Rykerr Medical's

Vent Management Guide

for Invasive Mechanical Ventilation in Transport

Version 1
May 2020

A to LLC logo

RYKERR MEDICAL

⇒ prior to do list or cover:

daveyvermeulen@yahoo.com

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Return to Contents

Table of Contents

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Return to Contents

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A Personal Intro

There are lots of good reasons why I thought it'd be good to put together a primer ~~of sorts~~ on vent management, but the main one is that my first vent experience was a near-disaster and I'd like to share what I've learned since then so that others can avoid what I had to go through. I also think there's some room for diversity in how we, as an industry, present material to each other and move forward with our understanding of complicated things. So my second hope is that this interactive style of writing can be of help to some folks and maybe inspire others to do the same and build on the whole idea.

But to start with the awful beginning story: I was brand new to an ambulance service in rural New Mexico, having moved from Pittsburgh about two years after I first got my medic. I was still green but felt like I had gotten a lot of experience back in the city and was maybe over-confident. Anyways, I started at this service in mid-November and this call I did was the day after Thanksgiving, so I had basically just arrived in NM and gotten settled in to the second EMS service I had ever been given medical control at. Things were different for sure. Five- and ten-minute transport times had been replaced by ones much longer in our 5,000 square mile coverage area, the ambulances were giant machines that could be rigged to carry three patients each and would never have made it in the city alleys, and protocols/ capabilities were a lot more lenient and included vents, surgical crics, hiking in to patients broken in the woods - that sort of thing that this city boy just hadn't done before.

Oh, and also two-patient interfacility transfers. Our flagship hospital was in Albuquerque, one hundred and eighty miles or two and a half hours away by bus, so it was hugely advantageous to load two patients in on a single truck to avoid and extra six-ish hours of that second truck being gone from the service area. So when I was asked if I was OK with a vent patient and a psych patient going up to Albuquerque at the same time I didn't say no and we started getting things together. Part of that prep process was another guy showing this guy how to use the LTV1200, as I hadn't gotten to that part in my orientation and didn't yet have the confidence to say "no" to things I wasn't comfortable with or ready for.

My five-minute vent lesson was subpar, to say the least, and then I was off to the big city with the vent guy on the stretcher and the psych guy on the bench seat, two EMTs up front just in case I needed anything. My first action when the vent started beeping was to press that handy "silence" button – per the lesson I had received on the machine's operation. When that didn't work I figure it might be because the patient wasn't listening to the vent settings we had dialed in before leaving, so I paralyzed him ~~with Vec~~ – also per the lesson I had received. And that worked for a little while. Then I started getting more alarms and a low sat, so I did what all good medics do and disconnected the vent, grabbed my BVM and had the EMTs up front pull over so that one of them could hop in the back and give me a hand.

Sats still stayed low, the alarms were yelling at me, the EMT was like "WTF, bro, get it together," and I didn't know what to do, so I turned the vent off, pulled the tube out and started over from the very beginning with BLS airways and the BVM. So that happened and we had the airway secured, sats came up and then I handed the bag off to the EMT and set my sights on restarting this vent machine the way I had been taught just a little while ago. It was during this process that I realized my connections from the machine to the circuit had come undone. I must have stepped on them or something during the shuffle… Nowadays I would have simply looked at which alarm I was getting and worked through a systematic process for addressing that alarm. The whole fiasco would have been avoided. But back then I didn't know a single thing about vents, to include that the text on the screen was relevant to getting the alarm to stop. Other than what I learned in my short pre-trip lesson.

Return to Contents

And that's just part of the story. One other part, don't forget, is that guy on the bench seat watching the whole damn thing and me hoping he stays cool enough that I don't have to try and manage two patients simultaneously. And another part is that even though I finally did get that alarm situation sorted, I still had trouble managing my vent settings. I couldn't maximize my SpO_2 or keep my $EtCO_2$ in range, my patient would get super agitated every time the Vec wore off, etc.… So I returned back to small town New Mexico late on the day after Thanksgiving, year 2012, and decided then and there that I was never, ever, going to be in that situation again.

My initial study list looked something like this:

The Ventilator Book by William Owens

The LTV1200 Product Manual (and the DVDs)

EMCrit Dominating the Vent Series

I later came across many other great resources and I will mention those as we get to them. And also, I got on the technology train. Which I think is a huge facilitator of learning when used in the right way and I hope that this little experiment can demonstrate that. If you have the print version of this badboy you can just scan the QR codes for any of the references to access them (if available for free) or to see where you can purchase them (if they want your money); if you have an electronic version, just click the links. And if you have a version where the links don't work because it isn't legit, that's cool too: go here[1] to get it all free and official.

So now let's jump into the weeds and see where we end up. Keep in mind that this is to be an ongoing project and my first foray into this type of thing – so if you have feedback, just send it my way and offer either to lend a hand or a valid suggestion. I'd love to get more folks involved in this and make it both better and more accessible for all involved :)

EMS VTF
NM ETT
BVM OG
BLS
SpO_2
$EtCO_2$
QR
(DVD)

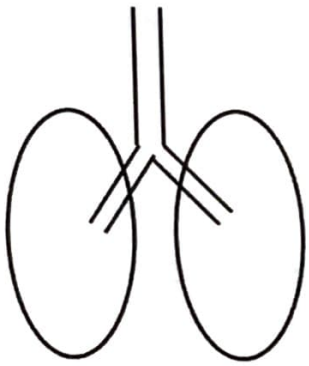
[1] Or follow the QR code on the cover to link to the website

Some Very Basic Physiology

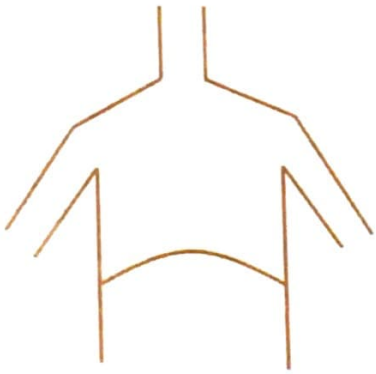
As a disclaimer: the stuff outlined here is super basic and intended to give a foundation for the fundamental concepts of vent management. One recommendation for looking into the details beyond this (much of which comes up later when we talk about specific conditions) is a good, solid, heavy Anatomy and Physiology textbook or any of the references listed at the very end.

The Normal Breathing Process

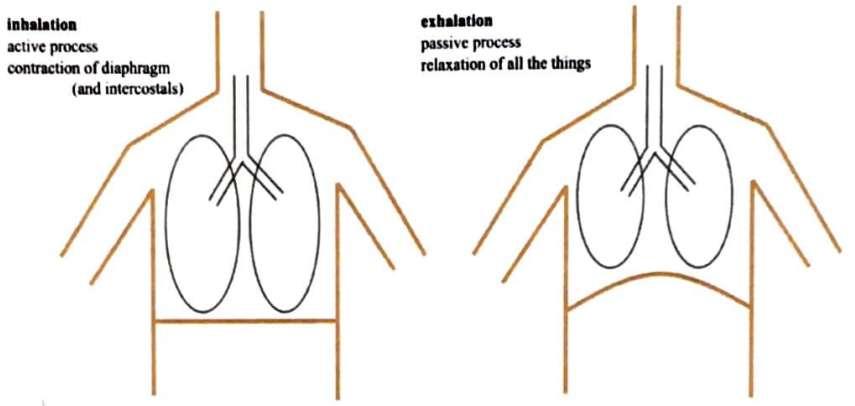
Let's start with a picture of what major components we are working with in normal inhalation and exhalation. At its most basic we have the lungs and the large airways:



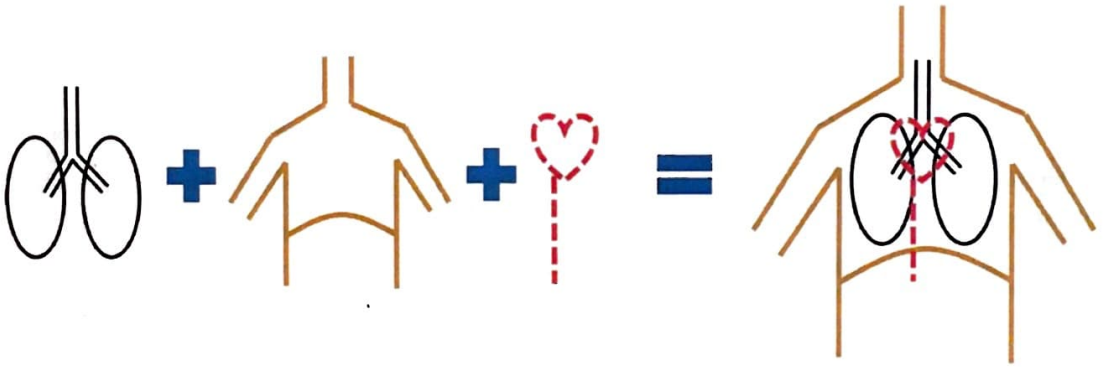
We also have the chest cavity and the diaphragm:



It's OK to consider the lungs to be "attached" to the chest cavity and diaphragm so that when the diaphragm contracts or flattens, the lungs expand – this sucks air into the plural space via a negative pressure:[2]



Inside this same cavity lie the heart and great vessels (and most importantly to our discussion, the inferior vena cava):

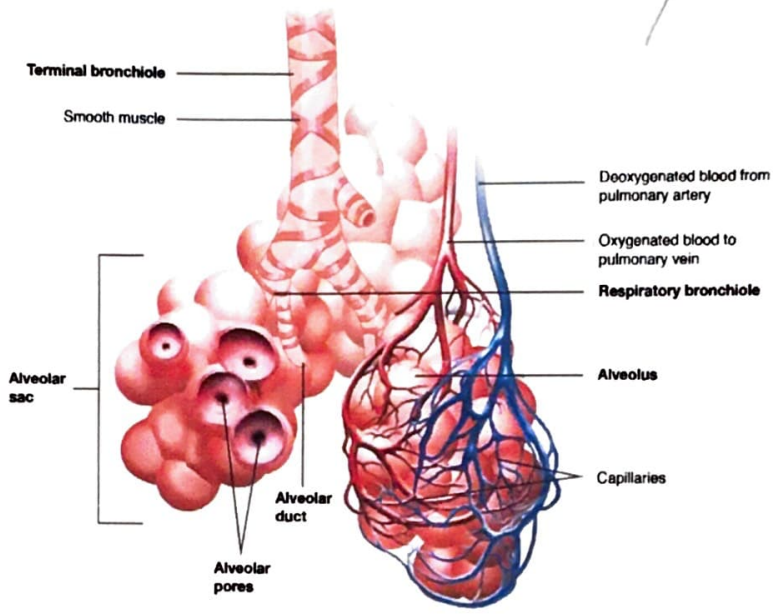


So now we have a system that normally functions by contraction of the diaphragm (with or without help from the intercostal muscles) to create a negative pressure, "sucking" of air into the lungs. Because this air movement occurs via a negative pressure, blood return via the inferior vena cava is facilitated by normal ventilation[3] – this will be important when we move on to talk about positive pressure ventilation in just a minute.

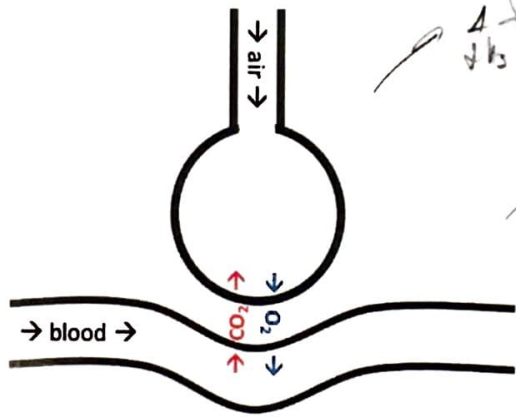
[2] This assumption mostly holds true for our need in the transport setting, so we won't take it much further than that here

[3] Azizov, 2017 – Video that explains how this mechanism works

From there we need to zoom in and take a look inside the lung tissue. The image below shows blood vessels encircling little sacs, known as alveoli, which are the homestay of ~~the all-famous~~ pulmonary gas exchange where oxygen goes into the blood and carbon dioxide goes out:[4]

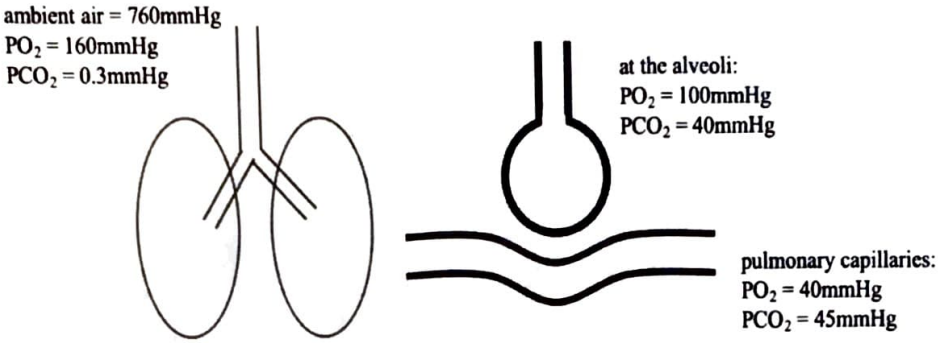


A simplified version of a single alveoli with a corresponding blood supply can help us understand the (patho)physiology of different situations:



[4] Betts & friends, 2013 (image) – This image is from a free online textbook ~~that we take a few other images from also~~

Next, let's add some numbers to that graphic of a single alveoli and its blood supply.[5] Note that in real life blood is continually moving past the alveoli and gases are constantly moving to reach equilibrium, so that as carbon dioxide is offloaded and oxygen is onloaded, there is a new supply of blood and a reset of the gradients across that membrane. Plus this diffusion of gasses from alveoli to pulmonary capillaries happens very quickly, so we generally aren't worried about the diffusion of gases as a function of speed being the limiting factor in this process:[6]



because there is an open system between the ambient air and the alveoli, the overall pressure at the alveoli is also 760mmHg, however the partial pressures of the components are different along the way

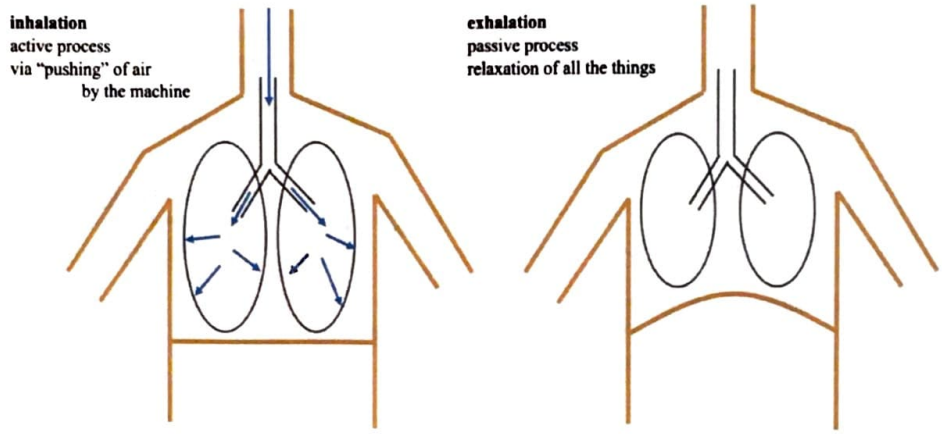
It's also worth mentioning that the pressure gradient or difference from alveoli to capillary is drastically different when comparing oxygen to carbon dioxide: oxygen has a pressure difference of about 60mmHg, carbon dioxide has one of just 5mmHg. While this may seem, at first glance, to put the body at risk of some sort of imbalance, carbon dioxide moves more easily through liquids, and thus the membrane between capillary and alveoli, (roughly twenty times so) and the net result is that oxygen and carbon dioxide exchange at about the same rate.

[5] Betts & friends, 2013 – They give all these values except for PO_2; that one is cited as 104mmHg, but we calculated it out in the **Appendix** and use the calculated value to maintain consistency throughout this text

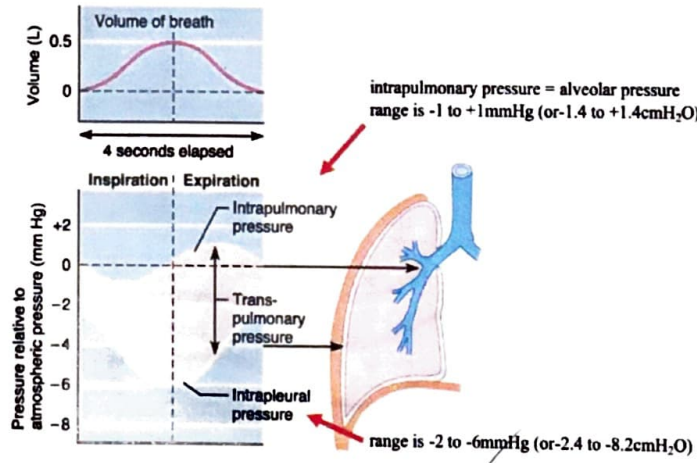
[6] Speller, 2018 – Outlines how both oxygen and carbon dioxide diffuse in the pulmonary system in the context of gas laws; do note, however, that certain states can slow this process down (and we'll get to those later on!)

How is Positive Pressure Ventilation Different?

Next we need to consider what happens when we bypass the whole negative pressure mechanism for ventilation and instead opt for a positive pressure approach.[7] Let's start at the top with the basic sketch of airways and lungs superimposed on the chest wall and diaphragm. When we ventilate by positive pressure ventilation (PPV) we have to physically displace the diaphragm and chest wall while simultaneously pushing air into the system – this requires a lot more pressure that we needed for that negative pressure, spontaneous mechanism:



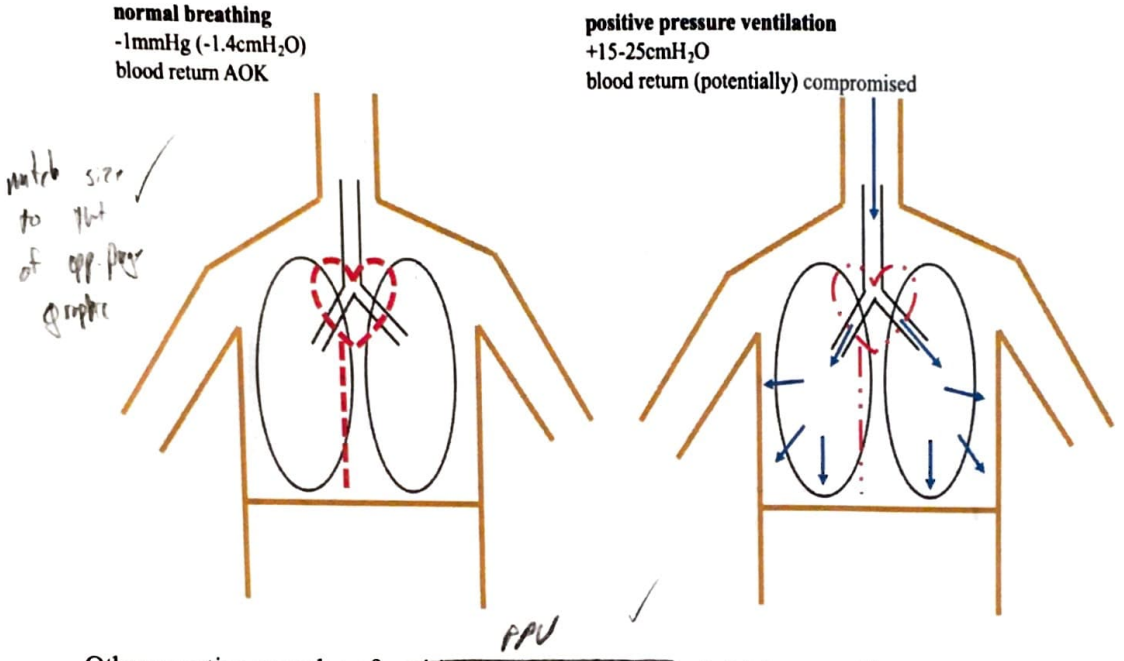
We will get to airway pressures and limits for them later on, but a normal plateau pressure (which reflects the average alveolar pressure in positive pressure ventilation) is in the range of 15-25cmH_20; compare this to the pressures represented in the following illustration:[8]



[7] This assumes that the patient is not contributing to this effort of breathing; to say it another way, this description is accurate for the patient who is not making any respiratory effort or is out of synch with mechanical efforts – in reality we can synch patient effort to machine effort to minimize the differences and effects discussed in this section (discussed more in **Comfort**)

[8] Kahathuduwa, 2013 (image) - Two things: we'll talk about the mmHg and cmH_2O conundrum at the end of the next section (in **Measuring Pressures**); alveolar pressure is the most relevant to our discussion for now, the concepts of transpulmonary pressure and intrapleural pressures are deferred here

The biggest impact of that increased intrathoracic pressure is the effect it may have on cardiac output (CO). Increased intrathoracic pressure can decrease blood return to the heart via pressure on the vena cava, resulting in decreased preload and, therefore, less output.[9] Let's represent it this way:



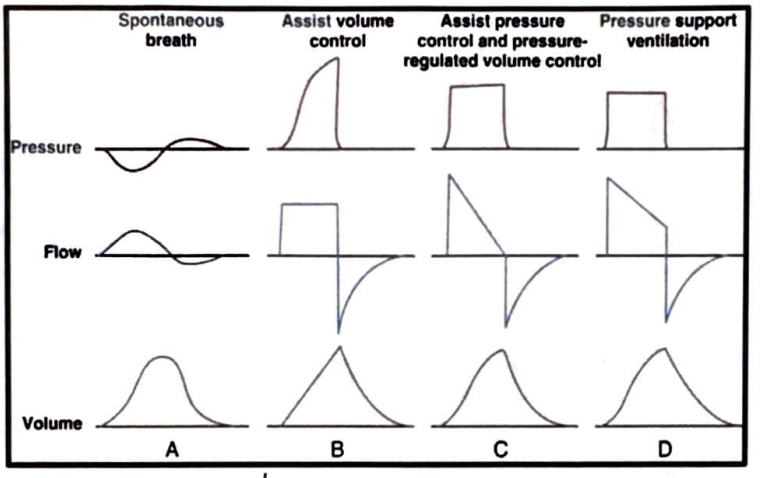
Other negative sequalae of ~~positive pressure ventilation~~ (which may still occur even if we have all the settings dialed in right!) would be patient discomfort, muscle fatigue/ weakening,[10] and physiologic changes to other body systems.[11] And then if we have things dialed in wrong on the machine or don't ventilate appropriately based on patient presentation, we can also cause things like direct injury to the lungs/ alveoli and hypoventilation (leading to shock). This is but a short list of the major things we'll worry about in this manual, just recognize that there is a lot of potential for bad and that's why we need to know how to manage the machine to the best of our collective ability and mitigate as many of these things as we can along the way.

[9] Strong, 2013; Mahmood & Pinsky, 2018 – Both this video and the article explain in more detail on how PPV (and particularly **PEEP**, discussed later) can affect CO, especially with concurrent hypovolemia; while it isn't always true that PPV decreases CO (sometimes the opposite can occur), the PPV/ PEEP → decreased preload → decreased CO sequence of events is most relevant to us in the transport setting

[10] Tobin & friends, 2010 – Outlines the idea that we can mitigate this consequence by adjusting vent settings to require that the patient make some intrinsic effort to breath; while their ending advice is to utilize an airway pressure waveform to monitor patient effort (something we don't routinely have in the transport setting), it still provides valuable insight on the whole concept

[11] Yartsev, 2019 – In fact, navigate to "Respiratory System" header at the top of this page and then down to the section on "Physiology of Positive Pressure Ventilation" for more detail on all of this stuff

We already saw how a pressure waveform might look over time with spontaneous, negative pressure breaths, so let's see how it looks with a machine delivered breath. Note that there are different types of machine delivered breaths in this diagram (plus some terms to discuss), and we haven't yet gotten there; that's totally OK, we just want to point out some general trends here. Big takeaway: the left set of patterns (the normal) looks nice and smooth, without any harsh changes or drastic swings in amplitude; all of the others have those things we don't want. Another thing is that the graphic representations of the types of breaths (i.e. each column of the three towards the right) are each slightly different - sometimes one mode will be more comfortable for a certain patient in spite of trying to do all the other things you know how to do, simply because how that patient's body responds:[12]



In an effort not to discourage anyone from ever putting a patient on a vent, there are ~~some~~ advantages ~~of positive pressure ventilation and mechanical ventilation~~. Most obvious of these is that it allows us to breathe for a patient in a relatively simple way when that patient is unable to do so on his or her own. More specifically, mechanical ventilation allows us to control and direct recovery with specific pathologies (such as acidosis, asthma, and ARDS; all of which we will discuss later on). Positive pressure can help move oxygen into the bloodstream more easily, managing ventilation can help that oxygen get delivered more effectively, manipulating time spent at different parts of the respiratory cycle can increase the amount of time that the body can participate in pulmonary respiration, etc. There are lots of good uses of the ventilator and we will get to all of them in due time, so don't worry if that got to be too much for a moment and know that in spite of its drawbacks, mechanical ventilation and ~~positive pressure ventilation do~~ have ~~their~~ place in the cosmos.

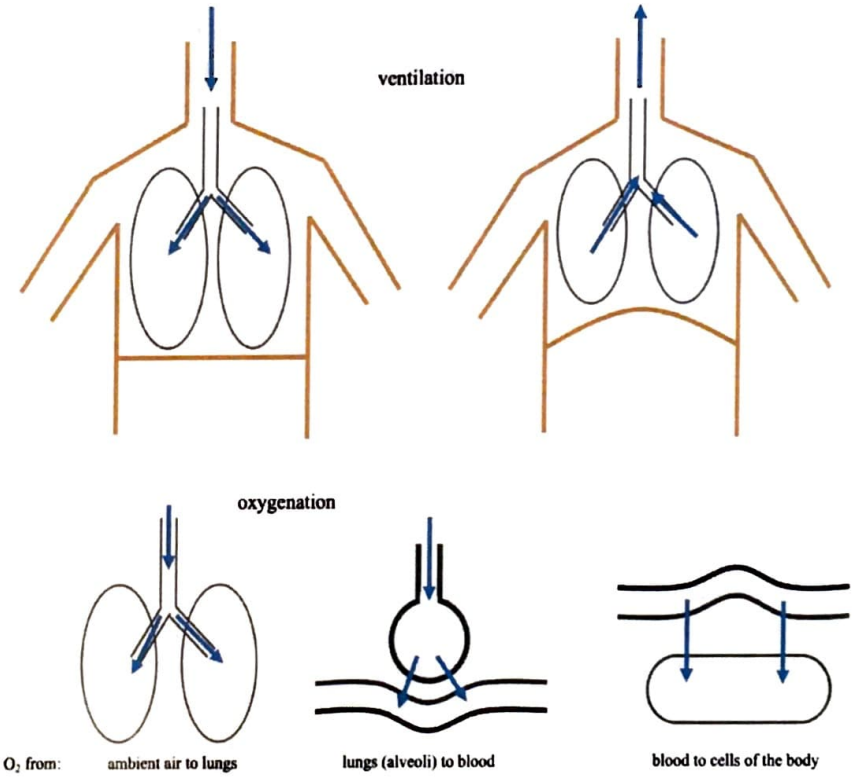
[12] Fuller & friends, 2014 (image) - This this assessment of what the body "wants" in terms of smooth waveforms and avoidance of harsh changes in amplitude is a subjective concept - it seems to make intuitive sense, but there may not be a good way to verify the idea

Return to Contents

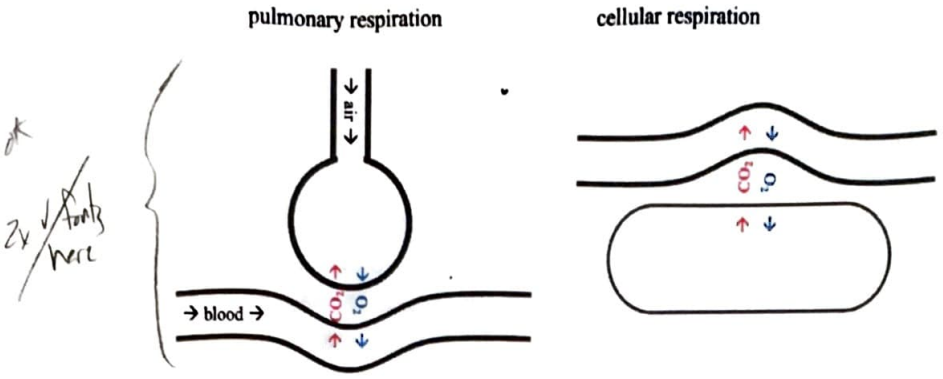
Other Important Concepts

Ventilation, Oxygenation, and Respiration

Just to differentiate the words that collectively describe breathing, let's chat about these three terms.[13] Ventilation refers to the gross movement of air as the body breathes in and out. Oxygenation refers to the transition of oxygen from the air outside of the body, through the respiratory and circulatory systems, and to the capillaries where it can be picked up by tissues for use. And lastly is respiration, which has two specific flavors. Pulmonary respiration refers to the exchange of carbon dioxide and oxygen in the alveoli of the lungs; cellular respiration refers to a comparable gas exchange at the tissues. To visualize it all, here are a few images to represent all of that:



[13] Betts & friends, 2013 – Explains in more detail the processes of ventilation (Section 22.3) and respiration (Section 22.4)



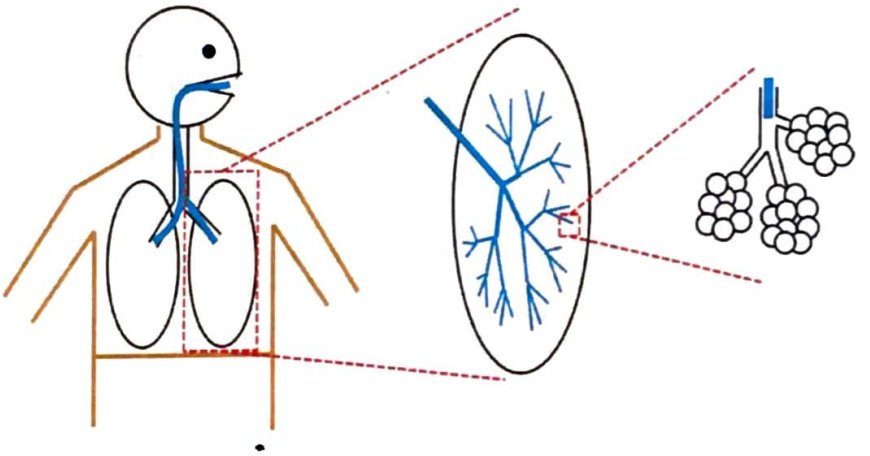
There is some overlap between oxygenation and pulmonary respiration in this context, but it helps to separate these ideas out. When managing the vent, we are most focused on the processes of ventilation and oxygenation. While respiration (in both forms) is very important, our ability to manipulate these processes isn't as straightforward as it is with ventilation and oxygenation; also, the part of respiration that we can impact, the pulmonary part, is covered in a roundabout way by our actions to address oxygenation. We will come back around to this idea in a bit when we talk about how to control both ventilation and oxygenation by changing different parameters on the ventilator.

Return to Contents

Dead Space[14]

Dead space can be an intimidating concept when it comes to vent management and we are going to try to both simplify it and identify specific situations in which it matters in the context of patient management. To start with, there are four types of dead space that we will discuss: anatomic, alveolar, physiologic and mechanical. We don't always see every one of these discussed in references, but we will include them all here to make sure that our understanding of dead space is complete. Dead space, as a term, can be used to describe any one of these subtypes, but it helps to recognize which type of dead space is of concern in a given situation.

To start things off, anatomic dead space is the air involved in the respiratory cycle that does not participate in gas exchange. As represented by the blue lines, it starts at the naso- and oro-pharynxes and extends down to the terminal bronchioles:



Another way to describe anatomic dead space, in light of this graphic, would be just about all the air involved in a respiratory cycle other than what ends up in the alveoli. Now this graphic isn't to scale, so it sort of seems as if dead space is the majority of the air involved in a respiratory cycle, but that isn't the case. There are tens of thousands of terminal bronchioles in a lung and hundreds of millions of alveoli,[15] so the majority of air ends up in the alveoli. It's also worth noting that this process is dynamic and that anatomic dead space refers to the air outside of the alveoli and respiratory bronchioles when those alveoli are fully inflated at peak of inspiration. As for quantifying this value: normal anatomic dead space is about 2ml/kg or about 1/3 of tidal volume.

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[14] Yartsev, 2019 – This is the best content we've been able to find on this subject, very thorough and with references to more information along the way

[15] Betts & friends, 2013 – And just to clarify some useless trivia: the terminal bronchioles (marked by the thick blue line in the far right side of this photo) are different then the respiratory bronchioles, which are the stems distal to that blue line that feed into each cluster of alveoli

Anatomic dead space is most relevant in our discussion of ventilated patients when we need to alter the amount of air that participates in alveolar gas exchange (i.e. ventilation). We will talk about this more later,[16] but we basically have two options when it comes to increasing the amount of air to the alveoli: increasing the frequency at which we deliver breaths or increasing the amount of air per breath delivered. If we add one breath to the equation, we must consider anatomic dead space and therefore the amount of air to the alveoli is less than the actual volume of that entire breath. On the other hand, if we simply add volume to breaths already being delivered, we actually get that additional volume at the alveoli because anatomic dead space has already been considered for each breath.

The next type of dead space is alveolar dead space. Alveolar dead space refers to the air in the alveoli that doesn't participate in gas exchange. This can be due to a few different things: decreased capillary blood flow, fluid in the alveoli, damage to the alveolar surface, etc. Regardless of cause, any time that alveolar air is limited in its ability to participate in gas exchange, we get alveolar dead space. In the normal human body, alveolar dead space is close to zero and we assume it to be negligible. In the sick or injured human body, however, we assume some alveolar dead space. ~~While there is a way to calculate this value (see Appendix), knowing that number doesn't routinely help in the transport setting. Instead, we assume alveolar dead space in all of our patients~~ and proactively take steps to accommodate that with our settings.

Interventions to address an assumed alveolar dead space would be ensuring adequate oxygenation, applying end-expiratory pressure,[17] utilizing appropriate ventilator settings by patient size, and proper patient positioning. All of these things will be discussed in sections to come, so no need to remember them here. Just know that the takeaway in regard to alveolar dead space is that we always assume it exists to some degree and we do what we can to mitigate it. Worst case scenario is that the lungs were healthy and that there was no alveolar dead space to begin with and that's totally fine – none of the interventions we do here would cause damage to the healthy lung when used appropriately. On the other hand, if we forget to make this assumption in a patient that does have some degree of alveolar dead space, we can increase mortality, delay recovery, and decrease the patient's ability to compensate for other threats that might come up during the clinical course (i.e. an infection along the way).

Next on the list is physiologic dead space. Physiologic dead space is the sum of anatomic dead space and alveolar dead space and represents all of the dead space before we introduce our devices into the system. In the healthy person, we often assume no alveolar dead space and therefore physiologic dead space is equal to anatomic dead space. Because of this relationship, the terms sometimes get used interchangeably. While there is a difference, the utility of knowing this fact doesn't much help our treatment of sick people, so from here on out we will refer to anatomic dead space and alveolar dead space and ignore the idea of physiologic dead space in an effort to be more specific with our discussion.

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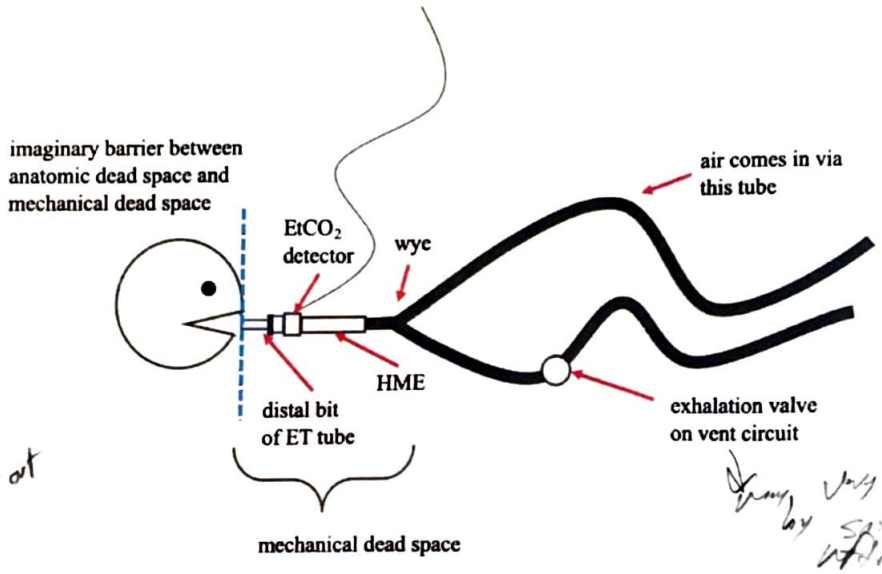
[16] In the section on **Ventilation (& $EtCO_2$)**

[17] While this does facilitate oxygenation, it also helps address the alveolar dead space situation via recruitment of more alveoli – these two ideas are discussed, respectively, in **Oxygenation (& SpO_2)** and **PEEP**

Last type of dead space is what we will call mechanical dead space. Mechanical dead space, which may also be noted as equipment or apparatus dead space, is the dead space that we add on to the system with our equipment: vent circuits, $EtCO_2$ detector, HME,[18] etc. To be a bit more specific, it refers to all the things from where anatomic dead space starts (oropharynx/ nasopharynx) to where exhaled air leaves the wye of the vent circuit:



Mechanical dead space is a problem because it increases the amount of "used up air" with which new air must be mixed before it gets to the alveoli. In the normal human being, fresh air is pulled into the airways starting right at that imaginary blue line in the above picture; in the ventilated patient, fresh air begins at that wye. We've discussed this effect in the **Appendix**, but suffice it to say that we should try to minimize mechanical dead space when possible (i.e. think about whether or not an in-line suction device or HME is needed rather than placing it blindly for all patients) and that the effect is more pronounced with smaller patients and higher respiratory rates (i.e. pediatrics).

One last thing about this concept is that there is a silver lining to our concept of mechanical dead space. The ET tube actually creates a narrow passageway from the teeth/ lips (where we drew that blue line) down to the trachea, essentially negating the dead space of the naso- and oro-pharynxes. So while the net change in overall dead space may be negligible as far as amount added versus amount taken away, we still want to maximize efficacy of ventilation and minimize unnecessary things in our vent circuit when possible. And we'll come back to this concept in the **Appendix**.

There is another related concept to consider in this discussion of dead space that doesn't quite fit any of the types above. We like to think of all of these volumes as fixed quantities of air, but the truth is that the containers that hold these quantities of air are flexible or have stretch and therefore we sometimes see differences in expected versus actual values. One example of this is that the amount of air we put into the system (tidal volume) doesn't always match up exactly with air out of the system (exhaled tidal volume). So where does that air go? Some of it stays in the alveoli (see upcoming discussion on recruitment), some of it leaks around our endotracheal tube (ETT) cuff, some of it is lost to the tissues and airway structures, etc. While this isn't exactly dead space per se, it helps to recognize that it is a thing that can cloud our understanding of air volumes.

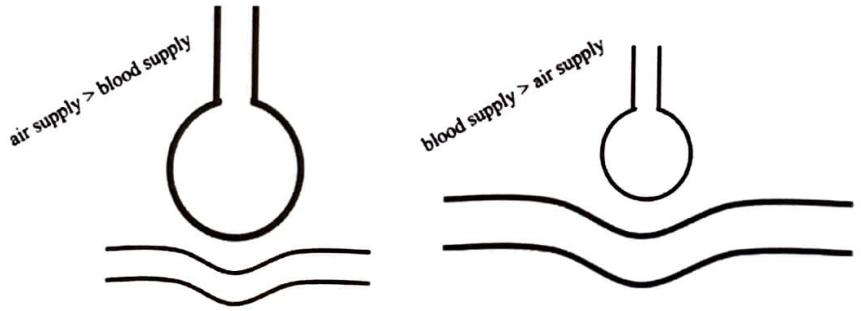
[18] Heat & Moisture Exchanger, discussed more in **Humidifiers**

Another place where this comes into play is with the vent circuits themselves. These plastic tubes are not rigid and do have a certain amount of stretch to them. If you look on the package of the tubing, there is a value that says how much volume of stretch a given circuit has per unit of pressure. We will revisit this idea again in later sections (once we discuss a few of the concepts mentioned here) but know that in volume control ventilation we may inadvertently overestimate the amount of air delivered if we ignore the stretch of the circuit. This is particularly relevant with little patients (i.e. infants), as the impact of this effect (ratio of misestimation to potential outcome) is more pronounced with smaller breaths (i.e. lower tidal volumes).[19]

Hypoxic Pulmonary Vasoconstriction[20]

Hypoxia in the pulmonary vascular bed results in vasoconstriction (thus the term, "hypoxic pulmonary vasoconstriction" or HPV), which is opposite of what happens in systemic circulation. This mechanism helps the lungs to avoid wasting blood supply to part of the lung that isn't getting enough oxygen – it's a mechanism to conserve resources and maximize efficiency of the system. Just as with other vascular beds in the body, the pulmonary capillaries are in a constant state of flux and respond to the needs of the system and the availability of resources (oxygen, in this case, being the driving force) by opening and closing.

Carrying on this conversation with a new term: HPV helps to avoid ventilation-perfusion mismatch (V/Q mismatch[21]), which could look like either of the following:



The left side type of V/Q mismatch demonstrates alveolar dead space. It shows that air supply (i.e. oxygen) in the alveolus is in excess of blood flow and therefore some of that oxygen won't get utilized or move into the bloodstream. The right side state is what we call a shunt. In a shunt, blood ends up passing through the pulmonary vascular bed without getting its full complement of oxygen. And it isn't always the case that the mismatch is due to volume of air in the alveoli as shown, it can also be related to some kind of impediment that prevents the movement of air out of the alveoli - examples of this would be pulmonary edema, ARDS, and pneumonia. In either of these cases, dead space or shunt, HPV is basically the body's mechanism for reversing this type of mismatch.

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[19] Bauer, 2018 – He discusses this idea in his book on vent management; we also demonstrate this impact in the context of managing a pediatric patient later on in the **Appendix**

[20] For more reading on the subject:
Dunham-Snary & friends, 2017 – Describes how this response can be inhibited my certain interventions; outlines the role of HPV in different pathologies
Lumb & Slinger, 2015 – Outlines the timelines discussed; also discusses a number of relevant pharmacological agents that contribute to the effect

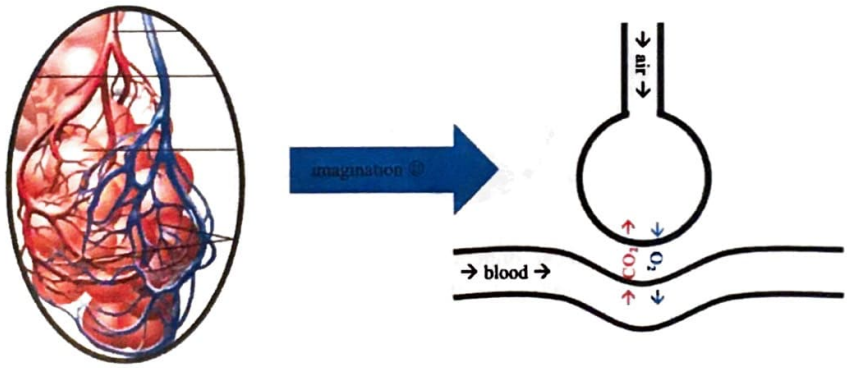
[21] Mason, 2019 - We just left out the idea of V/Q ratio in this discussion because our focus is on the general idea only, but take a look here for a quick explanation and overview of how this concept looks

Now one thing to know about this whole process is that it goes both ways: vasoconstriction is the response to hypoxia in the pulmonary capillaries and vasodilation occurs when oxygenation is adequate or that hypoxic state is resolved. We might consider these to be similar processes, just in opposite directions. There is a distinction, however, in the rate at which either change happens. The initial hypoxic vasoconstriction side of things happens on the order of second to minutes; the reverse process (vasodilation) typically also occurs quickly, but can happen much more slowly (up to hours) or incompletely (without complete reversal of the vasoconstriction) when the HPV response has been sustained for a while.

The HPV response and the fact that it may take quite some time to reverse helps to explain, in part, why we aren't always able to fix our vented patients as well as we want to in the short span we get to hang with them in transport. It also helps bring out the idea that just because a patient doesn't look awesome when we get there doesn't mean that the sending facility or crew has been doing things wrong – they may be taking the right steps and called us before enough time passed for the fix to work its way out. There are many more intricacies and effects of HPV on the body (see all those references on the previous page), but the main point at this juncture is that we may not be able to fix a super sick patient quickly. And that's just fine – we do what we can (as we will outline soon) and recognize that there are limits to what results we can expect.

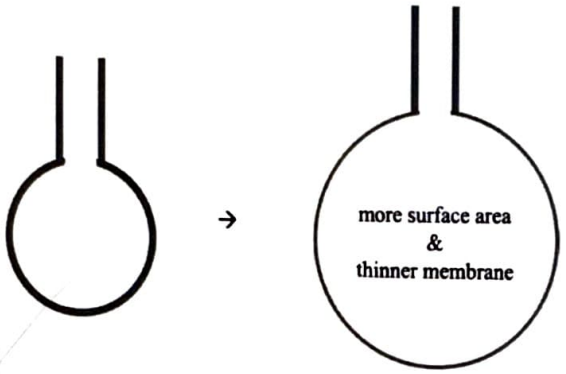
Alveolar Surface Area

Even though we have been demonstrating the alveoli-capillary interface as a single blood vessel running past the air sac, it is important to recognize, again, that this is a simplification of how things really are and that the surface of the alveoli are covered by a network of vessels:[22]

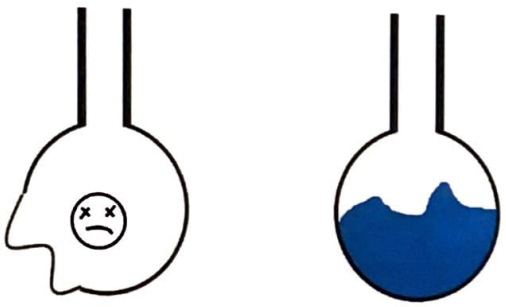


[22] Betts & friends, 2013 (image)

When we inflate the alveoli we get more surface area and that means more interface between air and blood. In addition, inflation of the alveoli causes the alveolar membrane to stretch and become thinner, allowing for easier diffusion of gasses:[23]



More surface area and thinner membrane makes it easier to move air from inside of the alveoli to the circulatory system,[24] so lots of our interventions with the vent are focused on this idea. That said, there are things that can get in the way of this improved gas exchange even if we do get the surface area up and membrane thinned out. Think of these as things that impact access to usable alveolar surface area:[25]



toxins can injure the membrane directly

fluid can impede gas exchange across the membrane

inflammation can also have a similar effect

All of this means that in order for efficient gas exchange to occur, we may have to manage multiple things simultaneously. We will get to all of these different things eventually, just know that the whole process isn't as simple as it seems at first glance.

