respiratory failure in the ED are likely to benefit from a V_T of 6 mL/kg IBW.

Obstructive Lung Disease and Auto-PEEP. The decision to intubate a patient with a COPD or asthma exacerbation is difficult because it often depends more on subjective factors, such as work of breathing, anticipated respiratory fatigue, or mental status changes. Once the patient is intubated, the

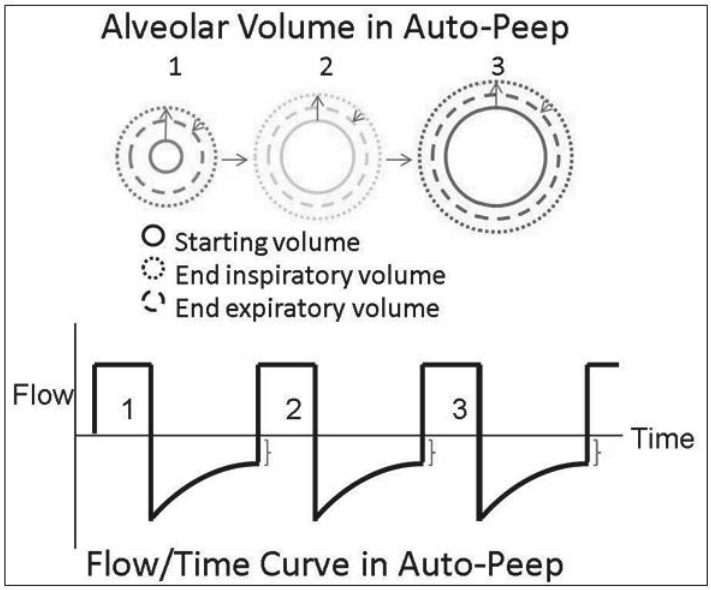
clinical management may become even more difficult. In the case of a COPD exacerbation, there is most likely going to be acute respiratory acidosis. Because of this and the need for close monitoring of V_E, a volume-controlled AC or SIMV is usually most appropriate. The provider can then set a minimum V_E in an effort to improve ventilation.

The obstructive nature of COPD and asthma makes these patients difficult to ventilate and also makes these patients prone to auto-PEEP[32] (also known as air stacking, breath stacking, or air trapping). When the end expiratory volume exceeds the relaxed lung volume (at the end of lung elastic recoil), there will be dynamic pulmonary hyperinflation. This represents the volume component of auto-PEEP. There is also a pressure component related to the buildup of air volume in airways.[33]

For most patients, an I:E of 1:2 is sufficient. However, in patients with obstructive lung disease who have expiratory flow limitation, an I:E of 1:2 may not allow for complete exhalation. For example, assume the patient is on a time-triggered volume-controlled mode that delivers a V_T of 500 cc every 5 seconds. The vent may deliver the 500 mL volume over 1 second, but with obstructive airway disease, it may take 5 seconds to fully exhale that volume. Monitoring of ventilator measurements may show inhaled V_T of 500 mL but measured expiratory V_T of only 400 mL. With each breath cycle, the problem is compounded and 100 mL of volume is added to the dead space. As this continues, there is increased alveolar distention and pressure. Eventually, the increasing airway pressures will inflict barotrauma, seen as pneumomediastinum or pneumothorax. The excess alveolar volume will overdistend and stretch the alveoli, causing volutrauma. Auto-PEEP also leads to a significant increase in intrathoracic pressure, which will cause cardiovascular compromise by impaired venous return to the RA and increased pulmonary vascular resistance. It is common to see hypotension or hypoxia as a result of these physiologic changes.

