Vent Management

A Guide by Rykerr Medical

Version 1
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Rykerr Medical's Vent Management Guide

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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 though. 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 text 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 (over-perhaps?) 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 Five- and ten-minute transport times had been replaced by ones much longer in our 5,000 square mile coverage area, the ambulances were giant machines that could be rigged to carry three patients each and would never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.

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 (~~after a few days of asking lots of questions and scouring the web~~) looked something like this:

- The Ventilator Book – William Owens (there was a kindle version at the time and I could take the kindle with me on my long transports to Albuquerque – this was in the day before I had a smartphone, so I couldn't just pull up videos on my phone like I can nowadays!)
- The LTV1200 Product Manual and DVDs (super exciting stuff… but I have since read the manuals for lots of the equipment I have worked with and it does provide some useful information)
- EMCrit Dominating the Vent Series (I had to watch these at the coffee shop down the street, as I didn't have internet where I lived – I was instead trying to grow food and tobacco in my yard back then, that and not get caught up in the "technology craze")

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 monies); 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 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 :)

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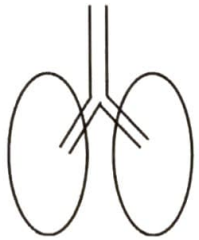
Some Very Basic Physiology

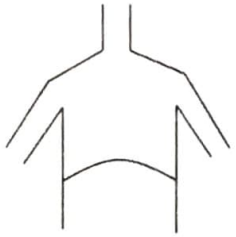
As a disclaimer: the stuff outlined here is super basic and intended to give a foundation for the fundamental concepts of vent management. One recommendation for looking into the details beyond this (much of which comes up later when we talk about specific conditions) is a good, solid, heavy Anatomy and Physiology textbook[1].

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:

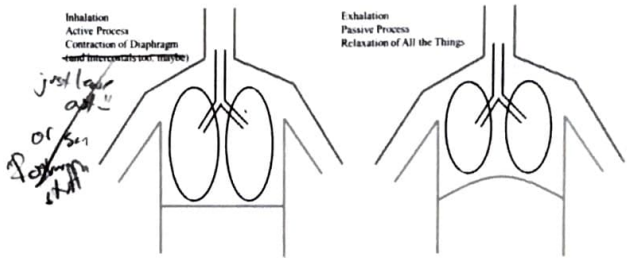


We also have the chest cavity and the diaphragm:

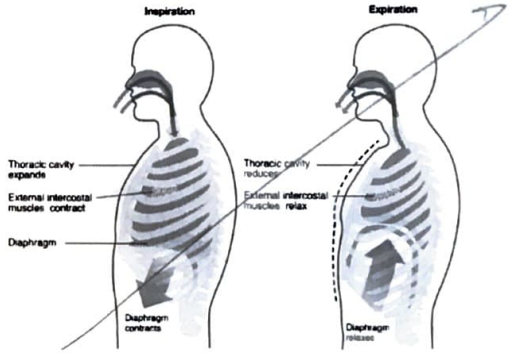


[1] Or this free one thanks to Openstax.org - also note that all the images in here that don't look like they were made by a four-year-old on Microsoft Paint are taken from this source (unless otherwise noted)

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]:

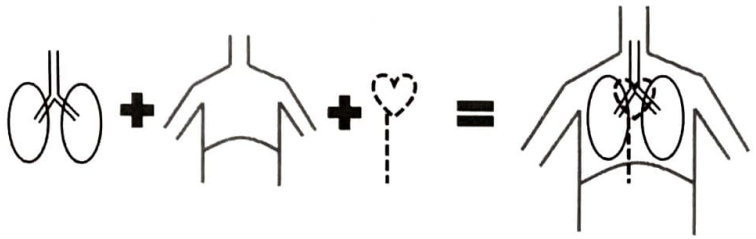


Here's another version that shows a bit more detail:



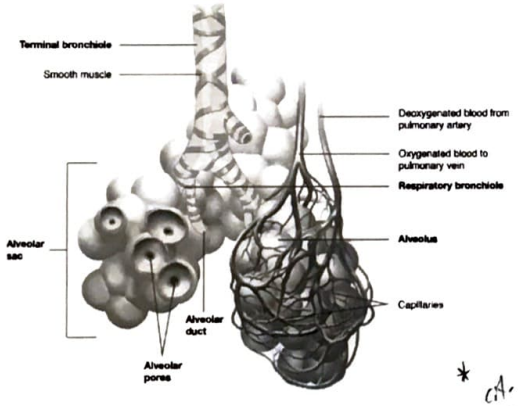
[2] This assumption mostly holds true for our need in the transport setting, but we've delved a bit further in to this idea in the **Appendix** to hash it out more clearly. There is also an article referenced in the **ALI/ARDS** subsection under **Specific Vent Strategies** that shows a specific case of this breakdown

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



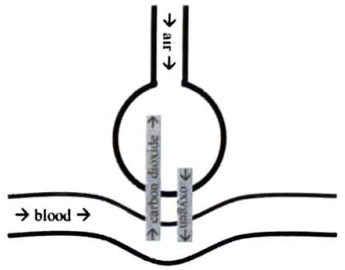
So now we have a system that normally functions by contraction of the diaphragm (with or without help from the intercostal muscles) to create a negative pressure, "sucking" of air into the lungs. Because this air movement occurs via a negative pressure, blood return via the inferior vena cava is facilitated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

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:

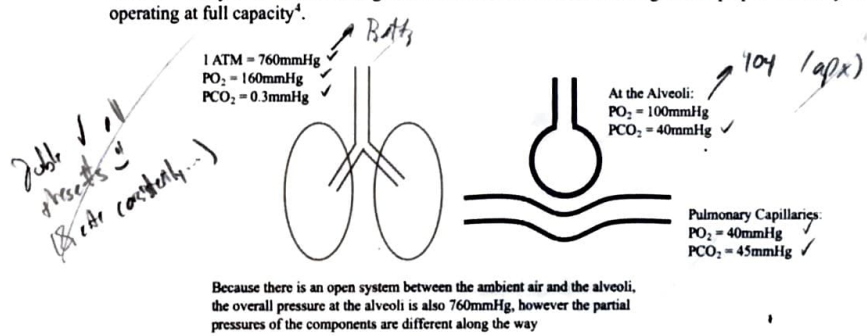


[3] Klabunde, 2008 – Second half of that page explains this concept in much more detail

A simplified version of a single alveoli with a corresponding blood supply ~~would look like this~~ (see below). ~~This is a representation that we will return to later on, as it~~ can help us understand the (patho)physiology of different situations:



Next, let's add some numbers to that graphic of a single alveoli and its blood supply. 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. It's important to know that in the normally functioning system, the body does not "outrun" this system – diffusion of gases and movement of blood is enough to keep up with a body running or operating at full capacity[4].



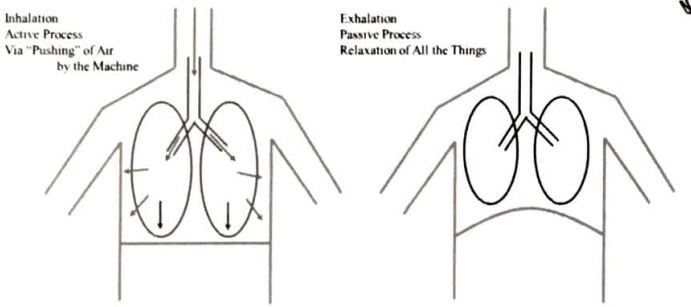
Because there is an open system between the ambient air and the alveoli, the overall pressure at the alveoli is also 760mmHg, however the partial pressures of the components are different along the way

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, ~~recall that~~ carbon dioxide moves more effectively 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.

