# Core Principles
# Of
# Respiratory Management

The Basics of Mechanical Ventilation

Michael C. Berrier, NREMT-P, FP-C, C-NPT, AAS

# ABOUT THE TEAM

Michael C. Berrier is has been active in public safety since 1987, and has been working and teaching as a paramedic for the past 19 years.  He has worked in rural/suburban 911, fire service, the offshore oilfield, EMS administration and most recently as a critical care transport paramedic in North Carolina.
Michael holds certifications from North Carolina, The National Registry of EMT's, University of Maryland-Baltimore County, Cleveland Clinic, Board for Critical Care Transport Certification and the Society of Critical Care Medicine.

Melissa Overton is a registered nurse and founder of MedicalTraining.me. Prior to her nursing career in critical care (ED, ICU), clinical education, and the cardiology office, she worked as an EMT, Nursing Assistant and Unit Secretary in the Emergency Department at Vidant Medical Center.

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# CONTENTS

Welcome to Core Principles of Respiratory Management: The Basics of Mechanical Ventilation.  If you work on a unit with , transport or just want to know more about ventilated patients, this course can help you understand this part of their care. And, while this program will not make you a respiratory therapist, it will introduce you to how people ventilate themselves, and how we as clinicians can use devices both simple and complex to take over when the natural process fails. Between the live course and this book, Core Principles of Respiratory Management will provide you with information about pulmonary physiology, ventilator terminology and the most common types of mechanical ventilation encountered in most emergency departments, ICUs and interfacility transports.

## COURSE OBJECTIVES

**Upon completion of the course, the participant will be able to:**

- **Describe basic pulmonary anatomy and physiology.**
- **List common indications for mechanical ventilation**
- **Describe ventilator types, modes and terminology**
- **Describe commonly used ventilator settings**

# Pulmonary A&P

CNS

When discussing the anatomy of breathing, we often forget to start at the top. In fact, the brain is the most important pulmonary organ, and the one with which we most frequently associate the dysfunction that requires some level of mechanical ventilation. Since the brain's number one job is to feed and oxygenate itself, it expends much of its work in the brainstem making sure the oxygen keeps coming. Specifically, there are three stimulation centers:

- Apneustic Center
- Ventral Respiratory Group (Expiratory Center)
- Dorsal Respiratory Group (Inspiratory Center)

and one inhibitory center

- Pneumotaxic Center

in the medulla and pons tasked with regulating the mechanics of breathing based on chemoreceptors and stretch receptors located throughout the body.

This nervous system control is communicated to the muscles and organs of respiration via several pathways, the most notable of which being the phrenic nerves. They innervate the right and left hemidiaphragm separately to carry voluntary or, most frequently, involuntary signals to the diaphragm to make it contract and cause inspiration.

What drives the respiratory centers and ultimately the mechanics of breathing is input from chemoreceptors and mechanoreceptors around the body which continually measure carbon dioxide and other chemical levels in the blood and adjust respiratory rate and depth accordingly.

Upper Airway

The primary functions of upper airway organs, especially in the nose and mouth, are to warm, humidify and filter the air. These structures, in conjunction with the lips, teeth, tongue, epiglottis and others combine to provide a small amount of physiologic positive end expiratory pressure, or PEEP which we will discuss later in the program. Lining the trachea are special cells known as goblet cells which produce mucous. Some respiratory diseases cause hypertrophy and excessive activity in the goblet cells which lead to pathologic amounts of airway mucous.

Larynx & Trachea

Starting at the vocal cords, the larynx conducts fresh air downward and into the trachea, which is about 11cm (4.3") long and between 21-27 mm in diameter. It is also interesting to note that the glottic opening (rima glottides) is only about 6-9mm wide, while the outside diameter of an 8.0 mm ET tube is 11.8mm.

# Pulmonary A&P

Lower Airway

At the distal end, the trachea bifurcates into the right and left mainstem bronchi. It is noteworthy that the right mainstem bronchus doesn't change direction very much from the trachea, while the left mainstem turns at a much sharper angle. This is important when troubleshooting endotracheal tube placement as a tube that has migrated distally seldom enters the left bronchus. After the mainstems, the bronchial tubes frequently split into branches that become progressively smaller until finally ending at the terminal bronchi that are connected directly to the alveoli. These tiny sacs, wrapped in capillaries, are the functional areas of gas exchange in the lung. The average human adult has about 300,000,000 individual alveoli which, if spread out in a single layer, would cover about half of a regulation tennis court. Between the alveoli and the capillaries is the alveolar-capillary membrane. Widening or destruction of this membrane accounts for many of the respiratory diseases that require therapy in the acute setting. The lungs are lined on the outside with a thin, two-part membrane called the pleura which helps to lubricate and protect the delicate lung tissue.

Vascular Anatomy

The visceral tissue of the lung itself is supplied with blood by the bronchial arteries. These should not be confused with the pulmonary arteries which carry blood from the right side of the heart to the lungs to be oxygenated. The blood returns to the left side of the heart for systemic distribution via the pulmonary veins. High pressures in the pulmonary vasculature often lead to problems with oxygenation and ventilation and ultimately circulation as a whole. When there is a portion of lung that is not reached by pulmonary circulation due to an obstruction (pulmonary embolism) or vascular interruption, this is known as a dead space. Conversely, if there is an area of lung with adequate pulmonary blood supply but no ventilation, this is called a shunt. We will discuss physiologic dead space and other meanings for that term later in the program.