There are clinical clues and ventilator measurements that may help providers identify auto-PEEP early. From a clinical standpoint, it is helpful to auscultate the lungs during several respiratory cycles. If the next inspiration occurs while still auscultating the previous exhalation, then the patient has auto-PEEP. It is also important to monitor the patient's respiratory effort and synchrony with the ventilator. There are different ventilator values that may aid in the diagnosis of auto-PEEP. Assuming no circuit or airway leak, the measured exhaled V_T should be approximately equal the inhaled V_T. If there is a significant difference between those two values, auto-PEEP is likely present. There are also ventilator graphics that show flow over time. If the negative flow (representing exhalation) deflection does not return to the baseline (V = 0) axis before another inspiration is initiated, then there will be air trapping.[33] (*See Figure 2.*)

Figure 2: Flow vs. Time Curve of Auto-PEEP



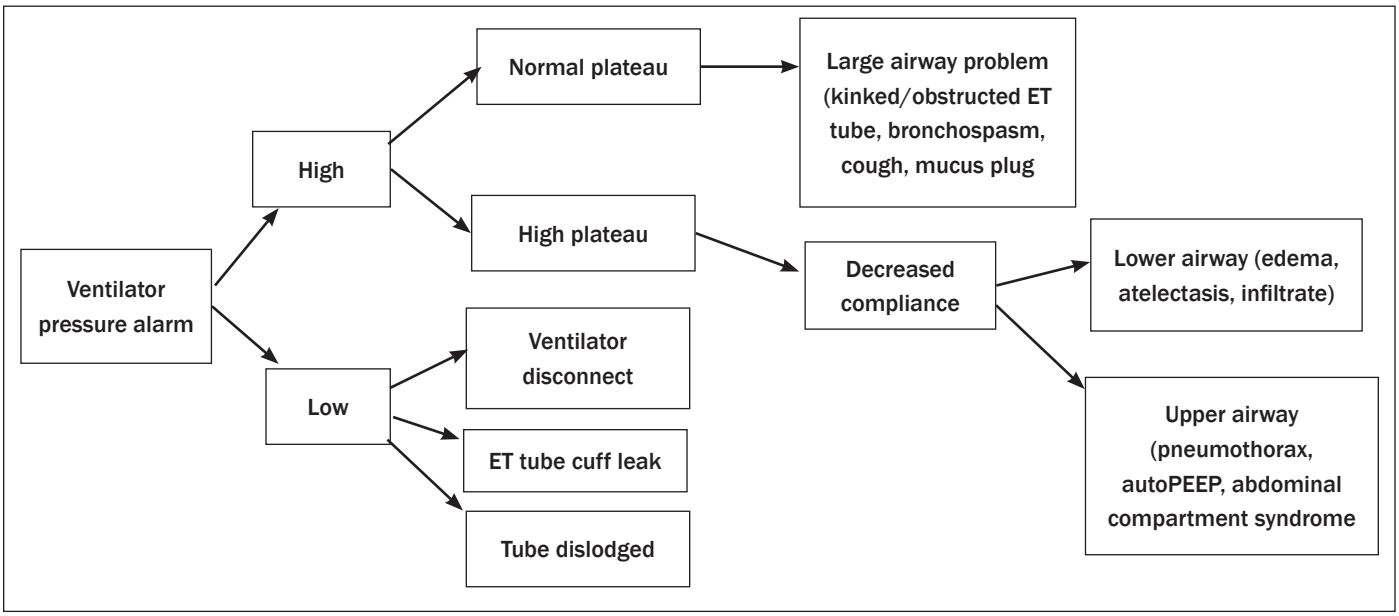
In the first two cycles, the expiratory flow does not reach 0 L/min before the next inspiratory phase starts. The retained volume outlined by the brackets represents auto-PEEP. The images below are a single alveolus acting as a model representing alveolar volume. With each inspiratory volume that has inadequate expiration, the alveolar volume is increasing. This volume stacking represents auto-PEEP. The third image shows alveolar volume after adequate expiration.

Troubleshooting the Ventilator

As with any piece of equipment, it is crucial to understand what to do when problems develop. Since the overwhelming majority of ventilated patients in the ED are critically ill, it is important to be able to quickly recognize and rectify problems with the ventilator.