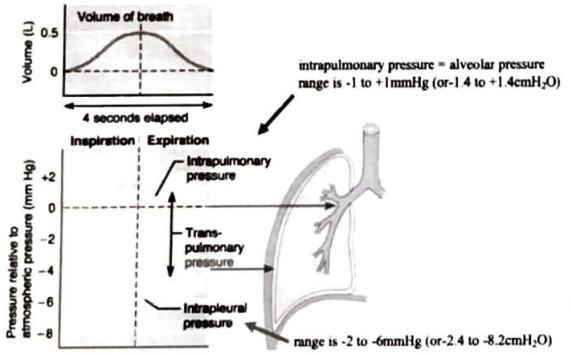
[4] Speller, 2018 – She also outlines how both oxygen and carbon dioxide diffuse in the pulmonary system in the context of gas laws; to note, however, that certain states can overtax this system to result in a situation – see discussion of A-a Gradient in the **Appendix**

How is Positive Pressure Ventilation Different?

~~Up to now we've covered the basics of spontaneous, negative pressure ventilation as occurs in the body at baseline~~. 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. 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 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:

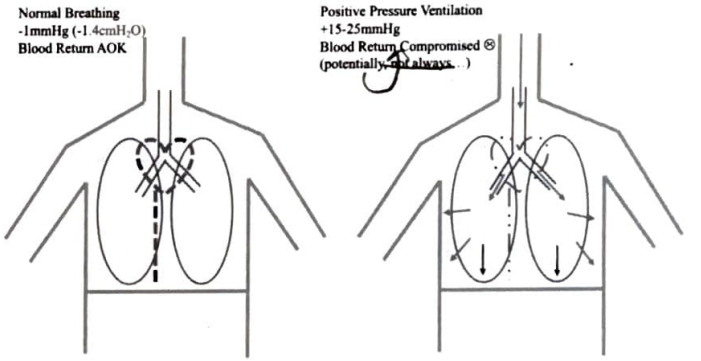


We will get to airway pressures and limits for them later on, but a normal plateau pressure (which reflects alveolar pressure in positive pressure ventilation) is in the range of 15-25cmH_20; compare this to the pressures represented in the following illustration[5]:



[5] Two things: we'll talk about the mmHg and cmH_2O conundrum at the end of this section (in **Measuring Pressures**); alveolar pressure is the most relevant to our discussion for now, the concept of transpulmonary pressure (and therefore intrapleural pressure) is deferred to the **Appendix**

The biggest impact of that increased intrathoracic pressure is the effect it may have on cardiac output. Increased intrathoracic pressure can decrease blood return to the heart via pressure on the vena cava, resulting in decreased preload and, therefore, less output[6]. 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[7], muscle fatigue/ weakening[8] and physiologic changes to other body systems[9]. 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[10] 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.

[6] Strong, 2013 – This video (which is just one of a great series on mechanical ventilation) has a section that explains a little more detail on how PPV (and particularly PEEP) can affect CO; while it isn't always true that PPV decreases CO (sometimes the opposite can occur), the PPV → decreased preload → CO sequence of events is most relevant to us in the transport setting

[7] 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

[8] 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

[9] 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

[10] ~~To be discussed at length later on~~...

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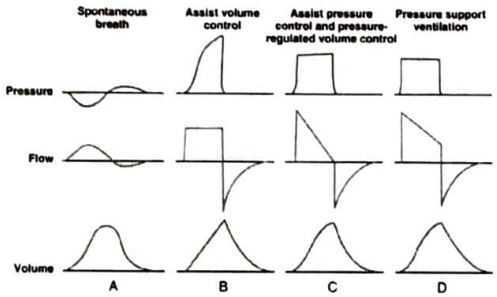
We already saw how a pressure waveform might look over time with spontaneous, negative pressure breaths, so let's see how it looks with a machine delivered breath. Note that there are different types of machine delivered breaths in this diagram (plus some terms to discuss), and we haven't yet gotten 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 worth mentioning is that the graphic representations of the modes (i.e. each column of the three towards the right) are each slightly different and 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[11].



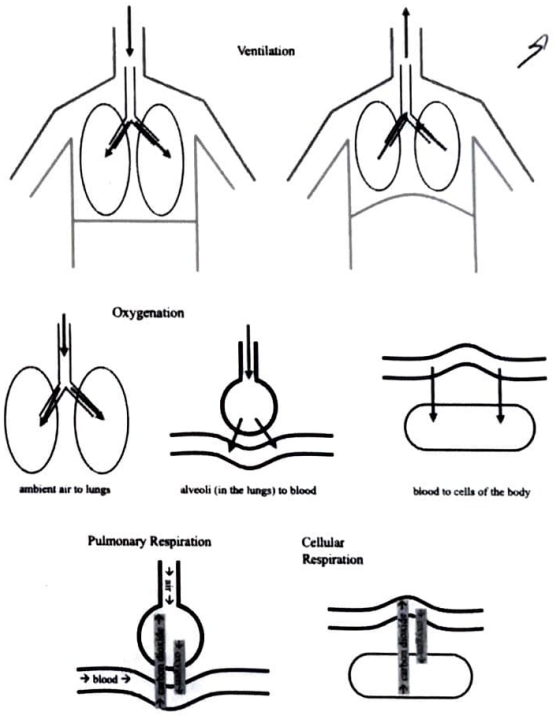
In an effort not to ~~be a Debby Downer and~~ discourage anyone from ever putting a patient on a vent, there are some advantages of positive pressure ventilation/ mechanical ventilation. Most obvious of these is that it allows us to breathe for a patient in a relatively simple way when that patient is 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 effectively, managing ventilation (and therefore acid-base balance) 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.

[11] This this assessment of what the body "wants" in terms of smooth waveforms and avoidance of harsh changes in amplitude is scientifically unfounded (as far as we know) and, rather, is a subjective concept. It seems to make intuitive sense, but there may not be a good way to verify the idea. And if anyone has evidence to the contrary, it would be greatly appreciated. ~~And if you have evidence to the contrary, that would also be of benefit for future versions of this handbook.~~ But for now we will roll with it.

Other Important Concepts

Ventilation, Oxygenation and Respiration

Just to differentiate the concepts that collectively represent breathing let's chat about these three terms[12]. Ventilation refers to the gross of air as the body breathes in and out. Oxygenation refers to the ~~movement~~ 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. If it helps, here are a few images to represent all of that:



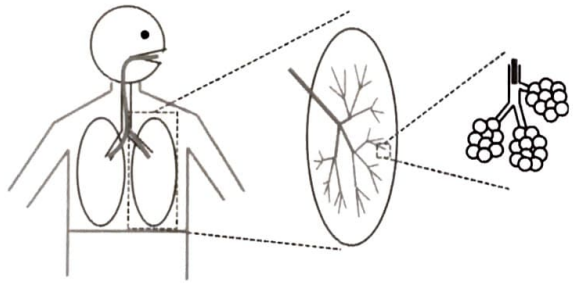
[12] Betts & friends, 2013 – Explains in more detail the processes of ventilation (Section 22.3) and respiration (Section 22.4)

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[13]. 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, the pulmonary part, that we can impact is covered in a roundabout way by our actions to address oxygenation.

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 flavors discussed in references, but we will discuss 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

[13] Whitten, 2015 – Goes into the difference between ventilation and oxygenation in the context of patient management

[14] All I can find on mechanical dead space is the vet article – keep looking and maybe use this as a ref at the Appendix? http://www.vasg.org/dead_space.htm

[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 in to each cluster of alveoli

inspiration. As for quantifying this value: normal anatomic dead space is about 2ml/kg or 1ml/lb (IBW) or approximately 150ml for the average adult male patient. We may also see anatomic dead space estimated at 1/3 (0.33) or 30% of tidal volume, but this only holds true if we have an appropriate tidal volume for that particular patient[16].