Muscular & Bony Anatomy

One of the more unique things about the lungs is that, for how absolutely vital their function is to human life, they don't actually DO anything. Everything a lung does, from filling with air and emptying that air to gas exchange is done entirely passively as a result of some other mechanism. To fill with air, the lungs are dependent on the muscles of inspiration and an intact bony housing, made up of the ribcage , sternum and spinal column. The muscles, chiefly the diaphragm, contract in such a way that intrapleural volume is increased, which decreases pressure in the lungs and brings air in from outside. When the diaphragm relaxes, the pressures equalize and air is forced out. Intercostal muscles, located between the ribs, have a limited role in normal respiration, but become more important during forced inhalation and exhalation. All of these things combine to maintain an "intact chest bellows".

# Pulmonary A&P

Physiology

Since we've already discussed the gross mechanics of breathing associated with the anatomic structures, the remaining physiology will be dealing with movement of air and gasses, and this is done mostly via pressure gradients. As stated before, when the lungs increase in size, the pressure inside the pleural space and alveoli drops, pulling air in from the outside, but how much? The average adult lung has a total capacity of about 6000 ml, or 6 liters. We don't move that much air in a single breath however. That is a much lower number, closer to 500 ml. The rest of the lung volume is taken up in residual volumes, some of which is available for use under extreme conditions (think Fight or Flight). We take some of these volumes into consideration when setting up a patient on the ventilator.

At the cellular level, movement of gas is still driven by pressure, but on a much smaller scale. This brings us to three very important terms associated with respiratory physiology that will be used extensively throughout the rest of the course:

- Ventilation:___________________________________
- Respiration:___________________________________
    - ______________________________
    - ______________________________
    - ______________________________
- Oxygenation:__________________________________

It is important to understand that, even though these words are often used interchangeably, they do not mean the same thing. We will discuss their clinical significance in few minutes.

Other terms you've probably heard before that we will use, are:

- Resistance:___________________________________
- Compliance:__________________________________
- Diffusion:_____________________________________

So, if ventilation, respiration and oxygenation aren't the same thing, then they must at least be dependent on each other, right? Related? Close friends? Follow the example in class about clinical testing for brain death, then think more about that question.

## Pulmonary A&P

Ventilation: Moving $CO_2$

Whether we are talking about normal, physiologic ventilation, or mechanical ventilation, we manipulate it the same way, and that is with ___________________________, which is calculated by multiplying ___________ X _________________. In order to change the amount of $CO_2$ in the blood ($PaCO_2$) or exhaled air ($PEtCO_2$), we have to change one of those two components. Which one depends on a lot of factors, such as peak inspiratory pressure (PIP) and physiological appropriate levels. $CO_2$ moves easily this way because it diffuses through water about 20 times faster than oxygen. Whatever changes you make to these factors will not significantly alter oxygenation.

Oxygenation

There are two main ways we can add or subtract oxygen from the blood stream and, by extension, the tissues, and those are __________________________ and __________________. One of these is wildly superior to the other, and we will talk much more about this later in the program.

## Ventilator Decoder Ring

One of the most intimidating parts of medicine in general is the language. There are so many new words and abbreviations and acronyms for every discipline, and respiratory care is no different. In order to work with ventilators and truly understand respiratory care, you have to speak the language. Most of the terms you need will be associated with ventilator indications, settings, or measurements.

Indications

When considering the indications for mechanical ventilation, a simple way to think about it is to ask "What do we want the vent to do?" If the patient has a neurological problem like a CVA or head injury but his lungs are healthy, then we can say that the vent is doing the thinking for him. On the other hand, if the patient has a primary respiratory problem like CHF or pneumonia, then the ventilator may need to add extra pressure or $FiO_2$ to oxygenate him. Finally, critically ill patients with sepsis or major illness may benefit from a reduction in oxygen and energy demand, so the ventilator can take care of some heavy lifting. For many patients the ventilator will need do only one of these jobs. But in truth, most patients will require more than a single function, even if it is a simple as supplemental oxygen and a mandatory rate.

# VENTILATION TERMINOLOGY

$FiO_2$: Oxygen content of inspired air. 21%-100%

Alveolar Volume: Air available for gas exchange, ≈350 ml

Deadspace Volume: Air in upper airway not available, ≈150ml

Tidal Volume: Air moved in single breath, ≈500ml, 6-8 ml/kg

Minute Ventilation: Air moved in a minute.

Frequency: Respiratory rate, mandatory + spontaneous

# VENTILATION TERMINOLOGY

Inspiratory Time: Time required to deliver inspiration.

Respiratory Cycle Time: Time to deliver a whole breath.

Flow Rate: Rate of gas entering lungs.

I:E Ratio: Relationship between Ti and Te.

Mean Airway Pressure: Airway pressure average

Peak Airway Pressure: Highest pressure during single breath

PEEP/CPAP: Pressure above atmospheric applied to airway during exhalation.

**Ventilator Decoder Ring**

# VENTILATION TERMINOLOGY

Trigger: Variable that makes the breath START

Control/Type: Variable that makes the breath STOP

Mode: Determines how ventilator initiates mandatory breaths, responds to spontaneous effort and supports spontaneous effort.

## Ventilator Type

A simple way to think about the ventilation type is this is the limit that terminates the mandatory breath, even though this doesn't tell the whole story. For the purpose of this course, we will look at three of the most common: volume control (VC), pressure control (PC), and pressure regulated volume control (PRVC).