Auto-PEEP. There are a number of interventions that may be used in patients with auto-PEEP. The first step is to lower the RR. If this doesn't work, lower the V_T. With less delivered volume, there is less to exhale. The next step is to shorten the inspiratory phase of each breath because it leaves more time for a longer expiratory phase. Rather than the typical I:E of 1:2, it may be necessary to set an I:E of 1:3 to 1:8. Lowering PEEP also improves exhalation. Other interventions, such as increased sedation and suctioning, may improve patient-ventilator interaction and maintain a lower RR and V_T. Since the RR and V_T are lowered in response

Figure 3: A Flow Diagram that Shows How to Systematically Assess Pressure Alarms and the Differentiation of Possible Etiologies



to auto-PEEP, hypoventilation is possible. Depending on the severity of the auto-PEEP, permissive hypercapnia, in an effort to protect from barotrauma and volutrauma, is reasonable.

Whenever a ventilated patient has a dramatic respiratory and cardiovascular decline, always consider auto-PEEP. Assess the pressures measured by the ventilator and look for clinical signs of auto-PEEP. If the patient has findings suggestive of auto-PEEP and has hypotension, immediately disconnect him from the ventilator and allow a prolonged exhalation. Keep the circuit disconnected until the full exhalation is complete. If this does not immediately resolve the cardiovascular decline, then the provider should rapidly evaluate for (and treat) a tension pneumothorax.

Air trapping may have dramatic effects on both respiratory and cardiovascular physiology. It is therefore imperative for providers to have a high index of suspicion and to frequently reassess their mechanically ventilated patients, especially those with obstructive lung disease or asthma. It is also important to keep in mind that MV does not treat the underlying disease. Parallel aggressive medical management aimed at bronchodilation is imperative.

Hypoxia. Hypoxia in the setting of MV is common and may often be difficult to treat. When an intubated patient has an abrupt change in clinical status, particularly oxygenation, it is important for the provider to first evaluate the machinery and circuit. A mnemonic (DOPE) is commonly used to guide this assessment. Look for a dislodged or displaced (D) ET tube. The patient may cough or tongue out the ET tube without warning. There are also times in which a position change may lead to the ET tube migrating either out of the trachea or down into the right mainstem bronchus, particularly in children. There may be an acute obstruction (O) in the ET tube or airway. Tube kinks, foreign bodies, or mucous plugging of airways are all potential obstructions that will cause hypoxia. With PPV, there should always be a heightened suspicion for pneumothorax (P), especially in patients with trauma, high peak airway pressures, or asymmetric lung sounds. In addition to a physical exam, it is often necessary to evaluate the patient with some form of imaging: bedside ultrasound, chest X-ray, or chest CT. There is always the chance that some component of the ventilator or other equipment (E) has malfunctioned. In this case, simplify the respiratory circuit by removing the patient from the ventilator and manually using a bag valve mask (BVM). This will allow time to address specific machine issues.

As discussed earlier, the two main ventilator settings that affect oxygenation are FiO_2 and PEEP. FiO_2 may be increased to 100%, but that alone is rarely adequate for patients that are difficult to oxygenate. The concept of PEEP and alveolar recruitment highlights the importance of intervening on the underlying V/Q mismatch. Incrementally increasing PEEP will improve oxygenation. When titrating PEEP, the beforementioned ARDS tables may be a valuable resource. There are times when patients with especially poor lung compliance require a PEEP of 20 to 24 cm H_2O. It is difficult to determine how quickly patients will respond to adjustments in PEEP. The oxygenation will typically improve within 10 minutes of increasing PEEP, but the full recruitment advantage at a given PEEP may not be seen for more than 60 minutes.[34] There will also be diminishing returns on PEEP as the level is raised. At higher levels, the compliant alveoli may stretch to the point that flow of the alveolar capillary bed is impaired. This will actually worsen

oxygenation as the perfusion of oxygen-rich alveoli decreases.