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. We will talk about this more later, 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. We will return to this idea again later (with a few illustrations), but it sort of makes sense to mention it now.

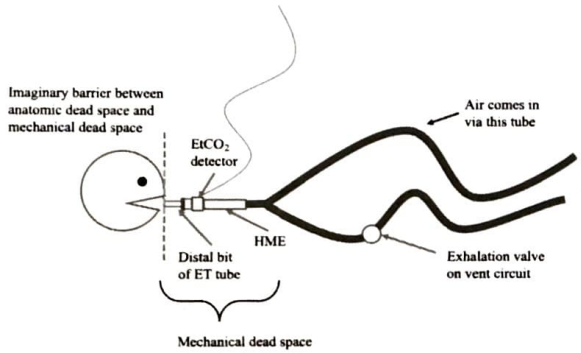
The next type of dead space is alveolar dead space. Alveolar dead space refers to the air in the alveoli that doesn't participate in gas exchange. This can be due to a few different things: decreased capillary blood flow, fluid in the alveoli, damage to the alveolar surface, etc. Regardless of cause, 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 basically 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 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, recruiting alveoli, 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. 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 ~~treatment~~ 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 a effort to be more specific with our discussion.

[16] Rather than going overboard on citations, here's a quick summary of two references for this section and the paragraphs that follow:
Quinn & Rizzo, 2018 – These guys give us the 2ml/kg and 1/3TV formula; they also define alveolar dead space and outline some clinical applications
Intagliata & friends, 2019 – And these guys cite the 150ml and 30% methods; they also review physiologic dead space
Also note: there is some overlap on these two articles (including an author), but they provide a brief overview of all this content in a slightly different fashion; neither refers to "mechanical dead space" as I have done, but they both mention the impact of this space via other terms
Brewer, 2008 - Now that we've given all these methods for estimating anatomic dead space, know that they may not be very accurate; all that said, the actual value doesn't much matter here, it's the application of this information that is relevant – so don't get too caught up in the nitty gritty!

Last type of dead space is what we will call mechanical dead space. Mechanical dead space is the dead space that we add on to the system with our equipment: vent circuits, $EtCO_2$ detector, HME[17], 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 quantified 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). Another point worth considering is that we can mitigate this "used up air" conundrum by pushing fresh air (i.e. oxygenated air) into the system throughout the exhalation portion of the respiratory cycle[18] (i.e. with PEEP or bias flow[19]).

There is one other 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 an expected versus actual value. 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 ETT cuff, some of it dissipates into the tissues, 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.

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 return to 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.

[17] Heat & Moisture Exchanger, discussed more in the **Appendix**

[18] De Robertis & friends, 2010 – These guys outline some strategies to avoid this "rebreathing" of CO_2 in ventilated patients

[19] Find a good discussion of bias flow

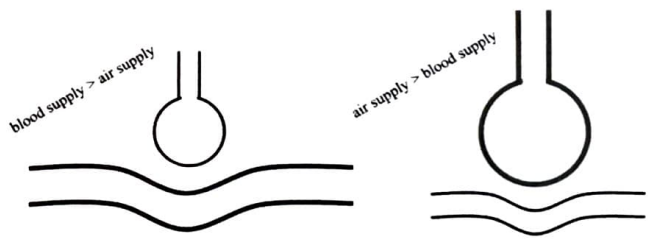
This is particularly relevant with little patients ~~(particularly infants)~~, as smaller volumes of air can have a much greater impact[20].

Hypoxic Pulmonary Vasoconstriction[21]

In systemic circulation, hypoxia causes vasodilation. If a part of the body isn't getting the oxygen it needs, the body opens up the blood supply to counteract the deficit and get blood (and thus oxygen) where it needs to go. The opposite occurs in the lungs: hypoxia in the pulmonary vascular bed results in vasoconstriction (thus the term, "hypoxic pulmonary vasoconstriction"). 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 oxygenation. Just as in the systemic capillaries, the pulmonary capillaries are in a state of flux and respond by opening and closing to the needs of the system and the availability of resources (oxygen, in this case, being the driving force).

Two terms related to this concept are shunt and V/Q mismatch. Shunt refers to when blood supply from the right side of the heart (unoxygenated) makes it over to the left side of the heart still without oxygen – something happened where it passed through a pulmonary vascular bed and didn't get any oxygen loaded on to it. This can happen when the alveoli are filled with fluid (as in pulmonary edema, pneumonia or ARDS) ~~or when blood cannot get to a region of the lung (as in a pulmonary embolism).~~ If there were no hypoxic vasoconstrictive response, the body would put even more blood flow into these regions and the patient's condition would worsen. Treatment here is to fix the cause of the shunt, i.e. move the fluid, gunk, whatever else is in that alveolar space out of there, so that air can get back in contact with the capillaries.

V/Q mismatch (or ventilation-perfusion mismatch) describes a state in which blood supply and air supply to the alveoli-capillary interface are out of balance, i.e. one or the other valuable resource is passing through the system without being utilized. This represents an inefficient use of resources and contributes to the previously mentioned idea of alveolar dead space. It can occur by either of two mechanisms: blood supply outweighs air supply or vice versa.



Going back to this hypoxic vasoconstrictive response, the V/Q mismatch represented on the left would lead to vasoconstriction – the body wants to redirect that blood flow to where it will be met with adequate air

[20] Eric Bauer podcast I think it was, find that

[21] For further 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
Tarry & Powell, 2016 – Discussed physiological factors that influence this response; also discusses role of catecholamines and vasopressin (among others, but these are noted to be relevant to the transport setting)

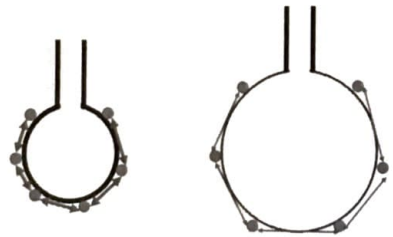
delivery to facilitate efficient oxygenation. Moving right, this V/Q mismatch is a step in the right direction and what we aim for with treatment: as we increase alveolar surface area (see next section) and add air in to parts of the system which have clamped down, we can reverse that vasoconstriction and improve the amount of lung participating in alveolar gas exchange. While a mismatch still exists at this point, the body will eventually respond to air supply and the vessel will dilate to meet that supply.

One last thing to note about this hypoxic vasoconstriction response is the timeframe on which it occurs in either direction. The vasoconstriction side of things happens on the order of seconds to minutes, while the reverse process of vasodilation happens on the order of minutes to hours. There is a lot of variability in these time frames based on all kinds of variables, but the important thing to note is that the downside happens fast and then it takes much longer to fix it afterwards. This is comparable to the idea of alveolar recruitment (discussed later) in that it is better to avoid the problem in the first place than to try and fix it after it has already occurred.

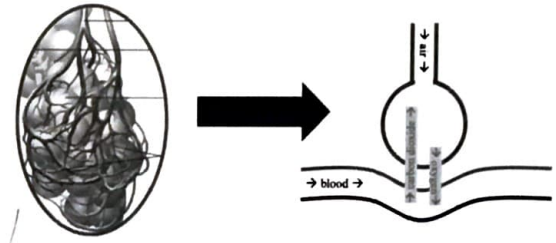
Last thing: there are ways to do mathy things and calculate the extent to which a shunt/ mismatch exists, but the actions to address the results of those calculations are things that we should arguably be doing anyways for our patients, so we will defer all of that nerding to the **Appendix**.