Volume Control (VC)

For this type of ventilation used in the majority of adult patients, the clinician selects a tidal volume (Vt) they wish to deliver to the patient. The Vt is chosen based on physiological appropriateness, usually between 6-8 ml/kg of ideal body weight. For the average adult male, this is somewhere around 500 ml. In addition to Vt, the clinician will also select a rate (F), FiO2, inspiratory time (Ti) and PEEP on most ventilators. In turn, the ventilator will measure peak inspiratory pressure (PIP) and exhaled tidal volume (Vte). VC is a very simple ventilation type as it has minimal settings and allows easy prediction of ventilation, depending of course on patient input. Since airway and lung pressures are variable, oxygenation is not guaranteed and barotrauma is possible, so close monitoring is still necessary.

Pressure Control (PC)

When setting up PC ventilation, the clinician will select a PIP limit and a Ti, and the ventilator will terminate the machine breath at the end of the set Ti. This type is most commonly seen in neonatal and pediatrics, but is also used in some more severely ill adults with oxygenation problems. Other settings in PC are, like VC, rate, FiO2 and PEEP. Since the Vt is variable, one of the most important measurements in this type is exhaled tidal volume, or Vte. This gives the most accurate approximation of how much air was delivered at the set PIP. While PC is thought to be a little more lung protective and responsive to changing lung mechanics, it requires a little more monitoring and finesse than does VC to ensure that ventilation and oxygenation are balanced.

A Word about Waveforms

Since most transport ventilators do not have graphics packages that allow the user to monitor waveforms, this is often an abstract concept to many people. But if the graphics are available, the three most common are the pressure, volume and flow waveforms. Looking at these can give some more clarity into how a breath is triggered and how the ventilator delivers that breath.

## Ventilator Type

Pressure Regulated Volume Control (PRVC)
PRVC attempts a "best of both worlds" approach to selecting a Vt or a PIP as the control for machine breaths. For this type, the clinician will select a desired Vt based on physiologic criteria, then the machine will do one or more volume oriented test breaths to find how much PIP the patient requires to reach the desired Vt. Then, the machine will switch to pressure oriented breaths, targeting the internally selected PIP to deliver the user defined Vt. The clinician will also set the same parameters as VC, including F, FiO2, Ti and PEEP. There is some debate about the utility or need for PRVC, but the blending of target thresholds is at least anecdotally beneficial. It may still deliver high pressures, especially if the lung mechanics change, and alterations of ventilation are possible. It is also worth noting that if the patient coughs, has a circuit disconnect or some other significant pressure variation, the ventilator will once again have to go through the test breath cycle, essentially starting ventilation over again.

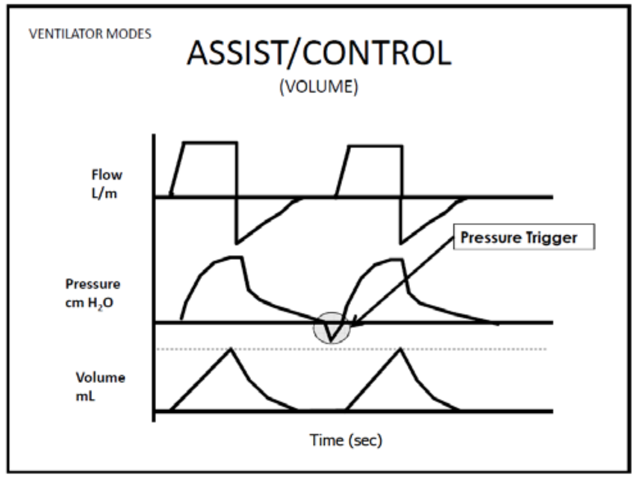
## Ventilator Mode

When we set the mode, we are telling the ventilator how we want it to initiate mandatory, respond to spontaneous effort, and support spontaneous effort. Put another way, the mode dictates what will happen when the vent is triggered.  We will review four of the most common: Assist/Control (A/C), Synchronized Intermittent Mandatory Ventilation (SIMV), Pressure Support (PS) and Non-invasive Positive Pressure Ventilation (NiPPV).

A Mode You Won't Find
First, let's briefly discuss a mode you won't find on just about any modern ventilator, and that's Continuous Mandatory Ventilation, or CMV. This mode is time triggered, and only time triggered. So, if we set the rate (F) at 12 breaths per minute, the machine will deliver 12 breaths per minute regardless of what the patient does. It will not allow additional spontaneous breathes, it will not support spontaneous effort, and it will not synchronize. As you might imagine, this mode is horribly tolerated by anyone but manikins and paralyzed patients. Modern ventilators will allow and support patient effort as well as taking on all the work when necessary.

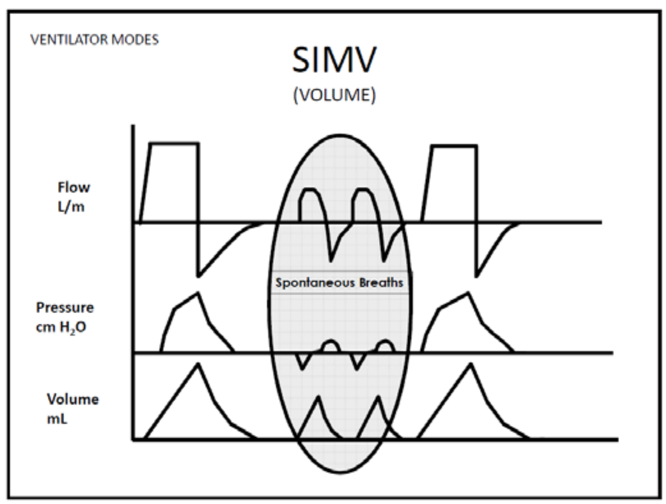
Assist/Control (A/C)
When selecting a mode, the decision is often based on the most appropriate response to a patient's spontaneous respiratory effort, and this is how most modes differ. Assist/Control