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[23] And we spell this out in much more detail in the section on **Oxygenation (& SpO_2)**

[24] Desai, 2012 – We cite this video in **Oxygenation (& SpO_2)**, but here it is now if anyone is curious before then

[25] George, 2015 – Check this out for a bit of extra detail on the difference between pneumonia and pneumonitis, both of which are included in this working list of things that can inhibit effective gas exchange

Return to Contents

Lung Size

Second to last thing related to underlaying physiology before we move on to talking about the machine: lung size is most strongly correlated with patient height. Because of this, we use a patient's height to calculate an ideal body weight (IBW)[26] when doing vent things. The idea is that a six-foot dude could weigh either 120lbs or 300lbs and the size of his lungs wouldn't change. There is a formula to calculate IBW for both males and females, often presented as a hybrid of metric and standard units:

$$IBW_{dudes}\ (kg) = 2.3(\text{height in inches}) - 60) + 50$$
$$IBW_{chicks}\ (kg) = 2.3(\text{height in inches}) - 60) + 45.5$$

For the metric enthusiasts, we also have it as so:

$$IBW_{dudes}\ (kg) = 0.91(\text{height in cm}) - 152.4) + 50$$
$$IBW_{chicks}\ (kg) = 0.91(\text{height in cm}) - 152.4) + 45.5$$

Or we can use charts like this:[27]

HEIGHT	PBW	4 ml	5 ml	6 ml	7 ml	8 ml
4' 0" (48)	17.9	72	90	107	125	143
4' 1" (49)	20.2	81	101	121	141	162
4' 2" (50)	22.5	90	113	135	158	180
4' 3" (51)	24.8	99	124	149	174	198
4' 4" (52)	27.1	108	136	163	190	217
4' 5" (53)	29.4	118	147	176	206	235
4' 6" (54)	31.7	127	159	190	222	254
4' 7" (55)	34	136	170	204	238	272
4' 8" (56)	36.3	145	182	218	254	290
4' 9" (57)	38.6	154	193	232	270	309
4' 10" (58)	40.9	164	205	245	286	327
4' 11" (59)	43.2	173	216	259	302	346
5' 0" (60)	45.5	182	228	273	319	364
5' 1" (61)	47.8	191	239	287	335	382
5' 2" (62)	50.1	200	251	301	351	401
5' 3" (63)	52.4	210	262	314	367	419
5' 4" (64)	54.7	219	274	328	383	438
5' 5" (65)	57	228	285	342	399	456
5' 6" (66)	59.3	237	297	356	415	474
5' 7" (67)	61.6	246	308	370	431	493
5' 8" (68)	63.9	256	320	383	447	511
5' 9" (69)	66.2	265	331	397	463	530
5' 10" (70)	68.5	274	343	411	480	548
5' 11" (71)	70.8	283	354	425	496	566
6' 0" (72)	73.1	292	366	439	512	585
6' 1" (73)	75.4	302	377	452	528	603
6' 2" (74)	77.7	311	389	466	544	622
6' 3" (75)	80	320	400	480	560	640
6' 4" (76)	82.3	329	412	494	576	658
6' 5" (77)	84.6	338	423	508	592	677
6' 6" (78)	86.9	348	435	521	608	695
6' 7" (79)	89.2	357	446	535	624	714
6' 8" (80)	91.5	366	458	549	641	732
6' 9" (81)	93.8	375	469	563	657	750
6' 10" (82)	96.1	384	481	577	673	769
6' 11" (83)	98.4	394	492	590	689	787
7' 0" (84)	100.7	403	504	604	705	806

PBW and Tidal Volume for Females

ARDSNet Studies

HEIGHT	PBW	4 ml	5 ml	6 ml	7 ml	8 ml
4' 0" (48)	22.4	90	112	134	157	179
4' 1" (49)	24.7	99	124	148	173	198
4' 2" (50)	27	108	135	162	189	216
4' 3" (51)	29.3	117	147	176	205	234
4' 4" (52)	31.6	126	158	190	221	253
4' 5" (53)	33.9	136	170	203	237	271
4' 6" (54)	36.2	145	181	217	253	290
4' 7" (55)	38.5	154	193	231	270	308
4' 8" (56)	40.8	163	204	245	286	326
4' 9" (57)	43.1	172	216	259	302	345
4' 10" (58)	45.4	182	227	272	318	363
4' 11" (59)	47.7	191	239	286	334	382
5' 0" (60)	50	200	250	300	350	400
5' 1" (61)	52.3	209	262	314	366	418
5' 2" (62)	54.6	218	273	328	382	437
5' 3" (63)	56.9	228	285	341	398	455
5' 4" (64)	59.2	237	296	355	414	474
5' 5" (65)	61.5	246	308	369	431	492
5' 6" (66)	63.8	255	319	383	447	510
5' 7" (67)	66.1	264	331	397	463	529
5' 8" (68)	68.4	274	342	410	479	547
5' 9" (69)	70.7	283	354	424	495	566
5' 10" (70)	73	292	365	438	511	584
5' 11" (71)	75.3	301	377	452	527	602
6' 0" (72)	77.6	310	388	466	543	621
6' 1" (73)	79.9	320	400	479	559	639
6' 2" (74)	82.2	329	411	493	575	658
6' 3" (75)	84.5	338	423	507	592	676
6' 4" (76)	86.8	347	434	521	608	694
6' 5" (77)	89.1	356	446	535	624	713
6' 6" (78)	91.4	366	457	548	640	731
6' 7" (79)	93.7	375	469	562	656	750
6' 8" (80)	96	384	480	576	672	768
6' 9" (81)	98.3	393	492	590	688	786
6' 10" (82)	100.6	402	503	604	704	805
6' 11" (83)	102.9	412	515	617	720	823
7' 0" (84)	105.2	421	526	631	736	842

PBW and Tidal Volume for Males

ARDSNet Studies

[26] May also be referred to as predicted body weight (PBW)
[27] NHLBI ARDS Nedtwork, 2014 (image)

As an aside, some people remember this formula for IBW as "inches over five feet" as shown below. Only problem with this is that it gets tricky if you have someone under five feet. But either way works:

$$IBW_{dudes}\ (kg) = 2.3(\text{every inch over 5'}) + 50$$
$$IBW_{chicks}\ (kg) = 2.3(\text{every inch over 5'}) + 45.5$$

When dealing with pediatric patients, our go-to reference ought to be the Broselow Tape. If that isn't available, we do have some formulas you can refer to:[28]

$$\text{Infant Weight (kg)} = 0.5(\text{age in months}) + 4$$
$$\text{Little Kiddo (1 – 4 years) Weight (kg)} = 2(\text{age in years} + 5)$$
$$\text{Big Kiddo (5 – 14 years) Weight (kg)} = 4(\text{age in years})$$

And note that the Broselow overlaps with the equation/chart above, so if we have a really small grownup or a big kiddo, we should still be able to get an IBW just fine. So no excuses! And very last thing: there are some apps out there that can help with this sort of thing, both for adults and for pediatrics.[29]

[28] Graves & friends, 2014 – There are lots of formulas out there, but we went with recommendations from these guys based on this paper they did comparing different methods

[29] Critical-Medical Guide & Pedi STAT – both are excellent resources to have on hand for quickly referencing relevant doses

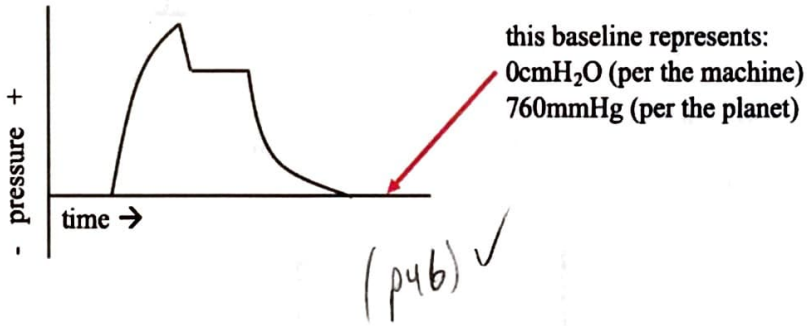
Return to Contents

Measuring Pressures

During mechanical ventilation we measure pressures in centimeters of water (cmH_2O). You may occasionally hear this pronounced as "sonnimeters of water" and know that a sonnimeter and a centimeter, in this context, are the same thing. So we have cmH_2O with mechanical ventilation, but we generally talk about ambient air pressures in other terms, such as mmHg, kPa, PSI, etc. We skimmed right on past this concept in a previous section when we said that 1mmHg is about $1.4cmH_20$ (this was when we talking about the fact that a normal negative pressure, spontaneous breath only talks -1mmHg of "sack" while a typical positive pressure breath via machine takes 15-25cmH_20 to move an equivalent amount of air), but let's now put it all down in a quick chart just to clear the water (or air):[30]

	ATM	PSI	kPa	mmHg	cmH_2O
ATM	1	14.7	101.3	760	1033
PSI	0.068	1	6.89	51.7	70.3
kPa	0.0098	0.145	1	7.5	10.2
mmHg (Torr)	0.0013	0.019	0.133	1	1.36
cmH_2O	0.00097	0.014	0.098	0.736	1

Also note that we assume that ambient pressure as it relates to airway/vent stuff is zero; so while true atmospheric pressure at sea level is 760mmHg (1 ATM), we call it $0cmH_2O$ to make things easier.[31] And then we have a way to represent breaths we give as waveforms showing pressure as a function of time with this new zero point (representing atmospheric pressure) as the baseline. For now we are going to ignore PEEP (since we haven't discussed that yet); we also don't have to worry about the specific components of the waveform – all those things will be discussed later on:



[30] We built this chart by Googling conversions for these values…

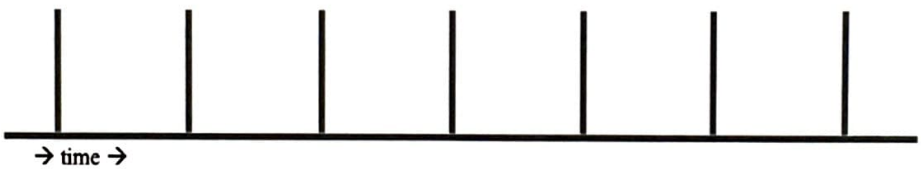
[31] Yartsev, 2019 – Scroll down to the section called "Airway Pressure" for some fun (and likely useless) trivia on why we measure/label pressures the way we do

Modes of Ventilation

This next section discusses how we organize the delivery of breaths to a patient. We've distinguished this concept of "mode" with that of "control" (see next section) in order to make things easier to conceptualize, but the terms sometimes get used with a bit of overlap. It helps us to think of mode as the overall pattern or organization and control as the specific way we choose to deliver breaths, ~~but we may see those ideas represented differently elsewhere~~. Now that we've clarified that distinction, we'll confuse it a bit more by starting our discuss of modes with one that includes the term "control" in the title. ~~We recognize that it gets a bit complicated, but hopefully it'll all make sense soon~~!

Controlled Mandatory Ventilation

Plain old control ventilation or controlled mandatory ventilation (CMV) is a mode of ventilation that isn't utilized much these days and doesn't exist as an option on many transport vents,[32] but it helps as a starting point to understand the other modes. In this mode we dictate how often we want to give breaths and how much of a breath to give on each of those instances and we ignore whatever the patient does ~~in response to that~~. Seems OK for patients with no inherent respiratory effort, but it can pose problems with those who do have some respiratory effort that doesn't quite mesh up with what the machine wants to do. To make this clear, let's assume a hypothetical timeline running left to right over an arbitrary amount of time with black hashes to represent machine-delivered breaths:



Now let's discuss what happens when the patient tries to breathe during this underlaying delivery scheme in each of these cases: more or less in the middle of two machine breaths (green), just after a machine breath (yellow), and just prior to a machine breath (red):

[32] That said, we can ~~generally~~ adjust settings in ~~either AC or SIMV~~ to ventilate the patient as if they were in CMV – it's just not a default option because we assume we want to support patient effort to breathe

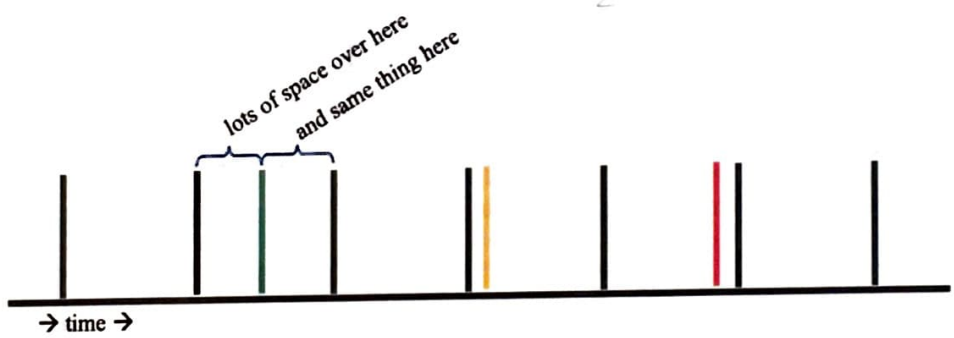
Return to Contents

In the green situation, the patient is free to take a breath if (s)he can and the machine-delivered breaths are likely to be unaffected. That said, the machine doesn't make any effort to facilitate the green breath, it just passively observes the patient struggling to breath. In the yellow and red situations the patient breaths and machine breaths can interfere with one another leading to discomfort, less effective air delivery, and possible damage due to increased pressures. None of this is of benefit to the patient, so the idea moving forward is that we need a strategy that works alongside the patient and helps meet an expressed need. Synching the machine with the patient improves comfort, conserves resources, facilitates recovery, reduce negative effects of positive pressure ventilation, and gives us more control over the management of the patient.

Assist Control (AC)

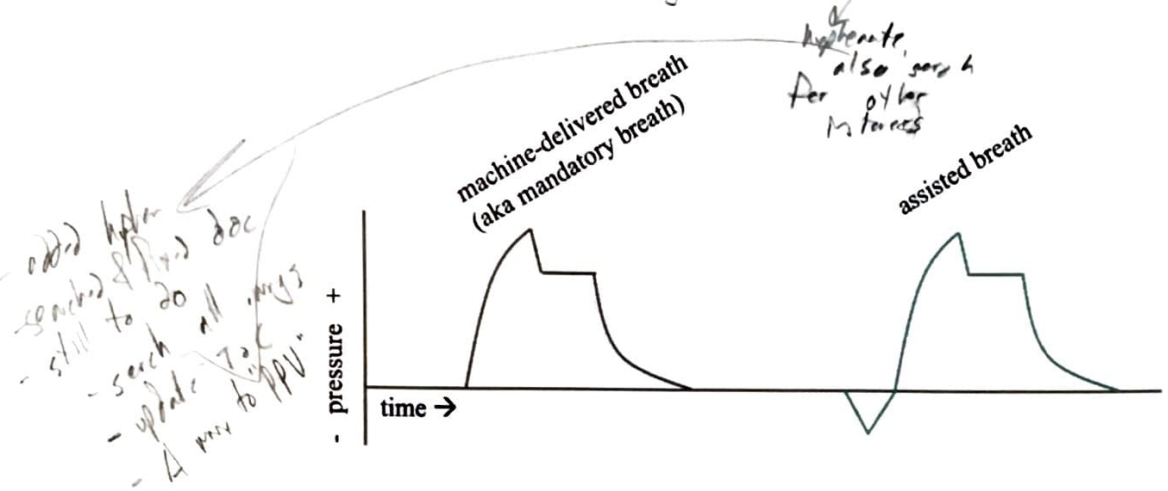
AC ventilation is a mode that augments a patient's spontaneous respiratory effort by delivering a preset amount of air when inspiratory effort is detected.[33] In the case with the green, yellow, and red patient-triggered breaths, the machine would recognize that the patient is trying to breath and then respond by giving a full breath on each of those occasions. The obvious advantage here is that the patient's expressed need for more breaths per unit time would be met. There is, however, a difference in how each of those breaths gets actualized.

With the green breath, there is space (in time) on either side of the breath, so the machine can assist the green breath without affecting other breaths in proximity ~~to the patient effort~~:



[33] A complete discussion of **Triggers** and how all that works is deferred until later on

The difference between those breaths can be represented via those pressure-over-time waveforms that we mentioned before. Note the dip in pressure at the start of the second waveform as the patient breathes in – this is the effort that gets sensed by the machine and then a full positive pressure breath is then given:[34]

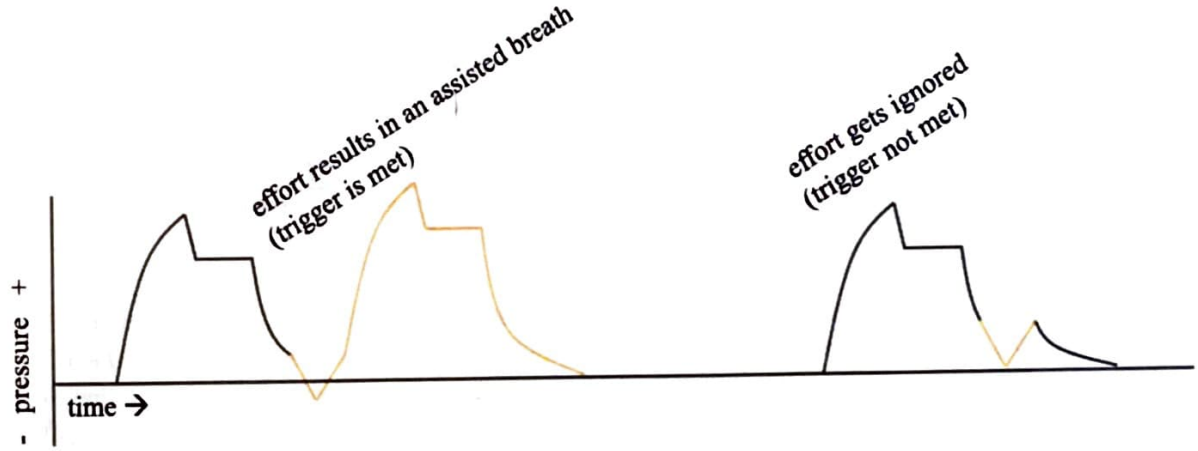


The ideal AC situation might look something like this where the patient's need for more breaths are met and that need, in the form of inspiratory effort or "pull," doesn't interfere or overlap with the scheduled breaths:

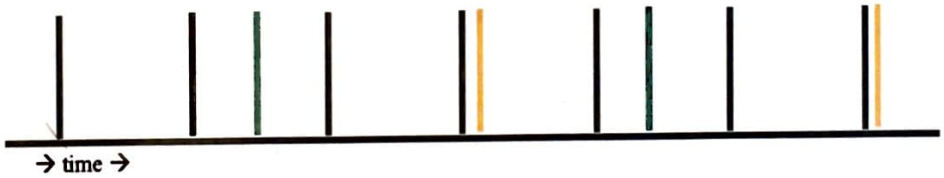
[34] Now this graphic makes it seem as if a pressure change detected by the machine leads to an assisted breath; while that could potentially be the case, the more common situation is a flow trigger; regardless of the trigger, however, the drop is pressure as shown in the graphic would occur in either case

Return to Contents

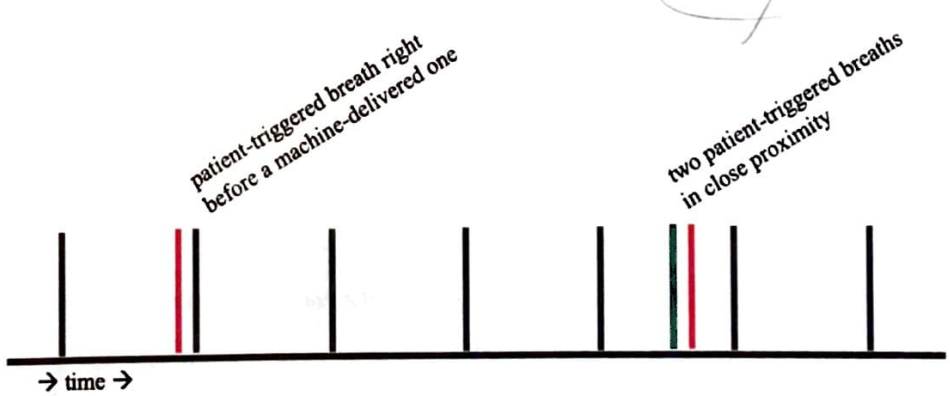
Moving forward, ~~however,~~ we have the proximity of breaths to consider. In the case of the yellow patient effort, the machine breath occurs just prior and, if ~~airway pressures haven~~'t had time to settle back to baseline, the breath may get missed or ignored. Now this depends on how the machine is set up and we can generalize it by saying that the further along the breath is or the closer the ~~pressure~~ breath has returned to baseline makes it more likely that the breath will "catch" and result in that full delivery. There are two possible outcomes: one in which the trigger results in an assisted breath and one in which the trigger does not result in a breath and the efficacy of the machine-delivered breath is simply altered somewhat:



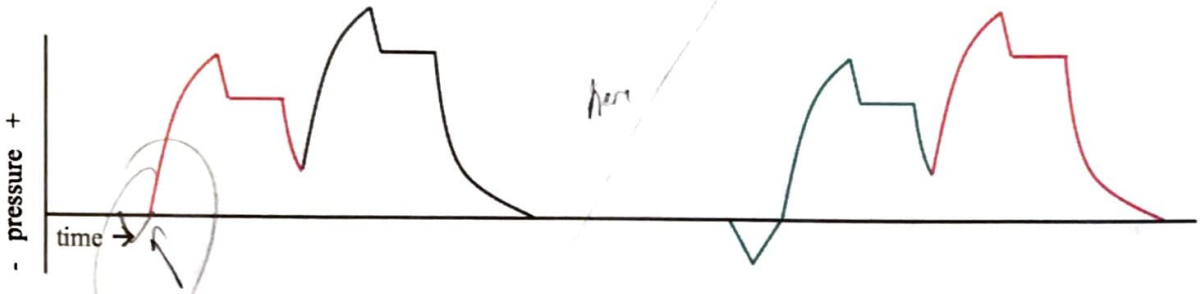
These yellow-effort situations are mostly safe for the patient, but may cause some issues related to slight higher pressures (left side, note the drift of maximum height on waveform) or discomfort (right side, due to an expressed need that goes unaddressed). That said, a combination of green and yellow effort is just fine for our patients in AC mode and allows the machine to adapt to what the patient wants in real time:



The issues with AC mode begin when we get those red-effort situations in which a patient-triggered breath precedes another breath. That other breath can be either a machine-delivered breath (as shown in the initial graphic) or another patient-triggered one (as in a sequence of patient ~~triggers~~ efforts in rapid succession):



If two breaths like this happen in close proximity, we run the risk that the first breath may not have time to cycle through before the next is delivered; we ~~might~~ get a breath on top of another or "breath stacking." This can increase pressure in the system and cause a complication known as AutoPEEP in which the pressure in the system doesn't get back to baseline before we add on another breath. We will discuss this further on down the line, but note that this is the primary drawback to the assist control mode. And here's ~~another way to~~ draw it out:

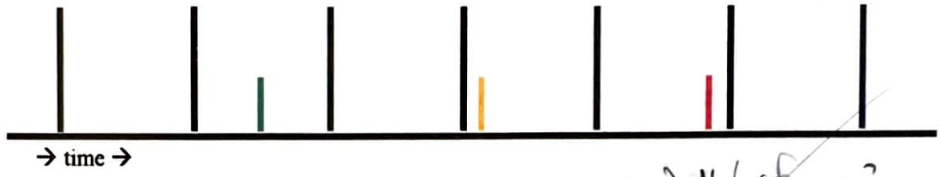


To summarize, AC mode machine-delivers breaths at a set rate and will supplement that with full breaths whenever a patient effort meets the trigger threshold. Upsides to this are that the increased needs of the patient are readily met, downsides are the risk for increased pressures and a move away from baseline (AutoPEEP, which we will get to later). As a general rule: anytime you have someone in AC mode you need to be vigilant and monitor both airway pressures and AutoPEEP.

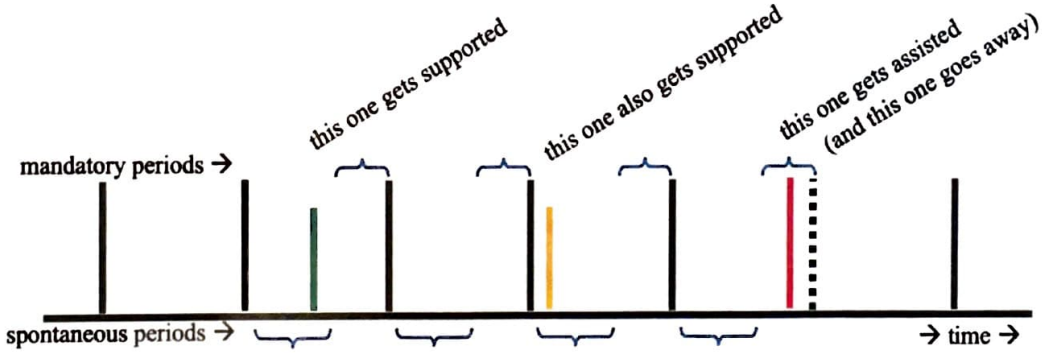
Return to Contents

Synchronized Intermittent Mandatory Ventilation (SIMV)

SIMV is ~~alternative~~ mode of ventilation that also seeks to mitigate the shortcomings of CMV. SIMV starts with idea of mandatory breaths or a guaranteed number of breaths to be given per minute. It then will support breaths taken in between these mandatory breaths. Furthermore, SIMV recognizes when patient effort is made in close proximity to a pre-scheduled mandatory breath and assists that effort in a way similar to how breaths were assisted in AC mode. Now there are more difference between these various **Types of Breaths** and we'll get back to that eventually, but let's focus on the timing aspect of SIMV first. ~~Let's go back~~ to our original idea:



SIMV's method for determining how to handle the instances of patient effort is to break the timeline into two alternating categories: mandatory and spontaneous periods. If a patient effort happens within a spontaneous period, it gets supported and that effort is facilitated by the machine in a manner that we will discuss real soon;[35] if an effort occurs within a mandatory period it gets assisted, a full breath is delivered, and the breath that had been planned for that mandatory period gets skipped:



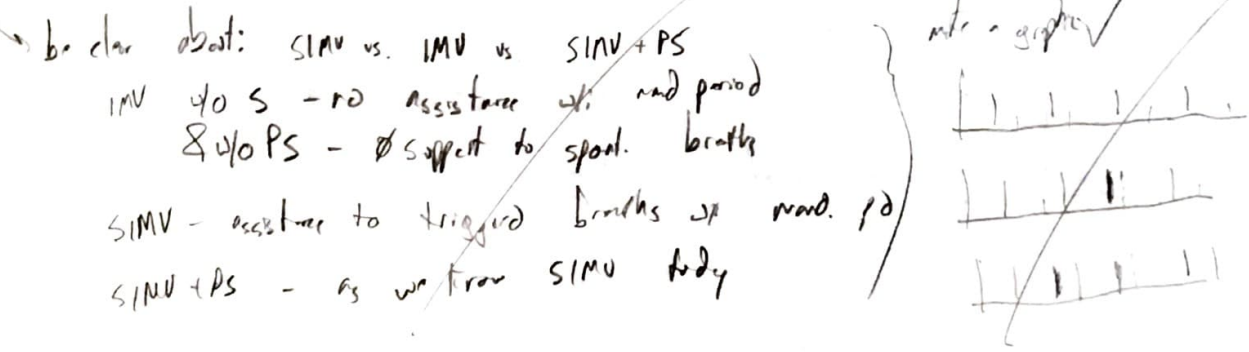
[35] Ollie, 2015 - This video demonstrates the idea in another way by way of a discussion about "IMV" ventilation (versus SIMV)

As far as the difference between supported breaths (green and yellow) and assisted breaths (red), the idea is that supported breaths only get a little bit of help from the machine and the assisted ~~(red)~~ breaths are fully facilitated by the machine to a target amount of air, just as in AC mode. Supported breaths are always supported via pressure, which basically helps the patient draw a breath a little bit easier;[36] assisted breaths are carried out to meet specific goals by the machine based on settings we input and can be either volume-targeted or pressure-targeted (which we will expand on in the next section). The practical difference is that pressure support (PS) breaths will give us a variable result that depends largely on the patient's contribution to that specific breath, while assisted breaths are more predictable.

At the risk of getting ahead of ourselves, PS breaths are often expected to be less than or smaller than mandatory and assisted breaths (in terms of volume of air). While it may make sense to titrate PS up so that supported breaths match the other ones in this regard, it isn't quite as simple as increasing the PS value on the machine. That said, there is no reason that the volume of air in a PS breath should be less than the other ones, it's more an issue that it often just happens to turn out that way because the nitty gritty details as to how these different **Types of Breaths** are brought into existence by the machine.[37]

And a few more things about SIMV mode. It originally came onto the scene as IMV mode (minus the "S") and did not include PS to breaths during that spontaneous period. You may see SIMV as we described it notated as SIMV + PS to better describe that difference. Another historical tidbit is that the mode was popularized as a method of weaning or getting someone transitioned from vent life to spontaneous breathing after an illness or intervention – the efficacy of SIMV for weaning has since been shown to be inferior to other methods. The result of all of this is that content on SIMV is often confounded by stuff that more accurately relates to IMV and that draws conclusions from a concept (weaning) that doesn't much matter in the transport setting.

To summarize, SIMV is a mode that both supports patient effort to breathe via PS breaths and avoids breath stacking by not delivering breaths in close proximity to others. ~~More specifically, SIMV~~ avoids the problem of AutoPEEP that we discussed in regard to AC mode. On the other hand, SIMV has been associated with ventilator asynchrony and can be harder to both conceptualize and monitor than AC ventilation (due to different the different **Types of Breaths** involved). In addition, SIMV is less able to meet a patient's expressed need for more air, as supported breaths are less predictable than assisted ones.



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[36] Loderserto, 2018 – This series provides alternative explanations to this concept as well as many of the others we will discuss along the way

[37] Hess, 2005 – That said, the primary function of pressure support breaths is to relieve workload required by the patient and facilitate intrinsic respiratory effort, this is fundamentally different that a pressure control breath (discussed soon) in which we utilize pressure to deliver a breath regardless of patient effort; this article discusses how additional PS may not correlate as expected with an increase in TV due to additional factors on the patient end of the equation and the breath is delivered

Return to Contents

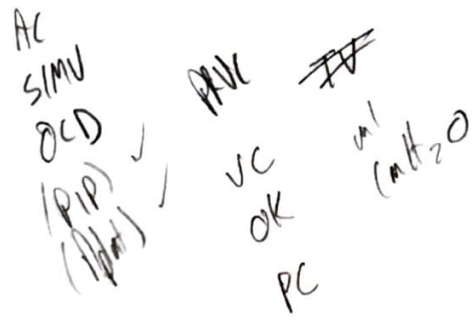
And Beyond…

Now that we know about both AC and SIMV modes, the decision becomes which mode to use for a given patient. While many folks have their preference and we could argue one over the other all day long until we are both blue in the face, the bottom line is that either mode could work for just about any patient type. Here's the general strategy we'll recommend (and we will revisit this idea again at the very end when we talk about building out a ~~protocol~~ guideline and putting it all together): if we have a patient already on the vent and all is well, just stick with whichever mode they are working with; if we are starting from scratch or reworking the settings altogether, try what our machine defaults to and then change modes if we need to down the line. That's about as simple as you can make it. All that said, there are cases in which one mode may be preferred over another and we will talk about those as they come up.

Control of Ventilation

We already discussed the first big choice in vent management: which mode (AC vs SIMV) to utilize for our patient. The next decision is to choose whether we want to control volume or pressure. If we choose to control volume, ~~airway~~ pressure will function as the dependent variable (i.e. we won't be able to directly control it); if we choose to control pressure, volume will function as the dependent variable. There is no right or wrong answer to this dilemma, but the general trend is that we use volume control in most cases and pressure control with pediatrics[38] or when they are especially concerned about airway pressures. Not saying this is the best decision, just saying that's how it's been done.

The reason for this is twofold. First (and arguably most relevant), the machines tend to default to volume control mode unless you do something to intentionally get out of it (such as choose "infant" on the patient type category). Second, volume control is a bit easier for some folks to wrap their heads around – it's a little more intuitive to think about set volumes and resultant pressures than it is the other way. But as we said above, there is no right or wrong; we can just as effectively and safely ventilate a baby in volume control as we can an adult in pressure control (even though this is contrary to what we normally do), as long as we know the underlaying concepts and keep an eye on all the important things along the way!

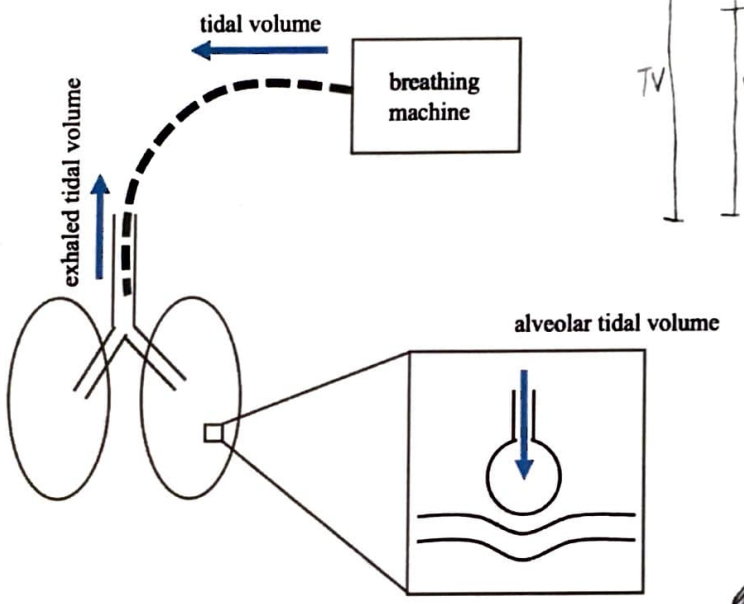


[38] Kneyber & friends, 2017 – Note that even the people who make the rules on pediatric ventilation don't endorse one method of control over another…

Return to Contents

Volume

In volume control (VC) ventilation we choose how much volume we want to push down the circuit with each breath delivered. This tidal volume that we put in goes to the lungs, does its thing at the alveolar level, and then gets exhaled out of the circuit. When we say "tidal volume" we are referring to the air going into the system from the machine; those other two concepts (alveolar tidal volume and exhaled tidal volume) vary from that value due to a number of different factors. Let's see how this looks in a graphic and then we'll hash out ~~a few~~ details ~~about all these terms~~:



Even though the actual definition of "tidal volume" is the amount of air moved during exhalation, we have a specific term, in this context, for exhaled tidal volume. And we need another term for the value we dial in to the machine, so it helps us to ignore the literal definition and break those two concepts up as we have just shown. To review what we discussed previously about dead space, ~~the~~ alveolar tidal volume is normally exhaled tidal volume minus anatomic dead space (which is about 2ml/kg or 1/3 of tidal volume), so about two thirds of what we push into the system.

Now what about those other kinds of dead space; mechanical and alveolar? As for mechanical dead space: this value doesn't actually alter volumes, rather it alters partial pressures of gasses within the volumes of air in question. Which means we don't have to worry about it for now. For this discussion, let's keep it simple: we already know that we want to limit mechanical dead space as much as possible, but in the context of tidal volumes and the physical amount of air moved during each breath we can ignore it. Alveolar dead space, on the other hand, can only partially be ignored. We can ignore calculating a value for alveolar dead space, but we need to take actions to address it just in case (as we discussed before and will discuss again).

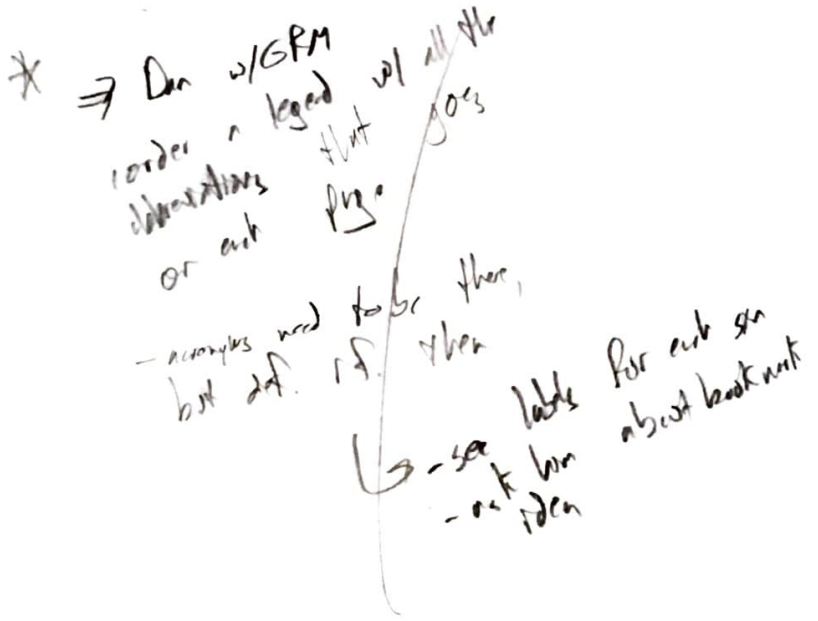
And what about that flexibility or stretch we mentioned in our discussion of dead space? We said then that the vent circuit has some give to it that can confound our approximation of the amount of air delivered. We factor that out by assessing volume by looking at exhaled tidal volume. To say it another way, when we want to know how much air we are giving to our patient, we look at the air leaving the lungs (that actual, textbook definition of tidal volume) and not at the air we push in from the machine, as there can be a notable difference between the two. In the event that exhaled tidal volume is not available on a particular machine, we do need to consider this difference and ensure that ventilation is adequate.[39]

[39] And as we already said, this is mostly a concern with pediatric patients in volume control – see **Appendix** for more on that

To summarize all of this: VC ventilation allows us to control the amount of air we put into the vent circuit. While we mostly care about exhaled tidal volume and alveolar tidal volume, dialing in a tidal volume on the machine is the closest we can get to controlling those values. Tidal volume is a precursor to both exhaled tidal volume and alveolar tidal volume and we should always make adjustments to the system using exhaled tidal volume to eliminate the effect of dead space when we can. While there isn't really a need to quantify dead space or calculate it out, it is important to keep in mind. This is particularly true when ventilating in volume control with a machine that doesn't measure exhaled volumes.

Next bit: when we dial in a tidal volume and move that air through the circuit to the lungs and alveoli, the result is an increase in pressure that is dependent on the amount of air going in and how that air moves. For now, we will defer a discussion of how we describe this air movement (i.e. its speed or flow and all that), just know that pushing a preset volume in means that pressure changes happen as a result of that air movement and that certain pressure changes (i.e. too much air too fast) can cause damage to the alveoli. At a certain point we can overinflate the alveoli, resulting in what we call barotrauma, and we for sure want to avoid that.

So the way to do this with VC ventilation is to monitor your airway pressures and adjust the volume input to avoid causing damage.[40] We will get to the specifics as to how we do that eventually, for now it's OK to leave it as so: in VC ventilation we control the amount of air going into the circuit at the expense of control over resultant pressures; that said, we always need to monitor airway pressures during VC ventilation in order to avoid causing damage to the alveoli. In addition, VC ventilation lends itself to an overestimation of alveolar tidal volume if we forget to factor in dead space.

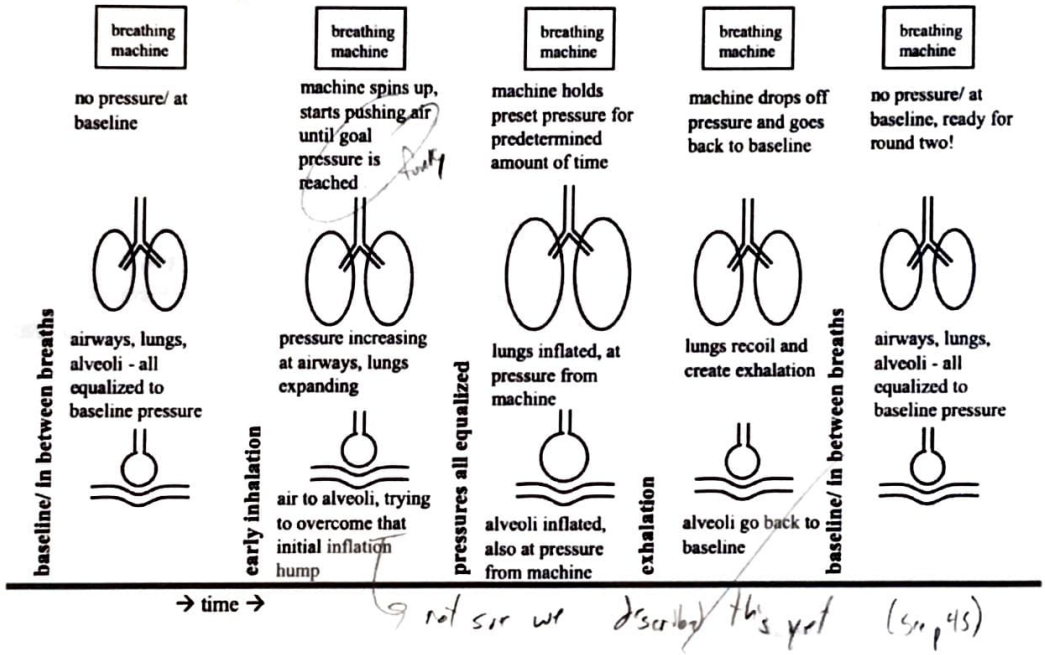


[40] While there are other parameters that we can adjust to avoid higher pressures (such as I-time and flow, if available), it helps to simplify things this way: more volume = more pressure

Return to Contents

Pressure

In the other corner of the arena we have pressure control (PC) ventilation.[41] In this mode, a breath happens as so: we have a dialed-in pressure, the machine spins up to maintain that pressure, the air all the way from machine to alveoli equalize to this pressure for a set time, then the breath cycles off and we go back to baseline. Because our input here is pressure, volume becomes our dependent variable (exhaled tidal volume,[42] to be exact; or textbook-defined tidal volume for the OCDers out there). Let's draw it out and see if we can make it a little ~~clearer~~:



In the fourth column, we see that recoil of the lungs (a passive exhalation) occurs when the pressure that had been keeping those lungs inflated drops off. This volume of air that gets pushed out of the circuit as the lungs "fall" back to normal is our exhaled tidal volume, which we then have to actively observe to make sure it meets the goal we have in mind for what volume this patient ought to be getting with each pressure breath we deliver. If this exhaled tidal volume is not what we want it to be, then we adjust the pressure in the system to get closer to our goal (more pressure means more volume, less pressure means less volume).

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[41] Meeks, 2018; Yartsev, 2019 - And we phrased it this way because there is much debate out there in vent world as to which strategy (volume or pressure) is superior; see referenced podcast and article for more information

[42] And if a machine is capable of pressure control ventilation it will most surely have a mechanism for measuring exhaled tidal volume; in the previous section we noted that some machines don't give us this value, but those machines tend to do ~~volume control~~ VC ventilation only

One thing worth pointing out here is that in PC ventilation we don't have to bother with considering that flexibility or stretch that we discussed when we talked about dead space (i.e. the compliance of the vent circuit), as the only way we have to measure volume is via exhaled tidal volume or what the patient breathes out (which is downstream of all that flexing/stretching nonsense). We do still need to consider anatomic and alveolar dead space, just as we did with VC, but the stretch factor we introduce in our circuit is eliminated. This is a big advantage of PC ventilation with small patients: forgetting to factor in 10ml (arbitrary number) in an adult is no big deal, forgetting to do so for a baby with small tidal volumes is huge. We'll discuss more later,[43] but just know that this is one advantage of pressure control.

Another advantage of PC is that we avoid the risk of over-inflation or high pressures at the alveolar level. The highest pressure those alveoli will see is whatever value we preprogram into the machine.[44] So as long as we follow some basic guidelines as to what a safe pressure is, there's not much risk of harm or barotrauma. The downside is that we don't have as good of control (compared to VC) over the amount or volume of air that we are putting into the system; instead we have to continually monitor exhaled tidal volumes and adjust to our goals.[45]

To summarize: in PC ventilation we control the pressure put into the system at the expense of control over resultant volumes; that said, we always need to monitor those volumes when we have a patient in PC mode in order to avoid hyper or hypoventilation. In addition, PC ventilation makes it a little more difficult to control ventilation (as opposed to oxygenation, ~~more or less referring to keeping the $EtCO_2$ within range~~ – again, one of those things we will get to later on), due to the breath to breath variability in volumes. The big advantage of PC ventilation is that we avoid the high pressures that can result from VC mode.[46]

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[43] In the **Appendix**

[44] For the most part this is true, but there are some exceptions that we'll chat about later in the section called **PIP and Pplat in Pressure Control?**

[45] Ashworth & friends, 2018 – What we've said here is a bit of a simplification, but it serves our purpose for now – refer to this article for a much more detailed discussion of how we can work towards our ventilation goals in PC ventilation

[46] There are more advantages (such as how PC breaths differ from VC ones in regard to flow waveforms), but we'll get to that stuff later on in **Types of Breaths**

Return to Contents

Pressure Regulated Volume Control

Pressure regulated volume control (PRVC) is one attempt to get at the best of both worlds when it comes to this volume vs. pressure conundrum. In this mode we dial in a goal for tidal volume and put a cap on pressure, then the machine tries to give breaths to the goal volume using the lowest possible pressure and without exceeding the max we have set. The machine makes adjustments to how it delivers each breath by looking at previous breaths and then adjusts delivery to add or take away volume working towards the preset TV goal. In the event that it can't reach the goal volume without exceeding the upper pressure limit, volume is sacrificed - think of the "pressure regulated" part as a hard stop.

Let's visualize this over a few breaths to see what it would look like:

goal TV: 400ml
pressure cap: 30cmH_2O

breath one
initial breath
25cmH_2O
300ml

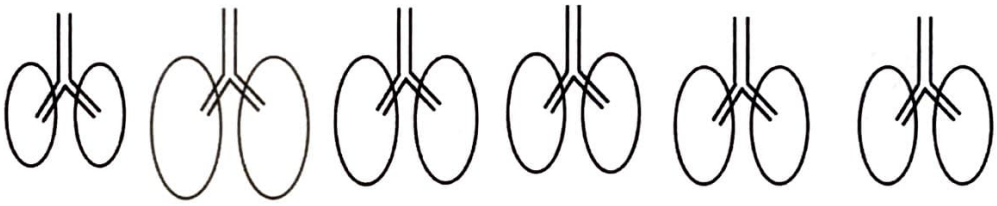
breath two
more pressure
30cmH_2O
450ml

breath three
a little less pressure
28cmH_2O
400ml

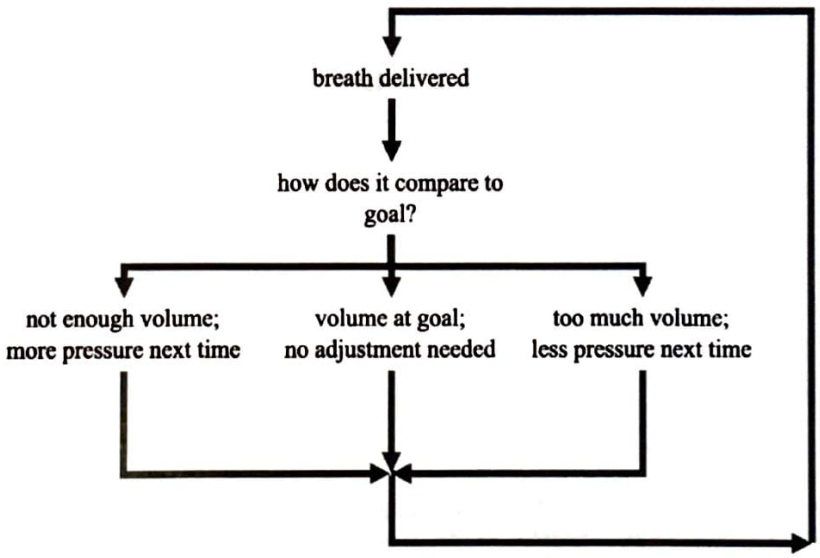
breath four
same pressure
28cmH_2O
375ml

breath five
a little more pressure
29cmH_2O
400ml

breath six
same pressure
29cmH_2O
400ml



If it helps, we can also think of this in an algorithm-style fashion where we decide where each breath ends up in relation to our goal and then adjust the subsequent breath in a cyclical manner:



This mechanism of decision-making one breath at a time doesn't quite describe the process accurately, but it gives the right idea. In reality the machine looks back at the last few breaths (varies by machine) and builds a small data set from which it decides how to deliver the next breath. So the system is actually a little more refined that our crude representation.