Aside from PEEP, there are other techniques that may be used to minimize the effect of V/Q mismatch. In patients with a focal consolidation or lateralization of the disease process, place the affected or bad lung up. This leaves the lung with better air exchange in a dependent or lower gravity position. Since pulmonary perfusion favors dependent areas, the lung with better ventilation will receive more perfusion. There has been recent evidence that this concept can also be applied to some patients with diffuse lung involvement. For patients with ARDS, especially those with posterior lung field involvement, outcomes improved when placing them in a prone position.[35] This technique has shown promise in the ICU setting, but it requires training, staffing, and equipment. These factors may hinder its application in the ED.

There are other situations in which a poor interaction between the ventilator and patient contributes to hypoxia. If a patient is agitated, he may be attempting to take breaths over controlled breaths or be trying to exhale against delivered breaths. This dyssynchrony with the ventilator inhibits air exchange and may contribute to hypoxia. Sedation may often alleviate this dyssynchrony and allow adequate breath delivery. The choice of sedation depends heavily on the overall clinical scenario. For most cases, treat pain with intravenous narcotic boluses and agitation with a non-benzodiazepine sedative, such as propofol. This regimen is typically effective and minimizes the delirium that has been seen with benzodiazepines.[36] In patients with ARDS and refractory hypoxia, heavy sedation may not be enough. Paralysis with neuromuscular blocking agents (for up to 48 hours) may eliminate patient-ventilator dyssynchrony, thereby improving survival and increasing ventilator-free days.[37,38]

Alarms. As mentioned before, the limits on the ventilator are in place as a safety net. They can be adjusted to accommodate specific clinical situations, but once a limit is crossed, the alarm sounds. The alarming ventilator warns providers that either targets are not being met or the patient is at risk for injury. The most common alarms will be related to high or low pressures. Figure 5 is a flow diagram that shows how to systematically assess pressure alarms and the differentiation of possible etiologies. When faced with pressure alarms without a clear etiology, remove the ventilator circuit and use a BVM. This minimizes variables by isolating the patient from the ventilator and allows for better clinical assessment of compliance.

There may also be alarms related to V_E. A patient with hypopnea or apnea may trigger an alarm for low V_E. This is a warning to providers that the patient is at risk for hypoventilation. Depending on the situation, the patient may need sedation decreased, an increase in the set RR on the ventilator, or conversion from PSV to a mode with a time trigger (for guaranteed breaths). A patient who has high V_E alarms may be agitated with tachypnea or may be compensating for a severe metabolic acidosis. Regardless of the alarm type, the provider should immediately assess the patient at the bedside.

Conclusion

In the current age of overcrowding, critically ill patients spend more time in the ED. This puts ventilator management in the hands of the EM provider. Intubation and PPV aid in stabilizing issues related to airway compromise, ventilation, oxygenation, and clinical course. The positive pressure circuit enables providers to optimize gas delivery while recruiting collapsed alveoli and controlling V_E. When choosing between the variety of modes and parameters, it is important to account for the patient's specific needs. Focus on identifying the underlying etiology and complicating features of the patient's respiratory failure. Keep in mind the physiology of ventilation and how titration of RR and V_T control V_E. From an oxygenation standpoint, consider the physiology of DO_2 and apply therapies that address the underlying problems. Use FiO_2 to maximize alveolar oxygen content while correcting V/Q mismatch with PEEP and MAP modulation. Frequently reassess the patient's tolerance of the ventilator as well as the effects of different settings and interventions. Alarms are warnings of possible patient danger and should be promptly evaluated. Remember that ventilators can stabilize but do not treat respiratory failure. The goal of ventilator management is to provide time for aggressive treatment of the underlying problem while minimizing physiologic disruption and airway damage.