will deliver machine (mandatory) breaths at a selected rate that terminate based on the selected control and with the selected $FiO_2$. For example, if you set the machine to deliver a tidal volume of 500 ml, 12 times a minute in A/C, then it will do that regardless of patient effort. In this way, the clinician can guarantee a minimum minute ventilation. Additionally, if the patient should spontaneously trigger the ventilator, it will deliver an additional breath at the selected tidal volume. To follow the example above, the set Vt is 500 ml, so any breaths triggered by the patient will also be delivered at 500 ml. This mode is useful for patients who are paralyzed and sedated since the machine is already set up to do all the work. And, if that patient should begin to exhibit some spontaneous effort, the machine will answer that effort with full tidal volume breaths, further guaranteeing minimum ventilation. The downside is, as patients become more aware and participate more in their own ventilation, they may become asynchronous with the ventilator which can lead to discomfort and possible hyperventilation.




Synchronized Intermittent Mandatory Ventilation (SIMV)

This mode is similar to A/C in that it will deliver a set rate at the set control (PIP or Vt) if there is no patient effort. Again, if you select a Vt of 500 ml to be delivered 12 times a minute, if the patient makes no spontaneous effort, then the ventilator will exactly what you asked. It is in the response to that patient triggering that SMIV differs greatly from A/C. Should the patient make enough spontaneous effort to activate the trigger, SIMV will only open the valve and allow the patient to inspire as much volume as he/she wants. So, instead of a guaranteed Vt for each patient triggered breath, Vt will be only what the patient pulls through the open valve. Also, the ventilator will synchronize the spontaneous effort with the time-triggered mandatory breaths so as not to over ventilate or "stack" breaths on top of one another. As you might imagine, if the patient is making a lot of respiratory effort, and it is of adequate depth, then this mode would be very comfortable. However, if that effort is not adequate to ventilate, but activates the trigger, then the vent may count that as a breath and not deliver a full mandatory breath, resulting in



hypoventilation. This mode is also very useful as part of ventilator weaning, as it can be combined with pressure support (see below) to augment patient effort.

Pressure Support (PS)

There are many patients who require ventilatory assistance but are still making some spontaneous effort that should be supported, and that's where pressure support (PS) is very helpful. PS applies additional driving pressure from the ventilator to spontaneous breaths to decrease the patient's work of breathing and augment the effectiveness of ventilation and oxygenation. PS differs from pressure control (PC) in that the driving pressure from PS is not sufficient to inflate the lungs and initiate a full breath, while PC delivers a set PIP on a mandatory, time-triggered scheme. The greatest value of PS in acute care is that it can be combined with SIMV to deliver a mandatory rate while offering support to the patient's effort to simultaneously encourage patient effort and decrease the work required to generate it. The greatest drawback of PS relates to the patient who is not yet breathing often or deeply enough and may rapidly decompensate without mandatory ventilation.

## Frequency

As you now know, ventilator frequency is just a fancy way to say respiratory rate. Depending on the machine you have, the setting for frequency may be labeled as Frequency (F), Breath Rate (BR), or Breaths Per Minute (BPM), but it all means the same thing...how often you want the ventilator to cycle. This setting is based on several factors, the most universal of which is the age of the patient:

| RESTING RESPIRATORY RATES(bpm) | |
|---|---|
| Birth - 6 weeks | 30-60 |
| 6 months | 25-40 |
| 3 years | 20-30 |
| 6 years | 18-25 |
| 10 years | 15-20 |
| Adults | 12-20 |

Other factors that influence the frequency setting are the current illness (metabolic acidosis, for example, would require a higher respiratory rate) and the clinical goal for ventilation. If the patient has a carbon dioxide derangement, then the rate may need to be higher, while oxygenation failure might benefit from a lower respiratory rate. Most machines will combine the setting and a measurement of spontaneous respirations to provide an accurate count of breaths per minute.

## Fraction of Inspired Oxygen ($FiO_2$)

We know by now that the primary function of a ventilator is to *ventilate*, that is to say, move air. But, since we also know that many of our patients will require at least some oxygenation management as well, modern ventilators will entrain supplemental oxygen at a percentage, or $FiO_2$, selected by the clinician between 21% (room air) and 100%. Unfortunately, oxygen is often applied routinely as part of a protocol for severe illness without an accompanying assessment to determine if it is actually indicated, and the truth is that the population who needs supplemental oxygen is a good bit smaller than we've been led to believe. The truth is that, unless your patient hails from a planet where the atmosphere is made up of more than 21% oxygen, applying more than that does not fix a problem, it only treats a symptom. Based on the evidence, in the absence of hypoxia, supplemental oxygen seems to do no good and may in fact do harm to patients with a variety of acute illnesses and injuries.