To flush out a few more details on this PRVC concept, let's look at another example of a few consecutive breaths. In this example something is causing an increase in pressure to the system, therefore breaths basically get cut short. The result of this would be a drop in minute volume or air moved per unit time.[47] It's important to keep this in mind with PRVC, as we can inadvertently drop minute volume pretty significantly in an effort to avoid high pressures:

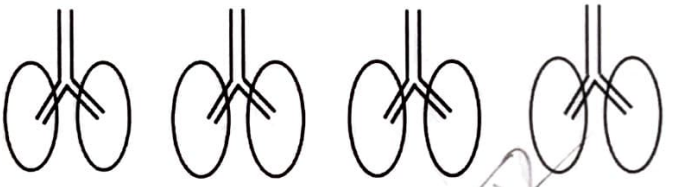
goal TV: 400ml
pressure cap: 30cmH_2O

breath one
initial breath
25cmH_2O
300ml

breath two
more pressure
30cmH_2O
325ml

breath three
can't give more
30cmH_2O
325ml

breath four
same pressure
30cmH_2O
325ml



and on and with the result that MV goes down (compared to our goal)

A few more things about PRVC: "pressure cap" is a make-believe term – the machine most often uses 5cmH_20 less than the set high-pressure limit for this value.[48] There are also restrictions on how much variation occurs from one breath to the next; to say it another way, the machine won't make crazy, drastic changes in response to one or two funky breaths. Another thing: the machine has a system to get this whole process started by giving "test breaths" via different methods when it first gets set up – no need to worry about that here, that's homework for us depending on the system and machine we use in the field.[49] Along that same idea, the machine doesn't actually know how much air (i.e. tidal volume) it gives with each breath until after the fact when it sees the exhaled tidal volume, that's why it can overshoot the goal. Last thing: PRVC is good when we are worried about barotrauma or giving too much pressure, but it is important to make sure we keep an eye on minute volume and match it to our calculated goal.

[47] Discussed in much more detail in just a few sections! (**Minute Volume**)
[48] And limits are discussed later when we get to **Alarms**
[49] Maher, 2019 – short video that describes this and gives a brief overview of PRVC (and it is just one video is a large series, so take a look at the rest of his content for more)

Return to Contents

Vent Parameters, Round One

Next step on our journey is to explain fully the ins and outs of some of the terms we use to describe different aspects of ventilation. Some of these have been mentioned already (and a few discussed in detail), but most of the complete explanations have been left out up until this point in an effort to better organize thoughts in a linear, stepwise fashion. If it helps to go back to previous sections after this discussion, go for it. Also, keep in mind that this is not an exhaustive list of all the terms, these are just the basics (~~with which you may have already been familiar with prior to getting into the manual~~), and more will come later.

Tidal Volume

Tidal volume per the textbooks is the amount or volume of air exhaled in a given breath. As previously discussed, it sometimes helps to break this concept up in to two distinct terms: tidal volume and exhaled tidal volume. Tidal volume, in this way of thinking, would be the volume of air we put into the system, while exhaled tidal volume would be the volume of air that comes out of the system. Tidal volume may be notated as TV or VT, exhaled tidal volume is notated at VTe.[50]

Tidal volume varies by the size of the patient and the normal range is 6-8ml/kg IBW. Recall the discussion we already had about ideal body weight (IBW) and the idea that lung size is best correlated to height. Also recognize that 6-8ml/kg IBW is just a framework from which we start when determining our initial settings and that tidal volume can range from 4-12ml/kg IBW, depending on the specific situation that we are up against. Enough on that for now though, we will talk further on that when we get into ventilator strategies.[51]

[50] You often see Vt and Vte instead of VT and VTe, but we've opted to do it our way so that there is consistent notation throughout – whenever you see a little "e" after a term it will refer to the "exhaled" version of whatever parameter it is attached to (i.e. MVe is exhaled minute volume, something we'll talk about shortly)

[51] Davies & friends, 2016 – And these guys offer a much more in-depth discussion of this general idea

Rate

Rate ~~is equivalent to the idea of~~ respiratory rate ~~and~~ describes how many breaths are delivered and/ or taken in one minute of time. It is also known as frequency and may be abbreviated by "f." You also may see rate abbreviated as "RR" to stand for respiratory rate.[52] Normal parameters vary by age, but the typical adult rate is 12-20 and pediatric rates are as outlined on your Broselow Tape or by this chart from the PALS Manual:[53]

American Heart Association. life is why

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

PALS

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)‡
Birth (12 h, <1000 g)	39-59	16-36	28-42‡
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	89-112	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

[52] While respiratory rate may semantically differ from frequency (i.e. patient's intrinsic rate versus overall rate), we've decided to keep it simple here and simply use RR to describe frequency in a general sense

[53] American Heart Association, 2016 (image) - As a quick disclaimer: these normal respiratory rates as outlined in PALS are not intended to be used for determining vent settings, rather they are outlined as such to identify normal and abnormal findings in an assessment; with that said, most transport clinicians are familiar with this reference and have ready access to it, so it makes sense to build our concept of vent management from a known source rather than introduce new values and numbers with which we may not be familiar

Return to Contents

For the detail-oriented people out there, there are some data points missing from this PALS chart. One strategy would be to guess based on available data (i.e. no listed rate for a 9-year-old, but you could assume a value that falls in between the School-aged Child range and that for Adolescents). Other option is to use this chart we've put together based on the existing data in the PALS Chart:[54]

Age Description	Age (yrs)	RR
Infant	.083 (1 month) – 1	30 – 53
Toddler	1 – 2	22 – 37
Preschooler	3 – 5	22 – 28
School-aged Child	6 – 7	18 – 25
Big Kiddos	8 – 9	17 – 25
Preadolescent	10 – 12	14 – 23
Adolescent	12 – 15	12 – 20
Adult	16 and up	12 – 20

Last thing: there are times that we set rate above or below what might be considered normal for the patient's age, but we'll get to those specifics when we discuss vent strategy for different situations later on.

[54] See **Appendix** for a discussion of how this chart was created

Minute Volume

Minute volume, also known as minute ventilation, is the amount of air moved in one full minute. It is the product of tidal volume and rate:

$$MV = RR \times TV$$

Minute volume/minute ventilation can be abbreviated as "MV" or "VE" and is the primary mechanism by which we control ventilation. We will discuss soon[55] how to manipulate both tidal volume and rate to address ventilation in just a bit, so don't worry about that for the moment. A normal MV for the adult patient is often cited at 4 – 8 liters per minute, but we prefer to use a weight-based calculation so that it applies to all patients sizes:[56]

MV = 100ml/kg (IBW) /min

As with rate or frequency, there are times that we use a different MV goal with specific patient types, but we will get to that later on. Last thing: just as with tidal volume, there can be different types of minute volume. "Minute volume" or "minute ventilation" typically describes what we dial in to the machine, then we tag "exhaled" on to either term (abbreviated MVe) to describe feedback the machine gives us about what the patient breathes out. Lastly there is alveolar minute ventilation (VA) which takes out anatomic dead space from the equation. While alveolar minute volume (another way of describing VA) is an important concept to consider, we base initial goals and calculations on MV or MVe and not on alveolar ventilation.[57]

Fraction of Inspired Oxygen

Fraction of inspired oxygen, or FiO_2, describes the amount of oxygen in the mix of gasses that we push into the patient's vent circuit when we give a breath. 100% oxygen would be an FiO_2 of 1.0; 21% oxygen or ambient air would be an FiO_2 of 0.21. Adjusting FiO_2 is often the easiest way we can address an oxygenation issue, but we'll discuss fixing things in just a little while. One thing worth mentioning at this point, however, is the idea that too much oxygen can be a bad thing.[58] While it may be tempting to dial the FiO_2 up to 100% on all patients, this isn't always warranted and can cause harm to our patients if they don't need it. At the same time, however, don't be skimpy: titrate FiO_2 to maintain an SpO_2[59] in the mid-to-high-90s. If there is good reason to suspect that SpO_2 isn't an appropriate measurement (such as with hemorrhage, CO exposure, etc.) or there is another greater worry (baby in the belly of mommy, traumatic brain injury, etc.), we can and should give 100%. And if we are ever in doubt, just give oxygen: most of the bad things take ~~a longer~~ time to cause damage and the risk of giving a little bit extra in transport likely outweighs the risk.

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[55] In our section on **Ventilation (~~and $EtCO_2$~~)**

[56] Weingart, 2010; Yartsev, 2019 – These guys cite a goal MV for the intubated patient as 120ml/kg/min and 70-110ml/kg/min, respectively; we've opted to go with 100ml/kg/min as a starting point due to ease of calculations and simplicity

[57] We do, however, make subsequent changes to address ventilation with these alveolar volumes in mind and we will get to that in **Ventilation (~~& CO_2~~)**

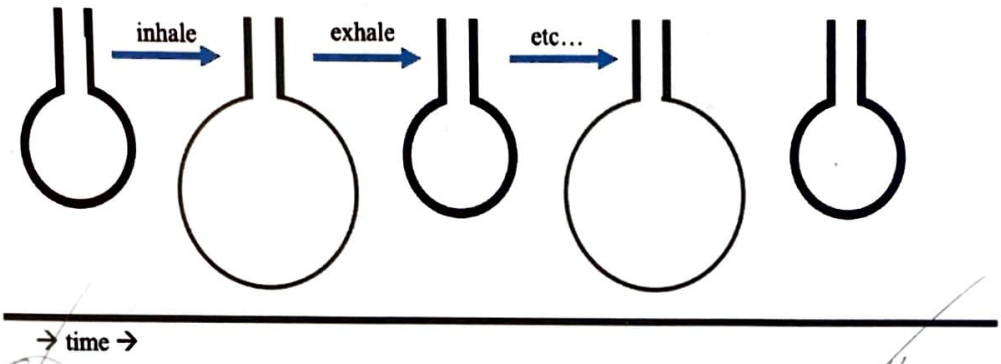
[58] Kallet & Branson, 2016 – Provides an excellent overview of both sides of the debate on whether or ~~not too much oxygen is a~~ thing

[59] And we will get into the details of SpO_2 in our section on **Oxygenation (~~and SpO_2~~)**

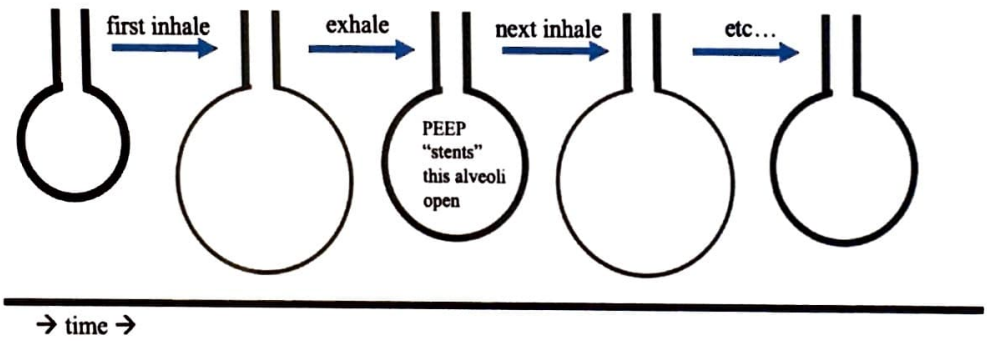
Return to Contents

Positive End-Expiratory Pressure (PEEP)

PEEP describes the positive pressure that remains in the alveoli at the end of expiration. And let's recognize that we basically explained a term using the words it's made up of, so we'll try it another way via a few steps. During mechanical ventilation we push air into the alveoli on inspiration, then that air moves out of the alveoli on expiration. We tend to conceptualize this (and have done so in all the sketches so far) as a net zero movement of air where the alveoli go from deflated to inflated and then back to deflated, as so:

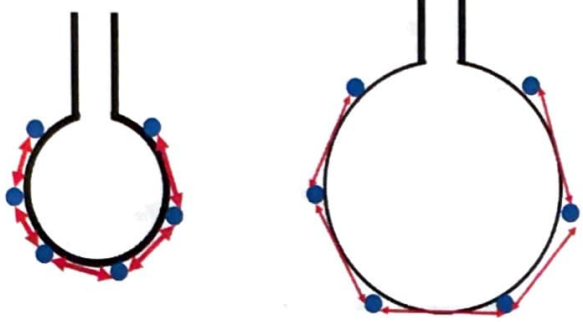


Now the truth is that we can put pressure into the alveoli and then leave ~~some of that pressure~~ there to hang out throughout exhalation (in the form of PEEP). So rather than the alveolar air sac deflating all the way back to its original size, it deflates only part way:



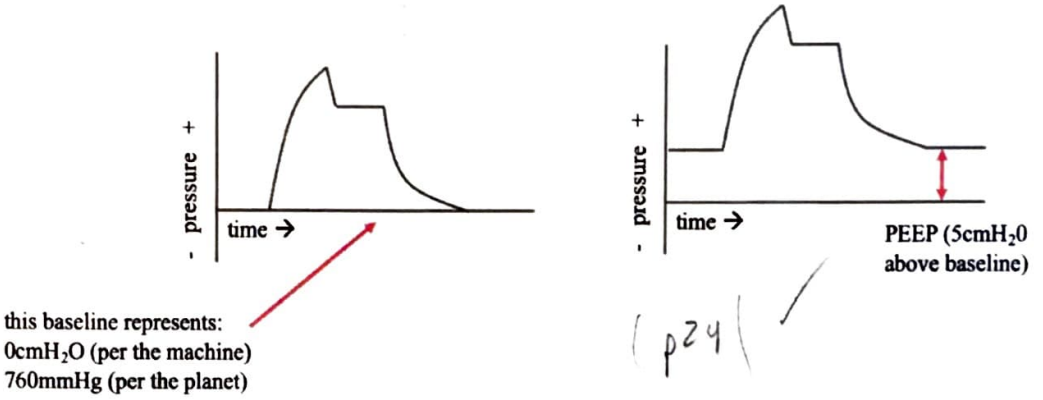
Recall our previous discussion of Alveolar Surface Area that the more inflated the alveoli are, the more they can participate in gas exchange. And this is due to both more surface area and a thinner membrane across which gas must diffuse. Next, add to that the idea that blood flow though the pulmonary capillary bed is continuous, it doesn't stop when inhalation stops. This means that pulmonary respiration or gas exchange across the alveolar membrane occurs throughout the respiratory cycle, both on inhale and exhale. PEEP helps facilitate this gas process on the exhalation side and then makes it easier to further maximize the effect during inhalation (i.e. a better starting point from which inhalation begins).

Another idea particularly relevant to this discussion of PEEP is that the "stenting" or opening-up of alveoli doesn't always happen in one breath as it's been depicted in the above drawing. Sometimes it takes time to get from a that left-most, deflated stage to a "recruited" or opened-up stage. Part of the reason for that is that there is fluid around the surface of the alveoli that resists expansion. Think of it as molecules on the alveolar surface that are holding hands with one another; as we increase size of the alveoli, we increase the distance between those hand-holders and make expansion easier:

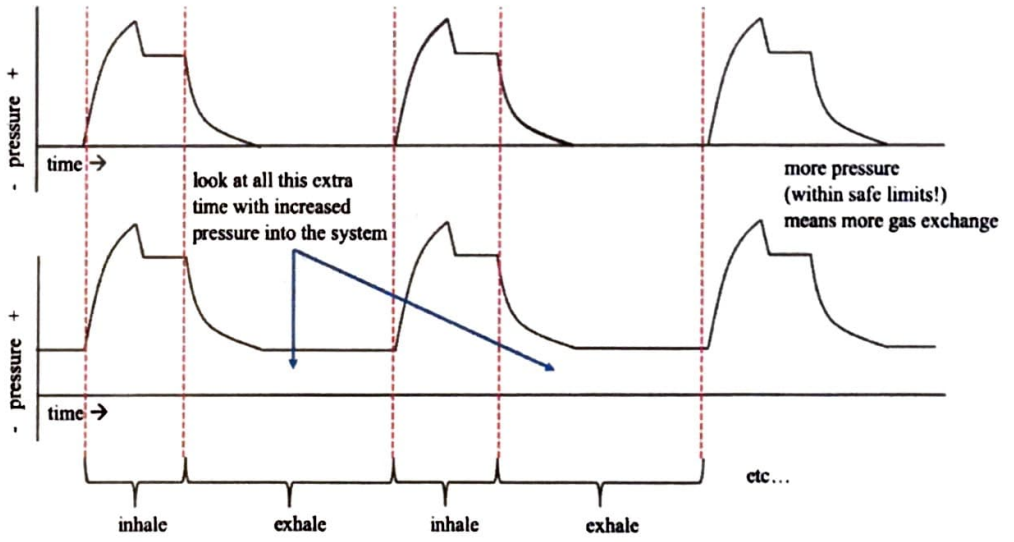


PEEP helps with this process by maintaining our progress along the way. As airway pressure increases on inhalation and the alveoli expand, PEEP essentially maintains that expansion on exhalation and prevents us from cycling back to that deflated, left-hand state in the above photo. An added benefit of this is that it reduces stress on the alveoli. Going from deflated to inflated to deflated to inflated and on and on can put stress on the alveoli; PEEP decreases the difference between those two states so that less net movement is required for each inhalation. We talk about this much more in the section on **Driving Pressure**, so no need for more detail at this point.

To summarize so far: PEEP is a residual pressure that we leave in the alveoli during exhalation to both maximize pulmonary respiration during exhalation and maintain recruitment of alveoli.[60] So now that we have that clarified, let's look a waveform representing pressure into the system as we deliver a breath. We've seen this image previously, but now we are going to add to it. The first breath is with no PEEP or zero PEEP or "ZEEP", the second one (right) is with 5cmH_20 worth of PEEP added in:



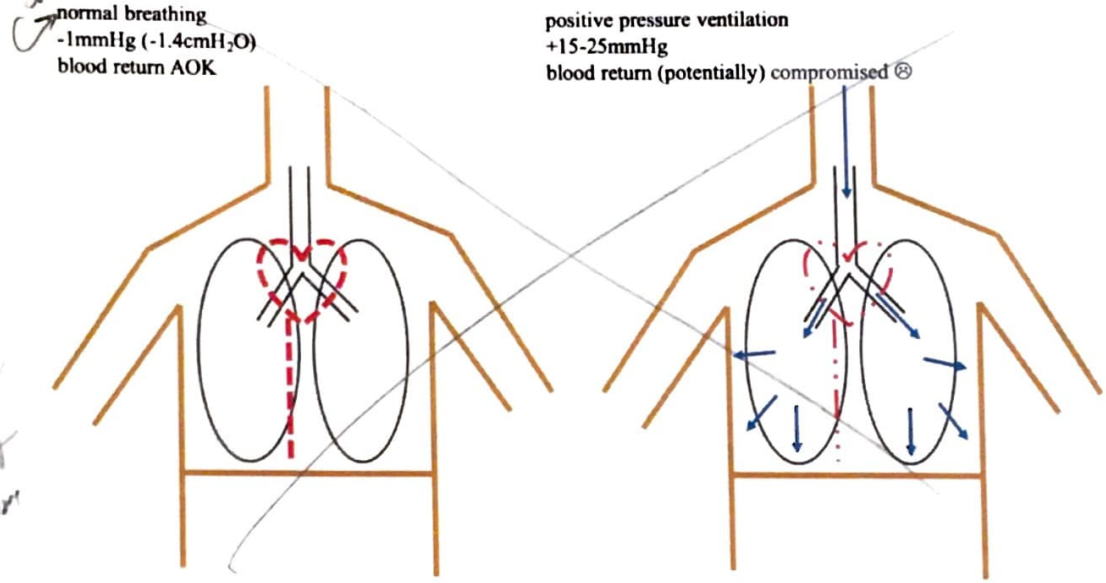
And to visualize this same idea over time, let's think of it this way:



Now this is not to say that gas exchange is nonexistent on exhalation in the first (no PEEP) case, just that it is augmented during the second one. There are also other mechanisms by which PEEP facilitates oxygenation, but those will come up shortly in the section on **Oxygenation ~~(and SpO2)~~**. The important thing to note for now is that PEEP basically acts to keep alveoli open during exhalation and that helps us utilize more lung volume while breathing for the patient.

[60] Kallet & Branson, 2016 – They explain that PEEP doesn't necessarily "open" the alveoli as we often hear it described, rather PEEP stents the alveoli open after inspiratory pressure changes (or recruitment maneuvers) open them up

Let's next take a look at downsides of PEEP. Most relevant one to mention is that PEEP can decrease blood return to the heart.[61] Recall from a previous discussion that any increase in intrathoracic pressure can impede blood flow back to the heart (~~and see image reproduced below~~). Because of this, normal PEEPs are less than $10cmH_20$. That said, we sometimes use PEEPs up to $20cmH_20$ in specific cases and we will talk about those later.



Other negative consequences of PEEP vary widely from things like worsening hypoxia and increased V/Q mismatch to decreased extra-thoracic organ function and decreased cerebral perfusion pressure.[62] That said, the important thing to ~~note~~ is that these negative effects typically manifest when the application of PEEP is taken beyond the level of therapeutic benefit. To phrase it a different way: use PEEP when needed, but don't assume it is without consequences and be sure to utilize it judiciously. And the specifics for how we go about that will be discussed shortly.

[61] Clinical Analysis Management, 2019 – And this effect of decreased CO due to PEEP isn't so much a thing with a ~~euvolemic~~ patient, so we can mitigate somewhat by fluids if our patient will tolerate it

[62] Coruh & Luks, 2014; Strong, 2013; Yartsev, 2019 – Refer to these sources for detailed explanations of all of those negative consequences of PEEP

Inspiratory Time (and I:E Ratio)

The next term to consider is inspiratory time, often referred to as "I-time." I-time is the amount of time over which we deliver a breath. A normal I-time varies by age as so:[63] [64]

Age Description	Age (yrs)	I-time (s)
Infant	.083 (1 month) – 1	0.3 – 0.6
Toddler	1 – 2	0.4 – 0.9
Preschooler	3 – 5	0.5 – 0.9
School-aged Child	6 – 7	0.6 – 1.1
Big Kiddos	8 – 9	0.6 – 1.2
Preadolescent	10 – 12	0.7 – 1.4
Adolescent	12 – 15	0.8 – 1.7
Adult	16 and up	0.8 – 1.7

One idea related to PPV is that the more time we spend pushing air into system, the more oxygen gets moved into the bloodstream. This means that more time spent on the inspiration side of the breath cycle (vs. exhalation) equals better oxygenation.[65] With that in mind, the most intuitive way to increase time spent at inspiration would be to lengthen the I-time. If we do that, however, we have to accommodate by decreasing time spent at expiration or by decreasing rate. Consider seventeen breaths over one minute of time:

60s ÷17 breaths ≈ 3.5 seconds per breath

if "in" or inspiration = 1.0 seconds,
then "out" or exhalation = 3.5 seconds – 1.0 seconds
"out" or exhalation = 2.5 seconds

if we lengthen inspiratory time to 1.5 seconds:
exhalation time = 3.5 seconds – 1.5 seconds
= 2.0 seconds

[63] See **Appendix** for how we got all these numbers
[64] Ashworth & friends, 2018 – There is a term called "time constant" in PC ventilation that we can use to quantify an appropriate I-time for a particular patient, but this isn't routinely available in the transport setting and we still need a value with which to initiate ventilation when we first get things rolling
[65] Discussed again later when we get to **Mean Airway Pressure**

We often represent this ratio between I-time and expiration time as an "I:E ratio" to describe the amount of time spent at inspiration in comparison to the amount of time spent at exhalation. A normal I:E ratio is anywhere from 1:2 – 1:3. Let's build an I:E ratio for the above examples:

in the first example, we have 1.0s : 2.5s, so our I:E ratio is 1:2.5

in the second example, we lengthened out inspiratory time to 1.5s;
So we now have 1.5s : 2.0s
we (almost) always write out I:E ratios with "1" as the first number,
So we need to simplify the ratio:
~~simply~~ divide both sides by the first number: $\frac{1.5}{1.5} : \frac{2.0}{1.5}$
and solve for our new I:E ratio of 1:1.33

So to bring it back home: we had a rate of 17 and an I-time of 1.0 with a resultant I:E ratio of 1:2.5. We wanted to increase time spent at inspiration, so we changed our I-time to 1.5 and ended up with an I:E of 1:1.33. For now we don't have to worry about the significance of these numbers, we just need to understand the math, how we get to these numbers, and the terminology associated with them. Let's try another example, but this time we will adjust rate instead of I-time:

per above: rate of 17, I-time 1.0s = I:E of 1:2.5
now let's increase our rate to 20 and recalculate the I:E ratio
60s ÷ 20 breaths = 3 seconds per breath

if "in" or inspiration = 1.0 seconds, then "out" or exhalation = 3.0 seconds – 1.0 seconds
therefore "out" or exhalation = 2.0 seconds

in this example, we now have 1.0s : 2.0s, so our I:E ratio is 1:2.0

now let's shorten our I-time to 0.8s and see what happens:
if "in" or inspiration = 0.8 seconds, then "out" or exhalation = 3.0 seconds – 0.8 seconds
therefore "out" or exhalation = 2.2 seconds

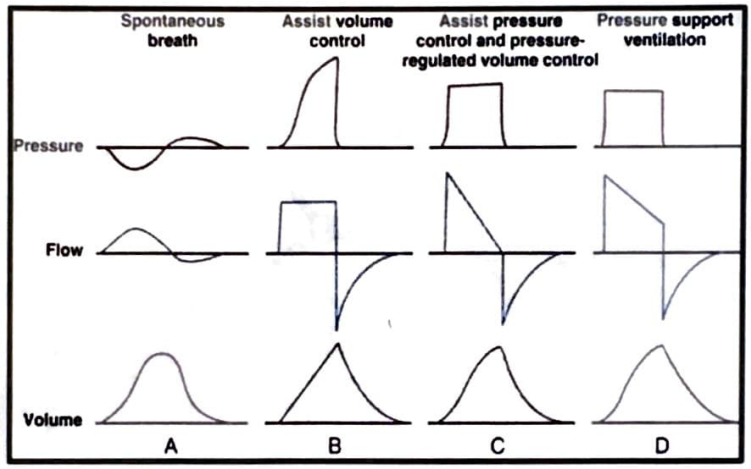
now we have 0.8s : 2.2s,
but we need to make this an I:E ratio with "1" as the first number:
$\frac{0.8}{0.8} : \frac{2.2}{0.8} = 1:2.75$

And let's summarize this all one more time and make a few generalizations: we can shorten our I:E ratio by either increasing I-time or increasing rate; we can lengthen our I:E ratio by decreasing I-time or decreasing rate. A shorter I:E ratio means less time (in relation to the whole in/out cycle) spent on exhalation, a longer or lengthened I:E ratio means more time for exhalation. We will talk about this later when we get to ventilator strategies, but know that some patients can benefit from a shorter I:E ratio and other can benefit from a longer I:E ratio, so it is important to know which changes affect the I:E ratio in which direction.

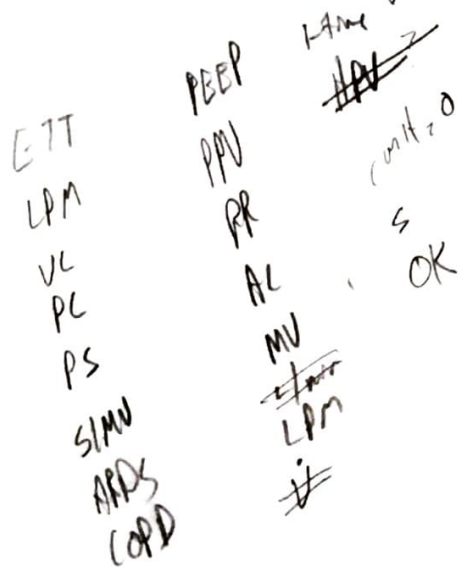
Return to Contents

Types of Breaths

Let's take a few minutes to discuss an image we presented towards the beginning of this manual. ~~The idea here is that~~ we want to explain in a little more detail each of the following types of breaths depicted below:[66]

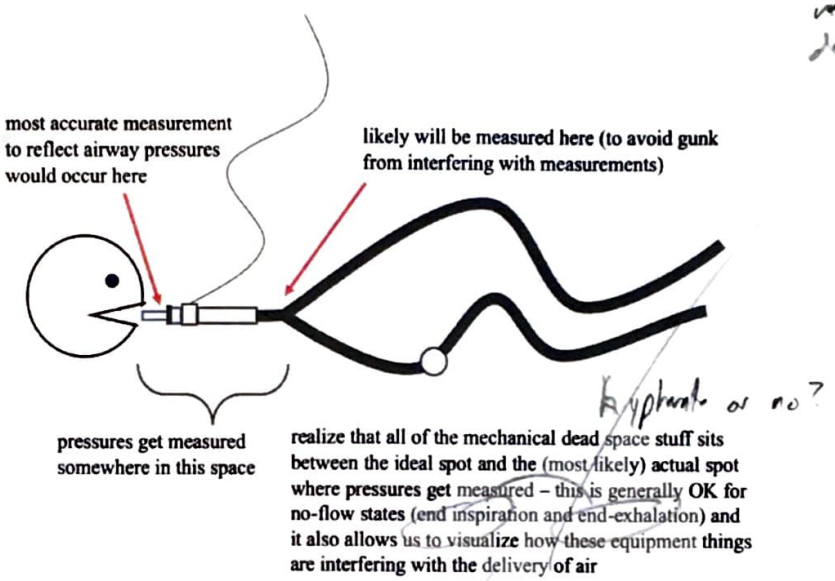


There are three waveforms depicted for each type of breath, but our focus for now is on the first two rows: pressure and flow, each shown over time. We sometimes hear these graphics of vent function ~~described~~ as "scalars," as in a "pressure time scalar" or "flow time scalar." The image above shows ideal scalar waveforms, real ones as produced by a vent may vary somewhat and will be less clean-cut than these guys. But enough on that for now, let's move on to each of these things: pressure and flow.



[66] Fuller & friends, 2014 (image)

Pressure is measured by the machine somewhere between the ~~endotracheal tube~~ (ETT) and the wye where the inhalation side of the circuit splits off from the exhalation side of the circuit:[67]

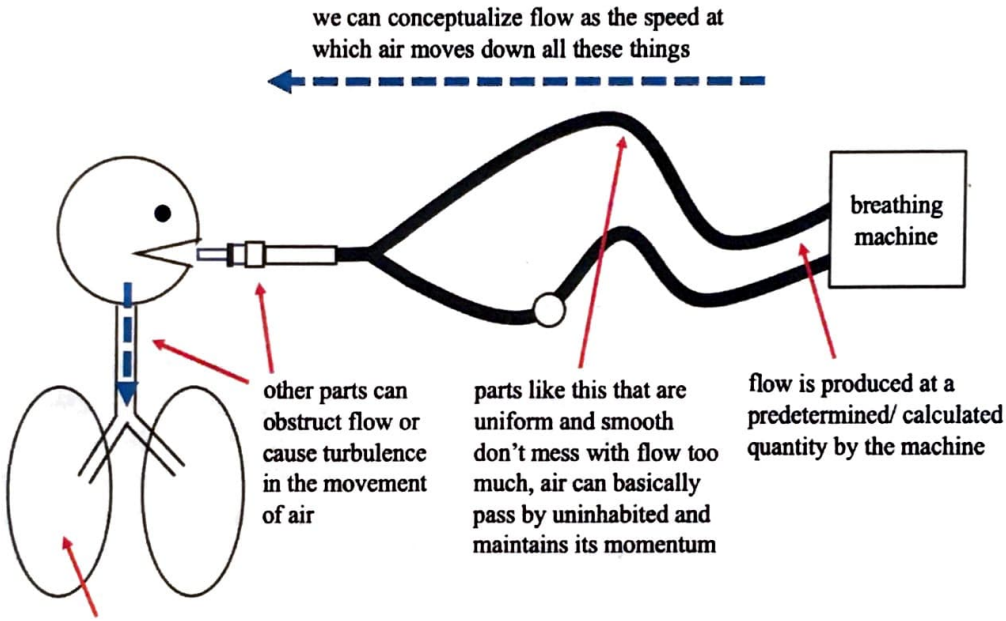


Another thing to mention is that the pressures we ~~"see" or~~ measure don't directly describe pressures at the alveoli or terminal ends of the airway, they reflect what's going on outside of the patient's body. That said, we can manipulate the system to approximate alveolar pressures (and we will discuss how to do that later) and we assume that the value we measure correlates with average pressure at the alveoli. Pressures experienced by individual alveoli vary throughout the lung and our measurement occurs outside of the lungs themselves, but this is the best approximation we have and therefore we base our treatment on the information available to us. ~~So~~ the waveform that shows pressure over time gives us a visual representation of how pressure changes at the mouth side of the system as we deliver a breath. And we already talked about how pressure is measured (in terms of units), so we are good on this general idea for now.

[67] Hess, 2014 – Also provides an overview of flow, which we discuss next

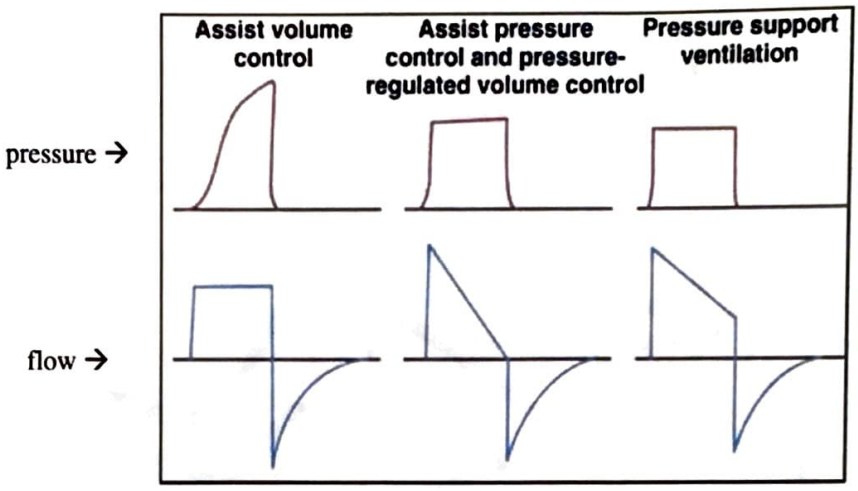
Return to Contents

Next concept to discuss is flow. Flow is ~~basically~~ a description of how fast we move air through the system and is quantified in liters per minute (L/min or LPM).[68] When we describe flow, we do so at the machine side of the system. As air moves away from the machine, however, different things can interfere with the speed at which the body of air is moving. But since we don't measure flow (rather we create flow and send it out into the universe via the machine), we see all of this interference indirectly via other parameters (such as pressures and volumes). Here's how it looks mapped out over top of the system:



[68] And sometimes notated by the symbol $\dot{V}$, but we also use that same symbol in Fick's Law stuff in the next section and don't want to get things confused…

Now that we are set on the basics of pressure (as measured in the system) and flow (as produced by the machine), let's look at a few of these waveforms again and see how we can deliver breaths in different ways:[69]



First thing to note is that there are three general categories: VC breaths (left), PC breaths (middle), and PS breaths (right).[70] In VC a breath is most commonly delivered via a "square wave" flow pattern in which the machine spins up right away to a set flow, holds it for a predetermined amount of time, then cycles off. With PC and PS breaths, however, flow is delivered via a "decelerating waveform" flow pattern in which the machine starts a breath by spinning up to a max pressure and then slowly maintaining that pressure by delivering less and less flow until the breath cycles off. To say this all another way: VC gives a constant flow for variable pressure, PC and PS give constant pressure at variable flow.

And let's follow this up with a series of sequential facts: There are some machines nowadays that can give VC breaths via a decelerating pattern, but those aren't commonly used in the transport setting. That means we can generally lump these three types of breaths in to two groups: volume/ square wave flow and pressure/ decelerating flow. Unless we are in VC and SIMV, we ventilate patients with one type of breath at a time. In very general terms: the pressure/ decelerating breaths are more comfortable for patients but take longer to deliver (i.e. not ideal when we need to give breaths fast or allow lots of time for exhalation).[71] [72]

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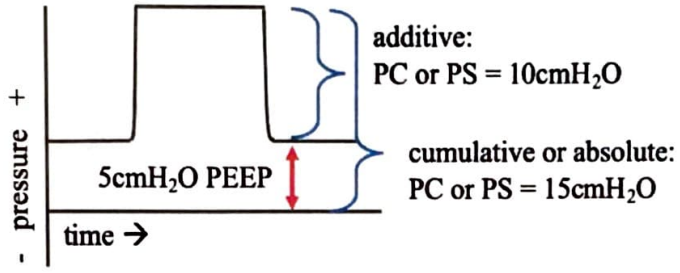
[69] Fuller & friends, 2014 (image)

[70] Our labels differ slightly from those in the image, but we'll hash all of this out soon

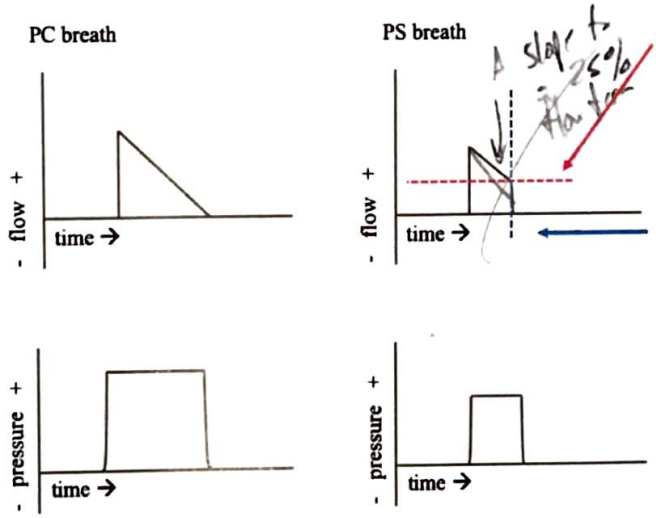
[71] Gonzales & friends, 2012 – Amongst many other fun things, these guys explain how pressure/ decelerating pattern may be best for ARDS patients and volume/ square wave may be best for obstruction related to COPD

[72] Iyer & Holets, 2016 – And in this presentation on vent waveforms, they describe how longer I-times may be indicated for patients vented with a decelerating waveform pattern

As for the two types of pressure/ decelerating pattern breaths (PC and PS), there are a few things to mention. First is that the pressure used to describe these breaths can either be referred to in addition to PEEP or inclusive of PEEP (and sometimes we describe the value as "cumulative" or "absolute" to include PEEP or "additive" to say it is added on top of PEEP).[73] This varies by machine, so just be aware of it:



The next thing to mention here is how PC and PS breaths differ. While both are given via a decelerating waveform pattern, the mechanism by which flow is ~~initiated and~~ terminated changes things. A PC breath is designed to deliver a full breath even with no patient effort, whereas a PS breath is designed to simply relieve some effort of breathing on the front end of a breath. Because of this difference, a comparable titration of pressure (i.e. a change of 5cmH$_2$O to both PC and PS) may result in different changes of volume in the very same patient. ~~Now~~ the mechanism by which this works is known as "termination," ~~some kind of~~ parameter at which the machine decides to stop supporting a breath and begin exhalation:



when flow drops below a certain threshold, the breath cycles off

this threshold is defined as a percentage of max flow for that breath (ex. flow termination of 25%)

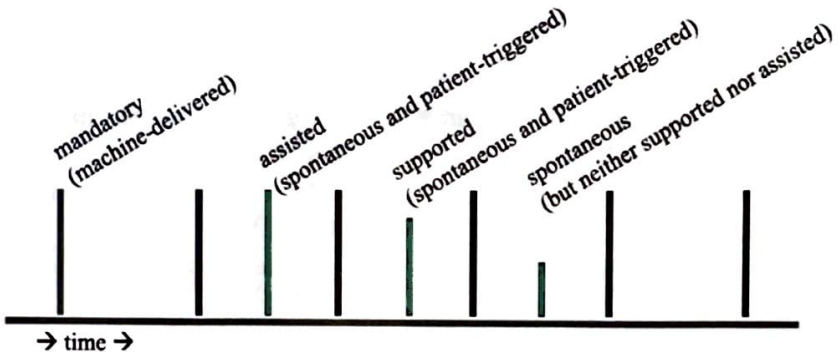
there can also be a time limit to terminate the breath; ~~or~~ if after a set time (ex. time termination of 1.0s) the breath hasn't terminated due to the flow trigger, then it gets cut short by the time trigger

we've shown this PS breath with a PS setting lower than the accompanying PC breath (lower amplitude in the pressure waveform), but recognize that this doesn't have to be the case

[73] Ashworth & friends, 2018, Bauer, 2016a – The first mentions this idea in passing in the context of PC ventilation; the second reviews this idea in the context of non-invasive PPV (which we don't get into here in this manual)

So to get more volume in a PS breath (represented by area under the flow time waveform), we either need more patient effort (i.e. don't "snow" all patients!) or we need to maximize our termination triggers (i.e. lower value for flow termination, longer time termination).[74] We don't typically get that far into the weeds with invasive ventilation and PS, but you will often see this idea discussed in terms of non-invasive ventilation (which we don't cover in this manual).

Last bit of this section: let's review different types of breath as it relates to mandatory, assisted, supported, and spontaneous breaths (which is slightly different than they were described in that first image in the section). We've touched on these in passing as we moved through the different modes, but let's just clarify a few things and show how they vary from one to another starting with a quick graphic:



Mandatory or machine-delivered breaths are the ones that we deliver via our set RR on the vent and to a specific goal, whether that be volume or pressure. Assisted breaths are triggered by patient effort and then the machine delivers a full breath to match the same goal as for the machine-delivered or mandatory ones. Moving right, supported breaths are also patient-triggered, but get delivered via pressure support and not to a set goal. Supported breaths are often smaller than mandatory or assisted ones, that's why they have been shown with a shorter green line.[75] And lastly are spontaneous breaths that don't get supported or assisted. These breaths basically get ignored by the machine and function solely via patient effort. We typically don't see these too often, as we ventilate patients in AC or SIMV modes, but they are shown for comparison.

And to take this discussion one step further, let's consider which types of breaths apply to which types of ventilation. In AC mode we have mandatory breaths and assisted breaths and neither supported nor spontaneous breaths (all breaths that meet the trigger will get supported). In SIMV mode we have mandatory breaths, assisted breaths (when a trigger is sensed within the mandatory period), and supported breaths (when a trigger is sensed in the spontaneous period). In neither mode do we routinely see spontaneous breaths. While there may be spontaneous effort that don't meet the trigger (and this theoretically could contribute some to MV), all noteworthy patient effort (defined by meeting whatever trigger threshold we have set) will get facilitated by the machine in some way in either mode.[76]

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[74] That said, the primary mechanism for terminating a breath will be the flow term and it may help to think of the time term as a backup in the event that the breath doesn't end via the flow term mechanism after a certain amount of time

[75] But again, this doesn't necessarily have to be the case – see section on SIMV for more on this idea

[76] And we realize that we've talked a lot about **Triggers** here, but the details of that has been deferred until later on

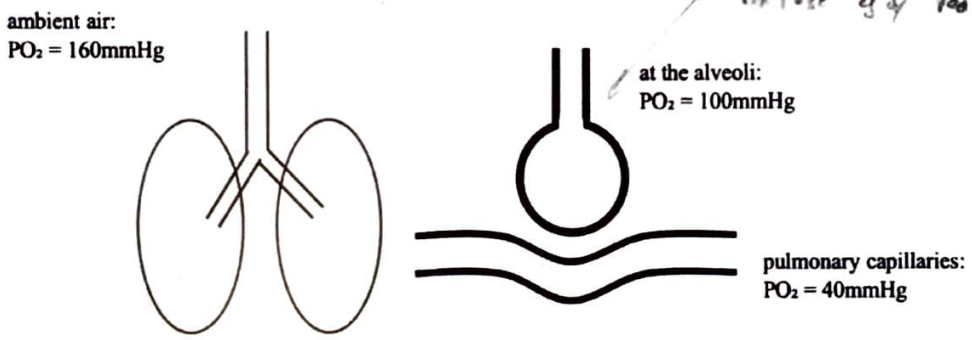
Return to Contents

Three Big Things

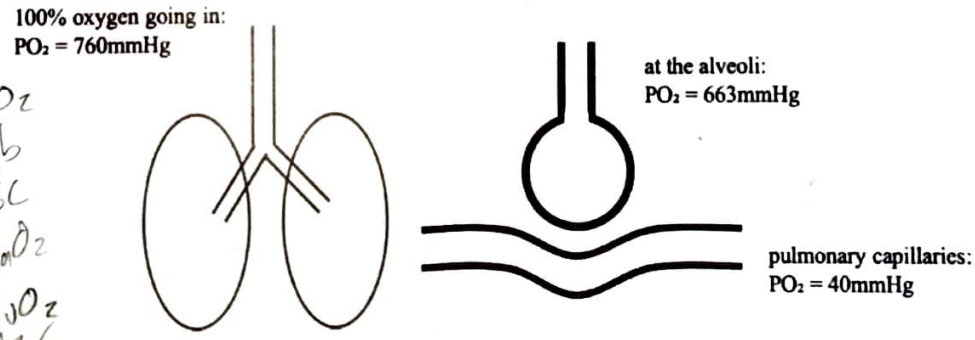
There are three super duper important things that need to be monitored and addressed for all ventilated patients, hands down and no matter what. The order in which we discuss them here is totally arbitrary, they all hold equal weight and are interrelated. The discussions that follow are in general terms and not specific to certain pathologies or patient types, that stuff will come soon.