Alveolar Surface Area

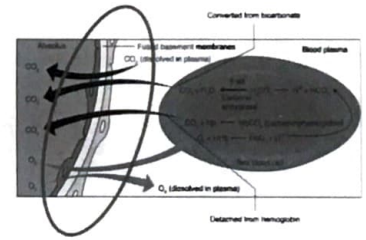
Without getting to far gone into the laws of physics, let's consider blowing up a balloon. At first it is a bit difficult to get it started, but once we get over that initial hump, it gets easier and we have a party. The reason for that is that as things stretch, they resist further stretching less - at least up to a certain point. We can consider the alveoli to be little balloons that fill with air in a similar fashion. That said, there is another characteristic that contributes to this idea: there is fluid around the surface of each alveoli that tends to resist expansion. Think of it as molecules on the alveolar surface that are holding hands with one another to resist movement away from one another; as we increase the volume of that alveoli, we increase the distance between those hand-holders and make expansion easier. Look at it this way and consider the strength of the hand holding to be proportional the thickness of the arrows:



So we have an alveoli that is difficult to open up at first, but then readily expands. Now there is a limit to this expansion, so it isn't a never-ending process – we will discuss that later on when we get in to measuring and addressing airway pressures, so let's hold off for now. Another super important thing to notice that changes from the left image to the right is that the available surface area on the inflated, right-sided alveoli has increased dramatically. This means that there is more alveolar surface area available for gas exchange. While we have been simplifying the interaction between alveoli and blood supply with a simple graphic representation, don't forget that the alveoli is actually covered by lots of vessels:



And the last important thing to take note of is that as that alveoli expanded and the surface area increased, the thickness of that alveolar membrane also stretched and got thinner. This makes it easier for gasses to diffuse across the membrane, particularly oxygen (which doesn't diffuse across liquids as readily as carbon dioxide):



Now we know that filling the alveoli up from an uninflated/ underinflated state is a bit difficult, but that the advantages are increased alveolar surface area and thinning of the space that impedes gas exchange, so it's worth the difficulty to make this happen. For now, suffice it to say that we need to make this happen and that we do that by increasing pressure in the system; we also know that there is a limit to how far these alveoli will stretch, but we'll get to both the mechanism for making this all happen and how to avoid causing damage in later sections.

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Lung Size

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) 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, typically 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, you also have it as so:

$$IBW_{dudes}\ (kg) = 2.3(\text{height in cm}) - 152.4) + 50$$
$$IBW_{chicks}\ (kg) = 2.3(\text{height in cm}) - 152.4) + 45.5$$

Or you can use charts like this:

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

Or you can use apps like this:

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, your go-to reference ought to be the Broselow Tape. If that isn't available, you do have some formulas you can refer to:[22]

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

Or you can use apps like this:

And note that the Broselow overlaps with the equation/ chart above, so if you have a really small grownup or a big kiddo, you should still be able to get an IBW just fine. So no excuses!

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

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 "suck" 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)[23]:

	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, we call it 0cmH_2O to make things easier.[24]

[23] We built this chart by Googling conversions for these values; not exactly sure how to cite that, but thanks Google!
[24] Yartsev, 2018c – Scroll down to the section called "Airway Pressures" for some fun (and likely useless) trivia on why we measure/ label pressures the way we do

Modes of Ventilation

Control

Plain old control ventilation or controlled mandatory ventilation (CMV) isn't utilized much these days and doesn't exist as an option on many transport vents[25], 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. 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, both just before (red) and just after (blue) machine delivered breaths:

In the red situation the machine would give the subsequent breath right in the middle of the patient's breath and in the blue situation the patient would be trying to take a breath in the middle of a machine-given breath. Neither situation is of benefit to the patient, as these patient-initiated breaths don't get actualized – the machine basically ignores the effort of the patient. This leads to discomfort, muscle fatigue and potential for increased airway pressures. The idea moving forward is that we need a strategy that works alongside the patient and helps meet their expressed needs. Synching the machine with the patient improves comfort, conserves resources, facilitates recovery and gives us more control over the management of the patient.

[25] 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

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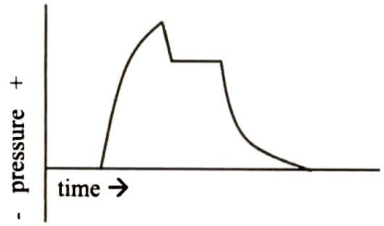
Assist Control (AC)

AC ventilation is a mode that supports a patient's spontaneous respiratory effort by delivering a preset amount of air (either by volume or pressure, but we'll cover that soon) regardless of the underlaying rate. So in this case (with the red and blue patient triggered breaths), the machine would sense inspiratory effort by the patient (a small negative pressure or "pull") and then respond by giving a breath as programmed. The result would be ten full breaths delivered in the timeline, rather than the underlaying eight breaths noted as black lines. The obvious advantage here is that the patient's expressed need for more breaths per unit time would be met.

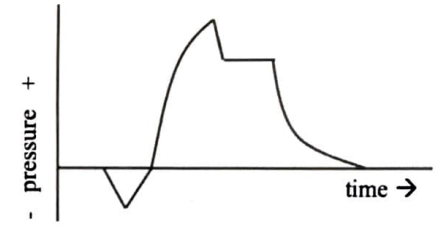
On the flipside, however, we have the proximity of breaths to consider. If a patient initiates the red breath just before a machine-programmed one, we run the risk that the first breath may not have time to cycle through before the next is delivered; we might get a breath on top of another, or "breath 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. Again, we will discuss this further on down the line, but note that this is the primary drawback to the assist control mode.

In the case of the blue patient triggered 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 to sense a patient trigger[26] and we can generalize it by saying that the further along the breath is or the closer the pressure has returned to baseline makes it more likely that the breath will "catch" and result in that full delivery. To represent these ideas graphically, let's start with a sketch of what airway pressures look like over time as a machine-delivered breath is delivered. We are going to ignore PEEP (since we haven't discussed that yet) and assume that baseline is "zero" or atmospheric pressure and that changes above and below the horizontal line are relative to that set point. We also don't have to worry about the specific components of the waveform at this point, all those things will be discussed later on:

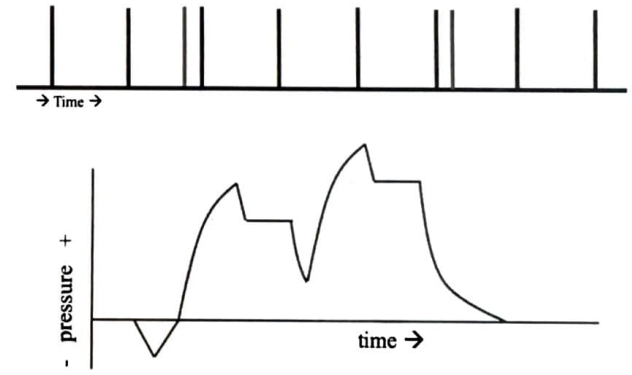


[26] A complete discussion of triggers and how all that works is deferred to the **Appendix**

Now the next step is to see what a patient triggered breath looks like. Note the dip in pressure at the start of the waveform as the patient breathes in and creates a negative pressure, this effort is sensed by the machine and then a full positive pressure breath is then given: *

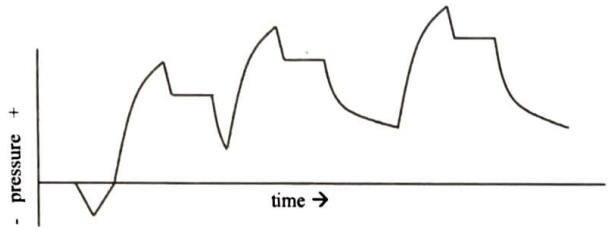


Let's go back to that initial sketch with red and blue lines representing the patient's attempt to breath and see what it would look like in the red line situation where the patient takes a breath and then the machine delivers a breath before that patient triggered one has a chance to return to baseline. Note that the end result is a higher pressure (greater overall amplitude) which can potentially cause damage:

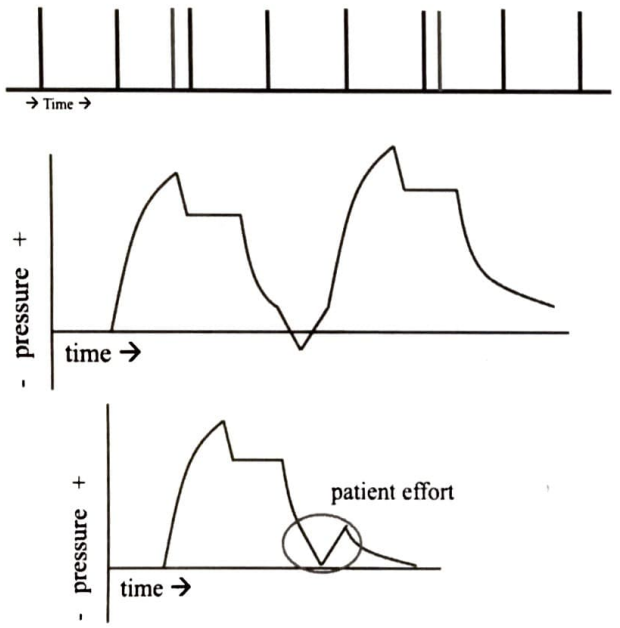


* now this graphic makes it seem that triggers are pressure related. We usually trigger flow; Ask (get more in later)

In addition, if we have multiple stacked breaths we might get in a situation where the downslope of the curve (which represents exhalation) never gets back to baseline and the pressure gets incrementally higher with each stacked breath:



In the blue line situation, where a machine delivered breath precedes a patient trigger, there are two possible outcomes: one in which the trigger results in a breath (shown first and with similar consequences of the above example) and one in which the trigger does not result in a breath and the efficacy of the machine triggered breath is simply altered somewhat:

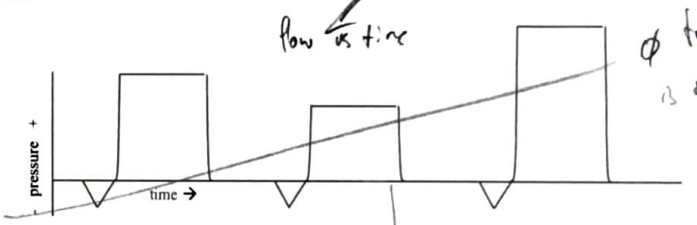


To summarize, AC mode machine-delivers breaths at a set rate and will supplement that with full breaths whenever a patient effort meets the trigger threshold. Upsides to this are that the increased needs of the patient are readily met, downsides are the risk for increased pressures and a move away from baseline (AutoPEEP, which we will return 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.

Synchronized Intermittent Mandatory Ventilation (SIMV)

SIMV is another mode of ventilation that also seeks to mitigate the shortcomings of CMV. While SIMV has historically been cited as a mode for weaning patients from the vent[27], it does have utility and is commonly used in the transport setting. SIMV assists patient effort by a similar trigger mechanism as is seen in AC mode; the difference is that rather than giving a full breath it provides a "pressure support breath" to augment patient effort. We haven't yet made it to the point of fully discussing the difference between pressure-delivered and volume-delivered breaths, but know that a pressure-delivered breath gives a variable amount of air (at a set pressure) and the resultant volume of air is dependent on how much the lung tissue expands in response to that set pressure.

One really important thing to consider here is that the volume that results from a given pressure can vary from breath to breath and is the function of many different things. Let's assume three consecutive patient triggered, pressure support breaths. The area of the space under the waveform represents volume delivered, so in each breath we see a different amount of air resulting from the same pressure support parameter dialed in on the machine:



This concept isn't, in and of itself, a bad thing, it is just something that we need to be aware of. If we dial in a pressure support of 15cmH_20 and get a few breaths of 400ml (measured by exhaled tidal volume, again a concept we will get to later on), we can't assume that this will hold true with time and, therefore, we need to keep an eye on it as we move forward. It is also worth noting that the pressure over time waveforms for these breaths are different than the other ones we have been using. The others were volume control breaths and these are pressure support breaths. It isn't worth getting caught up in the details at this point, just know that there was a reason for drawing them differently and that a more detailed explanation can be found in the **Appendix**.

[27] Citation

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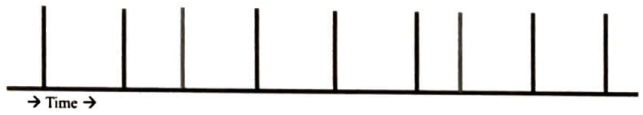
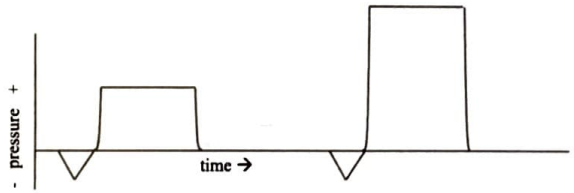
One other aspect of SIMV that sets it apart from AC is that it has a mechanism built in to prevent breath stacking or one breath being given on top of another. If we recall from our discussion of AC, one of the pitfalls was that you can stack breaths on top of one another and end up with both increased pressures and AutoPEEP. SIMV avoids this by two mechanisms: delaying machine delivered breaths after facilitating patient triggered breaths and not supporting patient triggered breaths in close proximity to machine delivered ones. Our initial dilemma with CMV was that we wanted to mitigate the negative consequences of breaths timed in close proximity to one another:

SIMV mode would respond to this as shown below. Note how the breath initially "planned" to get delivered just after the red line (which indicates a patient triggered breath) got pushed back in time to allow that red breath to cycle all the way through:



The downside to this, however, is that if the patient is triggering lots of breaths (because of inadequate sedation, increased need, or false triggering due to vibrations or a bumpy road), we could end up with something like this:

"Well," we might argue, "that doesn't seem too bad, the patient is breathing when he wants and we are simply supporting him with that." True story, but what if each of those breaths looks like these left sided breaths instead of those right sided ones?



If we get all left-sided breaths (with less area under the waveform, representing lower tidal volume), we may actually be ventilating the patient with less air per unit time (decreased minute volume). And that can be a bad thing, as we need to ensure adequate minute volume with all of our ventilated patients (another one of these concepts that we'll get to later…). Recognize that the size of these breaths is a function of both patient effort and what we dial in as pressure support.

Ideally SIMV would include a mix of machine-delivered and patient-triggered breaths and the resultant minute volume would be close to our therapeutic goal, but that doesn't always happen and we need to watch out for it. Last thing to mention about SIMV: while we program the machine-delivered breaths to give us a certain volume (whether in volume control or pressure control mode), it is standard practice that patient triggered breaths are not as big as machine-delivered breaths. So what we typically see are tidal volumes at goal for machine initiated breaths and lower tidal volumes for patient triggered breaths. While that is the status quo, such a strategy may not make sense in the transport setting and it may be worth considering a strategy in which we titrate pressure support up so that pressure support breaths are comparable to machine-initiated breaths[28].

To summarize, SIMV delivers patient-triggered breaths via pressure support and not a guaranteed volume; in addition, there is a delay mechanism built in that attempts to prevent breath stacking. Upsides to this are the avoidance of overinflation and AutoPEEP, downsides are that minute volume can suffer if there are too many triggered breaths being delivered. As a general rule: any time we have someone in SIMV mode we need to be vigilant and monitor exhaled tidal volumes (to compare machine-initiated and patient-triggered breaths) and minute volume.