So, why is supraatmospheric oxygen bad? It has been known for quite some time that newborns, especially those born prematurely, were are risk for damage to their eyes and other organs from increased oxygen exposure. Further research into children born with certain congenital heart defects (CHD) found that they were dependent on a patent ductus arteriosus (PDA) to circulate oxygenated blood to the rest of their body. Since increased oxygen levels in the blood ($PO_2$) lead to closure of the PDA, these patients are often given limited oxygen at atmospheric or sometimes even subatmospheric concentrations to maintain PDA patency until they can get a surgical repair. For adults, the dangers of supplemental oxygen are a little more nuanced with effects that are usually observed in the long term rather than acutely as noted in neonates. As oxygen surpasses adequate levels in the blood, arterial receptors send signals to reduce the vessel size, demonstrating oxygen as a powerful and fast vasoconstrictor. This mechanism is useful in the minutes after birth as a neonate converts from fetal to newborn circulation, but for adults this can cause problems. Incompletely metabolized or excess oxygen is also prone to increase production of oxygen free radicals, or reactive oxygen species (ROS). These molecules are necessary for certain function inside of cells, but an abundance of ROS can lead to cellular damage which is thought to exacerbate ischemia during heart attack and stroke.

Most current medical texts support initiating mechanical ventilation with 100% $FiO_2$ until you figure out what is wrong and what the patient's true oxygen needs are, then rapidly titrating downward to achieve the clinical goals. It is entirely possible that many patients will be adequately oxygenated at or near the atmospheric $FiO_2$ of 21%

## Positive End Expiratory Pressure (PEEP)

In order to maintain proper oxygen delivery for ventilated patients, even those with no suspected oxygenation failure, it is necessary to maintain a physiologic amount of mean airway pressure through application of PEEP. We actually do this as healthy, functioning humans with our upper airway structures (lips, teeth, tongue, nasal concha). Typically, this physiologic PEEP is about 5 $cmH_2O$. Once we intubate someone, however, those structures are bypassed by the smooth bore tube that provides no resistance and thus no physiologic PEEP. To maintain alveolar recruitment and MAP, we should at least provide a physiologic amount of PEEP to the ventilated patient either with a PEEP valve or through the ventilator. For those patients who do have oxygenation failure, PEEP can be lifesaving and is very often the most effective way to improve oxygen delivery. The most common type of hypoxia, hypemic hypoxia, results when oxygen cannot enter the bloodstream, usually because of an obstructive process like COPD or pulmonary edema. PEEP can help "push" oxygen through the alveolar wall and into the capillaries, increasing the partial pressure of oxygen in the plasma and, by extension, increase delivery to the tissues. Increased PEEP can also help maintain alveolar recruitment. PEEP demands must be balanced with hemodynamic status, as excessive PEEP will increase intrathoracic pressure and compress the great vessels, both decreasing preload and increasing afterload to inhibit cardiac output.

PEEP Compensation

This general term can refer to a couple of different ideas. First of all, when you set up the ventilator on PC, the control variable is the peak inspiratory pressure, or PIP. This is the highest pressure you want to achieve and maintain during the respiratory cycle, commonly set around 25 $cmH_2O$. This number usually means 25 $cmH_2O$ above zero, or a baseline pressure of 0 $cmH_2O$, or said another way, 25/0. However, this setup would assume that we had not set a PEEP, which is pretty rare. Rather, if we set a PEEP of 5 cmH2O (physiologic PEEP), when the respiratory cycle ends, the baseline pressure falls only to 5 $cmH_2O$ instead of all the way to zero, expressed as 25/5. This is important when setting up your ventilator based on a physician order because you have to know if the PIP setting on your ventilator *includes* the PEEP or is added to the PEEP. For many machines, the PIP that you select will be added to the PEEP, making the total peak pressure PIP + PEEP.
Secondly, PEEP compensation can refer to how the pressure trigger responds to PEEP. If the ventilator is set to respond to spontaneous effort based on the patient pulling a set negative pressure through the vent circuit, this is a pressure trigger. A typical setting for this is about –2 $cmH_2O$, but the trouble can lie in what that means. In other words, -2 $cmH_2O$ below *what*? If the ventilator is not PEEP-compensated, then the patient will have to pull that –2 $cmH_2O$ below zero, which means they will also have to pull through the PEEP, making a total of –7$cmH_2O$ worth of effort to trigger the vent. PEEP compensated ventilators will adjust the pressure trigger to relate to the set PEEP, meaning the patient will only have to expend the effort required to pull –2, regardless of the PEEP setting.

## Sensitivity

Most ventilator modes will either allow or depend on patient effort, so we must select the type of trigger and how sensitive that trigger will be. The most common triggers are pressure and flow. A pressure trigger works when the patient tries to inhale through the vent circuit and drops the pressure to the set limit, at which time the internal valve opens and the ventilator delivers air based on the mode and type. A flow trigger is similar, except that the patient effort is measured in liters per minute of flow through the circuit.

**Pressure Trigger: - cm$H_2O$**
**Flow Trigger: liters/minute**

The trigger sensitivity determines how much patient effort is required for the ventilator to initiate a machine assisted breath. The higher the sensitivity, the less work the patient must do to get the vent's help. The less sensitive the trigger, the more the patient has to do to get an assisted breath. Whether the vent we use has a pressure or flow trigger, we have to find the "sweet spot" for each patient and situation. If the trigger is too sensitive, then the ventilator is prone to auto-cycling. In other words, the machine may initiate a breath from the circuit moving or bumps in the road and cause hyperventilation. Conversely, if the trigger sensitivity is too low, then the patient's own respiratory effort may go unsupported because it was not adequate to activate the ventilator.

## Non-Invasive Positive Pressure Ventilation (NiPPV)

NiPPV is a broad term for positive airway pressure applied by some kind of external mask or other apparatus. Mostly, it refers to continuous positive airway pressure (CPAP) and Bi-level Positive Airway Pressure (Bi-PAP). NiPPV is delivered through dedicated machines or by transport ventilators that include a non-invasive mode. Either way, the goal is to apply additional pressure to the airway to increase mean airway pressure and stent open the airways and alveoli. The difference between the two is how they deliver that pressure.