References

1. Wood S, Winters ME. Care of the intubated emergency department patient. *J Emerg Med* 2011;40:419–427.
2. Chalfin DB, et al. Impact of delayed transfer of critically ill patients from the emergency department to the intensive care unit. *Crit Care Med* 2007;35: 1477–1483.
3. Walls RM, Murphy MF. *Manual of Emergency Airway Management*. Wolters Kluwer Health/Lippincott Williams & Wilkins, 2008.
4. West JB. Respiratory Physiology *Respiratory Physiology: The Essentials*. Lippincott Williams & Wilkins, 2012.
5. Chapter 50. Acid–base management. 2013.
6. Rincon F. et al. Association between hyperoxia and mortality after stroke: A multicenter cohort study. *Crit Care Med* 2013.
7. Rockswold SB, et al. A prospective, randomized clinical trial to compare the effect of hyperbaric to normobaric hyperoxia on cerebral metabolism, intracranial pressure, and oxygen toxicity in severe traumatic brain injury. *J Neurosurg* 2010;112:1080–1094.
8. Bullock MR. Hyperbaric oxygen therapy. *J Neurosurg* 2010;112:1078–1079; discussion 1079.
9. Chan B, Gaudry P, Grattan-Smith TM, et al. The use of Glasgow Coma Scale in poisoning. *J Emerg Med* 1993;11: 579–582.
10. Donald C, Duncan R, Thakore S. Predictors of the need for rapid sequence intubation in the poisoned patient with reduced Glasgow coma score. *Emerg Med J* 2009;26:510–512.
11. Davies A E, Kidd D, Stone SP, et al. Pharyngeal sensation and gag reflex in healthy subjects. *Lancet* 1995;345: 487–488.

12. Magder S. Bench-to-bedside review: Ventilatory abnormalities in sepsis. *Crit Care* 2009;13:202.
13. Hess DR. Ventilator modes: Where have we come from and where are we going? *Chest* 2010;137:1256–1258.
14. Bersten AD, Soni N. *Oh's Intensive Care Manual: Expert Consult: Online*. Elsevier Health Sciences, 2013.
15. Agasti TK. *Textbook of Anaesthesia for Postgraduates*. JP Medical Ltd, 2010.
16. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. *N Engl J Med* 2000;342:1301–1308.
17. Futier E, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. *N Engl J Med* 2013;369: 428–437.
18. Weingart S. EMCrit Podcast 3-Intubating the patient with Severe Metabolic Acidosis.
19. Kallet RH, Branson RD. Respiratory controversies in the critical care setting. Do the NIH ARDS Clinical Trials Network PEEP/FIO2 tables provide the best evidence-based guide to balancing PEEP and FIO2 settings in adults? *Respir Care* 2007;52:461–475; discussion 475–477.
20. Brower RG, et al. Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. *N Engl J Med* 2004;351: 327–336.
21. Luecke T, Pelosi P. Clinical review: Positive end-expiratory pressure and cardiac output. *Crit Care Lond Engl* 2005;9:607–621.
22. Vieillard-Baron A, Prin S, Chergui K, et al. Echo-Doppler demonstration of acute cor pulmonale at the bedside in the medical intensive care unit. *Am J Respir Crit Care Med* 2002;166:1310–1319.
23. Fanelli V, et al. Acute respiratory distress syndrome: New definition, current and future therapeutic options. *J Thorac Dis* 2013;5:326–334.
24. Mikkelsen ME, et al. The epidemiology of acute respiratory distress syndrome in patients presenting to the emergency department with severe sepsis. *Shock* 2013
25. Petrucci N, Iacovelli W. Ventilation with lower tidal volumes versus traditional tidal volumes in adults for acute lung injury and acute respiratory distress syndrome. *Cochrane Database Syst Rev* 2004;CD003844.
26. Stehman CR, et al. Bedside estimation of patient height for calculating ideal body weight in the emergency department. *J Emerg Med* 2011;41;97–101.
27. Hickling KG. Permissive hypercapnia. *Respir Care Clin N Am* 2002;8:155–169.
28. Bidani A, Tzouanakis AE, Cardenas VJ, Jr, et al. Permissive hypercapnia in acute respiratory failure. *JAMA* 1994;272: 957–962.
29. Kregenow DA, Rubenfeld GD, Hudson LD, et al. Hypercapnic acidosis and mortality in acute lung injury. *Crit Care Med* 2006;34:1–7.
30. Ni Chonghaile M, Higgins B, Laffey JG. Permissive hypercapnia: Role in protective lung ventilatory strategies. *Curr Opin Crit Care* 2005;11:56–62.
31. Curley G, Hayes M, Laffey JG. Can 'permissive' hypercapnia modulate the severity of sepsis-induced ALI/ARDS? *Crit Care* 2011;15:212.
32. Reddy VG. Auto-PEEP: How to detect and how to prevent — a review. *Middle East J Anesthesiol* 2005;18:293–312.
33. Laghi F, Goyal A. Auto-PEEP in respiratory failure. *Minerva Anestesiol* 2012;78:201–221.
34. Chiumello D, et al. Time to reach a new steady state after changes of positive end expiratory pressure. *Intensive Care Med* 2013;39:1377–1385.
35. Guérin C, et al. Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med* 2013;368:2159–2168.
36. Barr J, Pandharipande PP. The pain, agitation, and delirium care bundle: Synergistic benefits of implementing the 2013 Pain, Agitation, and Delirium Guidelines in an integrated and interdisciplinary fashion. *Crit Care Med* 2013;41:S99–115.
37. Needham CJ, Brindley PG. Best evidence in critical care medicine: The role of neuromuscular blocking drugs in early severe acute respiratory distress syndrome. *Can J Anaesth J Can Anesth* 2012;59:105–108.
38. Papazian L, et al. Neuromuscular blockers in early acute respiratory distress syndrome. *N Engl J Med* 2010;363: 1107–1116.