Oxygenation (~~and SpO_2~~)

It may have come up once or twice before, but oxygen is pretty important stuff. Oxygen gets to tissues via a few steps, some of those we can affect directly with the ventilator. There are also more complicated ways to manipulate oxygenation, but let's focus on the simple stuff for now, starting with a review of how oxygen gets from the ambient air to the tissues. The following is a version of a graphic we used earlier that shows partial pressures at a few steps along the way. These pressures are for the spontaneously breathing patient:



~~Also~~ recognize that gasses will diffuse from areas of high concentration (~~higher partial pressures~~) to areas of lower concentration. ~~So~~ in this baseline example, ~~we can conclude that~~ oxygen will move from the ambient air to the alveoli, and then into the pulmonary capillaries. The first way that we can speed this process up is by changing the partial pressure of oxygen at the start of the system. Instead of 21% of the gas mix or 160mmHg of oxygen, we can titrate that all the way up to 100% (FiO_2 1.0) or 760mmHg. This will increase the rate at which oxygen diffuses to the alveoli, resulting in a higher partial pressure of oxygen downstream and, subsequently, faster diffusion into the blood stream:



Let's recap this bit and do some math: PO_2 at the alveoli on ambient air is 160mmHg, PO_2 at 100% oxygen is 663mmHg. To quantify the result of this difference let's apply Fick's Law:[77]

$$\dot{V} = \frac{(P_1 - P_2) \times \text{Area} \times D}{\text{Thickness}}$$

$\dot{V}$ = rate of gas diffusion across a membrane (i.e. alveolar membrane)
P_1 = ingoing pressure (i.e. at the alveoli)
P_2 = pressure at other side (i.e. in the blood)
Area = self-explanatory…
D = diffusion constant
Thickness = also self-explanatory…

if $\frac{\text{Area} \times D}{\text{Thickness}}$ is constant and we call it "k,"
we end up with the following:

$$\dot{V} = (P_1 - P_2) \times k$$

and let's add in some numbers for the ambient air and 100% oxygen situations:

$$\dot{V}_{\text{ambient air}} = (100 - 40) \times k$$
$$= 60k$$

$$\dot{V}_{100\% \text{ oxygen}} = (663 - 40) \times k$$
$$= 623k$$

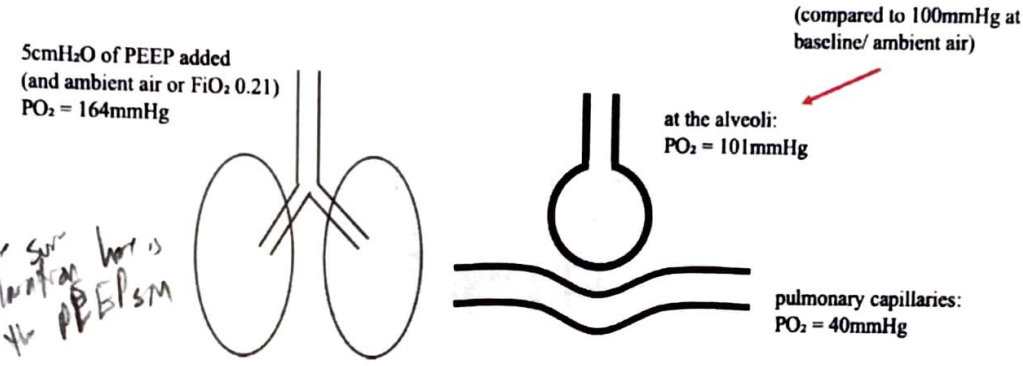
That means that oxygen diffusion occurs ten times faster at 100% oxygen (or an FiO_2 of 1.0) than at room air. Which is both nuts and a clinically significant thing to be aware of. The takeaway here is that whenever we need to increase the diffusion of gas across the alveolar membrane, FiO_2 is a heck of a way to get that done. The holdup is when other factors in the equation (area and thickness) are also issues, then we may need to augment this strategy with other techniques. And also remember that oxygen molecules can cause problems, so deliver them to the patient judiciously.[78]

To expand on this idea just a bit before we move on, one of the arguments against a high FiO_2 is the idea of absorption atelectasis – the closing of alveoli related to nitrogen washout and the fact that oxygen quickly diffuses into the blood stream leaving less gas in the alveoli.[79] While the clinical impact of this theoretical consequence is questioned by some,[80] it is worth keeping in mind. And if we do give credence to the idea, some ways to mitigate this effect would be maintaining a patient's spontaneous effort to breath (strategies for which are discussed shortly in **Comfort**) and performing **Recruitment Maneuvers** (discussed much later).[81]

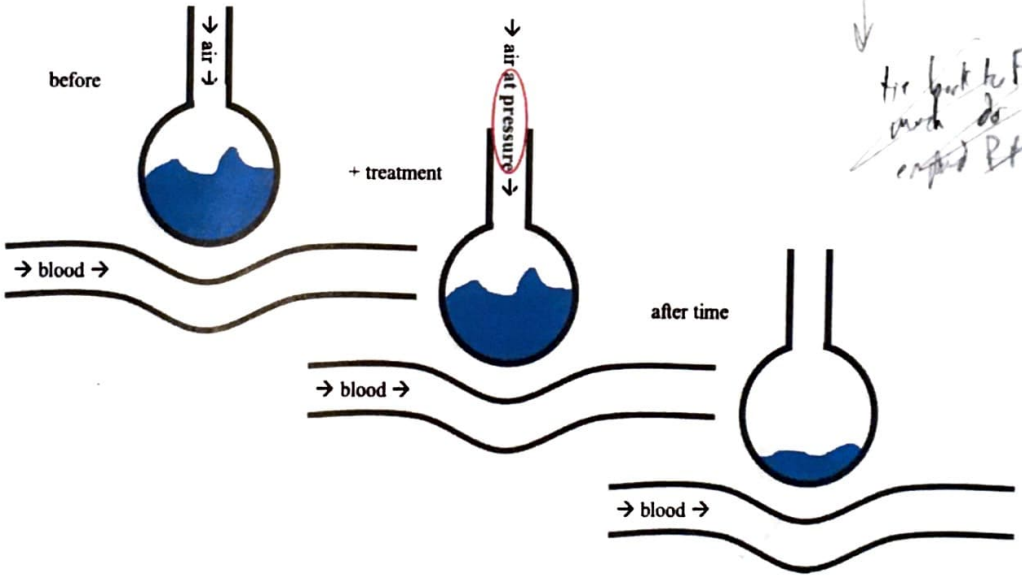
[77] Desai, 2012 – Best ever explanation of this concept courtesy of Kahn Academy
[78] Kallet & Branson, 2016 – We cited this first one earlier when we first discussed **Fraction of Inspired Oxygen**
[79] Dunphy, 2012 – Short video that explains this mechanism and how patient effort can mitigate this effect
[80] Yartsev, 2019 – Also describes some of the other mechanisms by which oxygen can adversely affect our patients
[81] Hartland & friends, 2015 – The article outlines an argument for the use of these maneuvers in certain patients (which seems reasonable to extrapolate to some of the patients we see in the transport setting)

Return to Contents

The next way we can increase oxygenation is via PEEP. Now PEEP doesn't quite work by the same mechanism, as the addition of PEEP doesn't much change the partial pressure situation as we saw with an increase in FiO_2:

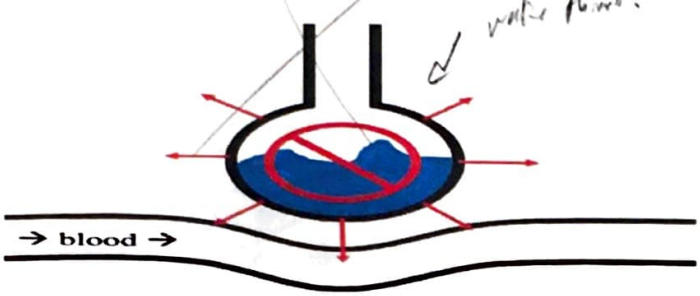


Instead, PEEP facilitates oxygenation primarily by increasing alveolar surface area, and also by extending gas exchange into the exhalation side of the breath. We discussed that first concept back in the section on **Alveolar Surface Area** and the second one just a moment ago in the section on **PEEP**, so no need to redo all of that here. One more mechanism by which PEEP helps oxygenation is that it cleans up the alveolar membrane, in a sense, by pushing out or displacing fluid that accumulates there. Think of it this way:



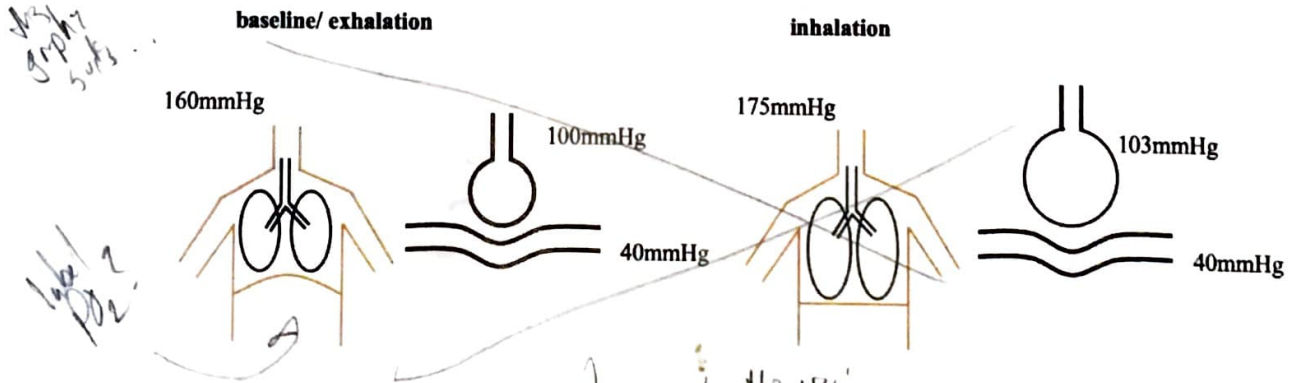
So we have three ways that PEEP helps with oxygenation: it increases the surface area of the alveoli, it extends gas exchange into the respiratory side of the breath, and it helps to physically displace fluid from the alveoli:

1. arrows emanating from alveoli = increased size/ more surface area
2. stretch/ space distortion = extension of gas exchange into exhalation side
3. no smoking X on fluid = displacement of all that stuff



Just a quick recap before pressing on: assuming ventilation and comfort are adequate (see next sections), initial steps to fix oxygenation are increasing FiO_2 and then adding PEEP. While it is totally OK to use a stepwise approach that titrates both FiO_2 and PEEP in line with one another,[82] recognize that FiO_2 is your most direct fix for improving partial pressure of oxygen at the alveoli and has very few consequences in the acute (i.e. short term) setting. PEEP is especially helpful in increasing alveolar surface area and driving fluid out of the lungs, but may decrease cardiac output by way of a drop in preload to the heart (especially if our patient is down on fluids). And lastly, both of these techniques (FiO_2 and PEEP) improve oxygenation throughout the respiratory cycle.

The next logical step in this discussion is to consider what happens during inhalation. Changes to both FiO_2 and PEEP affect oxygenation throughout the respiratory cycle, that is both on inhalation and exhalation, but lots of our oxygenation happens during inspiration. Here's a comparison of what pressures and alveolar shape would look like with an FiO_2 of .21 (ambient air) and no PEEP, both at baseline/ on exhalation (left) and on inspiration (right). We'll use an arbitrary added pressure of $20cmH_20$ or 15ish mmHg:



[82] We'll touch a bit more on this subject in the section on ALI/ ARDS later on

Note both the greater pressure difference between alveoli and capillary as well as changes to the alveolar surface (more of it and thinner) during inspiration. This leads us to conclude that more time spent at inspiration further maximizes oxygenation, therefore strategy number three to maximize oxygenation is to increase the I-time ~~to make use of this piece of knowledge~~.[83] If we extend I-time long enough, it will eventually become longer than exhalation and we end up with an "inverted I:E ratio" that might be written as 2:1. We previously stated that we "always" express an I:E ratio with a "1" as the first number, but we lied – the exception to that rule is when we have an inverted I:E ratio. Let's amend that previous rule to say that one of numbers in the ratio needs to be "1" and that it is always the first (inspiratory) number except in cases where we have an inverted I:E ratio.

The primary drawback of a really long I-time (and therefore of an inverted I:E ratio) is that it can be uncomfortable for our patients and we will need to get super aggressive to maintain patient synchrony with the machine. Comfort is one of the three super duper important concepts in this section, so enough said about that until we get there. An inverted I:E may also make it tough for the patient to exhale fully, predisposing us to that AutoPEEP issue. Summary up to this point is that there are three ways to improve oxygenation by adjusting settings on the vent: increase FiO_2, add PEEP, and lengthen the I-time.

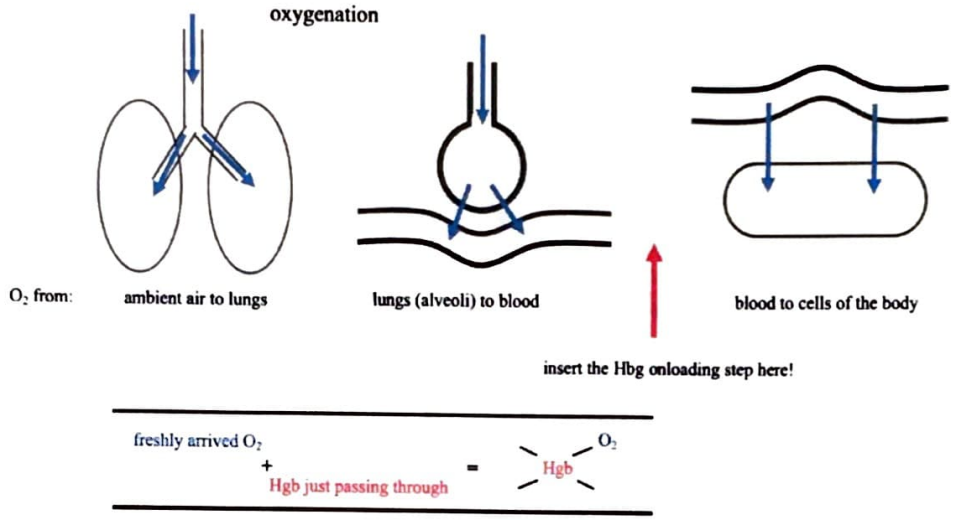
"Now why," we might ask, "do we not just fill the lungs up with 100% oxygen and keep them inflated – we'd have ~~a forever-long maximum diffusion of oxygenation~~ into the blood stream, right?" There are two reasons for this. One is that we don't want to ~~drop preload or blood return to the heart~~ indefinitely (as discussed above). Two is that it isn't all about oxygen – we also have to consider its partner in crime, carbon dioxide. Carbon dioxide doesn't diffuse so well in gas (as compared to oxygen) because it is a bigger, heavier molecule. The movement of carbon dioxide, ~~therefore~~, is partially dependent on movement of the body of air in which it hangs out. And that leads us into our next section on ventilation, but a few more things to cover before we get there.

Recall back to our previous discussions of both ~~the hypoxic vasoconstrictive response~~ and alveolar dead space. There are times where we are getting oxygen into the system just right, but components inside the system are out of whack and that oxygen is not being put to good use. One thing we, as clinicians, sometimes do to exacerbate this "~~things out of whack~~" concept is lay our patients flat. Unless you have good reason to do so, all vented patients should have their head of bed elevated somewhat.[84] And backboards (if you are still using those archaic torture devices!) are no excuse, just prop the whole head end up with something to get a comparable effect. The reason why we elevate the head of bed to improve oxygenation is multifaceted, but it has a fair amount to do with gravity and is beyond the scope of this discussion.

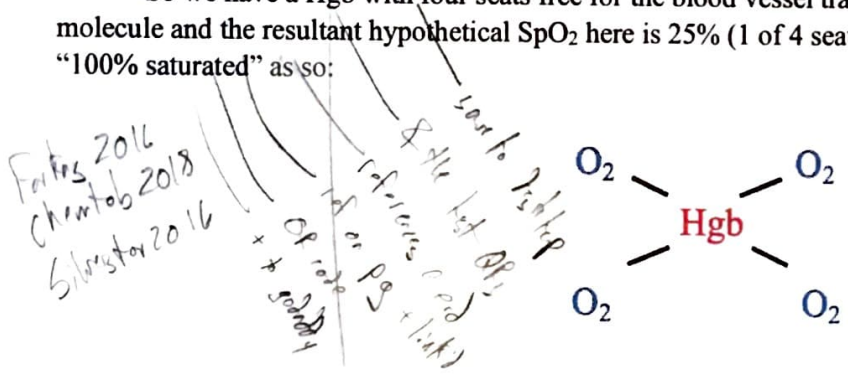
[83] While we could also make the argument that gong up on RR increases the amount of time spent on inspiration, doing so also impacts ventilation (next section) so we generally don't consider RR one of the variables by which we control oxygenation

[84] Spooner & friends, 2014 - This study provides evidence for head of bed elevation in all ventilated patients (except as contraindicated)

One more thing to consider is how we measure oxygenation. Our standard tool in the field is pulse oximetry or SpO_2. SpO_2 uses infrared to "see" to what extent our hemoglobin is saturated with oxygen (or oxygen-like things, but we won't worry about the tricky parts here). The process here goes like so: oxygen gets to the alveoli, it crosses into the blood stream via diffusion gradients of gas, then once in the bloodstream it gets picked up by hemoglobin (Hgb) for a ride down the blood vessel. Let's draw out the onloading process:



So we have a Hgb with four seats free for the blood vessel train, one of which is occupied by an O_2 molecule and the resultant hypothetical SpO_2 here is 25% (1 of 4 seats filled). Fill all four seats up and we are "100% saturated" as so:



Do note that Hgb doesn't cruise freely through the vessels, it comes attached to red blood cells (lots and lots of Hgb per each RBC), but the four seats per Hgb is a fair description. Also consider that we measure this saturation peripherally (hence the "p" in SpO2 versus an SaO_2 for "arterial" or an SvO_2 for "venous"). This means that if blood isn't getting to the periphery where we have our little probe attached, numbers may not be accurate (and one way around this is to always confirm a good qualitative waveform before believing a quantitative value the machine gives you).

One last summary before moving on from oxygenation. Oxygenation is one of these three super duper important things. We measure it via SpO_2, which tells us how filled up with oxygen the Hbg (attached to RBCs) in the blood are as they move past wherever we have attached the SpO_2 probe. To get a better number (or improve oxygenation) by moving numbers on the vent interface, we have three options (and we typically do them in this order): increase FiO_2, add PEEP, lengthen the I-time. All that said, let's not forget the basics: position your patient appropriately and make sure ventilation and comfort are addressed simultaneously (see next sections).

Ventilation (and $EtCO_2$)

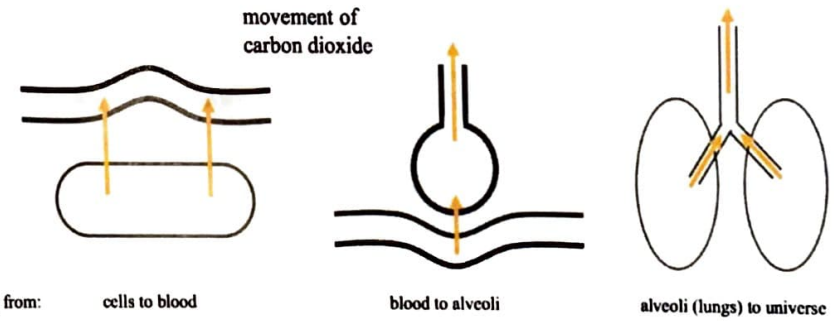
Next super-duper important thing is ventilation. Ventilation refers to the movement of air in and out of the system as we deliver breaths and allow exhalation. As discussed before, this is vitally important for the movement out of carbon dioxide. Too much carbon dioxide hanging out in the lungs with no escape is bad news, so we can't just focus on getting oxygen in. So how do we know if we are moving enough air for a given patient? There are two strategies here and we will discuss them both in turn: calculated minute volume and end-tidal carbon dioxide ($EtCO_2$).

If we math it out, our minute volume goal for the typical patient should be:[85]

$$MV \approx 100\text{ml/kg (IBW) /min}$$

This number varies a bit for patients with an increased need (i.e. acidosis), but it's a good place to start as written and is an appropriate minimum for most patients. Having a goal minute ventilation in mind and then assessing actual minute ventilation (typically measured and reported by the vent) is great way to ensure that the patient's minimum needs are met.

Concurrently, we also use $EtCO_2$ to monitor ventilation. When the body uses up oxygen at the tissue level it kicks back CO_2 into the blood stream. That carbon dioxide then makes its way up to the lungs where it passes into the alveoli and then is exhaled out. It looks about opposite to our previous sketch showing how oxygen moves through the system:



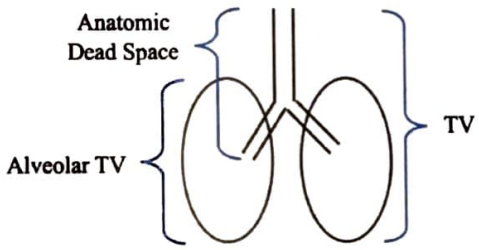
[85] And we discussed where this number comes from previously, in the section titled Minute Volume

So the value we get on our quantitative $EtCO_2$ reading is a function of all of these factors. It gets a bit complicated, but the standard approach to managing ventilation with $EtCO_2$ is to use a base range and adjust minute volume (which is a function of both RR and TV) to get the quantitative value within that acceptable range. Normal range for $EtCO_2$ is 35-45mmHg; values above range require an increase in MV to "blow off" more carbon dioxide, values below range need you to read the next paragraph carefully.

A low $EtCO_2$ can be caused by a few different things, one of which is hyperventilation or too much ventilation. This can be detrimental to a patient, as an alkalotic state (due to too much tidal volume and a low $EtCO_2$) can throw off the patient's homeostasis and lead to some bad stuff. In this case, it'd make sense to decrease MV (by lowering either RR or TV) to get the $EtCO_2$ (and therefore ventilation) back to normal. All that said, a low $EtCO_2$ could also be due to a breakdown somewhere else in the system (i.e. at any of those yellow lines in the previous drawing). For example, if perfusion is no good we may see a low $EtCO_2$ even though the issue is not necessarily a ventilation problem. In this case we could kill the patient by "chasing" their $EtCO_2$ or dropping MV to an unsustainable level.

We can navigate this whole situation by managing ventilation by looking at both minute volume and $EtCO_2$ instead of just $EtCO_2$ by itself. There are times when we will be a bit off with MV and others when our goal range for $EtCO_2$ varies, but this system of dual parameters to evaluate ventilation is a safety check to remind us of all the factors that go into ventilation. So to summarize: we measure ventilation using both a calculated MV goal and $EtCO_2$. MV goal is 100ml/kg/min; normal $EtCO_2$ is 35-45mmHg.

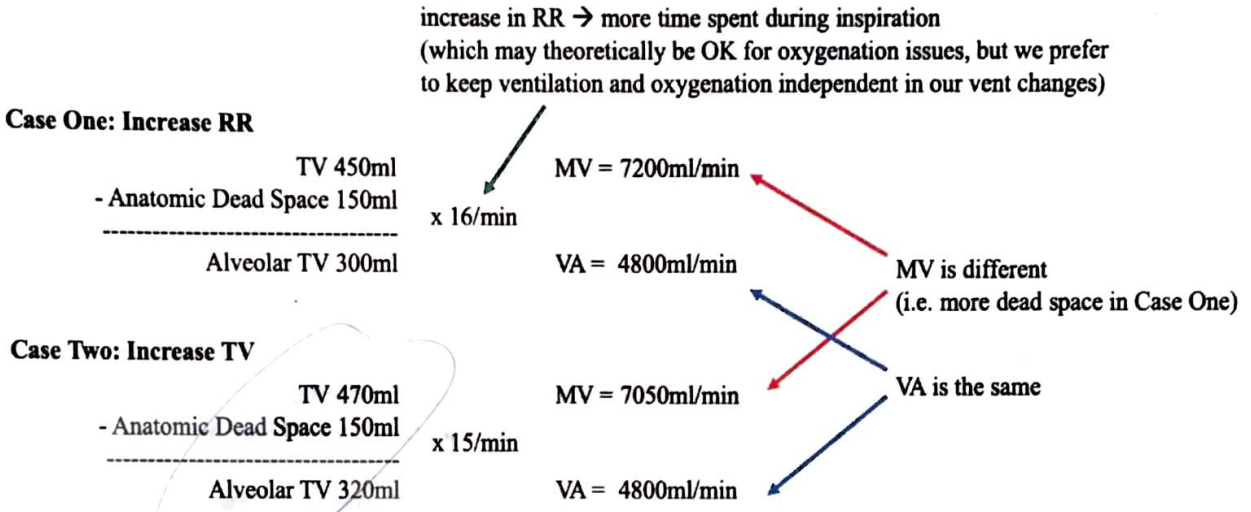
And one final point before we move on: when faced with the choice as to whether we should manipulate RR or TV to effect a change in MV, here's what we recommend: to increase MV, utilize TV first; to decrease MV utilize RR first. To explain why that is, let's say we have a patient breathing at a RR of 15 and a TV of 450ml:



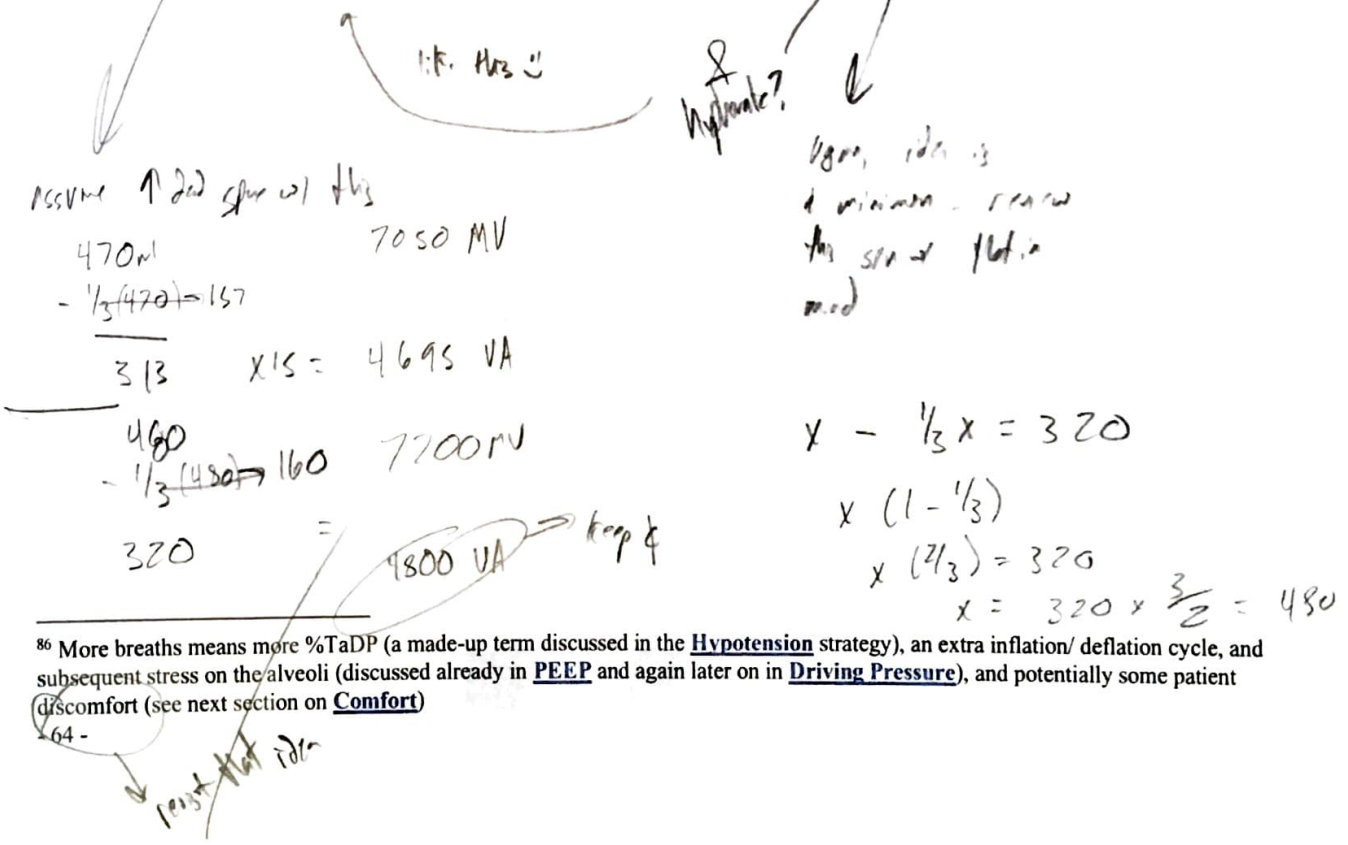
TV 450ml		MV = 6750ml/min
- Anatomic Dead Space 150ml	x 15/min	
Alveolar TV 300ml		VA = 4500ml/min

Return to Contents

Now assume we need to increase VA (~~which, just as a reminder, is alveolar minute volume~~) by an arbitrary value of 300ml. We could do this by either of two ways: increasing rate to 16 or increasing TV of each breath by 20ml. While either method is just fine mathematically, adding in an extra breath is a bit less efficient and puts more stress into the system. That stress comes in a few different forms, but we'll get to all of those later.[86] And here's how the math would look in either case:



Now on the opposite end of things, if $EtCO_2$ is low (which indicates too much MV), then we back off on RR first. That gives us the same differences, but in the reverse: less VA (which is what we want) accompanied by less time spent during inhalation and less dead space. As we said before, either strategy (titrating RR or TV) is fine to make a change to MV, it's just a bit more efficient to use TV to increase ventilation and RR to decrease ventilation. And we start our ventilation strategy using a weight based goal for MV (by way of an age-based RR rate and weight-based TV) and then titrate it as we go to an $EtCO_2$ goal.



[86] More breaths means more %TaDP (a made-up term discussed in the **Hypotension** strategy), an extra inflation/ deflation cycle, and subsequent stress on the alveoli (discussed already in **PEEP** and again later on in **Driving Pressure**), and potentially some patient discomfort (see next section on **Comfort**)

Comfort

The third super duper important parameter that we need to consider with vent management is patient comfort.[87] If your patient is not comfortable, (s)he may be "fighting the vent" or "out of synch" and the therapeutic effects that we want to achieve will be more difficult to attain. This asynchrony can also lead to increased airway pressures which leads to more problems downstream. And one more thing: it's kind of rude to shove a big plastic tube down someone's throat, take over their respiratory function in a way that goes opposite to normal physiology, and then load them up inside a small flying box with people crowded all around and lots of noise, vibration, weird lights, etc. So let's be nice people and keep our patient's feelings in mind.

We won't spend too much time on the subject of pharmacology, as the main focus here is on manipulating the vent itself, but recognize that analgesia and sedation are two different things and that we need to treat them both.[88] Also recognize that paralysis should be a last resort for nearly all ventilated patients, as it prevents us from actually assessing and evaluating our patients. And on that same note: while do want our patients to be comfortable, this doesn't mean that we "snow" them all or take away any inherent respiratory effort in order to achieve this goal. There is benefit to ventilated patients making some intrinsic respiratory effort and we like to maintain that whenever possible.[89]

When we manage comfort it is important to have a strategy for quantifying the idea so that we can gauge the efficacy of our interventions. Many agencies recommend scales or tools to use and here are some examples:

TABLE 1. NONVERBAL PAIN SCALE

	Category		
	0	1	2
Face	No particular expression or smile	Occasional grimace, tearing, frowning, wrinkled forehead	Frequent grimace, tearing, frowning, wrinkled forehead
Activity (movement)	Lying quietly, normal position	Seeking attention through movement or slow, cautious movement	Restless, excessive activity and/or withdrawal reflexes
Guarding	Lying quietly, no positioning of hands over areas of body	Splinting areas of the body, tense	Rigid, stiff
Physiological I (vital signs)	Stable vital signs (no change in past 4 h)	Change over past 4 h in any of the following: SBP > 20 mm Hg, HR > 20 beats/min, RR > 10 breaths/min	Change over the past 4 h in any of the following: SBP > 30 mm Hg, HR > 25 beats/min, RR > 20 breaths/min
Physiological II	Warm, dry skin	Dilated pupils, perspiring, flushing	Diaphoretic, pallor

Definition of abbreviations: HR = heart rate; RR = respiratory rate; SBP = systolic blood pressure.

Reprinted by permission from Reference 15.

[87] Rustam & friends, 2018 – This article is a lit review of lots of different papers on comfort in mechanically ventilated patients; while much of this stuff might be hard to relate to a patient we intubate in the field on a scene call, lots of it can translate to the interfacility transfer side of things

[88] Patel & Kress, 2011 – We've also taken the graphics for the NVPS and RASS scores from this article

[89] Mauri & friends, 2017 – Discusses how to navigate the benefits of spontaneous breathing in the vented patient with potential consequences

Score	Term	Description
+4	Combative	Overtly combative or violent; immediate danger to staff
+3	Very agitated	Pulls on or removes tube(s) or catheter(s) or has aggressive behavior toward staff
+2	Agitated	Frequent nonpurposeful movement or patient–ventilator dyssynchrony
+1	Restless	Anxious or apprehensive but movements not aggressive or vigorous
0	Alert and calm	
−1	Drowsy	Not fully alert, but has sustained (more than 10 s) awakening, with eye contact in response to voice
−2	Light sedation	Briefly (less than 10 s) awakens with eye contact in response to voice
−3	Moderate sedation	Any movement (but no eye contact) in response to voice
−4	Deep sedation	No response to voice, but any movement in response to physical stimulation
−5	Unarousable	No response to voice or physical stimulation

Procedure

1.	Observe patient. Is patient alert and calm (score 0)?
	Does patient have behavior that is consistent with restlessness or agitation (score, +1 to +4 using the criteria listed under Description)?
2.	If patient is not alert, in a loud speaking voice state patient's name and direct patient to open eyes and look at speaker.
	Repeat once if necessary. Can prompt patient to continue looking at speaker.
	Patient has eye opening and eye contact, which is sustained for more than 10 s (score, −1)
	Patient has eye opening and eye contact, but this is not sustained for 10 s (score, −2)
	Patient has any movement in response to voice, excluding eye contact (score, −3)
3.	If patient does not respond to voice, physically stimulate patient by shaking shoulder and then rubbing sternum if there is no response to shaking shoulder.
	Patient has any movement to physical stimulation (score, −4)
	Patient has no response to voice or physical stimulation (score, −5)

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Let's imagine a hypothetical scenario to get into the details on this: we pick up a vented human from a hospital; it's obviously uncomfortable and out of synch with the vent, so we address ventilation and oxygenation (per prior discussions) and then give our preplanned analgesia/ sedation combo and are on our way. Now we are cruising along, referring back to our chosen sedation scale reference card to find that our patient is becoming more uncomfortable - what do we do? Most obvious is pharmacologic intervention, that's often what we reach to first and is a totally acceptable move. But there are other things we can do on the machine that may not have the negative consequences/ adverse effects that the drugs do.

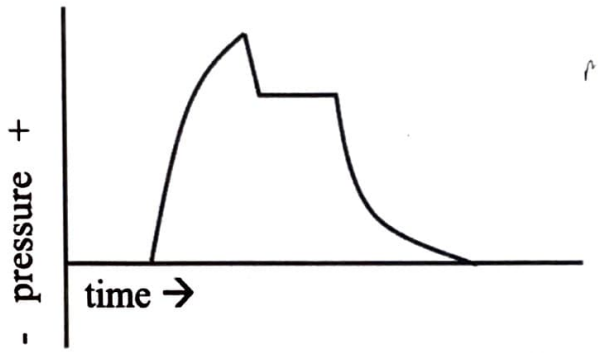
One parameter that we've discussed previously is I-time – occasionally a minor adjustment here can make a patient feel more comfortable. Not sure there's any evidence on this beyond the anecdotal, but as long as we aren't making large adjustments that impact other values, we should be good to experiment here. Switching modes may also help in this situation. We mentioned this already, but breaths are delivered differently in different modes and sometimes one may feel better to the patient for whatever reason. And lastly we can consider adjusting our **Triggers** to make it easier for the patient to take a breath when (s)he wants - more on that to come.

So last summary here and we'll include all three of these super-duper important parameters that we need to address on all of our patients, hands down and no matter what. Comfort should be assessed using an actual scoring tool and can be fixed with both drugs and vent manipulations. Oxygenation is measured by SpO_2 and gets fixed by increasing FiO_2, adding PEEP, and lengthening I-time/ Ventilation is evaluated by looking at MV (comparing it to a calculated goal) and $EtCO_2$, we make adjustments to RR and TV to manage ventilation; increase TV and then RR to increase MV, decrease RR and then TV to decrease MV.

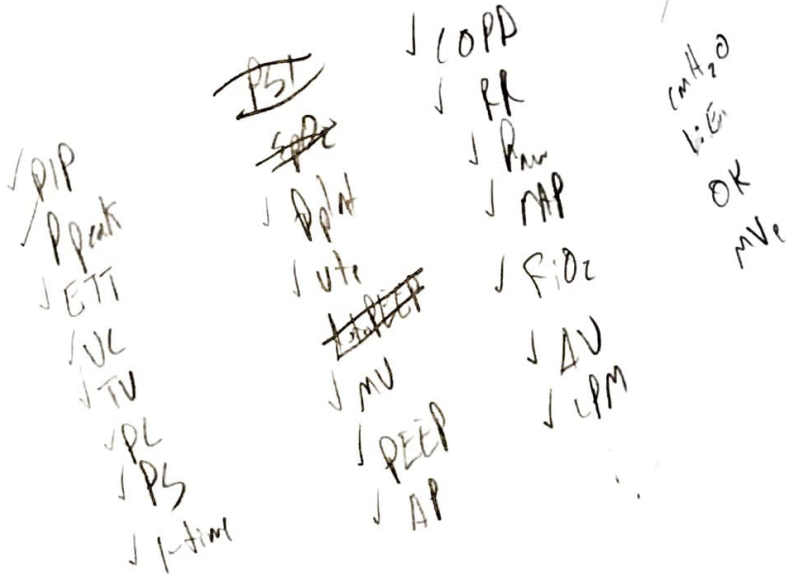
Return to Contents

Vent Parameters, Round Two

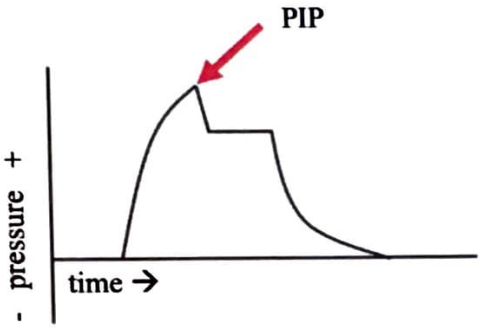
This next section discusses a few more vent parameters that we measure after the initial setup or taking over of a vented patient. They are considered separately than the values previously discussed because they are dependent on other things – we ~~can't typically~~ dial them into the machine, but rather we measure them to assess how things are coming along with the values we were able to control. To help clarify these ideas, which are all interrelated, let's refer back to an image we previously discussed. It shows pressure we put into the system over time as a breath is delivered in volume control ventilation:



We previously used this graphic to demonstrate a ~~few concepts in general~~, but it is now worth mentioning that this waveform and the two subsequent concepts (peak inspiratory pressure and plateau pressure) apply to ~~volume control~~ ventilation. Let's first get things clarified for ~~volume control~~ ventilation and then we'll talk about how these concepts carry over into ~~pressure control~~ ventilation.



Peak Inspiratory Pressure[90]



Peak Inspiratory Pressure (PIP) is the highest point on this waveform. It represents the maximum pressure as we deliver a breath into the system. It is also known as peak pressure (Ppeak). PIP is a function of both how we deliver a breath via the machine and how easily that breath can get from the machine down to the alveoli. A normal PIP is <35cmH_20. An isolated PIP that is too high generally won't cause damage to the patient, rather it likely indicates something gone wrong in the system. This is particularly relevant when we have a normal PIP that then become elevated – in these cases it is important to seek out the cause and fix the underlying issue.

On the machine end, PIP is the result of flow, which (recall from our section on Types of Breaths) essentially describes how fast we push air to achieve a breath. We generally don't manipulate flow directly on transport ventilators, so to decrease PIP by pushing buttons on the machine we have to make things happen in a roundabout way. Which isn't ideal and it gets complicated and the truth of it all is that most of the PIP issues we face are due to pathophysiology or equipment issues, so let's just skip right on ahead to how we can decrease PIP via other mechanisms outside of the vent itself.[91]

Causes of an elevated PIP include ~~many different~~ things like secretions in the ETT tube, bronchospasm, patient discomfort, mainstem intubation, pneumothorax, pulmonary edema, etc. Any time we see a high PIP we ought to try and identify a cause.[92] Once that cause is identified, then we can decide whether or not an action is needed. For example, a high PIP due to secretions should get suction and a high PIP due to a pneumothorax should lead to decompression; on the other hand, a high PIP due to a small ETT may be acceptable. The PIP in this case represents ~~pressure~~ due to the ETT and not the patient's anatomy, so we may decide to leave it alone (especially is there is good reason for that small ETT, such as airway swelling).

Another consideration here is patient comfort and the idea of laminar flow. Without getting too far into the weeds on this, recognize that air can move freely and efficiently through a uniform pipe or tube, but with movement or disruption to that tube airflow will be ~~less uniform and~~ more chaotic and ~~will~~ result in higher pressures. Keeping our patient comfortable and in synch with the vent leads to more uniform (i.e. efficient) air movement and lower PIPs. Morale here: make sure your vented patient is comfortable. And if you notice an increase in PIP, comfort ought to be one of the things to consider.

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[90] Nickson, 2019a – Short article that provides another good review of both PIP (this section) and Pplat (next section)

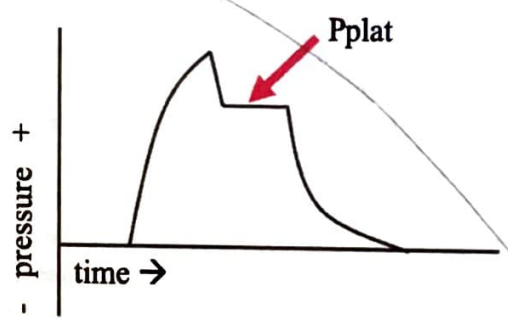
[91] But for the curious folks out there: in VC flow is determined from TV and I-time; in PC it is a function of pressure and I-time; with PS breaths it is a function of "rise profile" which we will hold off on discussing here

[92] And one part of how we do that is by assessing **Plateau Pressure** (next section) – and we have this all drawn out in a flowchart later, but first we need discuss all the terms and concepts first (see **Watching Pressures** if you just can't wait!)

To measure PIP we simply need to look at the vent display. Most machines will either give you the value of PIP or show a ~~little~~ barometer ~~of sorts~~ that fluctuates with each breath – PIP is always the highest value that comes up during a breath. Another way to keep an eye on PIP is by setting an alarm so that machine yells at you when a certain pressure is reached. ~~This is similar to the idea of setting your~~ SpO_2 ~~alarm during an RSI so that the monitor alarms when your patient desats and you know to stop the attempt and reoxygenate the patient.~~ That said, there is one critical difference with a high pressure alarm on the vent: yes it will tell you that the pressure has gotten too high, but it will likely (depending on model) also cycle off the breath it is giving in response to that high pressure alarm. This can potentially kill your patient and we will get in to that a bit more later on.[93]

So in summary, PIP represents the maximum pressure as a breath is delivered by the machine. A normal value is <35cmH_20 and we measure it by looking at the feedback on the vent interface. Potential causes include too much air, too much flow, small ETT, kinked ETT, patient discomfort, secretions, pneumothorax, mainstem ETT placement, bronchospasm, ~~and decreased compliance~~. While there are subtle ways to address PIP on the vent, interventions should focus instead on airway issues and comfort.

Plateau Pressure



Plateau pressure (Pplat) is the pressure in the system once the lungs fill with air and before the breath cycles off. It represents the average pressure at the alveoli as they are at maximum inflation during inhalation. A normal Pplat is less than 30cm H_20. Values higher than that can lead to direct damage to the alveoli which can subsequently cause issues with the whole respiratory process. There is no "too low" for Pplat but recognize that lungs that aren't being filled all the way (i.e. a low Pplat) may not be maximizing the surface area of alveoli and therefore oxygenation may not be at its best. And we will discuss this concept later on.[94]

The primary cause of a high Pplat at the start of ventilation is too much tidal volume. That said, it can also be present or develop over time due to patient discomfort, ~~decreased compliance,~~[95] pneumothorax, mainstem migration, and inhibition of chest wall expansion (such as in burns).[96] If we get a high Pplat, it is worth considering these other causes (and addressing them appropriately!) before dialing down TV, as we don't want to give up ventilation unnecessarily.[97] We do, however, want to avoid a sustained high Pplat over many breaths, as that will likely lead to damage to the alveoli.

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[93] Conveniently enough, this is in the section on **Alarms**

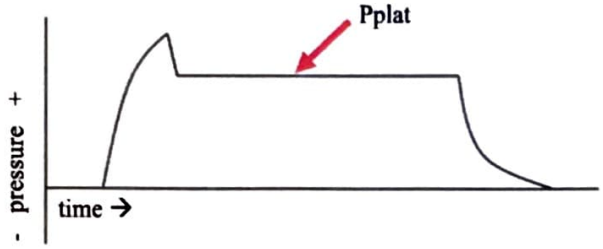
[94] See **Titrating Up on ~~Vte~~ VT**

[95] ~~Covered later in the section on **Compliance (and Resistance)**~~

[96] Recognize that some of these were also on our list of causes for high PIPs, but not all of those high-PIP things will cause a high Pplat

[97] And we will revisit this idea in an algorithmic fashion in the section called **Watching Pressures**

Measuring Pplat is little less direct that measuring a PIP and involves what we call a "maneuver." There are two maneuvers that we will discuss and this is the first of them. While we could theoretically watch the barometer on the machine and wait for that point during inspiration where pressure stays constant for a short spell, that amount of time is quite short and this is logistically difficult to accomplish. The workaround is to prolong inspiration via a maneuver called an "inspiratory hold" and allow the machine to measure that pressure accurately. It would look something like this:



We perform the inspiratory hold maneuver (in whatever way is appropriate for our particular machine) and the Pplat either pops up on the screen for us or we have enough time to read the value from the barometer. Easy enough, but when and how often do we do this thing? There isn't a universally accepted frequency for measuring this (or any of the other pressures discussed in this section), but it seems to make sense that we just add them on to our reassessment of vital signs (so every 5-15 minutes, depending on the program and patient acuity). While that may be overkill, it's better to measure too much than to miss things due to not checking often enough. At a minimum, Pplat should be measured after any increase in TV to make sure that we don't cause alveolar damage (and this includes after first putting the patient on the vent).

One last thing about Pplat is that the value we get is essentially an average of alveolar pressures across the lung. Some regions will experience higher pressures, others will experience lower pressures. The lung is not uniform throughout, but we can't rightfully measure alveolar pressures in specific lung regions or see to what degree this value would vary across the different parts. The safe limit of <30cmH_2O is a good guideline by which to limit our vent settings, but recognize that this doesn't mean that a pressure higher than that to one alveolus or a region of lung will always cause harm. Likewise, a Pplat <30cmH_2O is not a guarantee that damage will not be caused to some region of the lung.

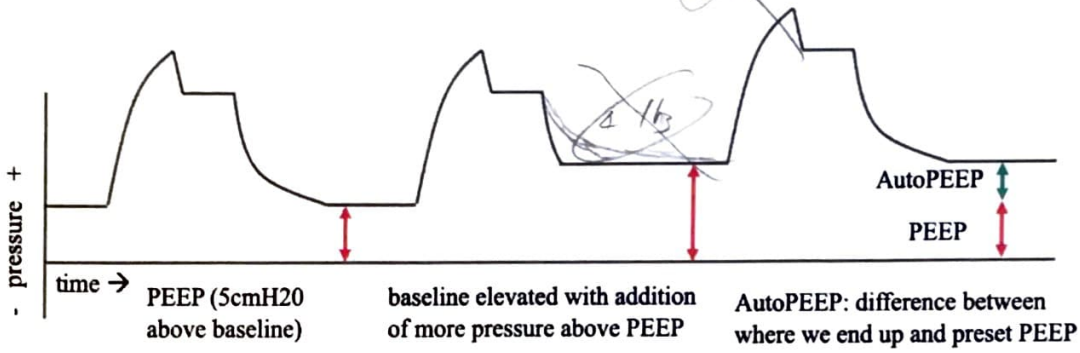
In summary, Pplat is the pressure seen by the alveoli when we deliver a breath in volume control ventilation. A normal value is <30cmH_20 and we measure it by performing an inspiratory hold maneuver. While there is no bottom limit to Pplat, it is important to recognize that we want to fill the lung and alveoli up with each breath delivered, so be wary of a super low Pplat and consider inadequate TV (and subsequently MV). High Pplat can be caused by too much TV, pneumothorax, restriction to chest wall expansion, mainstem intubation, and a few other things that we'll spell out later on.[98]

[98] See section on Watching Pressures

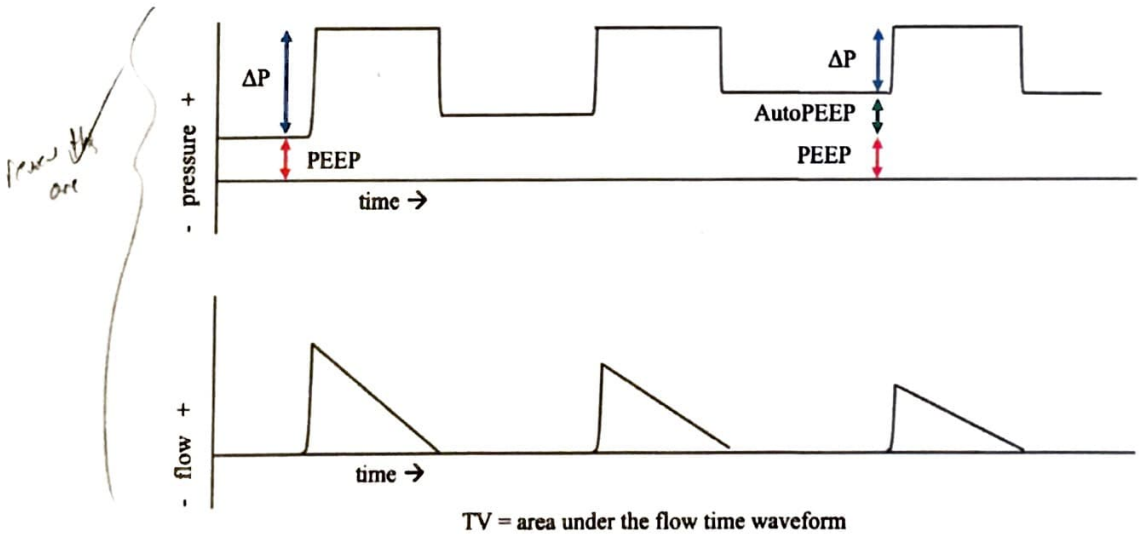
Return to Contents

AutoPEEP

AutoPEEP is the idea of PEEP being cumulatively added into the system inadvertently. Remember how we said before that we assume atmospheric pressure to be 0cm$H_2$0 as the starting point for our vent discussions and that PEEP is the addition of pressure on top of that (i.e. "adding 5cm of PEEP" to reset that baseline to 5cm$H_2$0)? Well, AutoPEEP is when that baseline starts to creep up from whatever we have set as PEEP to higher values because the patient isn't able to exhale all the way back to baseline before the next breath comes around. This idea is commonly referred to as "breath stacking" and might be represented like this:



Normal AutoPEEP is zero, i.e. we shouldn't have any AutoPEEP in the system at all. Presence of AutoPEEP in volume control can lead to an increase in other airway pressures, most importantly of which is Pplat; AutoPEEP in pressure control can result in decreased VTe and MV:

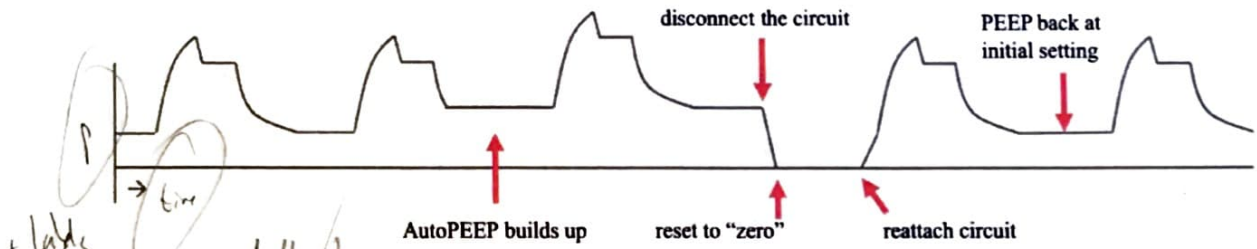


TV = area under the flow time waveform
less ΔP = less flow required = less TV

To measure AutoPEEP or to check its presence, we have to perform another maneuver called an expiratory hold.[99] Just as with the inspiratory hold for plateau pressure, doing an expiratory hold allows us to accurately see what the ~~real-time~~ pressure is when we expect the breath to have returned to baseline. Normally the machine will calculate an AutoPEEP for us by subtracting PEEP from whatever pressure it measured during the hold.