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.

[28] And if anyone has further insight on that, let's chat!

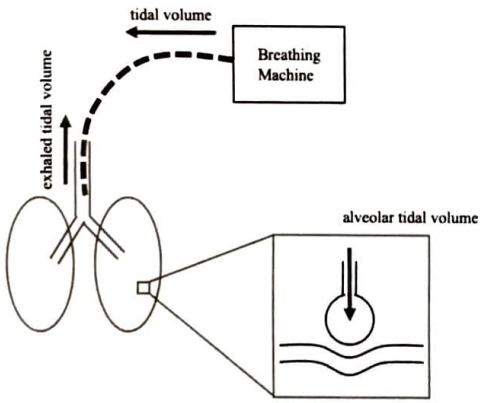
Control of Ventilation

We already discussed the first big choice in vent management: which mode (AC vs SIMV in the transport setting) 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 pressures will function as the dependent variable (i.e. we won't be able to directly control them); if we choose to control pressure, tidal volume will function as the dependent variable. There is no right or wrong answer to this conundrum, but the general trend is that folks will use volume control in most cases and pressure control with pediatrics[29] 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 around. 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![30]

Volume

In volume control ventilation we choose how much ~~tidal~~ volume we want to push down the circuit with each breath delivered. ~~Now,~~ this tidal volume that we put in goes into 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 of all these terms:



But wait a second, isn't the actual definition of tidal volume the amount of air moved during exhalation? That is true. But! 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 shown above. And to review what we discussed previously about dead space, the alveolar tidal volume is normally exhaled tidal volume minus anatomic dead space (which is 1/3 or .33 or 30% of TV; 2ml/kg or 1ml/lb; or 150ml-ish), so about two thirds of what we push into the system.

Now what about those other kinds of dead space; mechanical and alveolar[31]? 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 (and as we discussed before and will discuss later).

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. And in the event that exhaled tidal volume is not available on a particular machine, we just have to assume that volume in (tidal volume) is equal to volume out (exhaled tidal volume).

To summarize all of this: volume control 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 mechanical dead space (when that value is available to us). In addition, we need to remember that alveolar tidal volume is about two thirds of exhaled tidal volume (factoring out anatomic dead space) and that there may be some of that alveolar volume that doesn't get to play gas exchange (alveolar dead space). While this may have seemed like a bit of tangent, this is important!

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. Remember that balloon example and how we said that the easier-stretch superpower was self-limiting? At a certain point we can overinflate alveoli and we for sure want to avoid that.

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

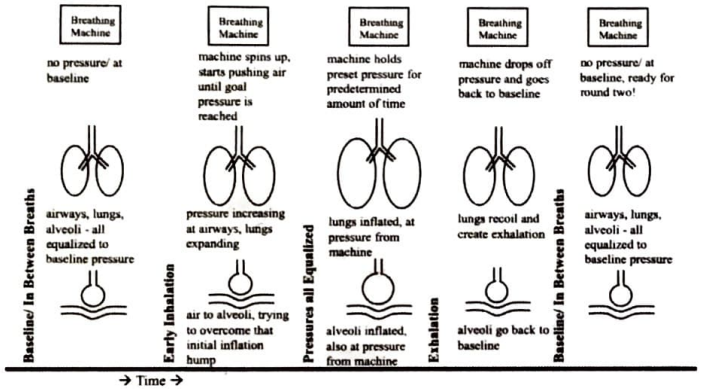
[29] Kneyber & friends, 2017 – Note that even the people who make the rules on pediatric ventilation don't endorse one method of control over another…

[30] Yartsev, 2018bc– This article discusses the pros and cons of VC and PC ventilation; while it may make more sense to read over it after the following discussion, we included it here for lack of a better place to mention it

[31] And as a reminder: both of these concepts are discussed in much more detail in the **Appendix**

Pressure

In the other corner of the arena we have pressure control ventilation. 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[32], 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).

One thing worth pointing out here is that in pressure control 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 volume control, but the stretch factor we introduce in our circuit is eliminated. This is a big advantage of pressure control ventilation with small patients: forgetting to factor in 10ml (arbitrary number) in an adult is no big deal, forgetting to do so in a neonate with tidal volumes of 25ml is huge. We'll discuss more later, but just know that this is one advantage of pressure control.

Another advantage of pressure control 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[33].

[32] And if a machine is capable of pressure control ventilation it will almost 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 ventilation only

[33] 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?**

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 volume control) 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.

To summarize: in pressure control ventilation we control the pressure put in to 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 pressure control mode in order to avoid hyper or hypoventilation. In addition, pressure control 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.

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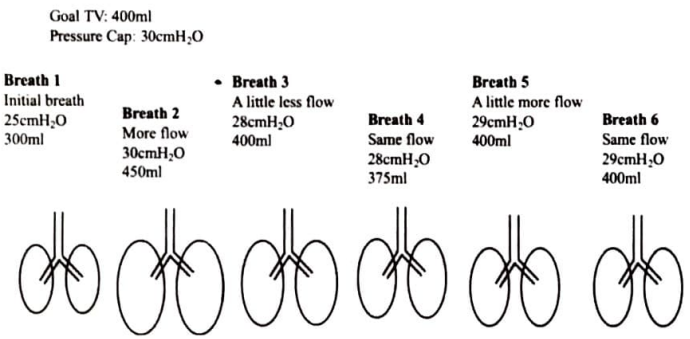
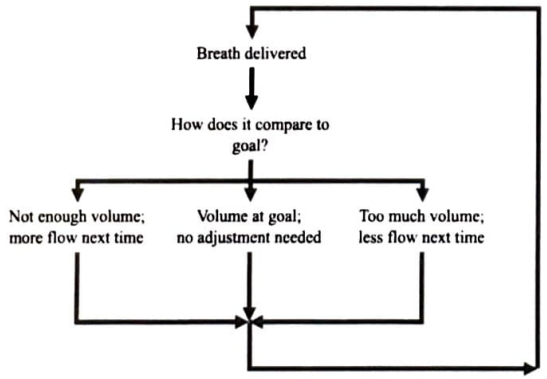
Pressure Regulated Volume Control

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 without exceeding the max pressure. The machine makes adjustments to how it delivers each breath by looking at previous breaths and then adjusts flow 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:

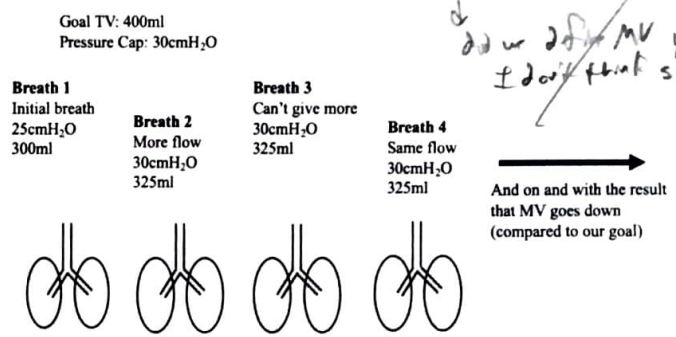


If it helps, we can also think of this in an algorithm-style fashion where we decide where each breath ends up in relation to our goal and then adjust the subsequent breath in a cyclical manner:



This flow 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, which is a good thing!

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 MV. It's important to keep this in mind with PRVC, as we can inadvertently drop MV pretty significantly in an effort to avoid high pressures.