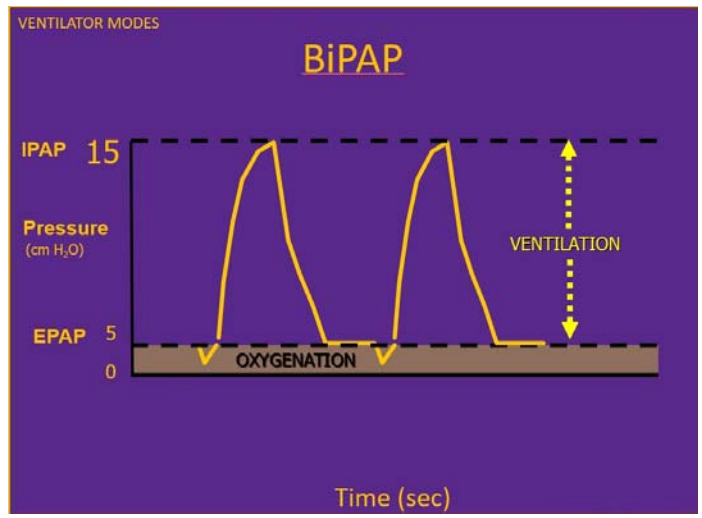
### CPAP

CPAP can be thought of as PEEP for breathing people, except that the pressure is applied evenly throughout the entire respiratory cycle. For this reason, the settings for CPAP are very simple: pressure and $FiO_2$. Some of the CPAP-only machines are oxygen driven, meaning that they always deliver 100% $FiO_2$. Aside from setting up the machine correctly, the other very important component of effective CPAP is a good, tight mask seal. Since it is non-invasive, generating the proper pressure is very dependent on making sure the mask is securely fitted.

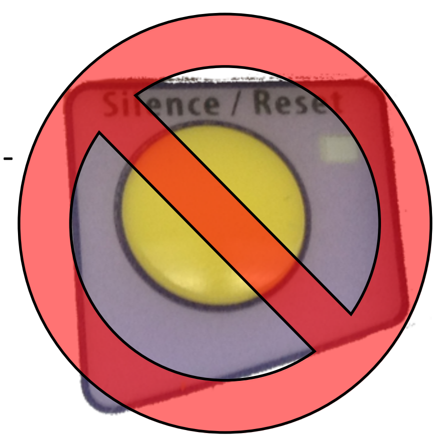
CPAP is widely used by EMS as a first line treatment for CHF with pulmonary edema and is widely credited with preventing many intubations and ICU admissions since its introduction into the prehospital environment CPAP is revered for it's simplicity and effectiveness in rapid application. It is usually well tolerated for patients well suited for CPAP, although contraindicated for patients with altered mental status or hypotension. Another critical failure with CPAP is waiting too long and allowing the patient to progress to respiratory failure.

<u>BiPAP</u>

Bi-PAP differs from CPAP in that Bi-PAP has two different pressure settings, Inspiratory Peak Airway Pressure (IPAP) and Expiratory Peak Airway Pressure (EPAP). The Bi-PAP machine will sense the pressure changes in the circuit and apply the appropriate pressure level. IPAP is higher to support inhalation and augment ventilation, while the EPAP is a lower pressure applied during exhalation to maintain alveolar recruitment and increase mean airway pressure.  This bi-level pressure scheme decreases work of breathing while maintaining oxygenation and ventilation, and allows for application to other conditions like COPD and restrictive airway disease. Because of the additional settings, Bi-PAP is a little more complex than CPAP, though it is just as dependent on a tight mask seal. And also like CPAP, if it is too late for Bi-PAP, it is too late.




## Alarms & Limits



Since ventilators either set or measure pressures and volumes, most alarms are designed to alert the clinician to deviations from normal values in one of those two things. Regardless of the level of alarm urgency, every one requires immediate attention and the prescribed correction to avoid patient injury or decompensation.

<u>High Peak Pressure</u>

A high airway pressure or PIP alarm is probably the one that requires the most rapid attention, since high pressures can lead to immediate injury and decompensation. The most common cause of a high pressure alarm is a cough or asynchronous spontaneous breath from the patient. Less common but more ominous causes of high pressure are tube obstruction, bronchoconstriction and pneumothorax. The high pressure alarm usually defaults to about 40 $cmH_2O$ for adults, and many clinicians will set the alarm around 10 $cmH_2O$ above the patient's average measured PIP. Attached to the high pressure alarm on most machines is a relief valve that will terminate a breath when the high pressure limit is Is reached to prevent that pressure from reaching the patient. If he or she continuously triggers the high pressure alarm, then most likely either a setting or the patient's level of sedation needs to be adjusted. Increasing the high pressure limit is seldom the answer.

Low Peak Pressure

The most likely cause of a low peak pressure alarm is a vent circuit disconnect, either from the ventilator, from the ET tube or, more ominously, from the patient with the tube still attached. A persistent low pressure alarm with an intact circuit may indicate an air leak or a deflated cuff. Another possibility is the patient initiated a spontaneous breath in the middle of a mandatory breath, momentarily dropping the pressure in the circuit.

Low Minute Ventilation

The low minute ventilation alarm is most important for spontaneous modes like pressure support, CPAP and Bi-PAP since ventilation is not mandatory in those modes. It is also useful for SIMV since the patient is capable of breathing almost entirely spontaneously without machine intervention. A persistent low minute ventilation alarm should prompt an assessment of the patient's readiness to maintain his or her own respiratory effort.