CME Questions

1. A patient with ARDS who is being ventilated with 6 cc/kg tidal volume at a rate of 24 has a blood gas that reveals pH 7.24 and pCO_2 60. The next best step in management is:
   A. increase tidal volume to 8 cc/kg
   B. increase sedation
   C. increase respiratory rate to a max of 35
   D. increase PEEP

2. A patient being ventilated with APRV has had increasing pCO_2 levels on the blood gas. Which modification to the ventilator settings will improve ventilation?
   A. increase time high
   B. decrease pressure high
   C. decrease time low
   D. increase time low

3. A patient with DKA has a decline in mental status and requires intubation for airway protection. An uncomplicated rapid sequence intubation using propofol and rocuronium is performed. His pre-intubation blood gas revealed pH 6.9 and pCO_2 12. What initial ventilator set up would be most appropriate?
   A. AC volume control with V_T 6 cc/kg and RR 30
   B. SIMV volume control with V_T 8 cc/kg and RR 16
   C. APRV with P high 28, P low 0, T high 5.4s, and T low 0.6s
   D. PSV with P inspiratory 10 and PEEP 5

4. When treating an intubated patient with a COPD exacerbation, what finding would be most concerning for auto-PEEP?
   A. worsening hypoxia
   B. elevated peak pressures but normal plateau pressures
   C. incomplete exhalation when auscultating
   D. increasing pCO_2 on serial blood gases

5. A patient with a history of asthma is intubated for respiratory failure. Within a couple of hours, his plateau pressures increase and his blood pressure dramatically decreases. What should be done first?
   A. order a portable chest X-ray
   B. disconnect patient's ET tube from the ventilator circuit
   C. decrease PEEP
   D. start vasopressors for blood pressure support

6. A patient presents with hypoxia and increased work of breathing. His chest X-ray shows a large left upper lobe consolidation and he is intubated for respiratory failure. He is on assist control

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

volume control. While in the ED awaiting admission, the patient's oxygenation worsens. Which of the following will most improve oxygenation?

A. increase FiO_2 and decrease PEEP
B. increase FiO_2 and increase PEEP
C. increase FiO_2 and increase RR
D. change to SIMV volume control + pressure support mode

7. The patient's oxygenation improves transiently with the above intervention, but he is now hypoxic again. What additional intervention will most benefit his oxygenation?