A few more things about PRVC: "pressure cap" in a make-believe term – the machine most often uses 5cmH20 less than the set high pressure limit for this value. There are also limits 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. Along that same idea, the machine doesn't actually know how much air (i.e. TV) 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 MV and match it to our calculated goal.

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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

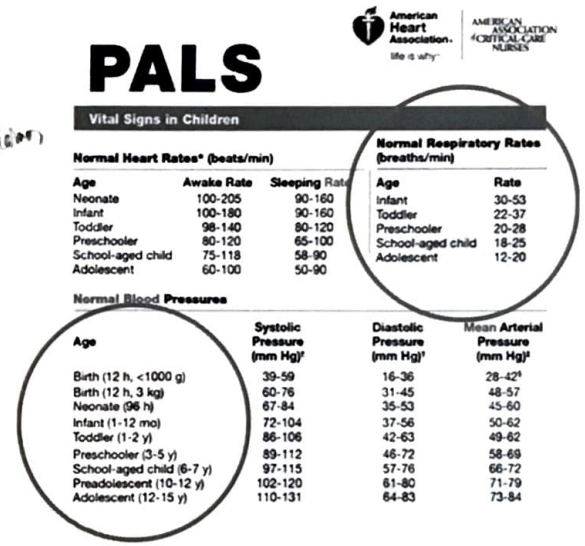
Tidal volume per the textbooks is the amount or volume of air exhaled in a given breath. As previously discussed, it sometimes helps to break this concept up in to two distinct terms: tidal volume and exhaled tidal volume. Tidal volume, in this way of thinking, would be the volume of air we put into 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. In this manual, we have tried to abbreviate things with initial letters of words if the term would be spelled out just to make things easier - this is just so you know what things mean if you see it abbreviated elsewhere.

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 4-10ml/kg IBW or more, 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.

We also previously mentioned the concept of alveolar tidal volume, but let's hold off on that one for now, as we will discuss it in a later section in more detail. For now we will focus on tidal volume as two distinct ideas (tidal volume and exhaled tidal volume) with a normal range of 6-8ml/kg IBW.

Rate

Rate is equivalent to the idea of respiratory rate and describes how many breaths are delivered 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. 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:[34]



American Heart Association. | 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

[34] Cite the PALS Book - 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.

For the OCDers out there, there are some data points missing from this PALS chart. On 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[35]:

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 here, here 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.

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[36]. We will discuss soon 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 minute volume 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 patient sizes:[37]

$$MV = 100ml/kg\ (IBW)\ /min$$

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 to describe the feedback the machine gives us about what the patient breathes out, and lastly there is alveolar minute ventilation which takes out anatomic dead space from the equation. While alveolar minute volume is an important concept to consider, we base goals and calculations on MV or exhaled MV and not on alveolar ventilation. As mentioned before, the reason for this is because alveolar dead space can be difficult to measure in the field and we ought to assume its presence and treat for it anyways.

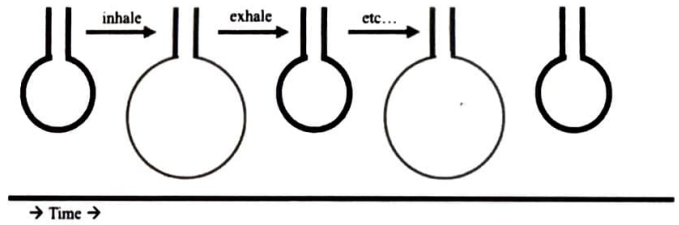
[35] See **Appendix** for a discussion of how this chart was created
[36] To be discussed at length in our section on **Ventilation (and $EtCO_2$)**
[37] Cite this

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[38]. 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[39] 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 just give 100%. And if we are ever in doubt, we 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 outweighs the risk.

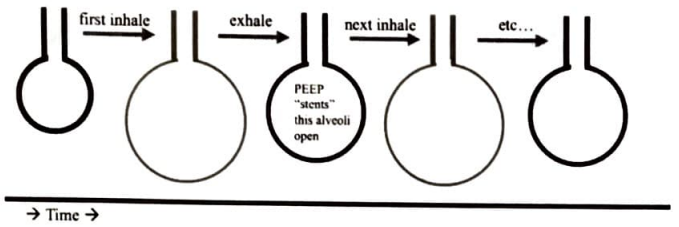
Positive End-Expiratory Pressure

Positive end-expiratory pressure (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:



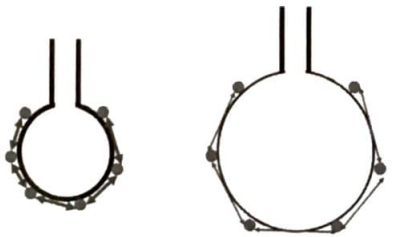
[38] Where did that article go…
[39] And we will get into the details of SpO2 in our section on **Oxygenation (and SpO_2)**

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. So rather than the alveolar air sac deflating all the way back to its original size, it deflates only part way:

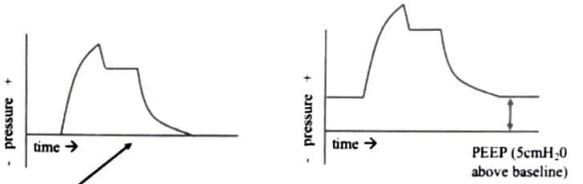


Recall our previous discussion of alveolar surface area (i.e. the more inflated an alveoli is, the more it can participate in gas exchange) and add to that the idea that blood flow though the pulmonary capillary bed is continuous, it doesn't stop when inhalation stops; pulmonary respiration or gas exchange across the alveolar membrane occurs throughout the respiratory cycle, both on inhale and exhale, and PEEP maximizes alveolar surface area during exhalation to make the exhalation-side of pulmonary respiration much more efficient.

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 a very long time to get from a that left-most, deflated stage to a "recruited" or opened-up stage ~~that provides some benefit to patient outcome~~. The reason for this is that initial "hump" that we must overcome when starting the inflation process. Remember that picture with the hand-holding surface tension molecules?



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[40]. So now that we have that clarified, let's look a waveform representing ~~alveolar~~ pressures as we deliver a breath. We've seen this image previously, but now we are going to add some numbers to it. The first breath is with no PEEP or zero PEEP or "ZEEP", the second one (right) is with 5cm$H_2$0 worth of PEEP added in:



One important thing to note is ~~that the area under the right-sided waveform~~ in this example breath is much larger than the area on ~~the left~~. Recall from our discussion of SIMV when we said that the area under the curve represents tidal volume – ~~while that was more or less true~~ in that SIMV example with no PEEP, the area under the curve actually represents the volume of air that participates in alveolar gas exchange. PEEP not only increases the amount of air that participates in gas exchange during inspiration, it also aplifies that gas exchange into the expiratory phase of the respiratory cycle.

We are getting closer to the end of our PEEP chat, but a few more tidbits before we move on. You may be wondering about this idea of "physiologic PEEP" and the idea oft-cited concept that all of us, at baseline, live with 3-5ish cm$H_2$0 worth of PEEP in our alveoli. "But how does this work," you may wonder, "when PEEP is a positive pressure and we normally breathe by a negative pressure mechanism and with very small pressure changes!?" The skinny of it is that it doesn't work, "physiologic PEEP" is not truly a thing. That said, there is some credence to the idea that intubating a patient and/ or strapping a vent circuit to their face increases resistance to the flow of air.[41] But this is a whole 'nuther animal and we'll leave it alone for now.[42]

Also review down sides of PEEP[43]

[40] Kallet & Branson, 2017
[41] Sagana, 2019 – re-read and get an explanation here
[42] Resistance is very briefly discussion in the **Appendix**
[43] Coruh & Luks, 2014 – go on… also the Strong Medicine Video, find that

Inspiratory Time (and I:E Ratio)

The next (and final, for now!) 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:[44]

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

We've already mentioned that during positive pressure ventilation 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. 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 ~~= 3.52941176 seconds per breath~~
~~≈ 3.5 seconds per breath~~
≈ 3.5 seconds per each in/out cycle

If "in" or inspiration = 1.0 seconds, then "out" or exhalation = 3.5 seconds – 1.0 seconds
~~Therefore~~ "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

We often represent this ratio between I-time and expiration time as an "I:E Ratio" to represent 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 as so: $\frac{1.5}{1.5} : \frac{2.0}{1.5}$
And solve for our new I:E Ratio of 1:1.33

[44] See **Appendix** for how we got all these numbers

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 return to this concept 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.