If we do have AutoPEEP this means that something is getting in the way of the patient exhaling all the way back to baseline before a subsequent breath is delivered. This could be due to patient discomfort or need for more MV, but it can also be due to obstructive processes that get in the way of effective exhalation (i.e. asthma and COPD) or even inadvertent triggering of breaths. The fix on the vent interface would be to shorten our I-time or decrease RR to increase the I:E ratio and allow more exhalation; otherwise we could consider more sedation/ pain control and make sure we aren't accidentally triggering.[100]

One other thing we can do to eliminate AutoPEEP and reestablish our baseline at actual PEEP is disconnect the patient from the vent circuit to allow a full and complete exhalation. This is one of those rare cases in which it is OK to disconnect the vent circuit from the patient during transport for therapeutic reasons. Simply allow the patient to exhale and then reattach the circuit (and most likely cancelling out a bunch of alarms in the meantime!). Just to make sure we understand how this works, let's draw it out as a waveform over time and label things along the way:



To bring it all home, AutoPEEP is a movement of the pressure baseline above whatever we have dialed in for PEEP. Issues with this are increased pressures (volume control) or decreased volumes (pressure control). Causes ~~include~~ inability to exhale fully, ~~agitation~~, and inadvertent triggering. Fixes include extending the amount of time spent in exhalation (shorter I-time,[101] lower RR), treating discomfort, and avoiding accidental triggers. In addition, we can reset AutoPEEP back to zero by temporarily disconnecting the vent circuit.

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[99] There are other ways to check for AutoPEEP, but they aren't typically available in transport unless we have access to scalars or waveforms

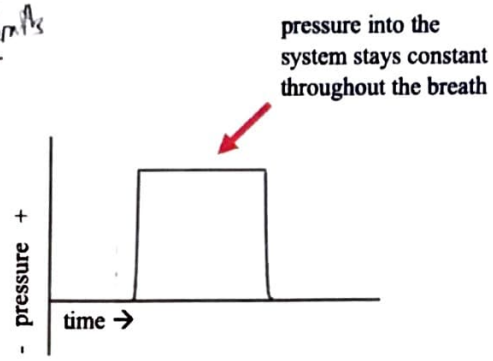
[100] There is also some discussion out there about using applied PEEP to mitigate AutoPEEP, but we will get to it when we discuss the **Obstruction** strategy

[101] And we mentioned in passing before that using VC ventilation may help us get to shorter I-times due to the nature of the "square wave" flow pattern – that was in **Types of Breaths** and we'll get back to it again in **Obstruction**

Return to Contents

PIP & Pplat in Pressure Control?

Up to this point we've discussed PIP and Pplat mostly in the context of volume control ventilation, but things are a bit different in pressure control. Let's start with what a pressure control breath looks like mapped out as pressure over time:



First thing to mention here is that PIP will only be above that flat line at the top of the square wave form (marked by the red arrow in the graphic) if something causes a disturbance in what the machine is doing – a hiccup, patient movement, speedbump, etc. The machine won't intentionally put more pressure than what we have dialed, but a PIP higher than the set pressure control can occur. So while we may still set a high pressure alarm and monitor PIPs in PC ventilation, our concern is more for being aware of disturbances to the system rather than being aware of changes to air flow (i.e. obstruction), as was the case in VC ventilation.[102]

Next thing: it generally happens that the average alveolar pressure eventually does equal that pressure represented by the top of the square waveform (towards the end of expiration), therefore we assume it to be true that PIP equals Pplat.[103] And because of this assumption that mostly holds true, it's OK that some machines don't let us do inspiratory holds in pressure control ventilation, as the data gleaned from the test just wouldn't provide any additional information. And also because the primary reason we want the Pplat (in volume control) is to rule out high alveolar pressures (to ensure the safety and wellbeing of the alveoli); in pressure control if Pplat doesn't match pressure control it's because true Pplat is less that the pressure control (which is a bummer, but not a safety concern for the alveoli).

Now the mechanism of it all is that it takes time for the alveolar pressure to rise up to match the pressure going into the system. Even though we start with a high pressure at the machine end of the system, it may take some time for that pressure to equalize down to the alveoli. If our I-time isn't long enough to allow that to happen, the alveolar pressure ~~(or plateau pressure)~~ may not ever get up to the level we have set for pressure control. We work around that in volume control by performing an inspiratory hold and waiting for as long as we need to in order to see that pressure even out. We don't always do that in pressure control because, as we said just a moment ago, the plateau pressure won't be above our pressure control value and so there isn't so much of a safety concern.

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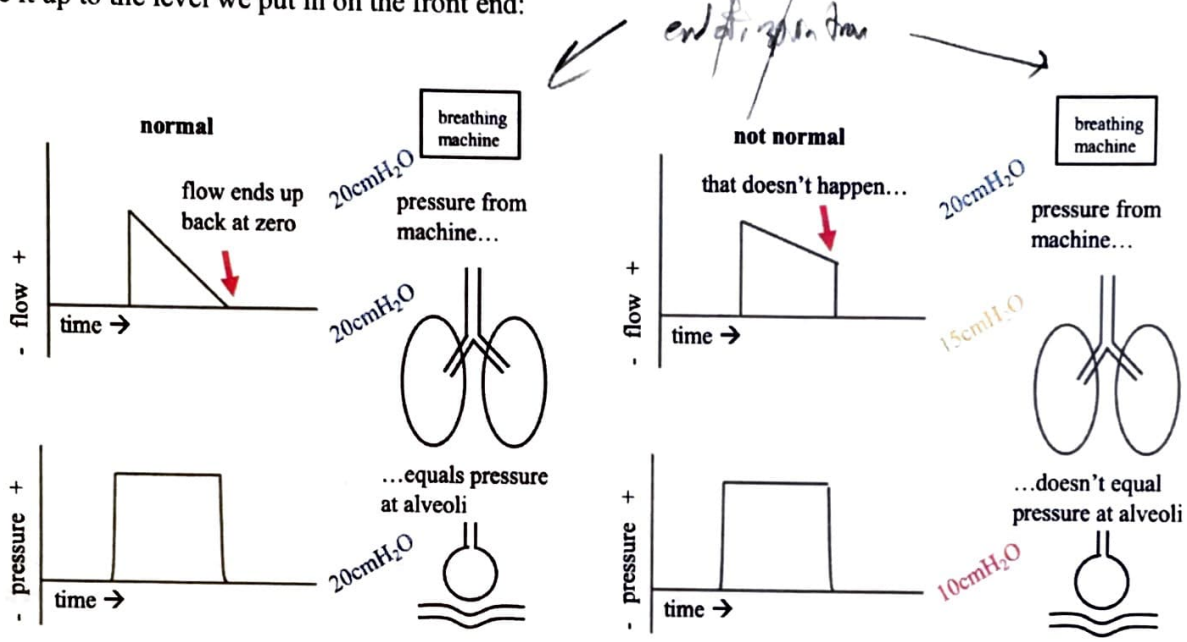
[102] In PC ventilation, we become aware of those obstruction issues by monitoring VTe and maybe flow (if available on our particular machine)

[103] Hess, 2014 – Another way to say this is that if flow gets to zero during the inspiratory phase, then PIP = Pplat

But if we wanted to know a little more about what's going on in the alveoli and we can't do an I-hold on our machine in pressure control, we can get a partial picture of things by looking at flow. Pressure control breaths start with a higher flow that then drops down towards zero flow throughout the breath. While it may be hard to see with quantitative values on your machine (unless you can view waveforms), if flow doesn't get down to zero before the breath cycles off, then we can consider that the pressure in the alveoli may not have made it up to the level we put in on the front end:



All that said, this isn't a great method unless you have waveforms to look at. And even then it's a binary thing – it says whether or not alveolar pressure got up to the value of pressure control, but it doesn't tell us what the alveolar pressure actually was. There are other ways to measure or approximate Pplat, although they are unlikely to be available to us in the transport setting.[104]

So what utility is there in knowing alveolar pressure (Pplat) in pressure control anyways? We said already that the usefulness of this information in volume control is to guarantee safety of the alveoli, but that isn't an issue in pressure control. Potential uses of knowing a Pplat in pressure control would be making sure our I-time is appropriate (i.e. that the inspiratory time is long enough to allow pressure going in to match pressure at the alveoli) and calculating things like compliance and driving pressure (both discussed later).[105] These are all cool things to work with, but it takes both time and effort and, therefore, may not be the best use of one's cognitive capacity when managing a sick patient in the transport setting. We will discuss this stuff, but know that Pplat is primarily a tool for ensuring alveolar safety in VC ventilation.

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[104] Mojoli & friends, 2015 –This short paper assesses the efficacy of these alternative methods of measuring Pplat (and also delta pressure)

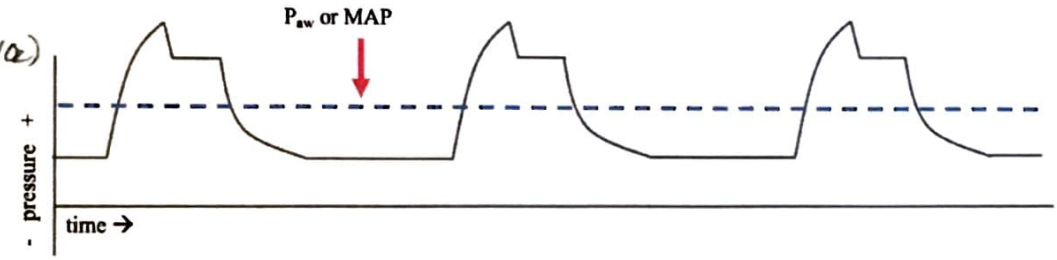
[105] In the sections **Compliance (and Resistance)** and **Driving Pressure**

Return to Contents

Mean Airway Pressure

Last pressure to talk about is mean airway pressure. It's typically represented as P_{aw} (stands for airway pressure) and less often as MAP (mean airway pressure). P_{aw} is the average pressure in the system throughout the respiratory cycle. There are formulas to estimate P_{aw},[106] but it's probably easiest to just read off of your machine (assuming it's there). We don't often use this pressure to guide treatment, but if we notice changes in the mean airway pressure we can then look into details as to what changed in the system to ~~result in that~~ outcome. For example, a high P_{aw} can result from all sorts of things, each of which is a totally different issue: an increase in either PIP or Plat, the presence of AutoPEEP, and increased rate. And same thing on the opposite end, lots of things can cause P_{aw} to drop and we then must work to identify a specific cause.

One other thing about P_{aw} is that it is strongly correlated with oxygenation.[107] Back in our discussion of Oxygenation (& SpO_2) we talked about how PEEP and I-time contribute to improved oxygenation. More of either of these things leads to a higher P_{aw}, so it can help to think of oxygenation in terms of this pressure and FiO_2. Just recognize that too much of this good thing can turn bad (i.e. too much pressure can have bad outcomes, as previously discussed). And while we commonly separate oxygenation out into multiple concepts (as we did previously), it may be worth keeping this in mind as we look for trends in patient presentation.



[106] Mentioned in passing in the section on Hypotension, then demonstration via calculations to justify that strategy in the Appendix

[107] Loderserto, 2018 – ~~Succinct~~ explanation of this relationship between P_{aw} and oxygenation

Compliance (and Resistance)[108]

Compliance is a measure of how much the lungs fill per unit of pressure put into the system. In math terms is looks like this:[109]

$$\text{compliance} = \frac{\Delta V}{\Delta P} = \frac{\text{TV or VTe}}{(\text{Pplat} - \text{PEEP})}$$

While a normal compliance (healthy and breathing spontaneously) is somewhere in the neighborhood of 100ml/cmH_2O, we often see values much smaller than that in our ventilated patients. The best way to utilize compliance during transport is to keep track of trends: increasing compliance is good, decreasing compliance is bad. If we do something that results in poorer compliance, maybe second guess whatever that change was; if we do something that results in better compliance, high fives are warranted. Acute causes of decreased compliance would be a worsening pneumothorax, inhibition of chest wall expansion, chest wall rigidity caused by certain medications, increasing VT or PC beyond the capacity of the lungs at that given time, etc.[110]

A related term is resistance. Resistance and compliance are often discussed together under the umbrella term of "respiratory" or "pulmonary mechanics" – that's why we talk about it here. Now the algebraic expression of resistance isn't quite as straight forward as for compliance and we often simplify it by making the assumption that flow equals 60LPM, so we're just going to skip on ahead and note it like this:

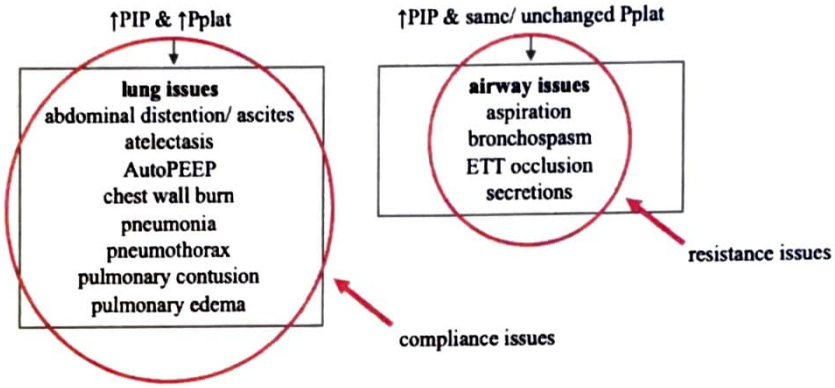
$$\text{resistance} = \text{PIP} - \text{Pplat}$$

[108] Trainor & friends, 2019 – This video reviews both of these concepts in a very succinct and straightforward way

[109] And to be more specific, this is what we would call static compliance and reflects changes at the alveoli; we won't get into dynamic compliance here

[110] And all of these high PIP, high Pplat situations will be discussed in the section on **Watching Pressures**

Resistance, in this simplified manner, is the limitation to air movement that must be overcome in order for us to arrive at a state in which air in from the machine gets to the alveoli. Assuming Pplat remains constant, resistance is represented by PIP. This means that we can approximate changes to PIP to signify changes to resistance. So things like kinks in tubing, biting on the tube, excessive secretions, etc. that are ~~potential~~ causes of increased PIP and unchanged Pplat correlate with an increase in resistance:[111]



And we mentioned already that the alternative strategy in PC ventilation when we don't have PIP or Pplat to guide us is to look at VTe and MVe to gauge when these ~~types of~~ things are happening (a drop in volume will indicate a change to resistance or compliance). We can also look at a quantitative value for compliance (if available to us on whatever machine we are working with) or see how flow is changing from breath to breath (most transport vents automatically adjust flow with changes to resistance and compliance; less flow equals more resistance and/ or less compliance).

[111] Cassone & friends, 2019 – And we will expand on this in **Watching Pressures**

A General Vent Strategy

In this section we are going to summarize general parameters in each type of ventilation (i.e. each combination of mode and control). ~~The idea here~~ to demonstrate what settings and goals are shared among all methods and which are specific to certain types of ventilation. This general strategy is similar to what is often described as a "lung protective" strategy that first came on the scene in regard to management of patients with Acute Respiratory Distress Syndrome – we've opted to present the two as distinct strategies and we'll come back to this idea when we get there.[112] We will also hash out a few of the differences in determining general settings for adults versus pediatrics. Let's start with a discussion of things that apply to most vented patients, regardless of mode or control:[113]

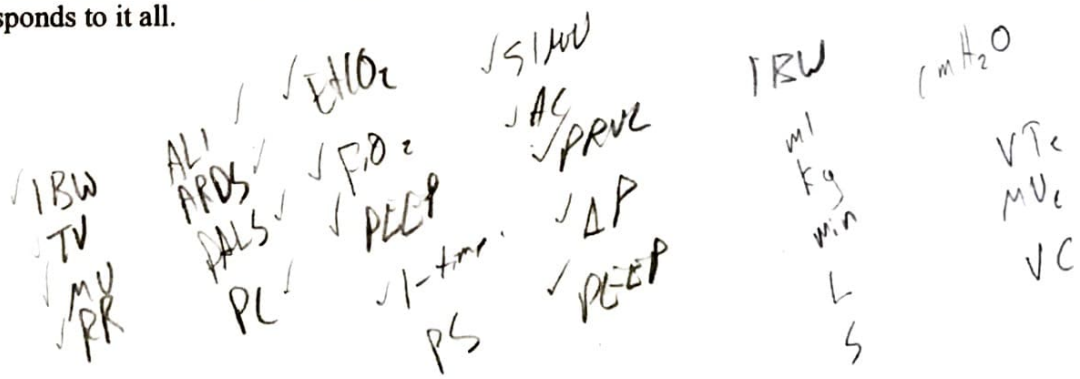
$$TV = 6 - 8ml/kg\ (IBW)$$
$$MV = 100ml/kg\ (IBW)\ /min$$

If we choose a TV of 6ml/kg and our goal is 100ml/kg/min, then our calculated rate is 17:

$$MV = RR \times TV$$
$$100ml/kg/min = RR \times 6ml/kg$$
$$100\cancel{ml/kg}/min \div 6\cancel{ml/kg} = RR$$
$$\sim 17 = RR$$

Likewise, if we go with 8ml/kg our initial rate (to match that MV goal) comes to 13 per minute. Although it's not uncommon to see recommendations for an initial rate of 10 to 12 with adults, calculating a RR based on a MV goal is our preferred strategy. There are often good reasons to use a lower RR, but we'll get to those later.

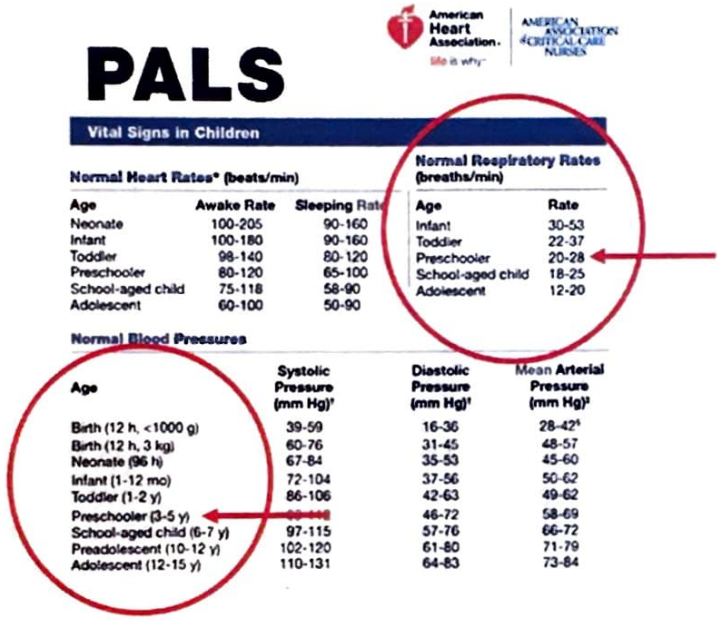
Moving forward, if we have a range of TVs to choose from, sometimes it just makes life easier to pick a nice, even number. For example, with an 80kg patient we end up with a TV goal range of 480-640ml and a MV goal of 8L; it's a totally legit move to choose 500 or 600 or any value in that range. Just recognize that if we pick a higher value for TV, we may want a lower value for RR just to keep our MV approximately the same. This does not have to be exact, as we will adjust these settings as we go and work towards our goals moving forward. So we may choose a TV of 500 and a RR of 16 (for a calculated MV of 8L). Or a TV of 600 and a RR of 14 (for a calculated MV of 8.4L). Either is cool for now and we'll dial in our settings once we see how the patient responds to it all.



[112] That will happen in the section on **ALI/ ARDS**

[113] Note that some patients do require different goals and we will discuss those shortly in **Specific Vent Strategies**; also, refer back to sections on **Tidal Volume** and **Minute Volume** for a ~~discussion~~ of these suggestions

As for kiddos, the ~~preferred strategy~~ approach is to choose a rate in line with a reference card and disregard the above suggestion of 13-17/min. While this will result in an overestimation of MV,[114] we can titrate values to address that later on. For example, let's assume a 4-year-old kid of 18kg. Based on this chart (again, from PALS) we want a RR in the 20-28/min range:[115]



PALS

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)†
Birth (12 h, <1000 g)	39-59	16-36	28-42‡
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	[illegible]	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

You can also use this chart based on the PALS data:[116]

Age Description	Age (yrs)	RR	I-time (s)
Infant	.083 (1 month)-1	30-53	0.3-0.6
Toddler	1-2	22-37	0.4-0.9
Preschooler	3-5	20-28	0.5-0.9
School-aged Child	6-7	18-25	0.6-1.1
Big Kiddos	8-9	17-25	0.6-1.2
Preadolescent	10-12	14-23	0.7-1.4
Adolescent	12-15	12-20	0.8-1.7
Adult	16 and up	12-20	0.8-1.7

per JA | ABW

[114] Because TV (or TV goal in PC) stays the same

[115] American Heart Association, 2016 (image)

[116] And see **Appendix** for an explanation of the amateur mathing that got us to this chart

And let's take these values and do a few calculations as so:

TV = 6 – 8ml/kg IBW
TV = 6 – 8ml x 18kg
TV = 108 – 144ml

MV goal = 100ml/kg (IBW) /min
MV goal = 1800ml/min
MV goal = 1.8L/min

MV calculated = RR x TV
MV calculated = (20 – 28)/min x (108 – 144)ml
MV calculated = 2160– 4032ml/min
MV calculated ≈ 2 – 4L/min

The result here is a MV goal that differs pretty significantly from the calculated MV, but what to do with this information? We will eventually want a MV (preferably measured as "exhaled") that matches our quantitative goal of 100ml/kg/min and also gives us an $EtCO_2$ in the normal 35-45 range, but let's start with 6-8ml/kg anyways and work towards that goal in the first little while after starting ventilation. This overestimation is particularly important and maybe even lifesaving if you decide to ventilate a kiddo in volume control mode. There is always some dead space that we introduce into the system and this overestimation will help to mitigate that.[117]

[117] To see this all spelled and drawn out in detail, refer to **Appendix**

Return to Contents

So we have TV, MV and RR all sorted, both for big people and small people; next we need to consider the other parameters that are constant between modes and control methods, then we will talk specifically about those things. Let's put it into a chart just to make it easier to visualize. And this chart is basically a summary of the section **Vent Parameters, Round One** – for review of the specifics of any of them, just refer back to that bit:

Parameter	Value	Pro Tips
TV	6-8ml/kg	Pick an easy number to work with that falls in that range
MV	100ml/kg/min	Just take IBW in kg and move the decimal over (75kg IBW = 7.5L MV goal)
RR	Adult: 13-17/min Kiddos: use a chart	Carry a reference card or have an app on a device[118] to quickly reference the pediatric values
FiO_2	1.0, then titrate down	You can titrate down in big jumps also, no need to go in small increments unless you have good reason to do so[119]
PEEP	5-6cmH_2O	For most vents this will be whatever the machine defaults to
I-time	Adult: 0.8-1.7 Kiddos: use a chart	Normal for the adult is 1.0

[118] Pedi STAT – Great resource for quickly referencing pediatric doses and equipment sizes

[119] Weingart, 2010; Lodeserto, 2018 – Both recommends starting at 100% and then dropping down to 40% to see how the patient does – we can always titrate back up if need be, but if all is well we just leave it there (or even keep titrating down)

Next step is to look at what extra parameters need dialed in on the machine depending on which mode and which method of control we choose for our patient. As we said before, we can ventilate any patient in any mode and via any method of control, so long as we know what to monitor for depending on what we choose. And if you are ventilating a patient in PC or SIMV (with PS), it's OK to just start out with the defaults on whatever machine we are working with and then titrating from there as long as we do so in a timely fashion and with our ventilation goals in mind. Let's draw it all out in another chart:

	Additional Parameters[120]
AC Volume	None
SIMV Volume	***Pressure Support*** – start at 5-10mmH_2O and titrate as needed
AC Pressure	***Pressure Control*** – start at 10-15cmH_2O and titrate to TV goal
SIMV Pressure	***Pressure Control*** – start at 10-15cmH_2O and titrate to TV goal ***Pressure Support*** – start at 5-10mmH_2O and titrate as needed
AC PRVC	***"Pressure Cap"***[121] – set to 25-30cmH_2O (often by setting high pressure limit to 5cmH_2O above what we want this to be)
SIMV PRVC	***"Pressure Cap"*** – set to 25-30cmH_2O (often by setting high pressure limit to 5cmH_2O above what we want this to be) ***Pressure Support*** – start at 5-10mmH_2O and titrate as needed

[120] It's a bit tough to identify specific starting points for both PC and PS in the literature and recommendations vary a lot, but these are points to start off at and then we should always titrate towards VTe and MVe goals as soon as possible. As for more insight into these initial settings:
Ashworth & friends, 2018 – They say start with PC at 5-10cmH_2O and limit ΔP (Pplat or PC – PEEP, which we will discuss later on **Driving Pressure**) to 16cmH_2O (which correlates with an additive PC of that amount – 16cmH_2O)
Kneyber & friends, 2017 – They recommend limiting a ΔP to 10cmH_2O for all (pediatric) patient types
Nagler & Chiefetz, 2019 – They suggest a starting PS of 5-10cmH_2O for kiddos
And just to be clear, all the pressures listed here (for PC and PS) are additive, not cumulative (and for a refresher on what that means, head back to **Types of Breaths**)

[121] Recall that this is a made-up term and is typically represented by 5cm less that what we set as the high-pressure limit



At the expense of being overly redundant, let's combine the last two charts into another one to summarize how we determine vent settings, in general and for the "normal" patient:

Step One: Set &/or Calculate		**Step Two: Make a Choice and Dial in Extra Stuff**	
TV[122]	6-8ml/kg	**AC Volume**	None
MV	100ml/kg/min	**SIMV Volume**	***Pressure Support*** – 10mmH_2O
RR	Adults: 13-17/min Kiddos: use a chart	**AC Pressure**	***Pressure Control*** –10-15cmH_2O
FiO_2	1.0, then titrate down	**SIMV Pressure**	***Pressure Control*** –10-15cmH_2O ***Pressure Support*** – 10mmH_2O
PEEP	5cmH_2O	**AC PRVC**	***"Pressure Cap"*** – set to 25-30cmH_2O (normally: set high pressure limit to 5cmH_2O above what we want this to be)
I-time	Adult: 0.8-1.7 Kiddos: use a chart	**SIMV PRVC**	***"Pressure Cap"*** – set to 25-30cmH_2O (normally: set high pressure limit to 5cmH_2O above what we want this to be) ***Pressure Support*** – 10mmH_2O

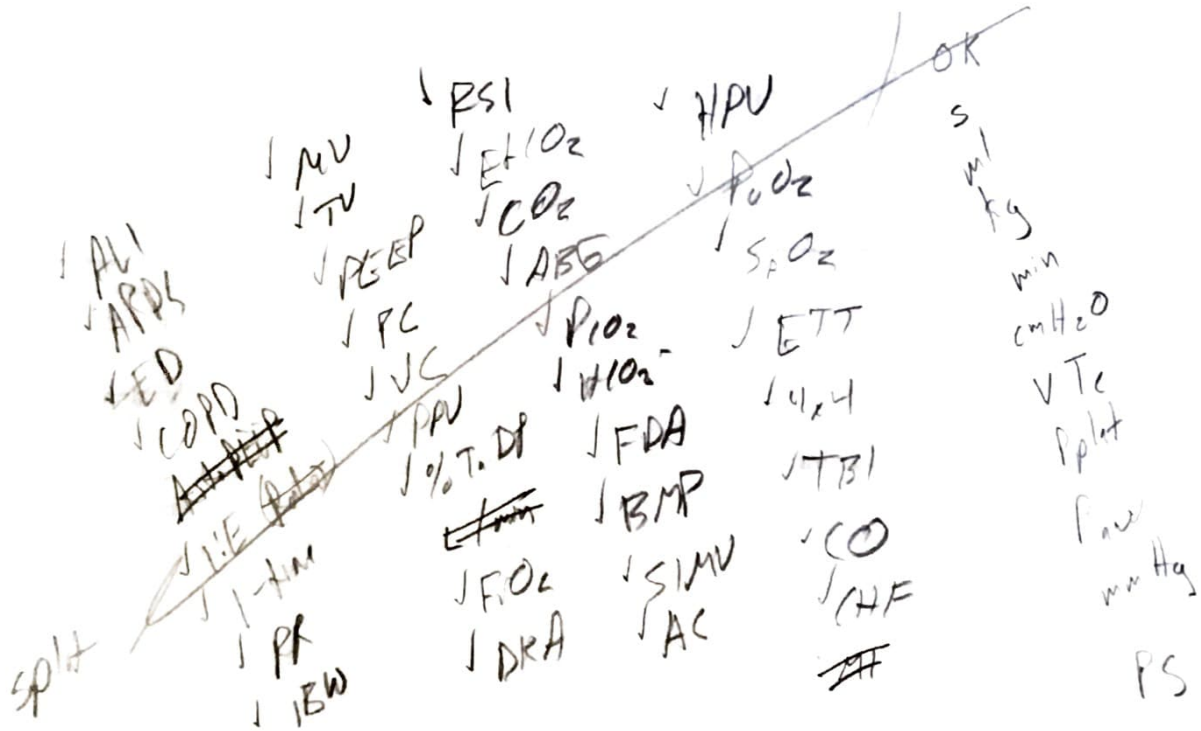
In the ideal world, that's how we get vent settings for a specific patient. In the actual world we have a few things to consider (and we'll frame them as questions): What pathophysiological changes affect the way this patient should be ventilated? What do we do with a patient already being ventilated if settings don't match what we come up with? How does this individual's body respond to all my theoretical stuff? The next few sections will answer these questions in turn. We will first look at specific situations that warrant alterations to this settings framework, then we will talk about setting up them vent in any scenario, and then we will ~~discuss~~ how to evaluate an individual's response to what we are doing with the machine and how we might adjust things to make him or her as happy as possible.

[122] In PC we don't actually set this guy, but we do need to have this value in mind and calculated out so that we can use it as a goal

Specific Vent Strategies

Now we have a chart that basically summarizes the initial calculations and choices we need to make for the average patient and depending on which type of breaths we want to deliver. Next step is to look at exceptions to the norm. To say it another way: sometimes a patient needs their breaths delivered in a specific way (different to what we ~~might call "normal"~~) due to a specific pathology. We sometimes take those normal parameters and alter them to meet specific needs and issues. It's totally OK to break the rules we've established so far, as long as we know when and how to do it and can justify a good reason. We will look at a few situations/~~etiologies~~ in turn to see how it all looks.

Vent strategies are often presented as a choice of two distinct categories: the "injured ~~lung~~" or "baby lung" approach and an "obstructive strategy." We've opted to present this decision making process as a set of five possible strategies from which providers can choose. First of those is the general strategy discussed just now, the other four include obstruction, hypotension, acidosis, and ~~what would be comparable to the "injured lung" approach~~. There is no right or wrong in this process, we just think it makes sense to take things a bit further as we have outlined in this following sections.[123]



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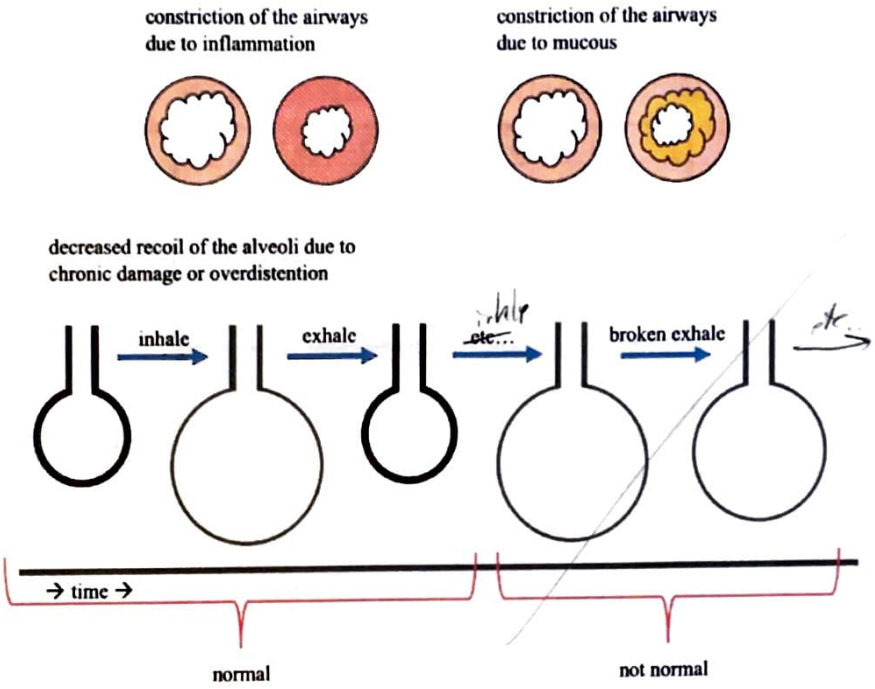
[123] To provide more context on this:
The Acute Respiratory Syndrome Network, 2000 – This was a major paper form ARDSNet that led the movement towards lower TVs with vented patients; while it focuses on a specific patient group (i.e. that "injured lung" cohort), it set the stage for further research into the idea of much lower tidal volume than were initially used
Weingart, 2010; Weingart, 2016b – A podcast series and paper, respectively and by the same guy, that outline this two-strategy approach to vent management; while directed towards ED physicians, the content is 100% applicable to those of us that work in the transport setting

Obstruction

In patients with asthma, COPD and/ or allergic reaction, we tend to run in to a problem of breath stacking or AutoPEEP because the patient is unable to exhale fully ~~in a normal amount of time~~. The pathophysiology is multifaceted and varies a bit depending on unlaying cause, but ~~they~~ can be summarized as some combination of the following:



~~Our~~ The fix ~~to this~~ is to adjust vent parameters to allow for more time at exhalation. We do this by extending or lengthening the I:E ratio. As we said before, a normal I:E ratio is 1:2-3 and we can adjust that by decreasing either the I-time or RR. In this patient population a good starting point is an I:E ratio of 1:5-6. The typical way to get here is to decrease RR (and also I-time) until we see an I:E ratio in that range that we want. The machine normally does this calculation for us, but just an example we'll show it all here:

With I-time 1.0s and RR 17:
60 ÷ 17 breaths ≈ 3.5s/breath
3.5s – 1.0s (I-time) = 2.5s
∴I:E ratio = 1:2.5

With I-time 1.0s and RR 13:
60 ÷ 13 breaths ≈ 4.6s/breath
4.6s – 1.0s (I-time) = 3.6s
∴I:E ratio = 1:3.6

With I-time 0.8s and RR 13:

$60 \div 13$ breaths ≈ 4.6s/breath

$4.6\text{s} - 0.8\text{s}$ (I-time) $= 3.8$s

$\therefore$ I:E ratio $= \frac{0.8}{0.8} : \frac{3.8}{0.8}$

I:E ratio = 1:4.8

So even if we drop both RR and I-time to the lower ends of our "normal" parameters, we end up with an I:E shy of what we want for these obstructed patients. Let's keep up with some of these calculations and put them all side by side:

I-time 1.0s		**I-time 0.8s**	
RR	***I:E***	***RR***	***I:E***
17	1:2.5	17	1:3.4
13	1:3.6	13	1:4.8
10	1:5	10	1:6.5
8	1:6.5	8	1:8.4

Now assume we choose an I-time of 0.8s and a RR of 8 (for a calculated I:E of 1:8.4), what does that do to our other parameters? Biggest thing that will be affected is MV. We'll do some calculations to demonstrate this impact on a 65kg IBW patient with a TV of 8ml/kg:

MV goal = 100ml/kg/min

MV goal = 100ml/kg/min x 65kg

MV goal = 6500ml/min

MV goal = 6.5L/min

TV = 8ml/kg x 65kg

TV = 520ml

MV calculated = TV x RR

MV calculated = 520ml x 8/min

MV calculated = 4160ml/min

MV calculated $\approx$ 4.2L/min

In fact, we'd have to go all the way up to a TV of 12ml/kg to get close to our MV goal:

TV = 12ml/kg x 65kg

TV = 780ml

MV calculated = TV x RR

MV calculated = 780ml x 8/min

MV calculated = 6240ml/min

MV calculated $\approx$ 6.2L/min

And at this point we run the risk of barotrauma or over-inflation injury (assuming a ~~volume control mode~~). That said, start at a TV of 10ml/kg and then titrate up if the patient's lungs allow for it (i.e. Pplat still below 30cmH_2O). If we can't reach our MV goal exactly, that's OK in the short term – we just want to try and get as close to it as possible while still allowing for full exhalation and avoiding the AutoPEEP issue.[124] We will simultaneously be doing pharmacological interventions (Albuterol, ~~Ipratropium~~, Magnesium Sulfate, Ketamine, Epinephrine – whatever ~~your~~ agency endorses) and hopefully the reason for this alternative strategy can get reversed to some degree and then we can go up on RR and work our way back to normal parameters.

In pressure control, we still drop the rate (and maybe I-time too) to lengthen I:E, but we also want as much volume per breath to try and get as close to our MV goal as possible. Instead of a PC at 10-15cmH_2O, consider going straight to the top and starting at 20-25cmH_20[125] to see what our VTe values look like. In addition, recognize that this Pplat upper limit is a generalization that may not be necessary for all patients, but we will expand on that more in pages to come.

Second to last thing to mention: it may be tempting to drop PEEP to zero in these cases to better allow the patient to exhale. The thought process goes like so: if they are breathing out while we are pushing air in, this has the potential to be problematic. That said, there is some thought that applied PEEP can help fix AutoPEEP, but we do want to keep applied PEEP lower than AutoPEEP. Just know that we may want to maintain PEEP at our minimum of 5cmH_2O to maximize oxygenation and help recruit more alveoli, but sometimes we let that go in order to avoid AutoPEEP. There may be a happy middle ground with a PEEP somewhere between zero and five, but there isn't much content on that and we'll leave it as a "maybe" in the overall scheme of things.[126]

Actual last thing to mention: if we have lengthened our I:E ratio to accommodate exhalation and we end up at a point where AutoPEEP is consistently zero, we can then titrate our I:E back to normal to make things more comfortable for the patient. This allows us to work back towards our MV goal that we started with, as it is likely that our MV will be below that goal with a much lower RR. If things change and obstruction recurs (and then we notice AutoPEEP all over again), we can go back to the longer I:E ratio. The idea here is that we are constantly reassessing what is going on with the patient and making these small adjustments to best ventilate the patient in a given moment. Just because a lengthened I:E was warranted at the start doesn't mean ~~they need~~ that forever.

To summarize our obstruction strategy: utilize a lower rate (and consider a shorter I-time also) to a goal I:E of 1:≥5. Consequently, we need to titrate TV (or PC[127]) up as far as the patient's lungs will allow. Know that we will likely be short on our MV goal and that's OK – as our pharmacological interventions start to work we can hopefully migrate back towards "normal" parameters to meet ~~the MV goal~~. Maybe consider dropping PEEP, but know that there isn't yet a good consensus on that. Also, be sure to check for AutoPEEP periodically and consider disconnecting the vent circuit to reset it back to zero if need be.[128]

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[124] Pruitt, 2007; Yartsev, 2019 – The first provides a more in-depth discussion of this "permissive hypercapnia" approach; the second gives way more information that we thought possible on the potential effects that such an approach may have (but of note, one of those effects may be bronchodilation)

[125] Which gives us the upper limit for a safe Pplat, assuming a PEEP of 5cmH_2O and an additive PC value

[126] Stather & Stewart, 2005 – In addition to explaining this part of things, these two also provide a ~~general~~ overview of a strategy for the asthmatic patient in general

[127] Just remember that it may be harder to get complete exhalation in PC ventilation (versus VC) due to differences in how those breaths are delivered (i.e. decelerating flow versus constant flow, see **Types of Breaths** to review this idea)

[128] Which we discussed in the section on **AutoPEEP**

Hypotension

In patients with hypotension (or the potential for hypotension) the primary concern is that mechanical ventilation can decrease preload to the heart and further contribute to the problem. We discussed this already in reference to both negative pressure vs. positive pressure ventilation and PEEP,[129] and we mentioned then that euvolemia seems to mitigate this effect. So first strategy here (since we are committed to PPV) is to restrict PEEP to whatever minimum value we need to maintain adequate oxygenation. Beyond that, however, we can limit the time spent at inspiration during the overall respiratory cycle. Think of it this way: preload drops further when we increase intrathoracic pressure, so if we decrease the amount of time spent pushing air into the system (i.e. increasing intrathoracic pressure), we can limit this affect.

To quantify the idea, consider two patients: one at a RR of 17 and one at a RR of 10. If we assume an I-time of 1.0s (norm for the adult patient), let's calculate how much time the patient experiences a state of decreased preload (i.e. inspiration):[130]

$$\%\text{ TaDP} = (\text{RR} \times \text{I-time}) \div 60 \text{ seconds}$$

$$\%\text{TaDP} = (17 \times 1.0\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 17\text{s} \div 60\text{s}$$
$$\%\text{TaDP} = 28\%$$

$$\%\text{TaDP} = (10 \times 1.0\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 10\text{s} \div 60\text{s}$$
$$\%\text{TaDP} \approx 17\%$$

We can further drop this percentage by decreasing I-time:

$$\%\text{TaDP} = (10 \times 0.8\text{s}) \div 60\text{s}$$
$$\%\text{TaDP} = 8\text{s} \div 60\text{s}$$
$$\%\text{TaDP} \approx 13\%$$

By dropping our rate to 10 (from 17) and dropping I-time to 0.8s (in the adult patient), we can cut the amount of time spent at decreased preload by over half. While we could keep dropping RR, we stop at 10 because we need to maintain MV in these patients. Let's look at what happens to MV if we drop RR to 10 and then come up with a strategy to address it. As before, we'll assume a patient with an IBW of 65kg and a TV of 8ml/kg:

$$\text{MV goal} = 100\text{ml/kg/min}$$
$$\text{MV goal} = 100/\text{ml/kg/min} \times 65\text{kg}$$
$$\text{MV goal} = 6500\text{ml}$$
$$\text{MV goal} = 6.5\text{L/min}$$

$$\text{TV} = 8\text{ml/kg} \times 65\text{kg}$$
$$\text{TV} = 520\text{ml}$$

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[129] See both **How is Positive Pressure Different?** and **PEEP**

[130] This is another one of those made up terms which we identify as %TaDP or "percentage of time at decreased preload"

MV calculated = TV x RR
MV calculated = 520ml x 10/min
MV calculated = 5200ml/min
MV calculated = 5.2L/min

Now 5.2L/min isn't super far off from 6.5L/min, but we need to remember that a hypotensive patient is likely at risk of shock and, therefore, we need to make sure we are matching blood flow to the lungs by delivering at least what our calculated MV goal is. This idea is in stark contrast to the obstruction strategy in which we decided it was OK to let MV fall below goal; in hypotension we need to maintain (or even exceed, especially with acidosis ~~or trauma~~ – discussion on that to follow) our MV goal. So let's titrate TV up to 10ml/kg and see where we end up:

TV = 10ml/kg x 65kg
TV = 650ml

MV calculated = TV x RR
MV calculated = 650ml x 10/min
MV calculated = 6500ml/min
MV calculated = 6.5L/min

If we drop RR to 10 (and I-time to low of normal by age) to minimize the percentage of time spent at decreased preload (i.e. inspiration) and increase TV to 10ml/kg, then we maintain our MV goal of 100ml/kg/min. Now that we've logically arrived at a strategy of decreased RR and increased TV, let's rewrite the order of the steps as so: increase TV first, then decrease RR to match MV goal. The reason for this is that we don't want to arbitrarily drop RR and then wind up in a situation where we can't titrate TV up to goal – that would result in a decreased MV (which we said is an important thing in the patient at risk for shock). So let's go up on TV as much as we can to a goal of 10ml/kg (or as close as possible with safe Pplats) and then drop RR afterwards. Even if we aren't able to drop %TaDP by half as in the example shown, we can at least move in that direction while ensuring adequate ventilation.[131]

[131] Another advantage of titrating TV first and then RR is that it allows the strategy to be applicable to both adult and pediatric patients without having to come up with more age-based recommendations; while this may or may not be a good reason in and of itself, it is worth keeping processes simple and applicable across the board…

Now there are other justifications for using a high TV and low RR strategy that don't include this %TaDP concept, we just find that this concept makes it easy to appreciate. An alternative justification would be that the strategy decreases dead space.[132] We talked about this idea back when we discussed making changes to address MV needs[133] and the idea is that dead space gets introduced with each breath given, so fewer breaths (with more volume each) means less dead space overall. Another rationale would be P_{aw}[134] – this high TV, low RR approach decreases average pressure into the system, especially when we consider lowering PEEP towards zero (i.e. using the bare minimum necessary to maintain oxygenation). While lowering P_{aw} can negatively impact oxygenation, we may be able to counteract that with higher FiO_2 to meet our oxygenation goals. The moral here is that there are multiple justifications for this strategy; one has been spelled out here and the other two are deferred until the **Appendix**.

To summarize: in the hypotensive patient we want to decrease the amount of time spent at decreased preload while maintaining MV at our weight-based goal. To do this, we drop I-time to low of normal, increase TV towards 10ml/kg IBW (in PC this may mean starting at 15-25cmH_2O), and then decrease RR to maintain our MV goal. We also want to be cautious of high PEEP while recognizing that oxygenation (facilitated by PEEP) is important in these patients with potential low perfusions states. Said one more time in the short and sweet manner of things: when ventilating the hypotensive patient, drop I-time, increase TV, drop RR (to ~~match~~ MV goal), and keep PEEP at a minimum.