Apnea

This is also an important alarm for spontaneous breathing modes, as it will alert you when the patient has stopped triggering the ventilator. In CPAP or Bi-PAP modes, most machines will supply a backup mandatory rate to rescue patients from apnea after a set time, usually about 20 seconds.

Low Oxygen/$O_2$ Pressure

If you selected an $FiO_2$ greater than 21%, any interruption in the flow of oxygen to the ventilator will usually result in a low oxygen alarm. However, some older, gas powered ventilators may not have this alarm, and will only signal low gas by failing to work.

Other alarms & limits

Some other common alarms include high and low PEEP, high F, and battery alarms. Depending on the ventilator and patient, these will have varying levels of urgency.

## Troubleshooting

Usually an alarm will prompt the clinician to try to fix a problem with the ventilator or the patient. It can be something as simple as finding a power or oxygen source, or something critical like replacing the ET tube or decompressing a tension pneumothorax. Whatever the reason, it is best to use a systematic approach to finding and fixing the problem while keeping patient safety at the forefront of your priorities. Many clinicians use a simple process to remember that the critical steps for ventilator troubleshooting start with the patient and outwards to the machine.

First things First!
If the ventilator fails to deliver a breath or the patient begins to clinically deteriorate, the very first step is to remove the machine from the patient and begin bag valve ventilations. If the patient immediately begins to improve, then you can easily determine that the problem is with the ventilator, or at least with the patient-ventilator interface. If they do not improve, this simple mnemonic can help you remember what to check.

| | | |
|---|---|---|
| D | DISPLACEMENT | **Displacement of ET tube** |
| O | OBSTRUCTION | **Obstruction/king in ET tube or circuit** |
| P | PNEUMOTHORAX | **Lung collapse, tension or otherwise.** |
| E | EQUIPMENT | **Lost power, low oxygen, mechanical failure** |

## Maneuvers

The available maneuvers will vary depending on the ventilator and the current mode.

$O_2$ Flush
Even if the patient does not require 100% $FiO_2$ for the duration of your encounter, certain events or procedures like suctioning or movement may necessitate increased oxygen delivery for a short time. Most machines have an $O_2$ flush feature which will increase the $FiO_2$ to 100% for a set time limit to prevent desaturation. If you find that you need this feature multiple times in a short duration, the patient probably requires either more $FiO_2$ or some other adjustment to improve oxygenation.

Inspiratory Hold

We have already discussed measurements and alarms for high peak airway pressure, which represents the highest pressure achieved during the inspiratory time. While this is somewhat indicative of pressure in the lungs, it is more telling of pressure throughout the ventilator circuit. In order to get an idea of how much pressure exists against the alveoli, and thus an idea of the true risk of barotrauma, assessment of a plateau pressure (PPlat) is much more useful. Most ventilators will do this by initiating an inspiratory hold, which is just a fancy, mechanical way of making the patient hold his breath. The vent will deliver a mandatory breath based on the selected control, then will not allow it to exhale. This effectively isolates the lungs from the circuit and then measures the pressure as air settles into the alveoli. Most sources suggest that PPlat never exceed 30 $cmH_2O$. Higher pressures are closely linked to ventilator induced lung injury (VILI) and barotrauma.

Expiratory Hold

In a similar way, most modern ventilators can assess the amount of pressure remaining in the lungs at the end of exhalation, or the effective auto-PEEP. To do this, the machine will allow the patient to exhale based on the selected mode and control, then prevents another inspiration. The ventilator will then measure the actual PEEP and compare it to the clinician set PEEP, and display the difference. A positive number indicates auto-PEEP, or that the patient is holding additional pressure above what the ventilator is maintaining. Too much auto-PEEP can increase intrathoracic pressure and possibly lead to hypoventilation.

## Summary

To understand how mechanical ventilators work, it is first necessary to understand how humans ventilate and oxygenate themselves. With the proper physiology, then pathophysiology in mind, it becomes much easier to know how to get the ventilator to do what the patient needs in order to first survive and then heal. Furthermore, knowing the terminology goes a long way to knowing what the limits, controls and modes are and how they work. With that knowledge, we can begin the study of how to participate in the respiratory care of our patients from the acute process that put them on the ventilator to the titration and weaning necessary to get them off of it.

# PATIENT #1

22 y/o 75 kg M s/p high speed MVC. RSI 2° to ↓LOC. No gross hemorrhage. BBS = & clear.

| VITALS | VENT SETTINGS |
| --- | --- |
| GCS: 3 | CONTROL__________________ |
| HR: 94 | MODE_____________________ |
| RR 20 (BVM w 100%) | $FiO_2$______________________ |
| BP: 138/88 | F__________________________ |
| $SpO_2$: 100% | Ti__________________________ |
| $EtCO_2$: 23 mmHg | $V_T$/PIP_____________________ |
| | PEEP______________________ |

NOTES

# PATIENT #2

59 y/o 100kg F s/p VFIB arrest. RSI 2° to persistent respiratory distress. BBS crackles throughout.

| VITALS | VENT SETTINGS |
|---|---|
| GCS: 3 | CONTROL__________________ |
| HR: 120 | MODE_____________________ |
| RR 20(BVM w 100% & PEEP 15) | $FiO_2$______________________ |
| BP: 192/110 | F_________________________ |
| $SpO_2$: 94% | Ti_________________________ |
| $EtCO_2$: 40 mmHg | $V_T$/PIP_____________________ |
| | PEEP______________________ |

NOTES

# PATIENT #3

74 y/o 60 kg ICU day #10, Dx: Sepsis. Levophed & phenylephrine infusing. BBS diminished & crackles.