A. reposition patient with left side down
B. perform needle decompression
C. reposition patient with left side up
D. increase head of bed height from 30 to 45 degrees

8. Which mode of ventilation is most appropriate in a patient who has severe mental status decline (GCS 5) related to acute spontaneous subarachnoid hemorrhage?

A. SIMV volume control
B. AC volume control
C. pressure support ventilation
D. APRV

9. Which patient is most likely to have hypotension in the immediate post-intubation period?

A. middle age male with status epilepticus
B. elderly female with acute COPD exacerbation
C. young male with acute narcotic overdose
D. female with severe sepsis from urinary source

10. A patient with ARDS sedated to a Glascow Coma Score of 5 is being ventilated with AC volume control with VT of 6 mL/kg. His ventilator is alarming for high pressures. An inspiratory hold maneuver is done and shows plateau pressures of 35 to 40 cm H_2O. What is the most appropriate next step?

A. change to PSV
B. decrease V_T to 5 mL/kg and then recheck plateau pressures
C. keep current V_T target but decrease inspiratory flow rate
D. increase sedation

In Future Issues

Epiglottitis

CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

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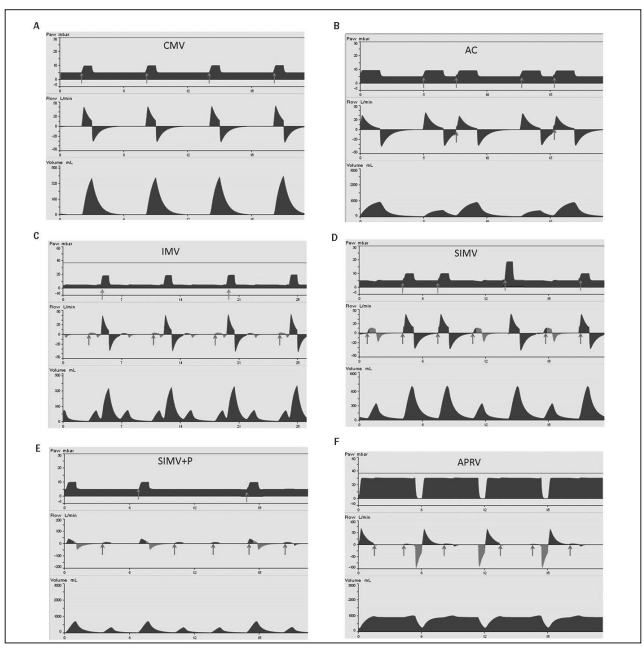
Ventilator Management in the Emergency Department

Waveforms of Different Ventilator Modes



Waveforms of different ventilator modes A) CMV: Note the arrows in the Paw represent controlled breaths. B) AC: In this diagram, the first and third arrows in the Paw are controlled breaths, the second and fourth are the assisted breaths. In the Flow section, the arrows are the patient's attempts to breathe. C) IMV: In the Paw section, the arrows represent controlled breaths and the arrows in the Flow section are the patient attempts to breathe. D) SIMV: In this diagram, the first, second, and fourth arrows in the Paw are assisted breaths, the third is a controlled breath. The arrows in the Flow section are the patient attempts to breathe. E) SIMV+P: In this diagram, the first arrow in the Paw is an assisted breath, the second is a controlled breath. The arrows in the Flow section are the patient attempts to breathe. F) APRV: The arrows in the Flow section are the patient attempts to breathe. (Images adapted from Dräger Evita V500 Product Demonstrator manual simulation available at www.Draeger.com.)