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[132] Bauer, 2015 – While the strategy discussed here is slightly different than ours (and includes decreasing PEEP all the way to zero), the basic idea is the same

[133] This was in **Ventilation (and ~~EtCO2~~)**

[134] And again, this is **Mean Airway Pressure**

Return to Contents

Acidosis

With acidosis one of our primary vent goals is to facilitate respiratory compensation against the underlaying acidosis. The classic example here is a DKA patient breathing at 30/min. Flight crew comes along, RSIs the patient, and then sets the vent up at a "normal" rate of 12. The patient had been compensating with an increased RR (and thus MV), but that compensation got taken away suddenly. As a result, the patient crashes and dies. So let's not do that. And just to quantify the extent to which our doing so changes the game for this hypothetical patient, let's look at the MV difference between a rate of 12 and 30 with an assumed TV of 500ml:

MV calculated = TV x RR
MV calculated = 500ml x 30/min
MV calculated = 150000ml/min
MV calculated = 15L/min

MV calculated = TV x RR
MV calculated = 500ml x 12/min
MV calculated = 6000ml/min
MV calculated = 6L/min

In an acidotic state, our MV goal increases a lot. While a bit tricky to pinpoint exactly what that goal ought to be, let's start with a goal double that of the normal patient: 200ml/kg/min.[135] To achieve that goal, we may need to increase both RR and TV. We said before that to increase MV (in an effort to get our $EtCO_2$ within a normal range) we typically start by changing TV first and then RR. The reason for this way that we get more bang for our buck, as adding a breath also adds in dead space to the equation. In the acidosis situation, however, the patient is likely already breathing fast, so let's just use a high of normal TV (i.e. 8ml/kg) and see what kind of RR we'd need to get to this increased MV goal of 200ml/kg/min:

MV goal = 200ml/kg/min
MV goal = 200ml/kg/min x 65kg
MV goal = 13000ml/min
MV goal = 13L/min

TV = 8ml/kg x 65kg
TV = 520ml

DKA RR RSI MV TV ml min L kg
CO_2 $EtCO_2$ ABG ETT PCO_2 HCO_3^- FDA mmHg
BMP AC SIMV PS

[135] Weingart, 2010 – Our suggestion vaguely resembles the one recommended here (double MV to drop CO_2 from 40 to 30, that's with a starting MV of 120/ml/kg); that said, this is a minimum starting point and we may need to take it further than that – the idea is that we initiate ventilation to prevent immediate deterioration and then go from there to work towards goals (as outlined later in this section)

$$MV\ goal = TV \times RR$$
$$13L = 520ml \times RR$$
$$13L / 520ml = RR$$
$$25 = RR$$

This means that a TV at 8ml/kg and a RR of about twice normal will get us the theoretical MV of 200ml/kg/min. In the normal patient, this would drive our $EtCO_2$ down significantly and create a state of respiratory alkalosis, but we said already that this compensatory respiratory rate is what we want – now we just need to figure out how to measure or quantify to what extent we are helping the patient. We said before in a footnote that this figure (the 200ml/kg/min one) is just a starting point, we then need to be a little more exact in how we go from there. There are a few strategies here and we'll talk about them stepwise in order of least exact to more exact.

First thing we can do is to match our set RR on the vent to the rate at which the patient was breathing before we took that respiratory effort away. This assumes that the patient was compensating adequately. And while this doesn't give us a quantitative goal to work towards, it is better than nothing. We can match the patient's effort on our machine, complete the transport, and then have the receiving facility check ABGs when we arrive to see how things have improved (or gotten worse, for that matter). Or if we can do gasses en route, we can always start this strategy and then evaluate progress along the way.

Another strategy is to measure the patient's $EtCO_2$ (perhaps via a nasal canula device or by cutting the ETT connector off a regular in-line attachment and sticking in the patient's mouth)[136] prior to taking the airway. We can then match the patient's RR (as above) or set RR to twice normal and then adjust to this $EtCO_2$ that the patient was at prior to us messing with things. Again, this strategy is similar to the above strategy in that it requires that the patient was compensating adequately on his or her own before we intervened.

A third approach is to utilize Winter's Formula to establish an $EtCO_2$ goal. The formula looks like so:

$$P_{CO2} = (1.5 \times HCO_3^-) + 8 \pm 2$$

The formula is designed to measure the respiratory component with a known metabolic acidosis (i.e. measured PCO_2 is compared to a calculated PCO_2 to determine adequate compensation if a mixed disorder is present)[137], but we can modify its use in the transport setting to guide our titration of EtCO2 (via MV):[138]

$$EtCO_2 \text{ should be} \leq (1.5 \times HCO_3^-) + 8$$

A few notes about all of this. $EtCO_2$ generally correlates with HCO_3^- fairly well, with $EtCO_2$ normally 2-5mmHg below PCO_2. That normal difference is due to anatomic dead space and will increase with additional dead space (i.e. alveolar dead space). That said, even with more dead space in play $EtCO_2$ and PCO_2 will move in stepwise fashion at the same rate.[139] So if we use this modified formula, adjust MV to that goal, and get our $EtCO_2$ right at the calculated value based on an HCO_3^- from labs, we still may be a bit shy of our MV goal.

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[136] For sure not FDA or manufacturer-approved and only to be used when no other options are available ☺

[137] Foster & Grasso, 2014 – Short video to explain the formula and it's use in a clinical setting

[138] Lodeserto, 2018 – See Part 3 of this series, it gives another perspective on how to manage the vented patient with concurrent (severe) metabolic acidosis

[139] Siobal, 2016 – And look here for more information on CO_2 monitoring in general



Just keep that in mind and know that's why we wrote it out as we did without the "±" and with the "≤." And the HCO_3^- can be from either the BMP or ABG for our use in the transport settings, but there are varying opinions on that.[140]

To bring it all home, we can do all of these strategies together: try to match the patient's RR and $EtCO_2$ as measured before we intervene, then compare both MV to our calculated minimum goal of 200ml/kg/min and $EtCO_2$ (both the patient's pre-intervention one and our subsequently-measured one) to an $EtCO_2$ goal derived from Winter's Formula. The only next best thing here would be to remeasure gasses en route to see how the patient is responding to treatment, but most of us don't have that capability in the field and we'll withhold a discussion of it here.

We went on a bit of a tangent here, but let's get back to our vent strategy for the acidotic patient: use a TV goal high of normal (8ml/kg) and increase RR (either to match patient's intrinsic rate or even just double normal for patient's age), then aim for a goal MV of 200ml/kg/min and an $EtCO_2$ of patient's baseline prior to intervention or as determined by Winter's Formula. Because we are shooting for high MVs in the acidotic patient, AC mode may be the best for these patients if they are triggering breaths spontaneously. If we do go SIMV and the patient has spontaneous effort to breathe, we may consider increasing PS so that patient-triggered breaths match machine-delivered ones (and this would avoid a drop in MV if we were following the normal SIMV strategy of PS breaths below TV goal).[141]

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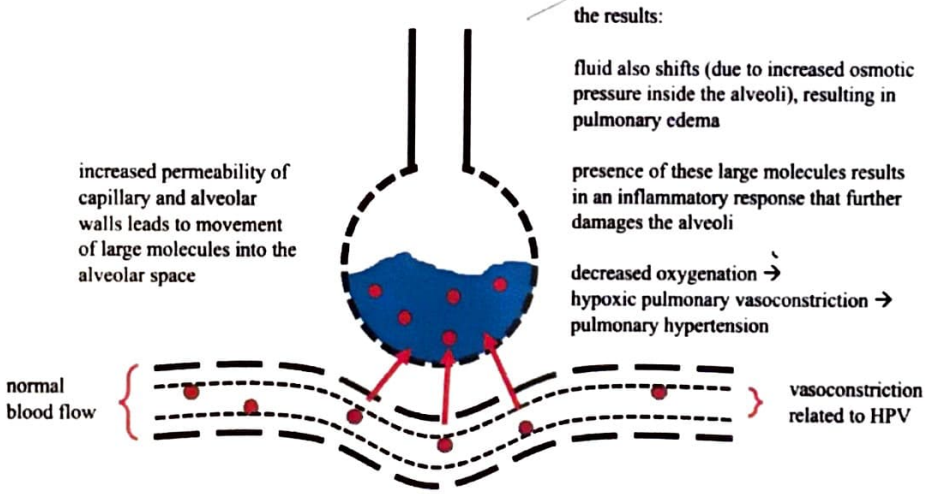
[140] Nargis & friends, 2015 – This is because in the BMP it is a measured quantity, in the ABG it is calculated and there can be some discrepancy between the two values; all that said, there is strong correlation between the two and it likely doesn't much matter in the majority of cases (and while this particular study was looking at the totally unrelated idea of cost-effectiveness related to blood gas analyzers in the developing world, the findings on correlation between the two values are still worthwhile)

[141] We talked about this idea way back in the section on **SIMV**

Acute Lung Injury/ Acute Respiratory Distress Syndrome

Another well-known and established strategy in vent management is the "injured" or "sick" lung strategy, also known as the "lung-protective" approach. These patients have lungs that are particularly susceptible to further injury and barotrauma and, as a result, we use less volume per breath in an effort to avoid over-inflation. We then have to increase rate to maintain MV or be OK with a higher $EtCO_2$. Another component of this strategy is higher than normal PEEP to improve oxygenation, maintain recruitment of alveoli, and physically displace stuff that has accumulated in the alveoli. We'll start by reviewing the concept of acute lung injury and discussing the pathophysiology of acute respiratory distress syndrome, then we'll get into specifics about vent strategy.

Acute lung injury (ALI) refers to a number of pathologies that inhibit normal pulmonary gas exchange.[142] Specific causes include sepsis, pneumonia, bleeding from a traumatic injury, inhalation of toxins or smoke, and aspiration. ALI is a concept that lives on a spectrum with acute respiratory distress syndrome (ARDS) being the end result if left alone to progress to the bitter end. While ALI, as a term, may also be described as mild or moderate ARDS, the underlaying pathology is the same. The main component of the disease process is that the alveolar and capillary walls becomes permeable to stuff that normal is normally sequestered in the blood:



There are quantitative criteria for ALI and/ or ARDS (depending on how we choose to define it), but that isn't necessary to our field treatment. Given our limited capabilities in the transport setting, we generally identify a patient who needs this vent strategy from a report per sending facility or suspicion based on clinical progression of the illness. There are also many recommendations to use this strategy for all patients who don't fit any other category.[143] The strategy includes low tidal volumes, higher than normal PEEP, maintaining recruitment, and permissive hypercapnia. Let's discuss each of these in turn and give some specific guidance.

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[142] Ragaller & Richter, 2010 – Not only do they provide a coherent and brief overview of ALI/ ARDS, they also discuss this whole vent strategy and summarize research to date (at least as of 2010)

[143] And in the case of two-strategy recommendations, it is either this or an obstruction strategy that make up the choices

Starting TV for these patients should be 6ml/kg IBW, but we may get as low at 4ml/kg eventually. This recommendation is from ~~one of~~ the ARDSNet studies[144] which compared TVs of 6ml/kg against 12ml/kg and determined that lower TVs resulted in significantly better outcomes for these patients. While it may seem that 6ml/kg and 12ml/kg represent two extremes and it could be tempting to rationalize that 8 or 10ml/kg probably isn't all that bad, we do know that 6ml/kg is OK, so let's just stick with the data and ventilate at 6ml/kg until the science people tell us otherwise.[145]

In addition to low TV, we go up on PEEP to improve oxygenation. Consider doing so in a stepwise fashion as recommended in these charts:[146]

→ give a ~~little more~~ context here

OXYGENATION GOAL: PaO_2 55-80 mmHg or SpO_2 88-95%
Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations such as shown below (not required) to achieve goal.

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

& the PrVENT trial?

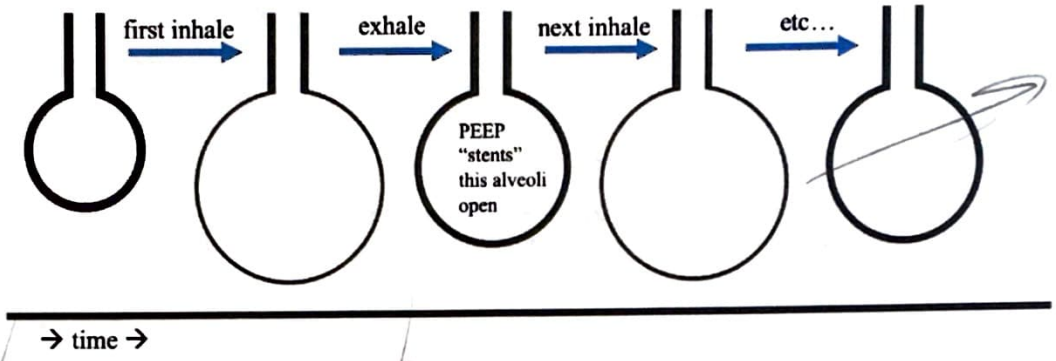
can I simplify this? & the other one too.

[144] The Acute Respiratory Distress Syndrome Network, 2000 – Much of the data we have on contemporary vent management comes from this group of researchers and subsequent investigations by other folks based on their research

[145] Sahetya & friends, 2017 – And for a more detailed discussion of this idea, take a look at this article

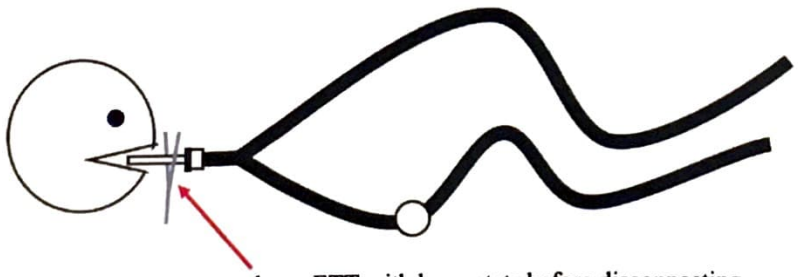
[146] NHLBI ARDS Network, 2005 (image); The National Heart, Lung, and Blood Institute ARDS Clinical Trials Network (2004) – The chart comes from that first reference sheet; the study cited shows that either of those two approaches is appropriate, in fact, they modified the study in process to test even higher PEEPs and that approach is also a legitimate choice (but we've left it out just to keep things a little more simple)

Another really important component of our ALI/ARDS strategy is alveolar recruitment. This is a concept that we haven't talked about much, but we'll get into it here.[147] Recruitment is the idea that we can actively re-inflate collapsed or underinflated alveoli as we depicted in our previous discussion of PEEP:



If we have a partially inflated alveoli stented open with PEEP and then disconnect the vent circuit, that alveoli goes back to where it was before we started. In a normal lung there are forces that maintain recruitment to prevent this loss and we can also re-recruit that alveoli on the order of seconds to minutes, so it isn't a huge deal for us to be worried about losing recruitment – we just get them back on the vent, add a bit of PEEP and we are back where we want to be with no real negative outcome. With the ALI/ARDS patient, however, it can take hours to recruit alveoli. This means that if we lose recruitment, we lose all of that progress towards better oxygenation and our patient can deteriorate very quickly.

With that in mind, it is important to keep the system that extends from the vent to the patient's alveoli intact at all times. When we do have to break the system, such as when we transfer the patient from our machine to the hospital's machine or vice versa, we can maintain recruitment by clamping off the ETT. The main point is to prevent pressure at the alveoli from dropping below PEEP, so it theoretically doesn't matter at which point in the respiratory cycle we clamp the tube and perform the swap. That said and just to be safe, let's do this clamping of the ETT during inspiration – that way if we leak some air out in the process, we have a cushion of safety. And here is what the technique looks like:



clamp ETT with hemostats before disconnecting (consider using a 4x4 to pad things so that the teeth on the hemostat don't damage the tube)

[147] And again in Recruitment Maneuvers

Last thing to mention with this ALI/ARDS strategy is MV. We mentioned already that we start at a TV of 6ml/kg and may need to go down to 4ml/kg. With higher PEEP we increase overall airway pressures and therefore that 6ml/kg TV on top of a higher PEEP (up to 20 in some cases!) means we might run in to high Pplats. So if we notice Pplat encroaching on our safe limit of 30cmH_2O, then we can dial the TV down to 5ml/kg and then to 4ml/kg (or if we are in PC we can titrate that value down and look at VTe). Dropping our TV to 4ml/kg will reduce MV and increase $EtCO_2$, but let's quantify that difference in MV:

MV goal = 6.5L

TV = 4ml/kg x 65kg
TV = 260ml

MV calculated = TV x RR
MV calculated = 256ml x 17/min
MV calculated = 4420ml
MV calculated ≈ 4.4L

And to maintain our MV goal, let's see what kind of RR we would need:

MV goal = TV x RR
6.5L = 250ml x RR
6.5L / 250ml = RR
25 = RR

So to maintain our MV goal with a TV of 4ml/kg we need a RR of 25 for the adult patient. Which is OK if we can comfortably get the patient there. If not, that's also OK. In fact, there is some evidence that hypercapnia (i.e. a high $EtCO_2$ related to a lower MV) is alright for these ALI/ARDS patients.* The data isn't super clear at this point, but rest easy knowing that if we can't attain our MV goal there may be a silver lining in this case. With pediatrics (when 25/min is too slow), we just go up on RR as much as we can to meet (or exceed if in volume control) our MV goal. Consider doubling RR or using the high end of normal for a given age range or just titrate up from a normal rate – the limiting factor will be comfort and exhalation (i.e. monitor for AutoPEEP to ensure full exhalation).

To put it all together: ALI/ARDS represents a spectrum of disease that primarily impacts the integrity of the alveolar walls and results in increased permeability, movement of large molecules and fluids into the alveolar space, and further damage from an inflammatory response. Vent strategy is focused on low TVs starting at 6ml/kg (and down to 4ml/kg if needed) to avoid barotrauma, high PEEP to both maintain recruitment of alveoli and displace fluid, maintenance of recruitment at all transfers in order to avoid rapid deterioration, and an increase in RR to maintain MV (possibly with a concurrent strategy of permissive hypercapnia).

And one last thing to mention about this strategy. We said just a moment ago that lots of folks recommend a two-strategy approach to ventilation in which we use either this ALI/ ARDS approach (termed "lung protective") or an obstruction approach.[148] We've said here that we have a general vent strategy for routine ventilation and then specific strategies for certain patient types. The differences between our general strategy (which is similar to a general "lung protective" one) and this ALI/ ARDS strategy is related to recruitment of alveoli (and being super careful to not lose it) and the idea that we may need to go down on TV to 4ml/kg. Both of these things are totally OK in the "normal" patient that we ventilate using the general strategy, it's primarily as matter of emphasis. If it makes things easier to default to this ALI/ ARDS strategy in all cases that don't warrant one of the others, that's completely acceptable.

Other Potential Strategies

The above list of vent strategies addresses four markedly different situations that we often come across in the transport setting, but there are other potential injuries or pathophysiologies that might also warrant specific adjustments to the normal list of settings ~~that we previously came up with~~. While we could theoretically compile a list of all the possible things and work out an algorithm to address each one in turn, that gets a little cumbersome and would result in a hefty protocol ~~of sorts~~ that might be difficult to navigate through when time is of the essence. As we said before, the idea is to work towards an understanding of how the body responds and how the vent does its thing so that we can make changes on the fly and anticipate the results that will come of any adjustment away from normal. But just to mention a few examples without going into the same level of detail as we did above, consider the following situations.

In the patient with a head injury/traumatic brain injury (TBI), we often choose to aim for an $EtCO_2$ low-of-normal to what we'd typically use for a standard patient.[149] While we don't necessarily ~~hyperventilate~~ these patients anymore, we could adjust MV to a tighter $EtCO_2$ goal of 35-40mmHg by going up on either TV (preferred) or RR. We also want to maximize oxygenation and, therefore, may be OK with an SpO_2 of 100% during transport (whereas we would normally titrate FiO_2 down in response). We may also make small adjustments to our settings in an effort to maximize patient comfort (and therefore avoid ~~any~~ increase in intracranial pressure), whereas we might not pay as close attention with other patients and simply use drugs to make them happy.

In the pregnant patient we might similarly utilize an FiO_2 of 100% to ensure maximize oxygen delivery to the fetus. Since many services don't have the capability of fetal monitoring during transport, this is a way to ensure that we don't have a hypoxic injury or put any undue stress on the fetus. We also need to consider an increased MV goal for the patient (which may mean an $EtCO_2$ goal low of normal, somewhere in the 30-35mmHg range),[150] as we have baby to consider as well. Another consideration is patient positioning – in the vented pregnant patient we not only have decreased preload due to PPV, we could see that drop in CO compounded by pressure of the fetus on the inferior vena cava. So either turn the patient to a lateral recumbent position or displace the gravid uterus over to the side.

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[148] Wright, 2014 - And for another review of that concept, take a read here

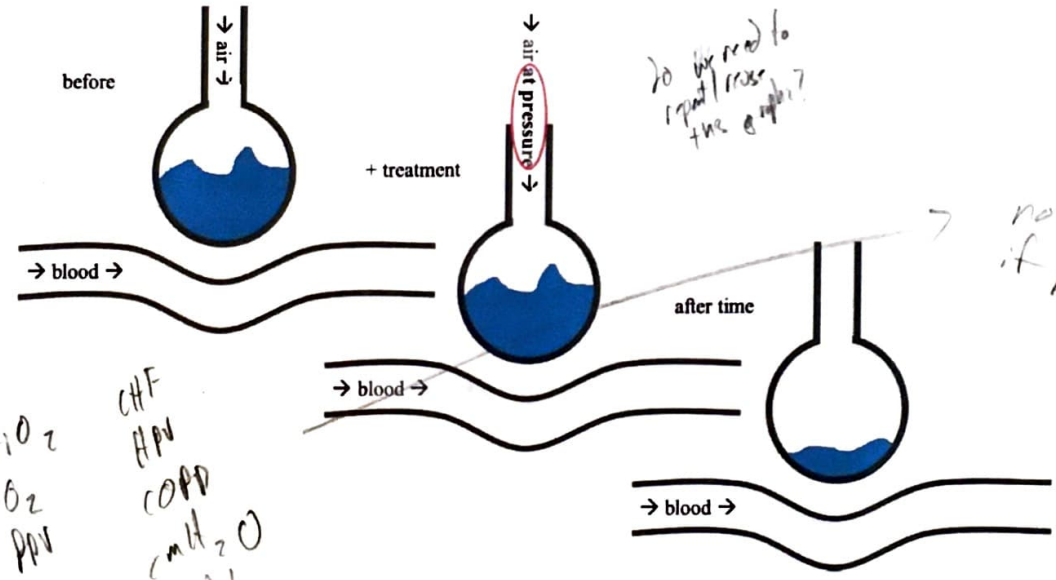
[149] Godoy & friends, 2017 – Detailed overview of this concept and research that has been done to date

[150] Wingfield, 2012; LoMauro & Aliverti, 2015 – The idea was suggested in a video by the first guy; the physiology behind it is discussed in an article by the other two

Significant chest trauma is another one. We'd like to treat these patients via the ALI/ ARDS strategy, but we may also be concerned with hypotension and ~~might~~ want to use the hypotensive strategy. Those two are at odds with one another (low TV and high RR for ALI/ARDS, high TV and low RR for hypotension). In this case we have to get creative. Maybe we forgo the hypotensive strategy and choose the ALI/ARDS one, but get aggressive early on with vasopressors and fluids/ blood products in anticipation that a hypotensive state may be precipitated by our strategy. Or maybe we go with a strategy more in line with the hypotensive strategy, but start out with ~~higher~~ PEEP and leave FiO_2 at 100%. There is no right or wrong here and it depends a lot on how the patient presents in that particular situation.

On a tangent to this chest trauma idea: if a patient develops a tension pneumothorax en route, best thing we can do is to take the patient off the vent.[151] Not take them off the vent and bag them, but take them off the vent and don't breath at all for them until we fix that problem. PPV can tension a pneumothorax very quickly and we want to avoid making things worse. So disconnect the vent, decompress (or place a chest tube/ perform a finger thoracotomy), and then get the patient back on the vent. Because of this, we may consider keeping all patients with the potential for pneumothorax on an FiO_2 of 100% - that allows us more time to perform the procedure in the event that a pneumothorax develops before the patient desaturates.

A patient with CHF or pulmonary edema may warrant more PEEP to facilitate the movement of fluid out of the alveoli:[152]



[151] Wingfield, 2012 – Haven't seen this idea discussed elsewhere, but it seems appropriate to discuss for all of us transport folks
[152] Perlman & friends, 2010 – While a Pplat up to 30cmH_2O is likely still just fine with these patients, just know that pulmonary edema can make the patient more susceptible to injury (and this article discusses why that might be via a unique experiement)

In addition, PEEP might help drop afterload to facilitate both perfusion and clearing of fluid from the pulmonary side of circulation. And while it may make sense that a high FiO_2 could mitigate the effects of an HPV effect in these patients, there is some risk to that strategy and treatment focused on adequate MV and PEEP are preferred for the CHFer.[153]

Folks with COPD may warrant different strategies due to potential effects of oxygen.[154] Same goes for an MI patient with the need for augmentation of cardiac output (i.e. right-sided MI).[155] We could even argue the case for a specific toxic-exposure strategy.[156] It quickly becomes evident that there are a number of cases that don't quite fit mold by which we try to simplify vent strategies. And that's totally OK. The templates are there as frameworks from which we then consider the specifics of each patient, one at a time. The important thing is to know what impact any vent change will have on the patient depending on how (s)he presents in a given situation. There are lots of cases in which there isn't a straightforward answer, but as long we don't make things worse by titrating things the wrong way, all is good.

[153] Kuhn & friends, 2016 – See discussion of these ideas here
[154] Swaminatahan, 2015 – Short and sweet discussion of whether or not these are even valid claims
[155] Mahmood & Pinsky, 2018 – They don't directly prescribe this approach, but they do lay out the framework of how it all might work
[156] Some combo of ALI plus or minus acidosis, depending on the agent and/ or route of exposure

Return to Contents

Make a (Calculated and Informed) Plan

This next section covers how we go about setting the patient up on the ventilator. In particular, it looks at how the process differs whether it's us initiating ventilation versus if we are taking over a patient in which ventilation has already been initiated. This may not seem like a big deal, but the taking over of a vented patient is a bit tricky. Even though we have these predetermined strategies for various different patient types, the truth is that there is a lot of variation in how patients respond to the vent: sometimes an asthmatic patient is happy with an I:E of 1:2, other times a hypotensive patient has a high RR and low TV for good reason, etc. Because of this, we need a method to determine when changes are needed and when we can leave things alone as we find them.

Getting ~~All the Numbers~~ Ready

First thing we do for any patient who needs to be or is already ventilated is listen. We listen to a report from whoever was hanging out with the patient before we got there. This is very important for all patients, as it can tell us how the patient has responded to or will respond to strategies we might have in mind. We then (as in *after* listening) decide on a strategy based on how we think that patient ought to be ventilated (i.e. hypotensive strategy, obstruction strategy, or some hybrid situation). Next we get an accurate patient height (either from a reliable healthcare provider or by measuring it ourselves) and perform three calculations: IBW, TV, MV.

Another component here is the patient exam. We'll discuss a few of the specifics when we talk about a patient already on the vent, but we for sure want to get an exam done before we start manipulating things or playing with ~~our~~ vent. ~~The idea here is that~~ our mental construct of a strategy based on the report we received should match what we see in the exam. If not, we need to clarify that amongst ourselves before moving forward. No need to elaborate on that here, we all know the importance of a good assessment. So once we have a report, have done an assessment, and are decided on a strategy, we move forward.

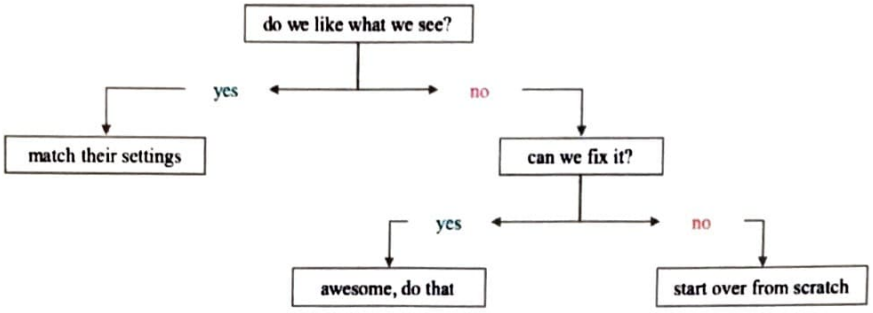
From Scratch

When we are the ones initiating the vent, it's fairly straightforward: we take the settings we've come up with based on presentation/ pathophysiology and plug them in to whatever mode and method of control we decide to use. We've already talked about the different strategies and why we may choose to use one mode/control over another ~~(and that a lot of this has to do with provider preference)~~, so we won't spend any more time on that here. But the easiest way to do this is to stick with whatever your machine defaults to and then adjust from there if need be. Once the patient is on the ventilator, we just need to confirm that everything is going as planned, beginning with the Three Big Things: oxygenation, ventilation, and comfort. Once we get those things sorted, we can then move on to some of the finer subjects (which will be discussed in the next section, **Keeping Things Going**).

It is worth reiterating at this point that the settings we conceptualize prior to initiating ventilation (and as discussed in the previous section) are starting points from which we then make adjustments. It may very well turn out that we end up with settings, based on patient need, that vary significantly from what we initially had in mind and that's totally OK. But the starting point ought to be based on both on calculated goals and settings founded in physiology. And if you have no idea which strategy to choose or if the patient fits too many categories all at once, just start with those basic settings we discussed in **A General Vent Strategy** and go from there.

Patient Already on the Vent

With someone already on the vent, it gets a little more complicated. We'll draw it out in a short, simple algorithm first and then we will expand on it ~~and discuss the specifics~~:



The first step in this little algorithm, "do we like what we see?" refers to a few different things: First of all are the Three Big Things: oxygenation, ventilation, and comfort – those for sure need to be addressed. Second is strategy: are the chosen settings at odds the with what we had in mind? In the case of a hypovolemic patient with a high RR, for example, we may say, "yes, this strategy may be detrimental to the patient." In the case of an asthmatic patient with an I:E of 1:3 we may decide, "this isn't what I would've set up from scratch, but let's see if it is working for the patient or not before deciding to change things." The idea here is to see what puts your patient at risk and what doesn't: a high %TaDP and hypotension does put a patient at risk, an I:E of 1:3 in an asthmatic with no AutoPEEP doesn't.

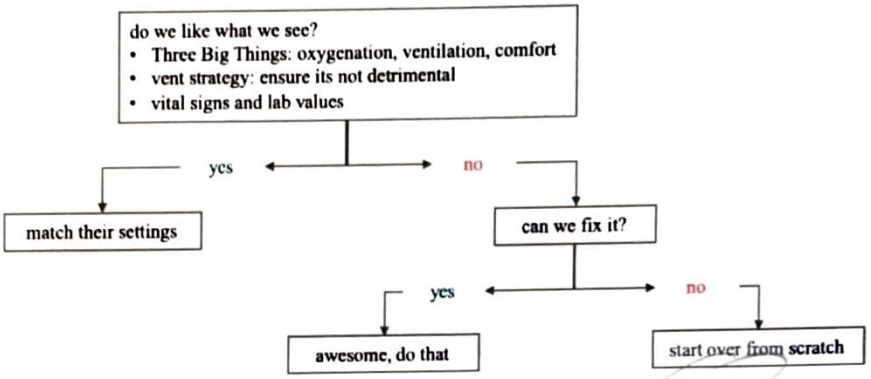
So we addressed the Three Big Things, we made sure the existing strategy isn't counterproductive based on what is going on with the patient, then we look at vitals and labs.[157] Again, no need to get into specifics here, but if all is well in each of those general three subject areas, then there is no reason for us to go messing with settings and we should match what they are using. The only exception here is if your machine can't do the settings they have. For example, the patient is on PRVC and you don't have that choice – then match as best you can in either volume or pressure control and go from there.

~~"But wait," we may hear from the audience, "~~what about checking a Pplat and AutoPEEP ~~and all of that!?~~" If your patient is alive and well and passes an assessment in all three categories we just discussed (the Three Big Things, vent strategy, vitals and labs), then those things can wait until we get them on to our vent. Some reasons for this: the delay here is only a few minutes at most, the measurements will likely vary by machine (i.e. how individual breaths are delivered), and we've already determined that the patient is stable via a number of different assessment parameters. And while scene time may or may not be a valid reason, we do want to use time efficiently and get patients moved unless we have reason to delay.

[157] And we don't really discuss labs in this manual, but there are some resources listed at the very end (under **Suggestions for Further Reading**) that can fill this gap

Return to Contents

Let's redraw that simple algorithm we started with and add in just a little bit of detail to include all of these ideas and then we'll move on to the next question and talk about it in detail:

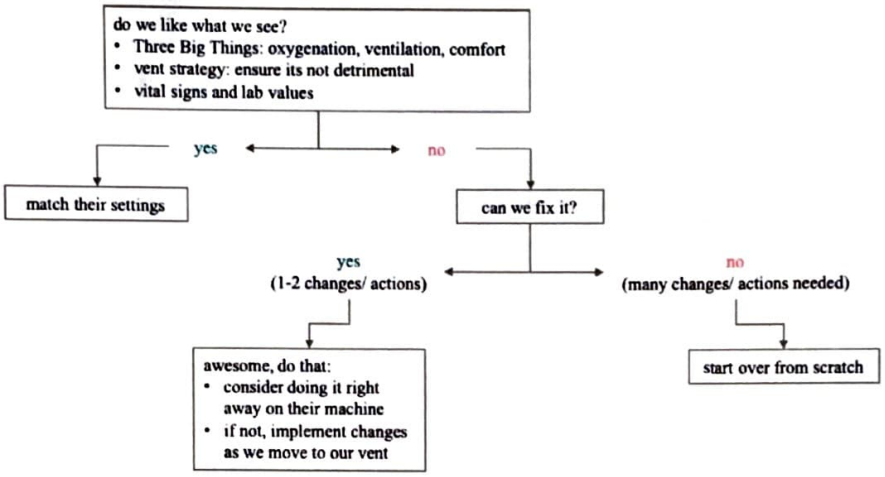


Next question to discuss further is, "can we fix it?" We'd like to fix whatever issues we have (as determined by our assessment in the first box of the algorithm) by way of one or two interventions and keeping the majority of settings as they are.[158] For examples: if the patient is uncomfortable and we can provide analgesia on top of the sedation they are already getting, that may be all that is needed; if we can fix a high $EtCO_2$ by increasing TV (or RR) a bit, no need to change mode or control; if we can address a potential for hypotension by decreasing RR and then increasing TV, all is good; etc. If, however, we are getting into a situation where it will take lots of changes to set things right, it may make the most sense to start from scratch with a whole new set of parameters. And in that case we may as well change a bunch of things and go with our preferred strategy.

One thing worth mentioning here is that it is sometimes cool for us to make these changes as the patient lies and on the sending facility's (or crew's) machine. Other times we just make the adjustments as we transition to our machine. We for sure want to avoid alienating the transferring staff by messing with their machine if that relationship doesn't exist, so just be cognizant that are two sub-options in the "Awesome, do that" course of action: do it right now and on their machine or do it as we transition on to our machine. Last thing and probably already obvious is that there is some middle ground here: we may make some changes/do some things right away and then defer other things until transfer, all as part of the same strategy. Example: give sedation now, adjust TV or RR during the transition.

[158] And for help in deciding this, consider using Critical-Medical Guide – it's an app that's got a nifty feature in which you simply enter in current vent settings and measured parameters and it spits out suggested vent changes to work back towards goals

And one more time, let's see how the algorithm would look with these additional details added in:



If at any time during this whole process things get too complicated, we can always skip ahead to the "start over from scratch" end of things, just recognize that the more changes we make, the less able we are to evaluate the efficacy of a single intervention. Just like a science experiment, it helps to isolate variables and know that the observed result can be attributed to a specific adjustment. And even though we mentioned it already, interpersonal dynamics also come in to play here: make changes based on necessity, not on personal preference – that will help ~~you~~ maintain positive relationships with referring staff and crews.

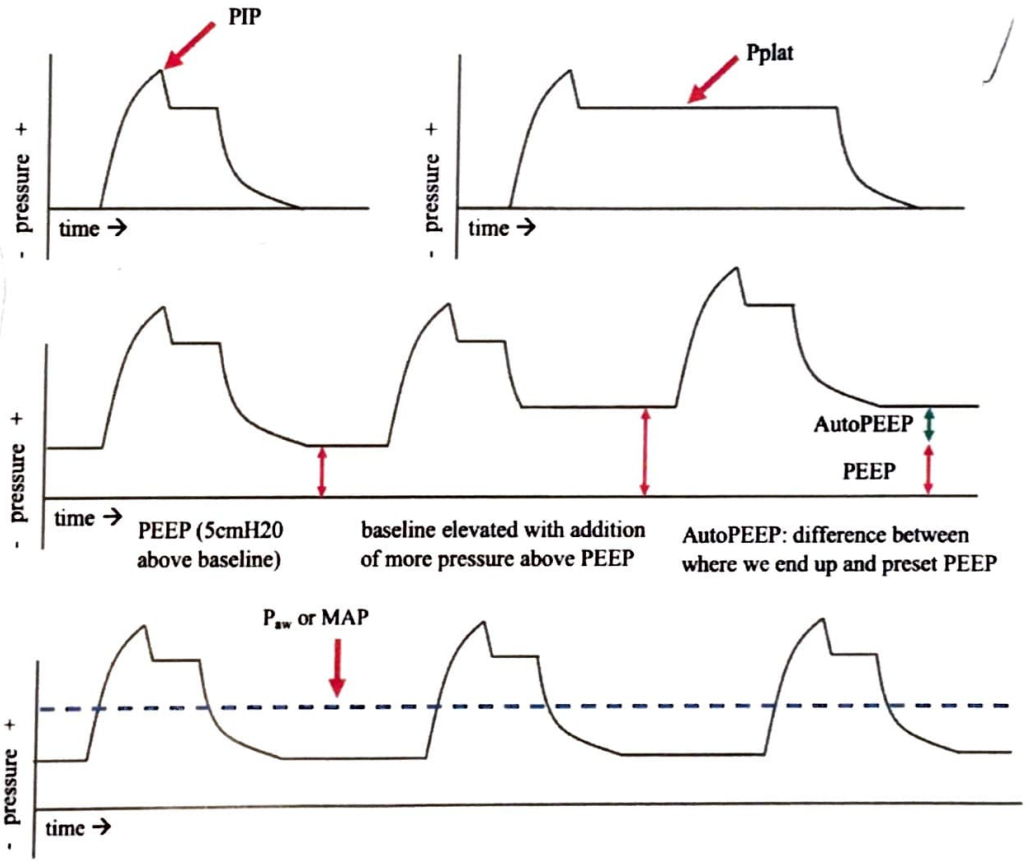
Return to Contents

Keeping Things Going

This next section goes over what we do once we have the patient on our machine and the Three Big Things (oxygenation, ventilation, comfort) have all been addressed. We talked already about how we sometimes vary from the settings we start out at and this section explains how that happens. We want to both avoid injury and optimize ventilation, so we slowly make adjustments to work towards those goals and ensure that things stay safe for our patients.

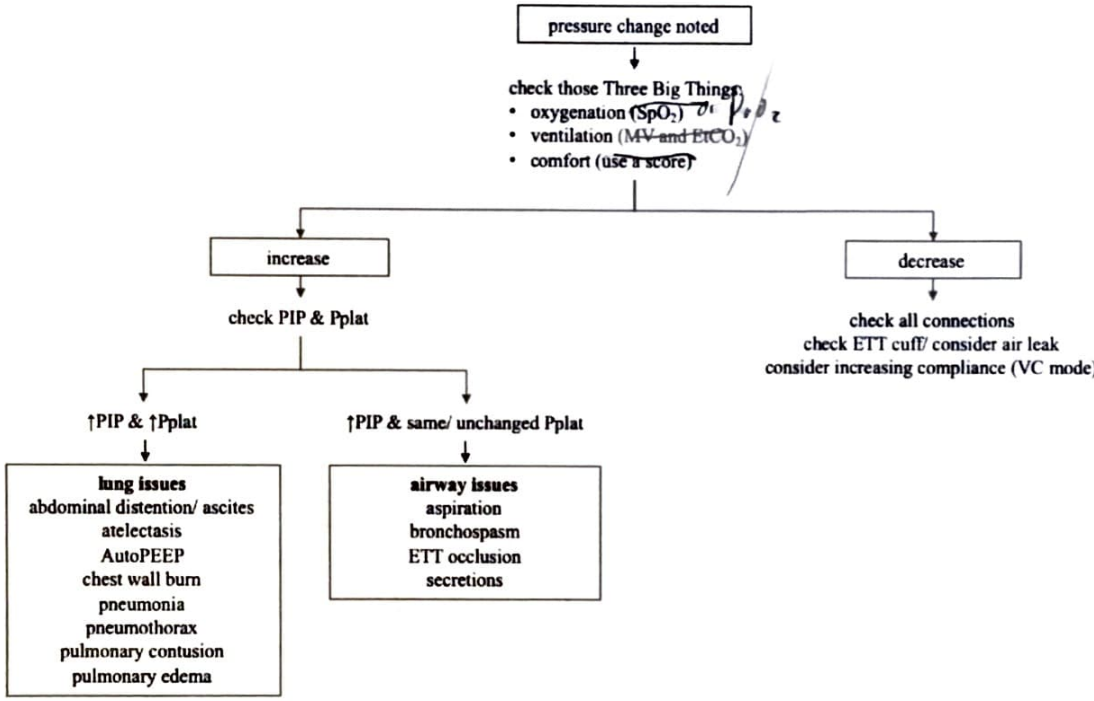
Watching Pressures

We talked about these ~~three~~ things already in the section titled Vent Parameters, Round Two, but here they are again: peak inspiratory pressure (PIP), plateau pressure (Pplat), AutoPEEP, and mean airway pressure (P_{aw}). And for visualization, in case we forgot, here's what they look like on a pressure waveform in volume control ventilation:



High for PIP is 35mmH_20, although we may go beyond that in certain situations (such as a small ETT). Pplat max is normally 30mmHg and we do try to stick by that one whenever possible. AutoPEEP is normally zero and we always take actions to address AutoPEEP when we see evidence of it. As for P_{aw}, we don't generally cite a normal range, but know that a change in this value can be the first indicator of a change somewhere in the system. All of these parameters should be checked (when possible, depending on control and patient's respiratory effort)[159] within the first few minutes after placing someone on our machine and then again periodically through transport. As we said before, if it may help to simply add these pressures on to a mental list of vital signs to reassess as we go.

As far as what to do with this information once we have it, here's a flowchart to help sift through the information and take action to address potential problems:[160]



[159] For example, if a patient is triggering lots of breaths, we may not be able to get a good AutoPEEP/ do an expiratory hold; if they are in PC ventilation, we may not be able to do an inspiratory hold (due to limitations of a particular machine)

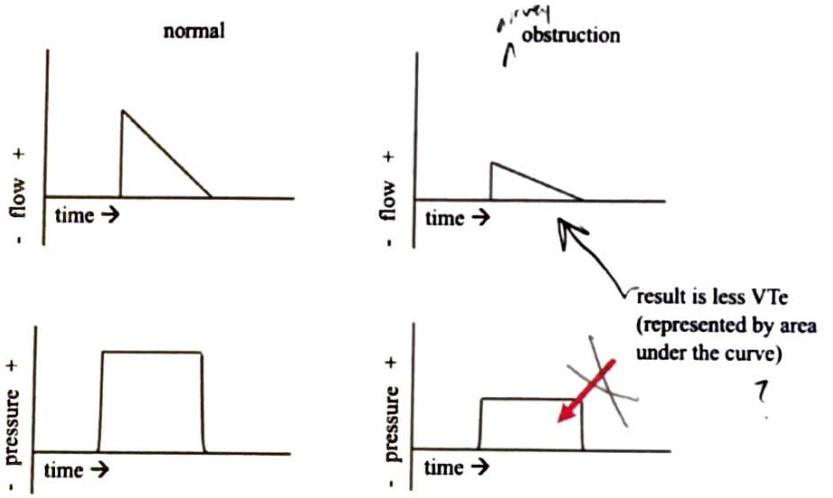
[160] Lodeserto, 2018 – The left bit of this chart is similar to one he puts forth

And then let's look at potential solutions for each of these cases:[161]

lung issues	
abdominal distention/ ascites	consider positioning
atelectasis	recruit some alveoli (via PEEP and/or recruitment maneuvers)
AutoPEEP	increase I:E ratio, trial VC, maybe adjust PEEP, disconnect circuit
chest wall burn	escharotomy
pneumonia	utilize PEEP to displace fluid
pneumothorax	remove from vent; needle decompression, chest tube, or finger thoracotomy
pulmonary contusion	consider ALI/ARDS strategy
pulmonary edema	utilize PEEP to displace fluid

airway issues	
aspiration	suction (prevent further aspiration), consider ALI/ARDS strategy
bronchospasm	fix with drugs, implement obstruction strategy
ETT occlusion	address comfort (biting), swap tube (something stuck)
secretions	suction

In pressure control ventilation when we may not have access to PIP or Pplat to identify these trends, there are other parameters we can look at. Most obvious is VTe – as compliance decreases,[162] VTe will drop (and vice versa). In the case of airway obstruction, often times we won't notice initially because the machine essentially accommodates for this increased airway resistance by using less flow initially:



[161] Briggs & Freese, 2018 – There are also lots of weird cases out there to explain things that can happen, the chart above should not be assumed to be an exhaustive list of causes or fixes; as an example, this referenced article from JEMS outlines a case of high airway pressures related to an ETT positioned with the bevel up against the wall of the trachea – the fix here was simply to rotate the tube 90 degrees

[162] As we mentioned in **Compliance (and Resistance)**

Since we don't typically monitor waveforms with transport ventilators, an airway obstruction may not get noticed in PC ventilation until it is severe enough to impact MVe.[163] The best way to catch these sort of things before they have an impact on patient outcome is by setting alarms appropriately so that we are notified right away as things change (see following section).

Alarms[164]

Next on our list of things to discuss are alarms. We won't talk about all the alarms that our machines might have, but we will talk about a few of the important ones. We can break alarms down in to two general categories: ones that are default on the machine and ones that we set. Those default ones may be different between machines, but deliver similar messages like, "hey man, your circuit got disconnected" and "oh snap, we ran out of oxygen." Those ones can be referenced and learned about in the manual for whatever machine we happen to be using. The other ones, the ones that we set, are the one's we'll focus on here.

One important alarm we set on the machine is the high-pressure alarm (which goes off when our high-pressure limit is reached). The reason this alarm is so important is because if it gets triggered, the inspiration cycles off (in most vents). That means that if we have a situation where we repeatedly trigger a high-pressure alarm, we may end up with a MV that bottoms out and a patient that quickly deteriorates. Imagine we place a patient on the vent who has either an untreated airway obstruction or poor compliance[165] – if we try to ventilate this patient in volume control and at normal settings, every breath that goes might trigger the high-pressure alarm and get terminated early with a net result of almost no MV. The reason this safeguard exists, in spite of this risk, is because we could for sure cause a lot of damage if we accidentally give too much pressure.

Moral of the story here: if we are in volume control ventilation and have a concern for increased airway pressures, we should consider going up on the high-pressure limit before putting the patient on the machine in order to avoid dropping our MV. On the flip side, in pressure control we need to vigilantly monitor MVe (and also VTe) to avoid the same issue (of decreased MV). Which leads us the next most important alarm we can set: low minute volume. We set this limit at a reasonable value below our MV goal so that if things get weird and MV starts to drop, we get notified right away before our patient suffers. In this way we utilize the high pressure and low MV alarms to simultaneously ensure both safety and adequate ventilation for our patients.

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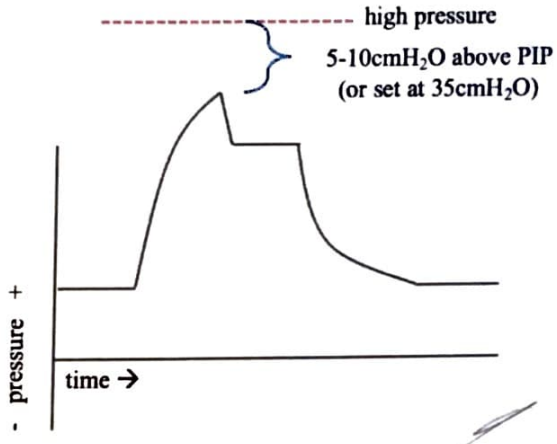
[163] We can also (again, this is in PC) look at flow as calculated and delivered automatically by the vent - higher flows mean less resistance, so even if we don't know ranges or normal values we can still use this concept to trend changes

[164] Disclaimer about this section: there isn't much out there in the universe to provide guidance on how we should set these alarms. There are studies out there that have collected data on alarm settings on in-patient units, but we don't feel it would be appropriate to apply those to the transport setting. Given that we move these patients one at a time with one or two well-trained providers (versus an ICU full of vented patients and lots of alarms at once!) we should arguably always have eyes on the machine and it makes sense to use much tighter limits for alarms that we might see in the hospital setting. That said, this is just one opinion on the whole thing…

[165] Again, as we talked about this in Compliance (and Resistance)

Return to Contents

As far as setting the high-pressure and low MV alarms, that is a bit dependent on our margin of safety and when we want to be notified of changes in the system. As a general rule of thumb, the high pressure limit should be no more than 10cmH_2O above your PIP. If, however, your PIPs are already high of normal, consider setting the high pressure alarm 5cmH_2O over that value or at our upper limit of 35cmH_2O:



In the event of one of those situations which may lead to repeated triggering of the high pressure alarm and sudden drop in MV, increase the high-pressure limit (even beyond 35cmH_2O if need be) to maintain MV. Note that this would be a short-term fix and we should start to consider other strategies right away: trial pressure control mode, consider pharmacological and procedural interventions, etc.