VITALS

GCS: 3
HR: 96
RR 12 (SEE VENT SETTINGS)
BP: 92/40
$SpO_2$: 93%
$EtCO_2$: 29 mmHg

CURRENT VENT SETTINGS

CONTROL: VOLUME
MODE: A/C
$FiO_2$: 50%
F: 12
$V_T$/PIP: 550
PEEP: 5

NOTES

NEW VENT SETTINGS

CONTROL____________________
MODE________________________
$FiO_2$_________________________
F_____________________________
Ti_____________________________
$V_T$/PIP________________________
PEEP_________________________

# PATIENT #4

4 y/o 17 kg F Dx: new onset seizures. RSI 2° persistent AMS and medications. BBS = clear

| VITALS | VENT SETTINGS |
| --- | --- |
| GCS: 6 | CONTROL___________________ |
| HR: 136 | MODE______________________ |
| RR 20(BVM w 100%) | $FiO_2$________________________ |
| BP: 88/50 | F___________________________ |
| $SpO_2$: 100% | Ti___________________________ |
| $EtCO_2$: 36 mmHg | $V_T$/PIP______________________ |
| | PEEP________________________ |

NOTES

# PATIENT #5

40 d/o 3.9 kg M Dx: RSV. RSI 2° respiratory distress and worsening ABG. BBS crackles throughout.

| VITALS | VENT SETTINGS |
|---|---|
| GCS: 3T | CONTROL__________________ |
| HR: 188 | MODE______________________ |
| RR 24(BVM w 100%) | $FiO_2$______________________ |
| BP: 76/46 | F___________________________ |
| $SpO_2$: 97% | Ti___________________________ |
| $EtCO_2$: 41 mmHg | $V_T$/PIP______________________ |
| | PEEP_______________________ |

NOTES

# PATIENT #6

24 y/o 95 kg F Dx: asthma exacerbation. RSI 2° respiratory distress and worsening ABG. BBS inspiratory/expiratory wheezes throughout.

VITALS

GCS: 5
HR: 108
RR 18(BVM w 100%)
BP: 118/78
$SpO_2$: 99%
$EtCO_2$: 51 mmHg

VENT SETTINGS

CONTROL___________________
MODE______________________
$FiO_2$________________________
F___________________________
Ti___________________________
$V_T$/PIP______________________
PEEP________________________

NOTES

# PATIENT #7

74 y/o 105 kg M Dx: CHF. $O_2$ via NRB @ 15 lpm.  BBS crackles and rhonchi throughout.

VITALS

GCS: 15
HR: 114
RR 14
BP: 198/104
$SpO_2$: 84%
$EtCO_2$: 48 mmHg

VENT SETTINGS

CONTROL___________________
MODE______________________
$FiO_2$________________________
F___________________________
Ti___________________________
$V_T$/PIP______________________
PEEP________________________

NOTES

# PATIENT #8

14 y/o 65 kg F Dx: DKA. RSI 2° severly altered mental status and intractable vomiting.  BBS = clear

## VITALS

GCS: 3T
HR: 134
RR 18(BVM w 100%)
BP: 98/64
$SpO_2$: 100%
$EtCO_2$: 17 mmHg

## VENT SETTINGS

CONTROL___________________
MODE______________________
$FiO_2$_______________________
F___________________________
Ti___________________________
$V_T$/PIP______________________
PEEP________________________

## NOTES

# PATIENT #9

51 y/o 135kg M ICU day 2. Dx: Respiratory failure

VITALS

GCS: 9
HR: 101
RR 18 (SEE VENT SETTINGS)
BP: 114/56
$SpO_2$: 91%
$EtCO_2$: 31 mmHg

CURRENT VENT SETTINGS

CONTROL: Pressure
MODE: A/C
$FiO_2$: 80%
F: 18
$V_T$/PIP: 20
PEEP: 5

NOTES

NEW VENT SETTINGS

CONTROL___________________
MODE______________________
$FiO_2$________________________
F___________________________
Ti___________________________
$V_T$/PIP_____________________
PEEP________________________

# PATIENT #10

34 y/o 85 kg F GSW x 2 ABD.  RSI 2° comabiveness.  BBS severe-ly diminished.

| VITALS | VENT SETTINGS |
|---|---|
| GCS: 3T | CONTROL__________________ |
| HR: 125 | MODE_____________________ |
| RR 12(BVM w 100%) | $FiO_2$______________________ |
| BP: 88/50 | F_________________________ |
| $SpO_2$: 76% | Ti_________________________ |
| $EtCO_2$: 22 mmHg | $V_T$/PIP_____________________ |
| | PEEP______________________ |

NOTES

# ABOUT THE INSTRUCTOR

Michael C. Berrier is has been active in public safety since 1987, and has been working and teaching as a paramedic for the past 15 years.  He has worked in rural/suburban 911, fire service, the offshore oilfield, EMS administration and most recently as a critical care transport paramedic in North Carolina. Michael holds certifications from North Carolina, The National Registry of EMT's, University of Maryland-Baltimore County, Cleveland Clinic, Board for Critical Care Transport Certification and the Society of Critical Care Medicine.

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www.ccellc.pro
919.766.0795
michaelberrier@critical-care-excellence.pro