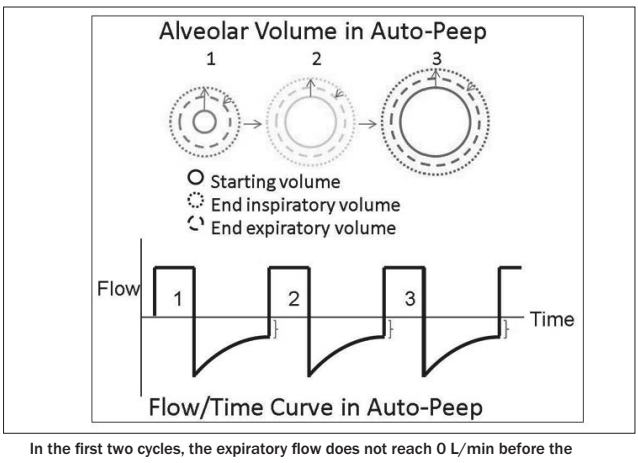
Suggestions for Initial Ventilator Settings Based on Different Clinical Pictures

	Airway protection	Hypoxia	COPD or Asthma	Increased metabolic demands
Key concerns or considerations	Apnea	Lung protection	Auto-PEEP	Matching respiratory alkalosis
Mode	PSV, AC, SIMV+P	AC, SIMV+P	AC, SIMV+P	AC, SIMV+P
V_T (mL/kg of IBW)	6	6	6	6
RR	10-14	12-20	6-8	18-24
PEEP (cm H_2O)	5	8	0-5	5
FiO_2	0.4	0.8-1.0	0.4-1.0	0.4
I:E	1:2	1:2	1:3-1:5	1:2
Inspiratory flow (L/min)	60	60	100	60
Each setting should take into account the underlying pathology and key physiologic consideration.				

Tables from ARDSnet Protocol Summary Showing Different Combinations of PEEP and FiO2

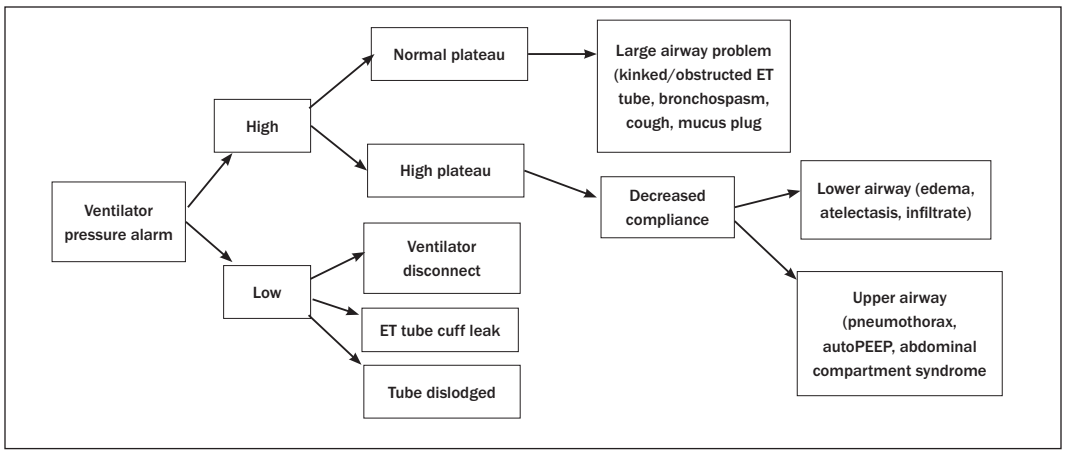
Higher PEEP and Lower FiO_2														
FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18-24
Lower PEEP and Higher FiO_2														
FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	5	8	10	12	14	14	16	16	18	20	22	22	22	24
The goal oxygenation is PaO_2 55-80 mmHg or SpO_2 88-95%.														

Flow vs. Time Curve of Auto-PEEP



In the first two cycles, the expiratory flow does not reach 0 L/min before the next inspiratory phase starts. The retained volume outlined by the brackets represents auto-PEEP. The images below are a single alveolus acting as a model representing alveolar volume. With each inspiratory volume that has inadequate expiration, the alveolar volume is increasing. This volume stacking represents auto-PEEP. The third image shows alveolar volume after adequate expiration.

A Flow Diagram that Shows How to Systematically Assess Pressure Alarms and the Differentiation of Possible Etiologies



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