As for the low MV alarm: set that within 25% of the MV goal that we calculated when we first started into this process of getting the patient on the vent.[166] If we have a patient breathing in excess of that goal and we want to know if that changes, we just set the low MV goal 25% below what they are currently at. In any case, the low MV alarm is just a catch to alert us when we've missed a change – typically we will be on top of these trends and notice things before the alarm even gets sounded, but sometimes we get distracted by other interventions and this backup system can keep us notified.

Other alarms that we can set to help us better keep track of what's going on with the vent and our patient are low peak-pressure, low frequency, high frequency, and low PEEP. Low peak-pressure alerts us when the PIP is lower than we would expect; this could indicate a cuff leak, increase in patient's respiratory effort (i.e. negative pressure produced with patient effort),[167] or a loose connection (an actual disconnection would probably trigger a disconnect alarm, one of those non-adjustable alarms consistent across machines, as the pressure would drop much more significantly). Low frequency can let you know if the patient's RR starts to decrease – this is good if the patient is consistently breathing above a set RR and we want to be aware if that intrinsic effort changes. And reasonably enough, the high frequency alarm advises us when the patient starts to breath faster or if some mishap is causing the machine to think that (s)he is.[168] Lastly, low PEEP lets us know if the end expiratory pressure drops below our set PEEP – This could indicate a leak or cuff deflation.

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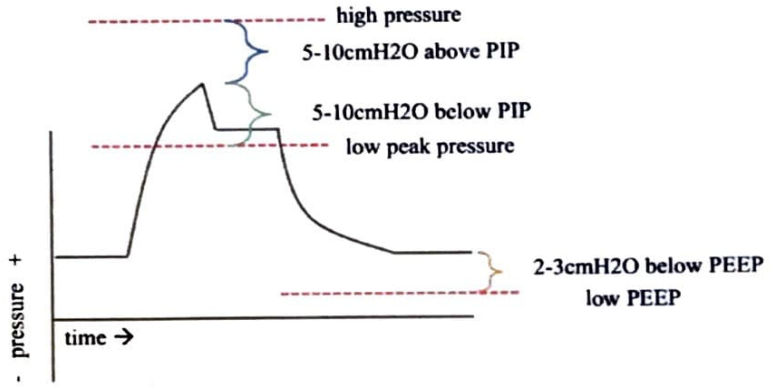
[166] And this 25% figure is an arbitrary number that we feel is appropriate, there aren't too many specific recommendations for this type of thing

[167] Weingart, 2019 – In addition to discussing four of the most significant vent alarms, this podcast proposes the idea that vent alarms ought to be addressed in the same way as a "code blue" in the hospital setting

[168] Which we call "auto-triggering" and will discuss shortly in **Triggers**

That's just a quick, short overview of alarms; recognize that the most important ones are high pressure and low MV, but that there are a number that can help us be aware of changes in the system as we work through a transport. Because there is so much variation between machines, the best way to get familiar with the alarms you will be working with is to read the manual that comes with the machine. Super fun reading, but it's good information and can help you fine tune the feedback from the vent so that you can better monitor what's going with the patient.

And we'll end with a graphic to show how some of these alarms would be represented on that pressure over time waveform in volume control ventilation:



low pop o/ cuffed ETT tubes to do. if tube is too small?

Titrating Up on TV?

Up to this point we've recommended considering TVs above that 6-8ml/kg range in just a few circumstances: to increase MV (in the **Ventilation (& $EtCO_2$)** section), with airway **Obstruction**, and as part of the **Hypotension** strategy. We also said that we want to limit our Pplat to a safe level <30cmH_2O whenever possible, which includes when we decide to go up on TV.[169] The idea here is that more TV is OK, but only to a certain limit. And the best tool we have to establish that safe limit in the transport settings is Pplat, so that's what we use. All that said, it is worth discussing this idea further to see what we know about increasing TV and some of the intricacies of the whole idea, because it gets a little more complicated.

One underlaying idea here is that TV is a component of P_{aw} and that this is a determinant of oxygenation.[170] So it might make sense to go up on TV as much as we can (~~and~~ within safe limits) to maximize oxygenation.[171] Increasing TV could also allow us to go down on RR (to keep MV constant). While this ~~would~~ take away from P_{aw}, it could help in other ways (i.e. by decreasing that %TaDP value we made up in the section on **Hypotension**). Now regardless of motive, this strategy of increasing TV is a bit at odds with the lower TV, "lung protective" approach pioneered by the ARDSNet studies.[172] That said, those studies looked at TVs of 6ml/kg versus 12ml/kg, so there may be some middle ground we just don't know much about.[173]

In light of this conversation, let's just say that we want to go up on TV for whatever reason. We've already said that our upper limit for Pplat is 30cmH_2O, so that's one limiting factor in the game. Another concept here is that we'd prefer to make changes slowly; rather than jumping from 6ml/kg to 10ml/kg (or whatever other arbitrary amount), we get there in a stepwise fashion in small increments.[174] And lastly, we can utilize compliance ~~(which we discuss later on, see **Compliance (and Resistance)** section)~~ to help guide us towards our goal. ~~Without getting too far into the idea of compliance, let's see how that might work~~.

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[169] Way back in the section on **Plateau Pressure**

[170] Lodeserto, 2018 – We cited this once already in **Mean Airway Pressure**

[171] That said, we typically use TV to effect change in ventilation instead of oxygenation (as we outlined in **Three Big Things**), but know that these things are interrelated and TV can actually impact both

[172] Wright, 2014 – And that "lung protective" strategy also includes limiting Pplat, utilizing PEEP to maintain recruitment, and limiting FiO_2 (in addition to lower TVs)

[173] Burrell, 2018 – This summary of a paper investigating this idea concludes that more data on this question is needed

[174] Felix & friends, 2019 – In a study on rats, these guys investigated this idea and determined that some of the harmful effects of high TVs can be mitigated by small and incremental changes; while this may or may not occur by exactly the same mechanism in humans, it seems likely that a similar approach would be warranted

In VC we could increase TV until we notice a spike in Pplat or a decrease in compliance; in PC we increase PC until we see a decrease in compliance or no increase in VTe after the adjustment. Once we hit either of these limits, we then titrate back the last increase (of TV or PC) to where things were just before the previous adjustment. To map it all out with steps in the chart representing reassessment during transport:

Volume Control Example				
Step #	**TV (ml)**	**Pplat (cmH_2O)**	**Compliance (ml/cmH_2O)**	**Action**
1	500	15	50	Increase TV
2	525	16	48	Increase TV
3	550	16	50	Increase TV
4	575	21	36	Decrease TV
5	550	16	50	No change, monitor
6	550	14	61	Increase TV

Note that even though Pplat doesn't get up to our previously established limit of 30cmH2O, we recognize that an increase beyond a TV 550 (line 4) gave us a spike in Pplat and drop in compliance, therefore we may titrate back a smidge and wait for the lungs to fill more before moving back up (line 6).

Pressure Control Example				
Step #	**PC (cmH_2O)**	**VTe (ml)**	**Compliance (ml/cmH_2O)**	**Action**
1	10	500	50	Increase PC
2	11	550	50	~~Increase PC~~
3	12	550	46	Increase PC (or stay)
4	13	550	42	~~Decrease PC~~
5	12	550	46	No change, monitor
6	12	600	50	Increase PC

It is worth mentioning here that VTe and compliance will likely vary from breath to breath and therefore it isn't quite as easy to recognize these trends in real time, but the general idea hold true. Also, this whole concept can be considered as an "icing on the cake" sort of thing – we may not get to this point in our vent management and that's just fine.

And to summarize: while increasing TV within safe limits for all patients may or may not be the best strategy, if we do decide to go that route we can use Pplat and compliance to guide progress and we ought to make changes in small increments. We will talk later on about another concept called **Driving Pressure** – this may be another one of the limiting factors in how much we decide to go up on TV, but we'll hold off on that for now.

Return to Contents

Acute Deterioration

The next thing to chat about is what to do if the patient begins to decompensate while on the vent. We will start with a common memory tool to address some of the major causes of acute deterioration of the mechanically ventilated patient:

The "DOPE" Mnemonic		
	issue	**action**
D	displaced tube	confirm tube placement
O	obstruction	suction, check for kinked ETT, consider bronchospasm
P	pneumothorax	remove patient from vent; decompress, chest tube/finger thoracotomy
E	equipment failure	check all connections

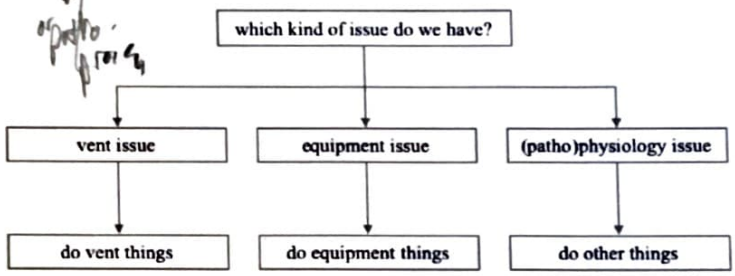
There are also some variations of this guy, so we may see it out there with an "S" at the end for stacking (i.e. AutoPEEP),[175] an "R" at the end for rigidity of the chest wall (a rare complication of Fentanyl administration),[176] or even with the "P" to represent pain and/ or (Auto)PEEP.[177] It also is sometimes accompanied by another mnemonic called "DOTTS" which outlines actions that can be taken to fix issues identified by "DOPE." Now "DOTTS" includes a step where we bag the patient with a BVM and we've crossed that step out – we don't recommend routinely taking someone off the vent unless we have good reason to and we'll get back to this idea in just a little bit. But just so we can see it in its true representation, here it is:

The "DOTTS" Mnemonic		
	action	**explanation**
D	disconnect the vent circuit	to fix AutoPEEP or decreased preload (i.e. pneumothorax or hypotension)
~~**O**~~	~~O_2 100% via BVM~~	~~to manually assess for issues (i.e. look, listen, feel)~~
T	tube position/ function	includes assessing placement and suctioning
T	tweak vent	consider decreasing RR, TV or I-time (i.e. with AutoPEEP or hypotension)
S	sonography	consider ultrasound to identify issues (if you have it)

The "DOPE" Mnemonic[178] (with or without "DOTTS") is easy to remember and can be used to guide the initial troubleshooting process when the patient starts to tank due to some unknown. Many of these occurrences can be tied to vent alarms or other assessment parameters, but that depends on which type of machine we are working on and what tools we have available. For example, a tube displaced too deep will likely result in a high pressure alarm (or eventually a low MV alarm) and a tube displaced out of the airway will likely result in a low pressure alarm. In regard to other assessments: a tube displaced too deep may result in a high P_{aw}, low VTe, patient discomfort, etc. and a tube displaced out of the airway can result in a low P_{aw}, drop in $EtCO_2$ with change in waveform, hypoxia, etc.

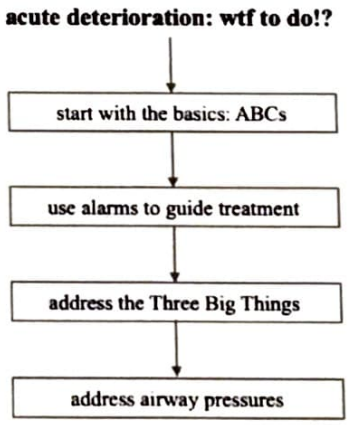
[175] Rezaie, 2018 – Also gives on overview of the "DOTTS" idea discussed below
[176] Thomas & Abraham, 2018 – While not all that common, it may be worth keeping in mind
[177] Wright, 2014 – A great read in general, but specific to this cause he's got a nice DOPE graphic that he got from another source
[178] Weingart, 2011 – For some useless trivia on where this mnemonic came from, take a look here

Because there are so many things to consider, building an algorithm to troubleshoot each possibility gets a bit difficult. But just for kicks we'll do it anyways. Before we get there, however, let's consider a few more things. First of all is that acute deterioration of the vented patient doesn't always mean that there is an issue with the vent – it could be some other issue related ~~to whatever else is going on or even an equipment issue~~ beyond the vent (i.e. ETT displaced). If it's a vent thing, then we mess around with the vent; but if it's another issue, our interventions should focus on drugs and procedures and that sort of thing. Think of it this way:

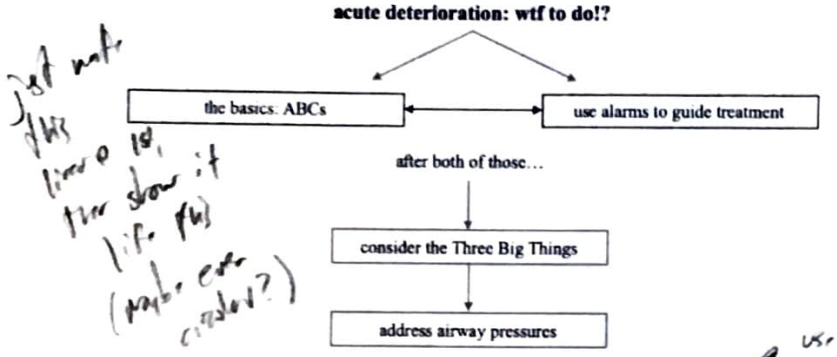


Now the reality is that it isn't always so cut and dry. There are times where we do both vents things and other things simultaneously. An example of this would be a patient already on the vent who experiences an allergic reaction to something – in this case we could simultaneously proceed with an obstruction vent strategy and give drugs to fix the problem. So while our little algorithm may be too simple, it often helps to take a moment to think about which sort of problem we have on hand and act accordingly.

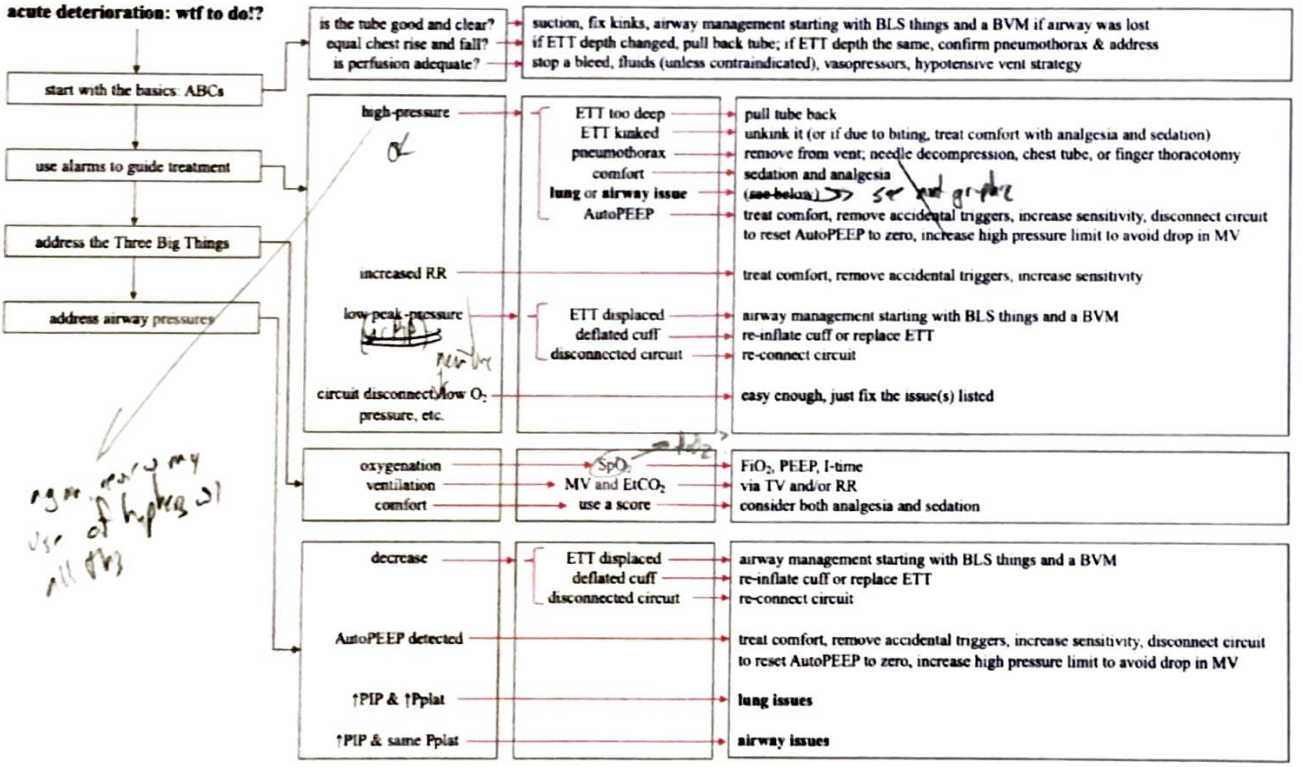
In light of the fact that there are so many variables involved, here's the stepwise approach we suggest for troubleshooting a crumping patient who is on the vent. And this approach takes advantage of feedback that we may have available to us from vent alarms and assessment parameters:



And in fact, one could argue that "using alarms to guide treatment" may even be a quicker solve than starting with the ABCs. While we recognize that this is blasphemy in the world of EMS and transport medicine, here's how that might look:



By working through each of these steps systematically, we hit all of the DOPE things and identify where in the system the issue lies (vent, equipment, physiology). Now it gets a bit more complicated when we add in specifics for each step along the way, but remember that the basic idea is a simple set of four steps:



There's no way to accommodate all possibilities in a single algorithm without getting too crazy with details, but that's the basic idea. But before moving on, just a few things to note. First is that a low MV alarm may also accompany acute deterioration, but it is likely tied to either a high-pressure alarm (with breaths cycling off due to that alarm getting triggered) or some kind of disconnect (which would likely be indicated by a circuit disconnect or low peak-pressure alarm). We also didn't include a low frequency or low PEEP alarm anywhere in this flowchart, as those probably aren't tied to an acute deterioration unless accompanied by one of these other trump cards. And then we already showed this once before (and recognize that not all of these are acute life threats), but just to clarify again the different lung and airway issues we might come across:

lung issues	
abdominal distention/ ascites	consider positioning
atelectasis	recruit some alveoli (via PEEP and/or recruitment maneuvers)
AutoPEEP	increase I:E ratio, trial VC, maybe adjust PEEP, disconnect circuit
chest wall burn	escharotomy
pneumonia	utilize PEEP to displace fluid
pneumothorax	remove from vent; needle decompression, chest tube, or finger thoracotomy
pulmonary contusion	consider ALI/ARDS strategy
pulmonary edema	utilize PEEP to displace fluid

airway issues	
aspiration	suction (prevent further aspiration), consider ALI/ARDS strategy
bronchospasm	fix with drugs, implement obstruction strategy
ETT occlusion	address comfort (biting), swap tube (something stuck)
secretions	suction

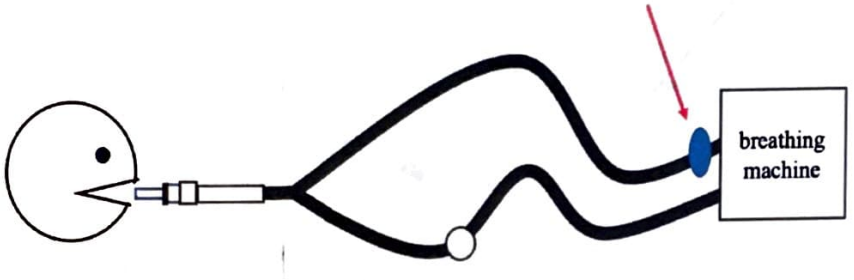
Now let's summarize what actions to take in the event of an acutely deteriorating patient on the vent. While there is a well-known memory tool (the "DOPE" mnemonic) to guide us through troubleshooting potential issues, that tool doesn't consider feedback from the machine (i.e. alarms) and, therefore, we suggest a simple sequence of four steps to work through potential issues: check your ABCs, look at and address any alarms, review the Three Big Things, then check pressures. If by then we haven't figured out our problem, we can consider taking the patient off the vent and bagging by hand (still not a great strategy though…) or getting out the ultrasound machine to try and identify an issue (if available).

Return to Contents

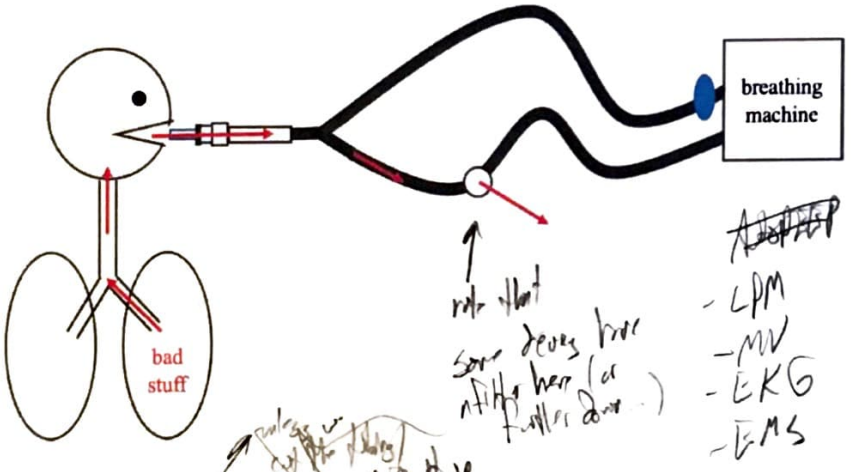
Other Random Things There May Be Questions About

Filters[179]

Filters are used in mechanical ventilation to prevent infectious gunk from transferring from one spot to another. In the transport setting we generally use in-line filters that simply fit in to the vent circuit. While there are a few possible options as to where we place the filter, it is most commonly put at the connection between the machine and the vent circuit (i.e. the inhalation side of the system):



The filter placed here essentially keeps bad stuff from the machine from getting to the patient. Which is fine, just recognize that it doesn't keep bad stuff from the patient from getting to us and our coworkers:



Now we could work around that by placing the filter at the patient's face/ ETT or even on the exhalation side of things, but the face option will increase mechanical dead space[180] and the exhalation side option may not be available with our transport vent. That said, placing a filter near the ETT may be warranted in certain cases (tuberculosis, flu, etc.), just know that in addition to the dead space issue it can also impede the movement of air (or flow) and that the fix for this is to increase air movement into the system (in VC this will probably happen automatically, in PC we may have to increase the pressure put into the system) and watch for adequate exhalation. But if you have a patient with some type of bad stuff that you don't want to breath in and neither of these strategies/placements is appropriate or possible, be sure to mask up!

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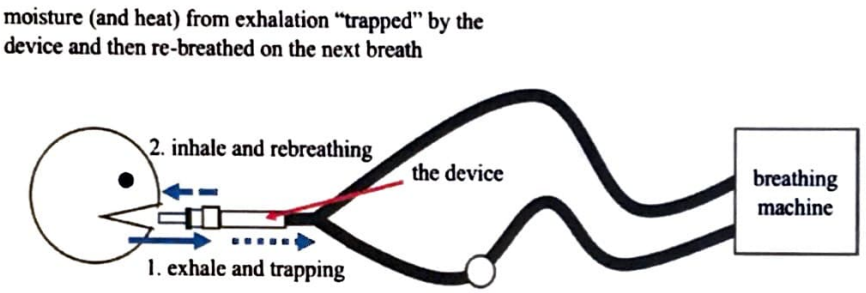
[179] Wilkes, 2011a & 2011b – He gives the most in-depth discussion of both filters (this section) and humidifiers (next section)
[180] Discussed in both Dead Space and Appendix

Humidifiers[181] [182]

Humidification of air is important in mechanical ventilation because dry air can cause damage to the lining of the respiratory tract. No need to get into the details here, just know that absent any contraindications we ought to try and add some degree of humidification to the air we push into the patient's lungs. We typically do this in transport by placing a humidification device called an HME (humidification and moisture exchanger) between the ETT and wye of the vent circuit. Placing the device further up on the inhalation side of the circuit would not work, as the device functions by trapping moisture (and also heat) from exhaled air and allowing it to be blown back into the patient's airways on the subsequent breath:



[181] Yartsev, 2019 – Excellent discussion of the passive style devices used in the transport setting

[182] Gillies & friends, 2017 – This Cochrane Review has determined that HMEs are comparable to actual humidifiers in providing therapeutic benefit and avoiding primary complications (airway obstruction, pneumonia, mortality) – while they admit that more research is needed, it's good to know that HMEs do have demonstrated value

It is worth mentioning that the HME is often the biggest contributor to our mechanical dead space (as outlined in the **Appendix**), but it ought to be used unless we have good reason not to. First (of two) good reasons not to would be small TVs, such as kiddos or ALI/ARDS patients.[183] In these situations, we want to minimize mechanical dead space as much as possible. Now there are smaller HMEs designed for littles and here's the basic idea on that: HMEs are rated to provide humidification for a certain amount of TV, higher value corresponds with more space needed within the internals of the device and, therefore, more dead space. To make this clear, let's look at info from one particular product line:[184]

Gibeck® Humid-Vent® HME

ITEM CODE	DESCRIPTION	TV RANGE (ML)	MOISTURE OUTPUT (MG H2O/L)	WEIGHT (G)	RESISTANCE (CM H2O)	DEAD SPACE (ML)	CASE QUANTITY
10011	Humid-Vent Mini	15-50	30, Vt = 20 mL	4.5	0.9, 10 lpm	2.4	30
11112	Humid-Vent 1	50-600	30.5, Vt = 0.2 L	9.4	0.3, 20 lpm	10	30
11132	Humid-Vent 1 port	50-600	30.5, Vt = 0.2 L	11.6	0.3, 20 lpm	14	25
13312	Humid-Vent 2 port	150-1500	28, Vt = 0.6 L	20.9	0.8, 60 lpm	29	20
14412	Humid-Vent 2S	150-1500	28, Vt = 0.6 L	19.8	0.8, 60 lpm	29	20
17731	Humid-Vent 2S Flex–sterile	250-1500	28, Vt = 0.6 L	26.4	0.8, 60 lpm	54	20
17732	Humid-Vent 2S Flex–clean	250-1500	28, Vt = 0.6 L	26.4	0.8, 60 lpm	54	20

we see here that more capacity for humidification correlates with more dead space

Second good reason not to use an HME would be the concurrent use of nebulized medications.[185] We want those drugs going into the patient, not getting absorbed by the HME. While we could theoretically place the in-line nebulizer between the ETT and the HME, that could also result in decreased medication administration unless we also added in a spacer. But then we'd have a huge amount of dead space and we already established that we want to cut down on that whenever possible. Also, the need for an HME is less with a nebulized medication because we are actively pushing moisture into the airways along with whatever medication is being given. One last time: no HMEs with nebulized medications. Don't try to rig it up to make it happen, as this will cause more problems. It is, however, OK to remove the HME for administration of a nebulized drug and then reattach it as soon as that is done.

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[183] Hinkson, 2006 – And we'll get back to this idea in the **Appendix** also

[184] Teleflex, 2019 (images) – Just to be clear, no relationship/conflict of interest here – it's just really nice how they lay out all the product info like this for us to talk about ☺

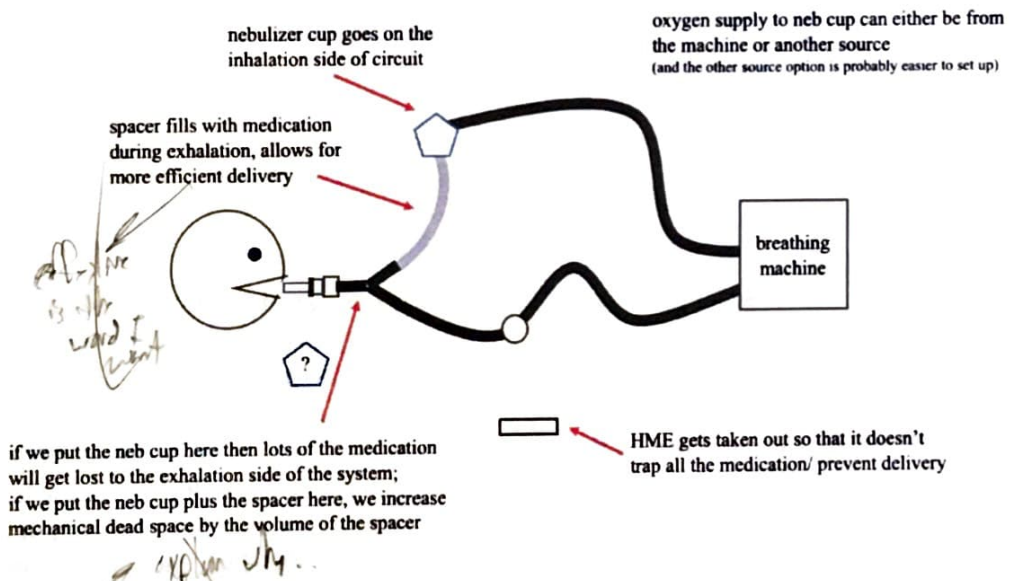
[185] And see the very next section for a discussion of **In-line Nebulization**

One other situation in which we ought to exercise concern with an HME would be increased secretions, as the HME can get clogged up to the point where it impedes air flow. This isn't a situation in which we never use an HME, rather it's one of those cases where we need to be aware of potential problems. Increases in PIP in VC or decreases in VTe in PC would likely be our first indication of an airflow problem of this sort.[186] If this happens and we are worried about an HME getting clogged up, we can either remove the device or replace it with a fresh one.

Very last thing about HMEs before moving on: while all HMEs provide some filtration of exhaled air, certain devices may even be classified as both filters and HMEs. This could potentially mitigate the escape of potentially infectious material from the patient into the ambient air via the exhalation side of the vent circuit as we drew out in the last section.

In-line Nebulization

Just to demonstrate a few things about why we do nebs the way we do, let's look at a setup of how the system looks when we nebulize a medication through the vent circuit. Recognize that there may be some variation between models, this is just the setup with which we are most familiar with and serves to outline the important stuff:[187]



That should be clear enough, but just to expand on a few points: we may need adapters and extra vent tubing to make this work, so we should plan ahead and have that stuff available in pre-built kits. The spacer is important, don't throw it away every time you open a circuit… Some machines recommend specific changes to settings to facilitate this process, read up on that and/ or have a chat with the manufacturer's rep for details about a particular machine.

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[186] Since we don't routinely monitor flow in the transport setting

[187] Dhand, 2017 – And for more info on placement of the nebulizer and bias flow (which we don't mention here) as it relates to this, take a read of this article

Return to Contents

Driving Pressure[188]

Driving pressure is a term to describe how much we inflate and deflate the alveoli with each inhale and exhale on the ventilator. The idea is that too much opening and closing (inflation and deflation, up and down – however we want to term it) can put stress on the alveolar walls and cause damage.[189] This damage, in turn, leads to decreased diffusion of gasses across the alveolar membrane. Driving pressure is the difference between Pplat and PEEP and is sometimes referred to ~~and represented~~ as delta pressure:

$$\Delta P = Pplat - PEEP$$

With our ALI/ ARDS patients, we try to limit driving pressure as much as we can to a max of 15cmH_2O.[190] Which is generally pretty reasonable, given that we use high PEEPs and low TVs in these patients anyways. And in the event that driving pressure is close to or above that upper limit, we can do **Recruitment Maneuvers** to try and utilize more lung, increase compliance, and drop driving pressure. This approach may sound familiar and is often referred to as "open lung" ventilation.[191] The idea here is that we keep the lungs as filled as possible (i.e. alveoli inflated) throughout as much of the respiratory cycle as possible. Again, this concept of limiting driving pressure and an "open lung" strategy are specific to the ALI/ ARDS population.

With that said, there may be a case for a comparable strategy in other patient groups, there just hasn't been much research on that to date. The one downside of this limited driving pressure/ "open lung" approach is that it can be tough to blow off CO_2 as much as we'd want. We said way back when that permissive hypercapnia is often a thing with ALI/ ARDS, but that may not be the case with other patient groups. Another consideration here is PEEP. PEEP is not a benign thing and we for sure need to consider all of the negative consequences of this approach[192] before applying it to all patients. For now we have pretty good evidence that limiting driving pressure and utilizing high PEEP is a good thing in the ALI/ ARDS population, but such a strategy may not be ~~best~~ for everyone.

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[188] Bugedo & friends, 2017 – Succinct overview of the concept of driving pressure and research done to date (as of a few years ago, at least!)

[189] Grune & friends, 2019 - While this is commonly accepted idea and we will assume it to be valid in our discussion, know that there is ongoing research on all of this (as shown in this article)

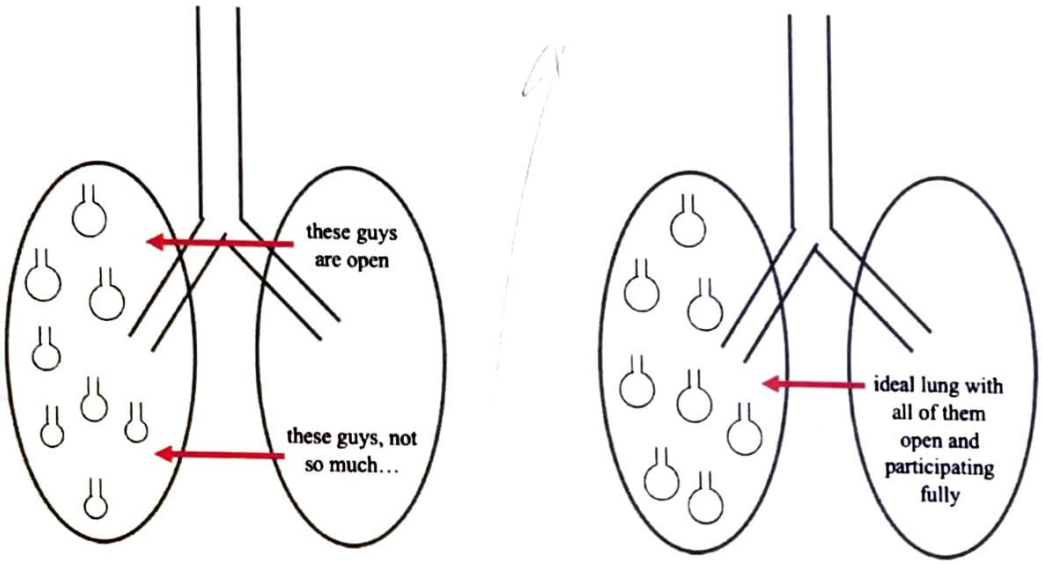
[190] Weingart, 2016a; Bauer, 2016b – Both podcasts look at a 2015 study on the subject

[191] Nickson, 2019b – Concise overview of the idea with many more resources cited

[192] ~~As discussed in **PEEP**~~

Recruitment Maneuvers[193]

A recruitment maneuver is a component of the "open lung" strategy that seeks to get more alveoli involved in the ventilation process. The idea is that there are portions of the lung that are open or participatory and others that are closed down or non-participatory (or maybe just less-than-optimally-participatory), and that we can do things to gain access to those clamped-down alveoli to improve both ventilation and oxygenation:

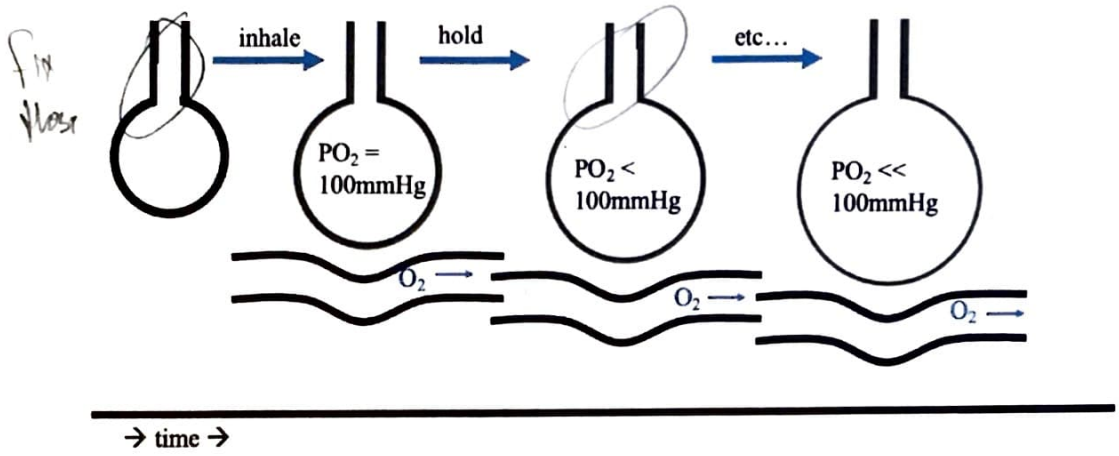


In a general sense, lots of things could qualify as recruitment maneuvers: prolonged inspiratory holds, higher PEEP, high frequency oscillation ventilation,[194] prone positioning, spontaneous breathing, etc. Basically anything that can help open those non-participatory alveoli falls into this category. Now in the transport setting (and, in fact, for most vent people), we tend to consider recruitment maneuvers to be either the prolonged inspiratory hold or the stepwise approach, so we will stick with those two ideas moving forward.

[193] Ragaller & Richter, 2010; Naik, 2015– The first is an overview of ALI/ ARDS management with one section on the idea of recruitment; the second is an article that also discussed recruitment, but particularly the idea that breaths of various sizes (whether intentional via vent management or spontaneous via patient effort) further contribute to recruitment

[194] Prost, 2011 - This is the only mention we have of this mode, as it isn't routinely available in transport: the referenced video is an overview of high frequency ventilation

We posed a hypothetical situation at some point earlier on in this manual about why we don't just blow up the lungs and alveoli with oxygen and let it sit like that for a while; we said then that we still have to consider the ventilation/~~CO_2~~ side of things, but the idea itself does have some merit. That said, the value of a recruitment maneuver (again, this is as a prolonged inspiratory hold) is more in the ability to open alveoli past that difficult-to-open stage than in the inflow of oxygen for a sustained amount of time, as the amount of oxygen in that air quickly begins to drop as oxygen diffuses in to the bloodstream and we don't replenish the supply:



A recruitment maneuver in this sense can be used to gain recruitment in any patient group, but has been most studied with ARDS patients. And while it has been shown to increase oxygenation, outcomes in terms of mortality and days on the vent seems to be unaffected or even worse.[195] To further complicate things: when we do try and get into the weeds as to how we should perform a recruitment maneuver, techniques vary significantly and there are potential adverse effects. So here's where we stand on this: more data is clearly needed, but there is low quality evidence that some benefit exists from performing recruitment maneuvers in ARDS patients; particularly as part of an overall "open lung" strategy.[196] Translating that to the non-ARDS patients who are simply hypoxic is a bit tough, as there isn't much data out there and we can often fix the issue by way of thigs we've already talked about (FiO_2, PEEP, and I-time) and ensuring adequate perfusion.

[195] van der Zee & Gommers, 2019 – Describes lots of the research that has gone into understanding this whole concept
[196] Hodgson & friends, 2016 – Cochrane Review that gives way more detail on this

But let's say we do want to do a recruitment maneuver for whatever reason. Maybe we are struggling to oxygenate a patient, or we forgot to clamp the ETT on transfer of an ARDS patient to our vent, or we want to try for better compliance/decreased driving pressure, etc. First thing to know is that the maneuver can cause hemodynamic problems and we ought to be on the lookout for those to avoid decompensation. Just as we discussed back when we first got into **How is Positive Pressure Different?** and **PEEP**, an increase in intrathoracic pressure can drop preload and subsequently impact cardiac output. So monitor all the things and have hard limits in place for abandoning the maneuver.[197] Also recognize the risk for causing a tension pneumothorax and consider that a floppy ETT cuff or uncuffed pediatric tube[198] will render the maneuver less effective.

The simplest way to do a recruitment maneuver is the prolonged inspiratory hold option that we mentioned above.[199] While this was often taught in the past, it is becoming less common in deference to more gentle and stepwise strategies. But to make it happen, here's how it would work: put your patient in PC mode, set PC to get a goal Pplat, then perform an inspiratory hold for as long as we think is appropriate. As far as specific on pressures and time, the data varies widely on that and we can't make specific recommendations on how that might look. Same goes for how often to perform the maneuver – most of the data out there discusses vented patient in an in-patient setting, so it is difficult to translate that to the transport setting in which we are only with the patient for a short amount of time.[200]

We mentioned already[201] that whenever we put more air into the lungs it seems advantageous to do so incrementally. Same goes for performing a recruitment maneuver. An alternative to the prolonged inspiratory hold would be a stepwise approach in which we put a patient in PC mode and establish a driving pressure (Pplat minus PEEP) that yields our goal TV, then slowly titrate up on PEEP in small steps and over time.[202] There is a rendition of this approach called the Staircase Recruitment Maneuver that titrates PEEP back down to a maximally beneficial level as determined by SaO_2 monitoring[203] – perhaps a modified version with SpO_2 monitoring and longer times between titrations (to accommodate a potential lag in SpO_2 readings) would be appropriate in transport.

In any event, the utility of recruitment maneuvers is to get more alveoli involved in ventilation. This allows us to ventilate to our TV goal with lower driving pressure, improves compliance, and works to correct V/Q mismatch across the lung.[204] While there are risks involved and the data is a bit vague when it comes to long-term benefits, it seems fair to conclude that if we mitigate those risks by using a stepwise approach and monitoring for patient decompensation along the way there is likely some use in the transport setting.

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[197] Claire & friends, 2019 - And for suggestions on these limits and an explanation of the next technique (the stepwise recruitment maneuver), take a look at this short guide

[198] Chambers & friends, 2017 – This study primarily examined how VTe differed from delivered TV with cuffed and uncuffed tubes

[199] Metz, 2016a – Video that shows this type of recruitment maneuver

[200] And we recognize that the lack of concrete suggestion here might be frustrating, but this is one of those things better answered by the agency or medical director that we work for…

[201] In the section, **Titrating Up on TV?**

[202] Metz, 2016b – Another video by the same guy as above, this one is a version of the stepwise recruitment maneuver

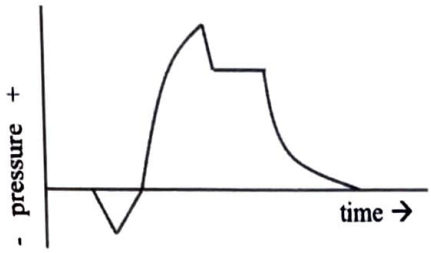
[203] Hess, 2015 – Take a look here for a discussion of this technique and others

[204] Hartland & friends, 2015 – We cited this study back when we discussed absorption atelectasis in **Oxygenation (and SpO_2)**; while it looks at a specific group of patients we don't often encounter in transport (those undergoing abdominal surgery), the findings are consistent with this conclusion

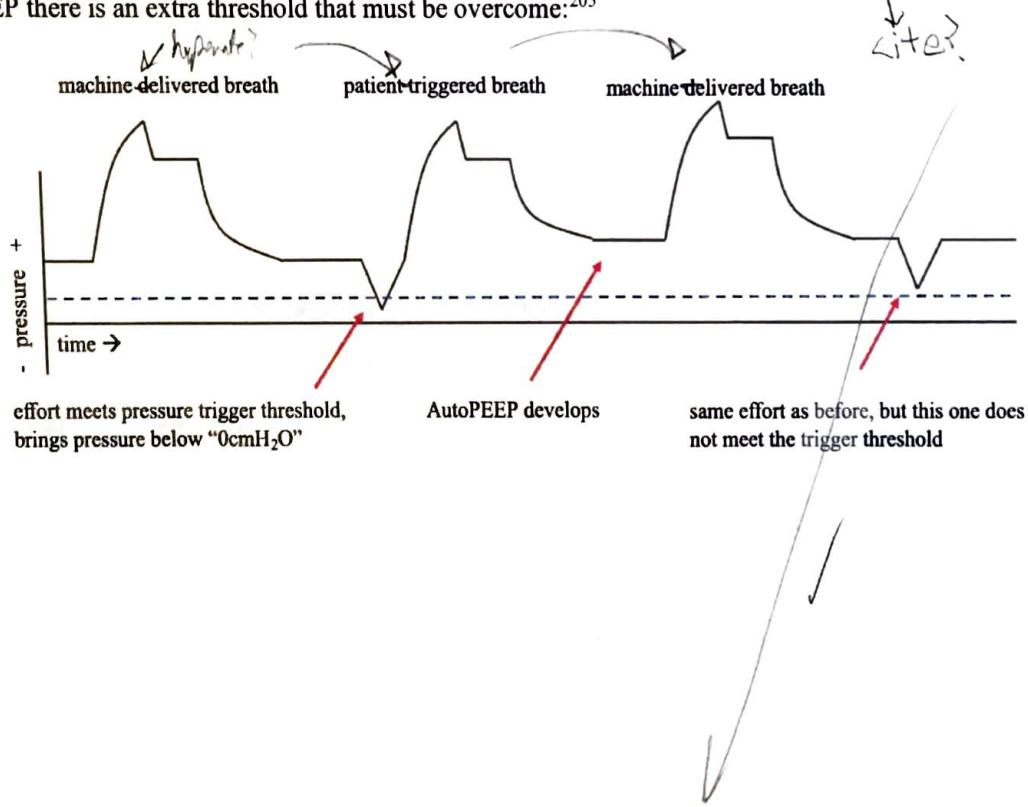
Return to Contents

Triggers

Triggers are the thresholds by which the machine knows when a patient is trying to breathe on his or her own. We first tried to communicate this idea via the following graphic:

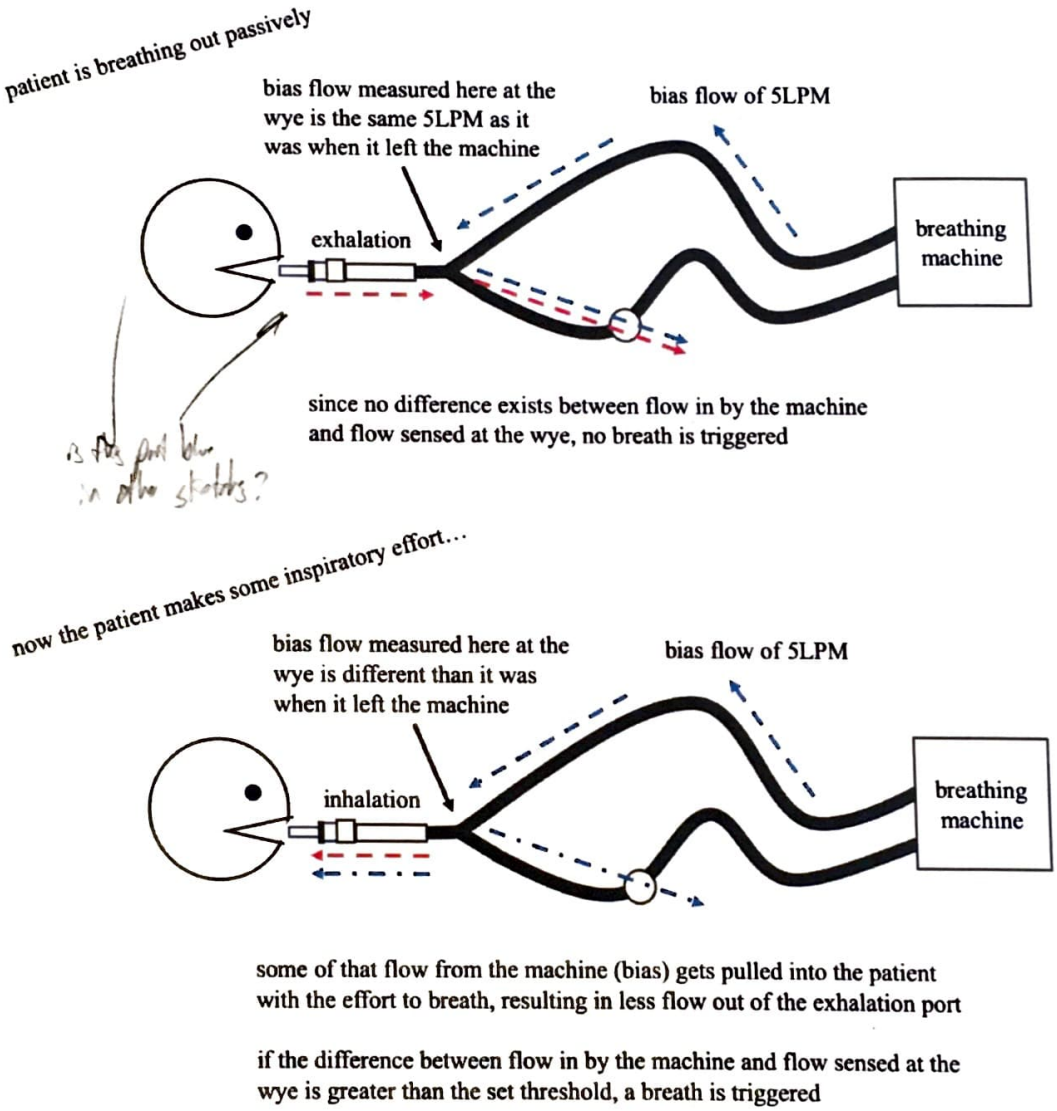


And then we footnoted the idea that that downward dip in pressure at the start of the waveform is more a sketch of convenience than an accurate representation of how things ~~actually~~ occur. In most cases the trigger that makes the machine recognize patient effort is based on flow rather than pressure. While some machines will allow you to use pressure triggers (normally around -1cmH_2O), this isn't commonly used. Pressure triggers have been shown to be more difficult for patients to overcome (at least with older model ventilators). In addition, the pressure trigger is relative to what we have dialed in for PEEP – this means that in the event of AutoPEEP there is an extra threshold that must be overcome:[205]



[205] Hess, 2005 – This explains how switching to a pressure trigger may mitigate breath stacking

So pressure triggers are a thing as we initially drew it out, but not the most common thing. We sometimes do use pressure triggers in cases of auto triggering (i.e. when we see too many triggered breaths due to things other than patient effort,[206] such as bumpy roads in an ambulance or turbulence in an aircraft), but for the most part we stick with flow triggers. To measure flow changes against a zero reference (i.e. we assume the pause between breaths to be a zero-flow state) the machine uses a concept called bias flow. Bias flow is a baseline flow of air into the system against which changes are measured. So when the machine says there is no flow going into the system, there is actually some flow going in, but it gets factored out ~~by the machine~~. Let's draw it out with an assumed bias flow of 5LPM just to see how it works:[207]



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[206] While we could utilize pressure triggers to mitigate worsening AutoPEEP with increased patient respiratory effort (assuming an initial flow trigger), we prefer to address the cause of discomfort or meet the patient's demands rather than ignoring it altogether

[207] Yartsev, 2019 – For more information in these triggers, others, and some of the stuff discussed in the rest of this section

The machine does this bias flow thing because it makes it easier to measure patient effort. It also allows for things like PEEP and the delivery of nebulized medications.[208] But enough on that. The point worth knowing is that a flow trigger cannot be set to a value greater than the machine's bias flow. So in the case where we have lots of accidental triggers (i.e. auto triggering is happening) and our trigger is set at 5LPM and we know our machine has a bias flow of 5LPM, we can do one of two things on the machine: switch to a pressure trigger or change (increase) bias flow to accommodate a higher trigger threshold.

And while we are on this point, it is worth discussing things we can do to address auto-triggering other than manipulating settings on the vent. First is to try and identify what input is causing the triggers. If it is a bumpy road or turbulence, perhaps getting the vent circuit off of the floor of the vehicle can alleviate the issue. If it is one of us crewmembers kicking the circuit, just stop doing that. Sometimes we get down a rabbit hole trying to accommodate a situation that can be avoided in the first place by taking a step back and seeing what is going on beyond the machine itself. That said, we should always attempt to address the cause of strong patient triggers, particularly discomfort and need for more MV.

Let's summarize triggering ~~up to this point~~: triggers are thresholds we set ~~for when~~ the machine knows that the patient wants to take a breath. We most commonly use flow triggers, but some machines allow for pressure triggers as well. Flow triggers are based on and limited by bias flow; normal bias flow is 5LPM, that gives us a range of 1-5LPM for setting our flow trigger. And for reference, 1-2LPM is commonly used in a hospital setting. Auto-triggering happens when the trigger is inadvertently met by movement other than patient effort to breath. Fixes to auto-triggering include mitigating the cause of the inadvertent trigger, increasing the trigger threshold, or trialing a different type of trigger.

Prone Ventilation

Prone ventilation is when we lay our ventilated patient face down on the bed or stretcher. Arguments and evidence in favor of prone ventilation include things like better V/Q match, decreased shunt, improved oxygenation, better ventilation, etc.[209] That said, prone ventilation isn't for everyone, studies are shrouded a bit by bias, and efficacy seems to be related to early implementation, time of application each day (16 hours per day!), and severity of hypoxemia (i.e. proning has benefit when oxygenation is a major issue).[210] ~~And~~ when we are called to transport a pronated patient, there are some logistical limitations to the process. Much of what we do requires access to the patient's front side and many of the tools we use in medicine are designed with the supine patient in mind. All that said, it is likely that we will see more of this in years to come so it made sense to do a quick survey as to where things are at in regard to prone ventilation in the field.

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[208] Dhand, 2017 – We cited this article previously in the section on **In-line Nebulization**

[209] Koulouras & friends, 2016; Henderson & friends, 2014 – And for details on any of those concepts, take a look at either of these articles

[210] Bloomfield & friends, 2015 – That said, when proning has been initiated it is likely for good reason and we transport people can help continue the strategy





Prone ventilation has been mostly studied in patients with ARDS. Given that ARDS isn't something we commonly diagnose or come across initially on scene runs, it seems likely that most of our prone ventilation will be done in the context of interfacility transfers. Which is good, because the process of getting someone pronated with an ETT and vent in place isn't the fastest thing we could do and managing an airway on an already pronated presents its own complications. So interfacility transfers of ARDS folks seems to be where we will most likely be using this technique as critical care transport providers.

We mentioned before in our section on **ALI/ARDS** that recruitment of alveoli is very important. So while it may be tempting to simply flip a pronated patient over for transport and then let the receiving facility re-pronate them, this could potentially set progress back quite a bit, so we want to do what we can to keep our actions in line with overall clinical course. That said, many treatment guidelines/ algorithms for this sort of thing include cyclical proning on some sort of schedule,[211] so it may be worth scheduling these transfers in line with transport capabilities (i.e. with no capacity to transport a prone patient, simply wait until it's supine time and make it happen then).

When it comes to the physical process of flipping someone over, there are a number of techniques and tools than run the gamut from a RotoProne bed[212] to simply using a flat sheet or slider.[213] Proning can also be performed at the time of transfer from one bed or stretcher to another (for example, let's say we are going from a hospital that doesn't do this to one that does – we could facilitate this at either end of the transfer).[214] This means that even if we don't transport a patient in a prone position in our vehicle, we may still get caught up in the process at some point.

A few considerations about transporting a pronated patient: access to the airway may be difficult or impossible, access to the anterior chest wall (for EKGs, assessment of heart and lung sounds, needle thoracostomy, etc.) will be limited, and stretcher/sled configuration may dictate that the patient be horizontal. For all of these reasons (and probably a great many others), it may be quite some time (or eternity…) until certain programs and crews decide to attempt this, but rest assured that it has been done already[215] and will likely become more common in years to come.

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[211] Olveira & friends, 2017 – And as one example of that, take a look at this protocol; also goes into detail on how to carry out the physical maneuver and discusses many of the concerns that could potentially arise along the way

[212] Arjo, 2020 – Manufacturer's content on this product, just for those who are curious about it

[213] Critical Care & Major Trauma Network, 2015; Critical Cardiff, 2017 – Two YouTube videos that demonstrate proning a patient

[214] Hospital Direct, 2017 – Another YouTube video that shows the maneuver while moving a patient between surfaces

[215] Boon & Boon, 2018 – These guys have both done it and provide a good overview of the application of proning in the transport setting, as well as a bit of an overview on the ALI/ ARDS pathology we already discussed; they also have a video at that same link that shows a one-person technique for flipping a patient on an EMS stretcher



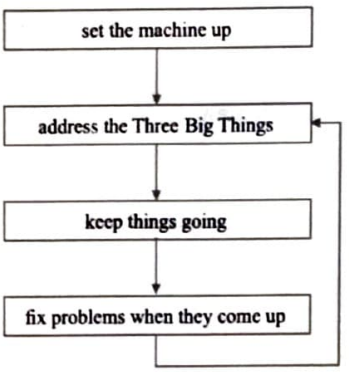



Return to Contents

A Proposed Protocol/ Flowchart

We said already that the goal of this learning experience is to know enough about vents that we can break out of the "cookie cutter" approach to management and understand why/make changes and how that impacts our patients. That said, it may help to have a framework to work with while we move towards that goal. We've tried to create an algorithm that covers all we've talked about up to now, that is generic enough to apply to different machines, and that fits on two opposing pages so that it can easily be utilized as a reference in the field. It's here to help folks work towards a higher level of competency or to simply take some of the load off of one's mind when things get busy on scene or in transport.

The basic idea of the flow is something like this:

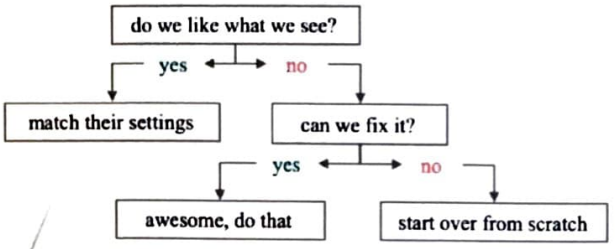


How to do Vent Stuff

Set the Machine Up

1. Prep
   a. Get a report from sending
   b. Do some arithmetic: IBW, TV, MV
   c. Assess the patient
   d. Consider a strategy
   e. Check circuit, attach $EtCO_2$ and ~~HEPA~~ filter, consider need for HME and/or suction

IBW_{dudes} (kg) = (2.3(height in inches) – 60) + 50
IBW_{chicks} (kg) = (2.3(height in inches) – 60) + 45.5
TV = 6-8ml/kg
MV = 100ml/kg (IBW) /min



2. Determine Settings
   a. Patient Already on Vent (see algorithm, right)
   b. From Scratch
      i. Turn on machine and leave at default mode and control
      ii. Dial in desired TV for 6ml/kg (or PC at 10-15cmH2O)
      iii. Adjust rate
         1. Adults: to MV goal
         2. Kiddos: using a reference range
      iv. Adjust I-time using a reference range
      v. Leave all other settings at machine defaults unless one of these considerations applies:

Age Description	Age (yrs)	RR	I-time (s)
Infant	.083 (1 month)-1	30-53	0.3-0.6
Toddler	1-2	22-37	0.4-0.9
Preschooler	3-5	22-28	0.5-0.9
School-aged Child	6-7	18-25	0.6-1.1
Big Kiddos	8-9	17-25	0.6-1.2
Preadolescent	10-12	14-23	0.7-1.4
Adolescent	12-15	12-20	0.8-1.7
Adult	16 and up	12-20	0.8-1.7

Strategy	Things to Do
Obstruction	increase I:E (≥1:5) by decreasing RR (and maybe I-time also), then titrate TV (or PC) up to maintain MV as able; consider less PEEP
Hypotension	limit PEEP; increase TV and then decrease RR to maintain MV
Acidosis	use high end of TV (goal): 8ml/kg IBW; increase RR: pre-intubation rate, to get prior/goal $EtCO_2$, or double normal value
ALI/ ARDS	higher PEEP

3. Initiate Ventilation (clamp tube if concerned with de-recruitment)

Address the Three Big Things

Parameter	Assessment	Normal	Actions
Oxygenation	SpO_2	93-99%	*Low:* consider position & suction, increase FiO_2, increase PEEP, increase I-time, consider pathophysiology/ medications *High:* decrease FiO_2 (unless contraindicated, i.e. pregnancy, anemia, hemorrhage, TBI, shock, etc.)
Ventilation	$EtCO_2$	35-45mmHg (low end for TBI)	*Any abnormal value:* consider etiology/ patient compensation for acid-base imbalance (may be best left alone) *Low:* consider perfusion status, decrease RR (monitor MV), then consider decrease in TV *High:* increase TV (max 10ml/kg, monitor Pplat), then consider increase in RR
	MV	100ml/kg/min (2x with acidosis)	*Low:* increase TV and/ or RR *High:* consider patient comfort, decrease TV and/ or RR, consider SIMV
Comfort	Ramsey, ANVPS, etc.	at provider discretion	analgesia & sedation, consider settings (MV and I-time), also consider accidental triggering

Return to Contents

Keep Things Going

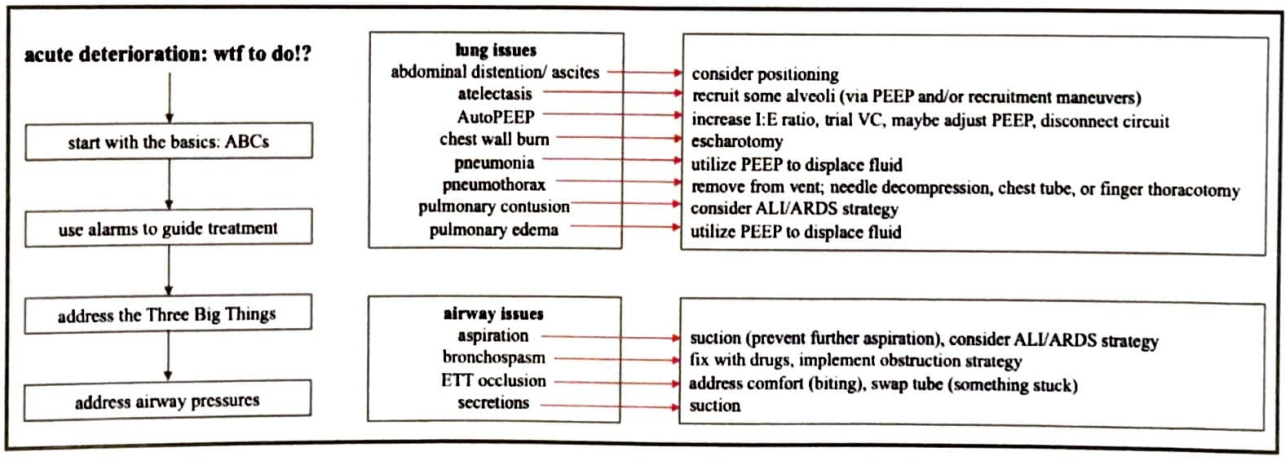
1. Set (and Troubleshoot) All Alarms
2. Consider Pressures (every time vitals get reassessed)

Parameter	Normal	Actions
Peak Inspiratory Pressure (PIP)	<35cmH_2O	consider potential causes (**lung** and **airway issues**) by checking Pplat decrease TV (or PC)
Plateau Pressure (Pplat)	<30cmH_2O	consider potential causes (**lung issues**) decrease TV (or PC)
AutoPEEP	none	increase I:E (lower RR, shorter I-time) consider inadvertent triggering, trial VC if in PC, avoid high PEEP disconnect circuit to allow exhalation
Driving Pressure (ΔP)	<15cmH_2O	decrease TV or PC consider more PEEP and permissive hypercapnia consider recruitment maneuvers
Mean Airway Pressure (P_{aw})	not applicable	monitor for trends and investigate further

3. Make Adjustments Moving Forward

Strategy	Things to Do
General Stuff	if oxygenation is all good, go down on FiO_2 (maybe all the way to 0.40) and reevaluate consider increasing TV to safe Pplat and acceptable ΔP
Obstruction	use drugs (in-line neb treatment, consider Ketamine for analgesia/ sedation, etc.) ensure no AutoPEEP develops if hypercapnia develops and no AutoPEEP noted, consider moving towards normal I:E
Hypotension	use caution with PEEP to improve oxygenation consider fluid and/ or pressors if perfusion improves, consider working towards normal settings to avoid higher Pplat and ΔP
Acidosis	maintain increased MV goal (minimum 200ml/kg/min) also consider Winter's Formula to guide treatment
ALI/ ARDS	consider titrating TV down to 5ml/kg, then 4ml/kg to maintain ΔP <15cmH_2O increase PEEP to maximize oxygenation, consider stepwise approach consider recruitment maneuver if hypoxia persists

Fix Problems When They Come Up



Suggestions for Further Study

Just some suggestions based on what kind of medium someone is looking for. This is not an exhaustive list, but just some places to start for getting better at the management of vented patients. Also recognize that each of these references has way more to offer than just the specific content linked – browse them all for more intel on many of the specifics we've discussed in this manual.

Audio/ Podcast

EmCrit Dominating the Vent Series
Part 1, Part 2


FlightbridgeED Vent Series
Part 1, Part 2, Part 3



Video, Vent Specific

Strong Medicine Series on Mechanical Ventilation

Thoracic.org Videos on Mechanical Ventilation

Video, Physiology

Ninja Nerd Science, section on Respiratory

Kahn Academy, section on Advanced Respiratory System Physiology

Text, Web-Based

Deranged Physiology, section on Respiratory

RebelEM, Simplifying Mechanical Ventilation
Part 1, Part 2, Part 3, Part 4, Part 5




Text, Books to Buy

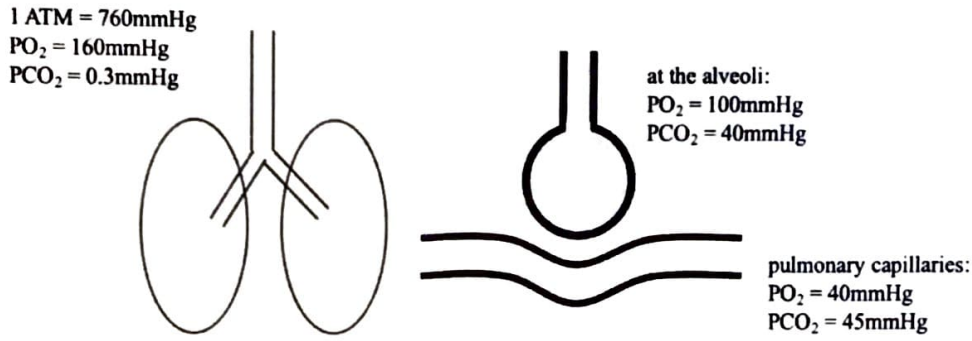
Ventilator Management: A Pre-Hospital Perspective by Eric Bauer

Vent Hero: Advanced Transport Ventilator Management by Charles Swearingen

Appendix

Alveolar Gas Equation

The alveolar gas equation allows us to calculate the partial pressure of oxygen in the alveoli in a given set of circumstances. We used this equation to get values listed in some of the graphics throughout this manual:



because there is an open system between the ambient air and the alveoli, the overall pressure at the alveoli is also 760mmHg, however the partial pressures of the components are different along the way

The equation goes like this:[216]

$$PAO_2 = FiO_2(P_{atm} - P_{H2O}) - (PaCO_2/RespQ)$$

PAO_2 is partial pressure of alveolar oxygen
FiO_2 is fraction of inspired oxygen, 0.21 for ambient air
P_{atm} is atmospheric pressure
P_{H2O} is partial pressure of water vapor at the alveoli, 47mmHg
$PaCO_2$ is as measured by ABG (or approximated from $EtCO_2$), we'll say 40mmHg
RespQ is respiratory quotient and is assumed to be 0.8[217]

Given that Resp Q = 0.8, we sometimes see the equation simplified as so:

$$PAO_2 = FiO_2(P_{atm} - P_{H2O}) - 1.25(PaCO_2)$$

And since P_{atm}, P_{H2O}, and $PaCO_2$ are all held constant in our thought experiments:

$$PAO_2 = FiO_2(760 - 47) - 50$$
$$PAO_2 = FiO_2(713) - 50$$

---

[216] Yartsev, 2019 – He's got a good graphic that shows the alveolar gas equation with all parts labeled, maybe makes a bit more sense to the visual learners than how it is represented here

[217] Patel & Bhardwaj, 2018 – These guys describe the details behind this "respiratory quotient" idea; maybe not relevant to our discussion of vent stuff, but good nerdy details for those who want more (another option would be to find yourself an exercise physiology textbook, likely to be some ~~intel~~ there)

But back to our original equation:

$$PAO_2 = FiO_2(P_{atm} - P_{H2O}) - (PaCO_2/RespQ)$$
$$PAO_2 = 0.21(760 - 47) - (40/0.8)$$
$$PAO_2 \approx 100mmHg$$

Other iterations of the alveolar gas equation that we demonstrated in the manual are shown here:[218]

PAO_2 at 100% or FiO_2 1.0 (no PEEP)
$$PAO_2 = FiO_2(760 - 47) - 50$$
$$PAO_2 = 663mmHg$$

PAO_2 with 5cm PEEP[219] (room air)
$$PAO_2 = FiO_2(760 (+ 4) - 47) - 50$$
$$PAO_2 \approx 101mmHg$$

PAO_2 during inhalation (20cmH_2O of pressure, no PEEP)
$$PAO_2 = FiO_2(760 (+15) - 47) - 50$$
$$PAO_2 \approx 103mmHg$$

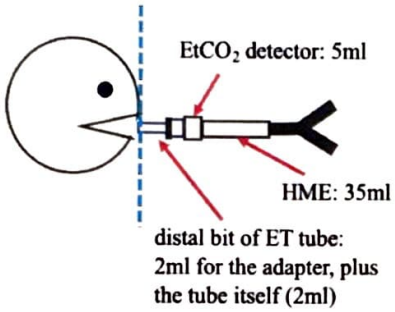
So we can use the alveolar gas equation to solve algebra problems in an effort to show how things like FiO_2 and PEEP affect PAO_2. And then if we know how much oxygen should be getting to the alveoli and can measure how much oxygen made it into the arteries (PaO_2 from a blood gas), then maybe we can understand something about the efficacy of that exchange. To say it another way, the idea is that we can use values for PAO_2 and PaO_2 to inform us on what is going on with a patient in reference to the movement of oxygen from the input of our vent system into the bloodstream. Values like "A-a Gradient" and "a/A Ratio" attempt to do just that. Now there are some limitations to both of these values and their application may be limited in the transport setting, so we won't get into the detail here.

[218] And this was back in the section on Oxygenation (& SpO_2)
[219] Just a friendly reminder that 5cmH_2O is roughly 4mmHg

Return to Contents

Mechanical Dead Space

In order to determine the effect of mechanical dead space, we first need to know how much volume each of the extra components takes up. This varies a lot depending on which specific device we use and can be found on the product labels that come with those devices, but we'll just generalize it here:



other potential things
in-line suction contraption/ angle: 5ml
flexible angle adapters: 10ml
filter: 50ml or more!

total estimate: 50ml
(excluding the filter, since we often put it elsewhere)

Now let's say we want to figure out to what effect 50ml of added dead space impacts ventilation (and our perception of that ventilation) in our patients. No this gets a little weird and the math takes a few leaps of faith along the way, but let's follow along and then compare what we come up with to data after the fact. Also note that we are going to introduce a few new ideas here and that we will get more into those in the very next section:

assume a patient of 65kg IBW
being ventilated at TV 6ml/kg (390ml) and RR of 17
MV calculated = 6630ml/min

now we already said a few things about this:
Alveolar TV = TV – Anatomic Dead Space
and this dead space is approximately 1/3 of TV
so Alveolar TV = 260ml
Alveolar Minute Volume (VA) = RR x Alveolar TV
in this case VA = 4420ml/min

and if we add 50ml more of dead space into the situation
Alveolar TV = TV – Anatomic Dead Space – Mechanical Dead Space
so alveolar TV = 210ml
VA = RR x (Alveolar TV – Mechanical Dead Space)
in this case VA = 3570ml/min

We already know that there can be a discrepancy between these two versions of VA, the one with mechanical dead space left out and the one with it included. But now let's consider the idea that the amount of CO_2 produced per minute doesn't change from case to case, rather it's simply the case that less of that CO_2 gets exhaled. So how much CO_2 gets left behind in the system what and kind of effect does that have on the body? To answer the first question, let's look at the following relationship:

$$\frac{V_D}{TV} = \frac{EtCO_2 - PECO_2}{EtCO_2}$$

Now there are two versions of this formula that use $PACO_2$ and $PaCO_2$ rather than $EtCO_2$, but it has been proposed that this representation might be of value in calculating dead space in practice.[220] So simply for the sake of this example, we will go with that. Now that $PECO_2$ value is something we haven't discussed yet, it is the mean partial pressure of CO_2 during exhalation. A normal value is around 30mmHg and it could also be calculated based on the idea that a normal fraction of expired CO_2 ($FeCO_2$) is about 4%:[221]

$$PECO_2 = FeCO_2 (P_{atm} - P_{water})$$
$$PECO_2 = 4\% (760mmHg - 47mmHg)$$
$$PECO_2 \approx 28.5$$

now if we use that value and the previous equation,
we can solve for an expected $EtCO_2$ in either of the dead space cases in question
only anatomic dead space:

$$\frac{130}{390} = \frac{EtCO_2 - 28.5}{EtCO_2}$$
$$EtCO_2 \approx 43$$

with mechanical dead space added in:

$$\frac{180}{390} = \frac{EtCO_2 - 28.5}{EtCO_2}$$
$$EtCO_2 \approx 53$$

[220] Siobal, 2016 – This is a theoretical thing and would require further experimentation, but it serves the purpose of showing to what extent dead space might impact quantitative measures of $EtCO_2$, all other things being equal
[221] ScyMed, 2018 – Good reference for calculations and normal values for all things physiology

Now a difference in $EtCO_2$ of 10mmHg doesn't necessarily mean that a corresponding quantity of CO_2 remains in the blood and impacts the body. The purpose of this exercise was simply to show that the potential exists for a buildup of CO_2. In the transport setting where $EtCO_2$ monitoring is routinely used to assess ventilation, we would simply increase MV to bring that second value into a normal range. But let's suspend that idea for just a moment longer and consider what impact this might have if we failed to do that. ~~In a study published in 2006,~~ [222] researchers looked at this very problem and determined that removing 115ml of dead space from a circuit resulted in decrease in $PaCO_2$ of 11mmHg and an increase of pH from 7.30 to 7.38. Furthermore, they were able to do that with less MV. Now this was in patients with ARDS in which one of our concerns is the amount of air needed to maintain ventilation and consequences of that air on the patient's pulmonary system, but the findings are pretty significant.

~~Now~~ back to our discussion and application to the transport setting: We said just a moment ago that we could potentially avoid this increased CO_2 retention by monitoring $EtCO_2$ and increasing MV to accommodate. But the truth is that doing so isn't always a benign thing. Going up on TV or PC will increase pressure (Pplat and ΔP), while going up on RR has the potential to cause discomfort and increase that %TaDP concept.[223] ~~So~~ if we can promote CO_2 removal while simultaneously avoiding all of those things, this seems like a pretty good reason to be conscious of adding unnecessary things into the vent circuit whenever possible.

One last thing about all of this in regard to kiddos on VC ventilation. We mentioned way back when[224] that it's OK if our calculated MV is larger than our goal MV because of some complications posed by dead space. We want to revisit that to show why that is and how we can mitigate it all. The example was a 4 year old kiddo of 18kg:

$$TV = 6 - 8ml/kg\ IBW$$
$$TV = 6 - 8ml \times 18kg$$
$$TV = 108 - 144ml$$

$$MV\ goal = 100ml/kg\ (IBW)\ /min$$
$$MV\ goal = 1800ml/min$$
$$MV\ goal = 1.8L/min$$

$$MV\ calculated = RR \times TV$$
$$MV\ calculated = (20 - 28)/min \times (108 - 144)ml$$
$$MV\ calculated = 2160 - 4032ml/min$$
$$MV\ calculated \approx 2.2 - 4L/min$$

---

[222] Hinkson & friends, 2006 – Small sample size, but significant findings that support the idea of limiting mechanical dead space

[223] And refer back to those respective sections for more: **Plateau Pressure**, **Driving Pressure**, **Comfort**, and **Hypotension**

[224] In the section **A General Vent Strategy**

Just as with the adult patient, we have anatomic dead space that is always there and then mechanical dead space that we add in. But we never did consider that the vent tubing itself has some flex to it. If you look closely on the label of your vent tubing, it may say something like "compliance 0.0008L/cmH_2O." So let's take that hypothetical example and run with it:

let's go with a TV of 6ml/kg (108ml) and a rate of 24
MV calculated = 2592ml/min
VA = RR x (TV – Dead Space)

to summarize all the dead space components:
we know we have about 36ml (1/3 of TV) anatomic dead space
let's say 20ml of mechanical because we have a pedi HME and $EtCO_2$ detector
and let's assume a ΔP 12cmH_2O to get to our TV goal
0.0008L/cmH_2O x 12cmH_2O ≈ 10ml
total dead space = 36ml + 20ml + 10ml
total dead space = 66ml

VA = 24/min x (108ml – 66ml)
VA = 1008ml/min

Now in this case the VA is probably a smidge low (MV goal was 1.8L/min), but we could then look at VTe and $EtCO_2$ to titrate up to an appropriate level. But what if this had been an 10kg one-year-old?

TV 6ml/kg = 60ml
total dead space = 66ml
which basically means no actual ventilation!

and even if we drop the HME and assume no mechanical dead space in that sense
total dead space = 36ml + 10ml = 46ml
MV goal = 1000ml/min
VA = 30/min x (60ml – 46ml)
VA = 420ml/min
we are still cutting it ~~pretty~~ close and will have to titrate up on MV pretty quick

So the moral of the story here is that we should either ventilate these kiddos in PC mode (to bypass this vent circuit stretcher dead space concept) or start at a higher end of normal TV (~~8ml/kg~~) and be ready to quickly go up on MV as soon as initiating ventilation in VC mode (based on VTe and $EtCO_2$). As we said before, there is no right or wrong to this, so long as we know the consequences and correct actions associated with whatever choice we make.

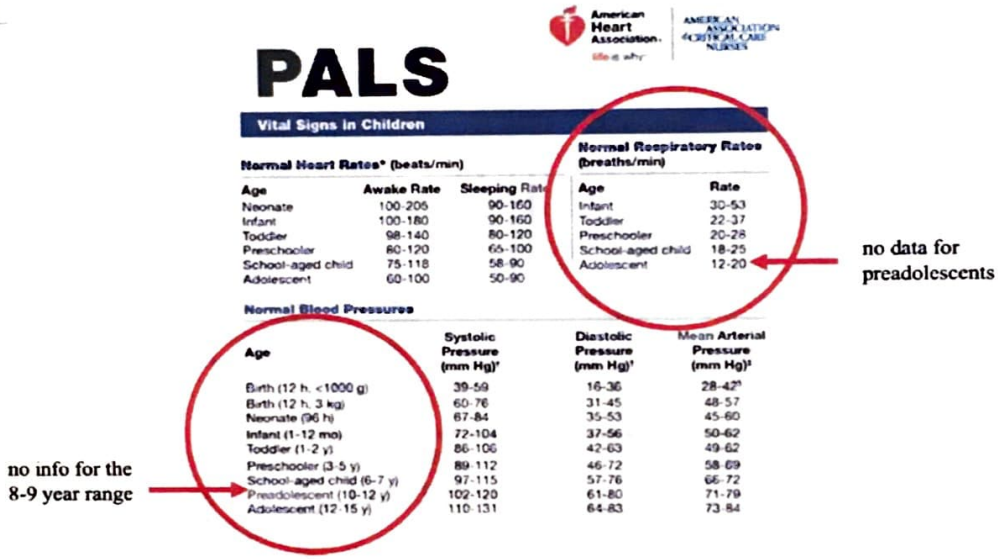
Return to Contents

Age-Based Settings

In an effort to make recommendations about vent settings for specific age groups, specifically RR and I-time, here's how the process went:

1. Make assumptions:
   a. "Normal Respiratory Rates" as outlined by PALS are good enough to work with[225]
   b. Normal RR range for an adult is 12-20 (cited in many, many sources)
   c. A normal I:E at rest/ spontaneous respiration is 1:2, but we often work with a ratio of 1:3 for vented patients[226]

2. Fills the gaps in the PALS "Normal Respiratory Rates" data set:
   a. What gaps?[227]



PALS

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rate
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-aged child	75-118	58-90
Adolescent	60-100	50-90

Normal Respiratory Rates (breaths/min)

Age	Rate
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-aged child	18-25
Adolescent	12-20

no data for preadolescents

Normal Blood Pressures

Age	Systolic Pressure (mm Hg)†	Diastolic Pressure (mm Hg)†	Mean Arterial Pressure (mm Hg)†
Birth (12 h, <1000 g)	39-59	16-36	28-42‡
Birth (12 h, 3 kg)	60-76	31-45	48-57
Neonate (96 h)	67-84	35-53	45-60
Infant (1-12 mo)	72-104	37-56	50-62
Toddler (1-2 y)	86-106	42-63	49-62
Preschooler (3-5 y)	89-112	46-72	58-69
School-aged child (6-7 y)	97-115	57-76	66-72
Preadolescent (10-12 y)	102-120	61-80	71-79
Adolescent (12-15 y)	110-131	64-83	73-84

no info for the 8-9 year range

PALS
RR
I:E
I-time
s

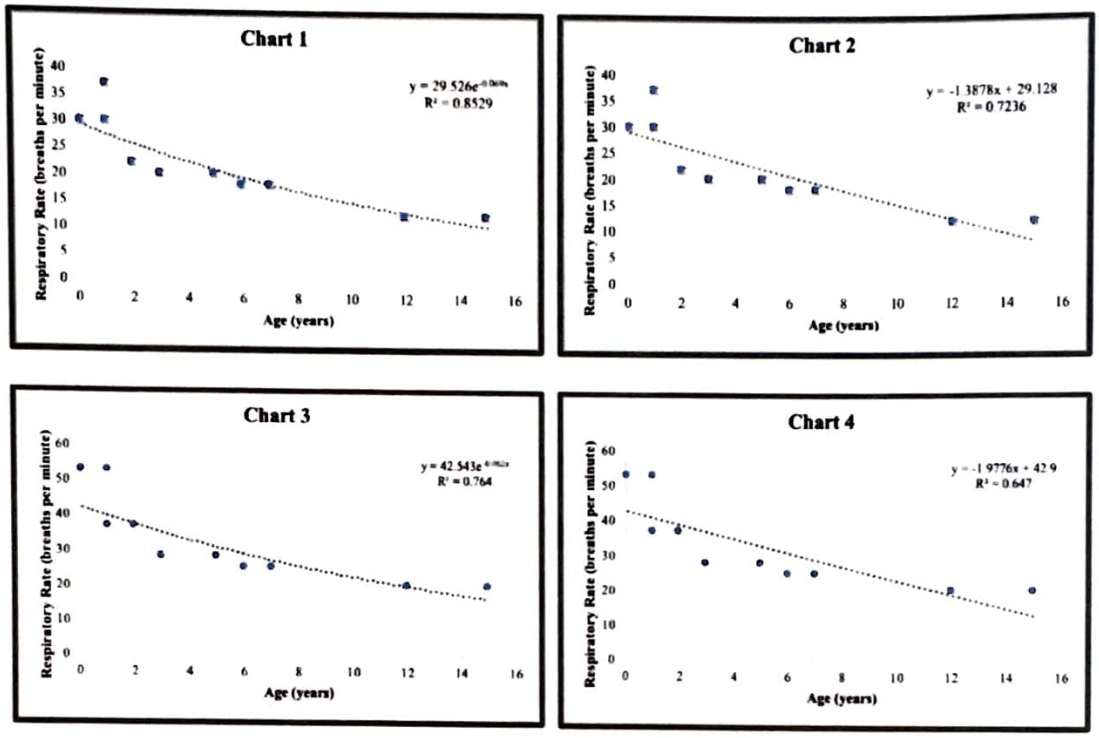
---

[225] And while there are gaps in their data, we can fill that in

[226] And this may be by convention of leaving I-time set at a given value, not necessarily because that's the thing we ought to be doing; but regardless, we'll get a range of possible values using both 1:2 and 1:3

[227] PALS, 2016 (image) – And we said already (section on **Rate**) that we chose to use these values not because they are intended for use with vent management, but because they represent normal values by age and are from a reference that most of us are familiar with and have access to

b. Plot the existing data using both high and low ends of RR by age, make charts, then add lines of best fit:



c. Using the better fits (exponential regression), solve for the missing data points in the PALS chart, then add those values in to a new chart (noted in blue):

Age Description	Age (yrs)	RR
Infant	.083 (1 month) – 1	30 – 53
Toddler	1 – 2	22 – 37
Preschooler	3 – 5	20 – 28
School-aged Child	6 – 7	18 – 25
Big Kiddos	8 – 9	17 – 25[228]
Preadolescent	10 – 12	14 – 23
Adolescent	12 – 15	12 – 20
Adult	16 and up	12 – 20

[228] Range here was calculated to be 17-26, but we went with 25 since range for School-aged Child was to a max of 25 – this was an arbitrary decision, but makes the final product flow a bit better

Return to Contents

3. Do a lot of calculations (for I-times):

60s ÷ RR = time per each respiratory cycle
Ex. For adult (low end RR): 60 ÷ 12 = 5s
Ex. For adult (high end RR): 60 ÷ 20 = 3s

I-time = time per each respiratory cycle ÷ number of parts in that cycle
Ex. For adult (low end RR, 1:2): 5s ÷ 3 ≈ 1.7
Ex. For adult (high end RR, 1:3): 5s ÷ 4 ≈ 0.8

Therefore I-time range for adults is 0.8 – 1.7s

4. Put all the data (both RR and I-time) into a chart:

Age Description	Age (yrs)	RR	I-time (s)
Infant	.083 (1 month) – 1	30 – 53	0.3 – 0.6
Toddler	1 – 2	22 – 37	0.4 – 0.9
Preschooler	3 – 5	22 – 28	0.5 – 0.9
School-aged Child	6 – 7	18 – 25	0.6 – 1.1
Big Kiddos	8 – 9	17 – 25	0.6 – 1.2
Preadolescent	10 – 12	14 – 23	0.7 – 1.4
Adolescent	12 – 15	12 – 20	0.8 – 1.7
Adult	16 and up	12 – 20	0.8 – 1.7

Hypotension Strategy Math

In the section where we outlined the **Hypotension** strategy, we introduced a concept which we labeled as %TaDP (percentage of time at decreased preload) and the idea was that if we decrease the overall amount of time spent pushing air into the system (i.e. inspiration) then we can mitigate the exacerbation of a hypotensive state. The result was a strategy that included a shorter I-time, higher TV, and lower RR. We also mentioned that there are other rationales for this strategy: less dead space and lower P_{aw}. We are going to calculate out these differences here just to give some more legitimacy to the argument.

But before we get there, one other thing to mention. PEEP is also a contributing factor to hypotension in the susceptible patient,[229] so we want to keep that to a minimum. While it may seem like a good idea to drop PEEP to zero in the hypotensive patient (especially in light of the P_{aw} calculations we'll show in just a moment), recognize that oxygenation is also super important and PEEP is one of our tools to maintain that. Other specific benefits of PEEP that'd we'd like to maintain in these patients include ease of triggering spontaneous breaths, alveolar recruitment, and decreased left ventricular afterload.[230] Last thing: the PPV/ PEEP → decreased preload → decreased CO sequence of events[231] can be mitigated by euvolemia or fluid resuscitation (to maintain a ~~CVP greater than PEEP~~).

Moving forward, recognize that is totally OK to drop PEEP all the way to zero if need be, but there may be consequences and there may be other relatively simple strategies (i.e. fluids and other vent changes) to mitigate the negative consequences while maintaining the benefits. It's also just fine to drop PEEP to zero in an emergency, then work back up to a beneficial level after the acute threat has passed and other interventions have been put into place – vent management is dynamic and we can adjust strategy as we move forward with patient care. So while we are going to show how eliminating PEEP can significantly reduce P_{aw}, which theoretically lessens the negative consequences of PPV, just know that there are multiple variables involved in this practice.

Now for the math, starting with how the lower RR, higher TV strategy decreases dead space. Let's assume another 65kg patient and see how it looks:

General Strategy
(6ml/kg TV)
TV 390ml
- Anatomic Dead Space 130ml

Alveolar TV 260ml

x 17/min

MV = 6630ml/min
(Dead Space = 2210ml/min)
VA = 4420ml/min

Hypotension Strategy
(10ml/kg TV)
TV 650ml
- Anatomic Dead Space 130ml

Alveolar TV 520ml

x 10/min

MV = 6500ml/min
(Dead Space = 1300ml/min)
VA = 5200ml/min

MV is basically the same

Dead Space is more in the General Strategy

VA is greater in the Hypotension Strategy

which means better efficiency with the Hypotension Strategy

note that anatomic dead space per breath is the same in both cases

[229] Discussed way back at the beginning in **PEEP**
[230] Yartsev, 2019 – He also discusses the idea of mitigating the effects of PEEP, discussed below
[231] We mentioned this very same idea in **How is Positive Pressure Different?**

This idea of better efficiency refers to the ~~concept~~ that in the hypotensive strategy we push less wasted air into the system. We already know that positive pressure, whether in the form of a breath being delivered or PEEP, has potential negative consequences, so if we eliminate any part of that (i.e. reduce dead space) while maintaining ventilation then our patient is better off. To say it another way, we want to try to make use (in the form of VA) of as much of the total air (MV) that we put into the system in an effort to eliminate pushing air in unnecessarily (dead space).

The next concept to discuss is P_{aw}. The airways and lungs live inside the thoracic cavity, so if we put pressure into ~~that system~~ then we see changes to pressure in the thoracic cavity. The idea is that P_{aw} directly correlates with a concept called intrathoracic pressure and intrathoracic pressure, in turn, is the thing that causes all those hemodynamic changes associated with PPV.[232] Now it gets exponentially more complicated than that, as pressure at specific components within that thoracic cavity, all of which are tied to hemodynamic function, vary significantly (in terms of influence on function, not necessarily quantitatively),[233] but the simple interpretation of the idea is that pressure we put in via the vent can disrupt hemodynamic function and result in less CO. So theoretically, if we limit P_{aw} we can minimize these potential negative consequences. At least that's the idea.

P_{aw} is normally measured by the vent itself, but there is a formula to estimate it using values for I-time, PIP and PEEP (and also T_{total}, which is the amount of time per breath or 60s ÷ RR):

$$P_{aw} = 0.5 \times (PIP - PEEP) \times (\text{I-time}/T_{total}) + PEEP$$

Using this formula, we built a spreadsheet of possible P_{aw} data points for each strategy on different values for PIP and PEEP:

General Strategy							
P_{aw}		PIP					
		10	15	20	25	30	35
PEEP	0	1.42	2.13	2.83	3.54	4.25	4.96
	1	2.28	2.98	3.69	4.40	5.11	5.82
	2	3.13	3.84	4.55	5.26	5.97	6.68
	3	3.99	4.70	5.41	6.12	6.83	7.53
	4	4.85	5.56	6.27	6.98	7.68	8.39
	5	5.71	6.42	7.13	7.83	8.54	9.25
	6	6.57	7.28	7.98	8.69	9.40	10.1

Hypotensive Strategy							
P_{aw}		PIP					
		10	15	20	25	30	35
PEEP	0	0.67	1.00	1.33	1.67	2.00	2.33
	1	1.60	1.93	2.27	2.60	2.93	3.27
	2	2.53	2.87	3.20	3.53	3.87	4.20
	3	3.47	3.80	4.13	4.47	4.80	5.13
	4	4.40	4.73	5.07	5.40	5.73	6.07
	5	5.33	5.67	6.00	6.33	6.67	7.00
	6	6.27	6.60	6.93	7.27	7.60	7.93

[232] Chiefetz, 2014 – Similar discussion to some of the other references cited previously, but specifically focuses on this idea of P_{aw} and the balance between oxygenation and the negative consequences

[233] Luecke & Pelosi, 2005 – Very detailed discussion of the physiology involved in all of this

Barring the most drastic possible scenario (excellent compliance and very low PIP per the general strategy, poor compliance and high PIP with transition to the hypotensive strategy; paired with keeping PEEP constant), we can see that the hypotensive strategy tends to give lower numbers for P_{aw}. While it is likely that overall compliance will decrease and thus PIP will increase as we move from left to right (due to higher TV with the hypotensive strategy), guesstimating to what degree that will happen seems unfair without actual experimental data. There may also be a mathematical model based on this idea that could identify cases where P_{aw} isn't actually decreased with the hypotensive strategy, but given that this is just one of three reasons to use the strategy (the other two being lower %TaDP and less dead space), it seems OK for now.

Just to demonstrate an arbitrary example, if we had a patient vented per the general strategy with a PIP of 20 and transitioned them to the hypotensive strategy and ended up with a PIP of 30, we'd get a small drop in P_{aw}:

General Strategy

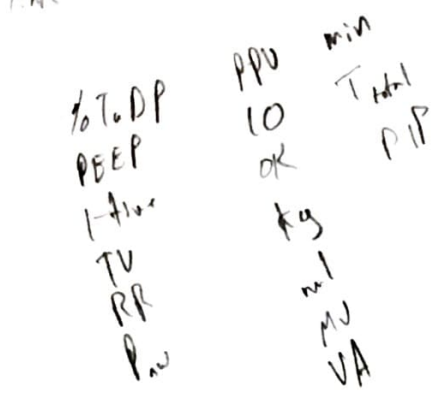
P_{aw}		PIP					
		10	**15**	**20**	**25**	**30**	**35**
PEEP	**0**	1.42	2.13	2.83	3.54	4.25	4.96
	1	2.28	2.98	3.69	4.40	5.11	5.82
	2	3.13	3.84	4.55	5.26	5.97	6.68
	3	3.99	4.70	5.41	6.12	6.83	7.53
	4	4.85	5.56	6.27	6.98	7.68	8.39
	5	5.71	6.42	7.13	7.83	8.54	9.25
	6	6.57	7.28	7.98	8.69	9.40	10.1

Hypotensive Strategy

P_{aw}		PIP					
		10	**15**	**20**	**25**	**30**	**35**
PEEP	**0**	0.67	1.00	1.33	1.67	2.00	2.33
	1	1.60	1.93	2.27	2.60	2.93	3.27
	2	2.53	2.87	3.20	3.53	3.87	4.20
	3	3.47	3.80	4.13	4.47	4.80	5.13
	4	4.40	4.73	5.07	5.40	5.73	6.07
	5	5.33	5.67	6.00	6.33	6.67	7.00
	6	6.27	6.60	6.93	7.27	7.60	7.93

At this point there's no experimental data (at least that we are aware of) to show to what extent this type of thing has on CO or other parameters of hemodynamic function, but given the logical sequence of events that we already outlined it seems like a step in the right direction for the patient who is hypotensive or at risk for becoming so.

Just to summarize things for this section: the hypotensive strategy includes shorter I-time, increased TV, lower RR, and keeping PEEP to the lowest level needed to maintain oxygenation. We discussed the idea of %TaDP back in the section on **Hypotension** and then we added to that just now the idea that this approach results in both less dead space and a generally lower P_{aw}. And while PEEP is a major contributor to P_{aw}, it also serves to maintain oxygenation; this means we ought to use caution in titrating it all the way back to zero.



Return to Contents

A Personal Reflection

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✓ Press. & Vol.
✓ Oxygen
✓ PPV & CV

to all videos in my folder: { Thoracic.org Videos, Ninja Nerd Resp, ~~Khan Acad Resp~~, ~~Vent Aro Amazon~~ }
↓
but they aren't ref'd in text, just at end for further reading

✓ Betts 2013 Sec 22.1
✓ 22.3
✓ 22.4

~~Certified Medical App~~
~~Dani std App~~

~~Labgenuity~~ → done
~~Weingart~~ → see above
~~Brown~~ → done