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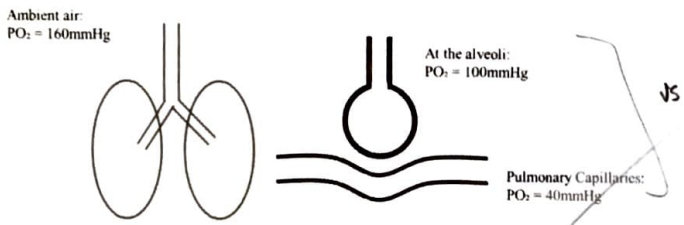
Three Big Things

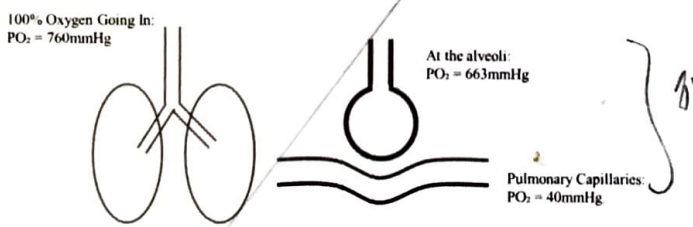
There are three super duper important things that need to be monitored and addressed for all ventilated patients, hands down and no matter what. The order we discuss them here is totally arbitrary, they all hold equal weight and are interrelated. The discussions that follow are in general terms and not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

Oxygenation (and SpO_2)

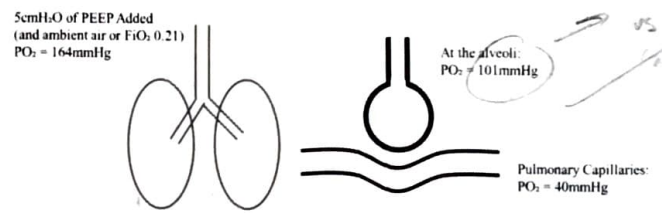
It may have come up once or twice before, but oxygen is pretty important stuff. Oxygen gets to tissues via a few steps, some of those we can affect directly with the ventilator. There are also more complicated ways to manipulate oxygenation, but let's focus on the simple stuff for now, starting with 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, ~~just FYI~~:



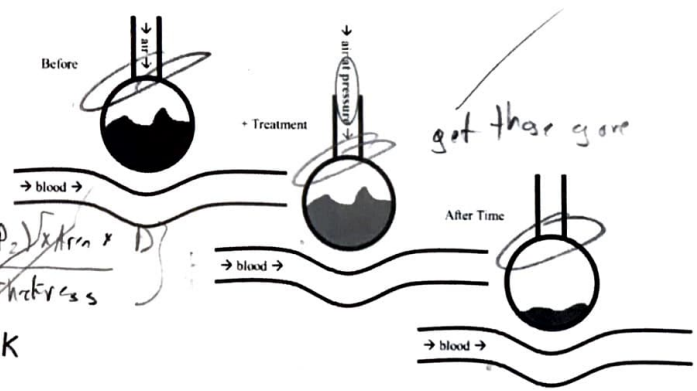
We also mentioned that gasses will diffuse from areas of high concentration (~~represented by higher partial pressures in this graphic~~) to area of lower concentration. So in this baseline example, we can conclude that oxygen will move from the ambient air, to the alveoli and then in to the pulmonary 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:



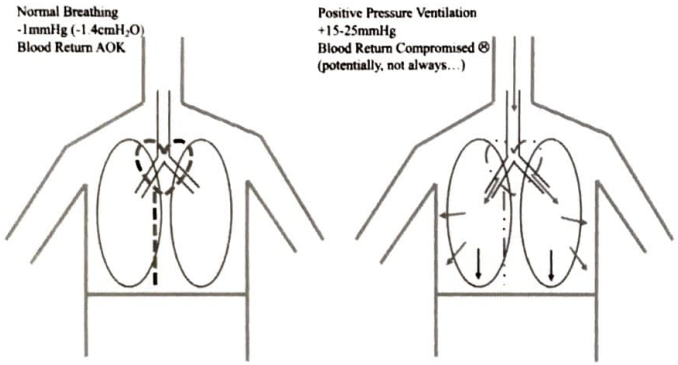
The next way we can increase oxygenation is via PEEP. Now PEEP doesn't quite work by the same mechanism, as the addition of PEEP doesn't much change the partial pressure situation as we saw with an increase in FiO2:



Instead, PEEP facilitates oxygenation by increasing alveolar surface area and 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 **Positive End-Expiratory Pressure**, 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 and gunk that accumulates there. Think of it this way:

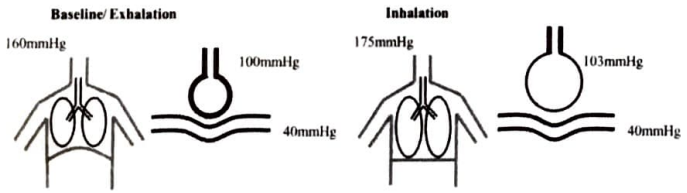


PEEP is really good at increasing oxygenation, but at the potential cost of decreasing blood return to the heart. It does its oxygenation thing by increasing alveolar partial pressure of oxygen, increasing alveolar surface area, and by helping "push" fluid out of the alveoli. Recall from a previous discussion 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:



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[45], 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 setting. PEEP is especially helpful in increasing alveolar surface area and driving fluid out of the lungs, but may decrease CO by way of a drop in preload to the heart. 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 most 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:



[45] We'll touch a bit more on this subject in the section on **Acute Lung Injury/ 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. 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 before 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.

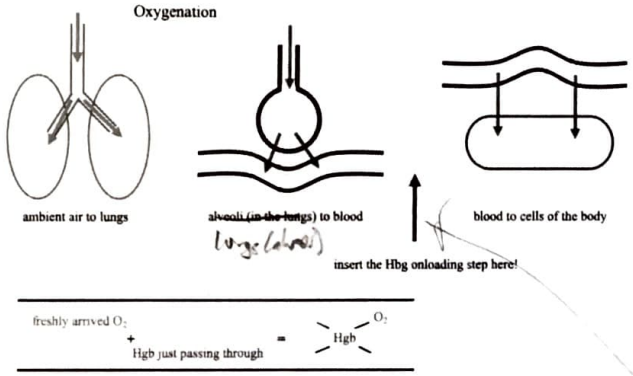
The primary drawback of a really long I-time (and therefore of an inverted I:E ratio) is that it is extra uncomfortable for our patients and we will need to get super aggressive to maintain patient synchrony with the machine. Comfort is one of the three super duper important concepts in this section, so enough said about that until we get there. An inverted I:E may also may 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 spinning dials 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.

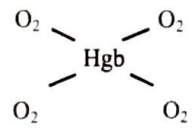
Recall back to our previous discussions of both the hypoxic vasoconstrictive response and alveolar dead space. There are times where we are getting oxygen into the system just right, but components inside the system are out of whack and that oxygen is not being put to good use. One thing we, as clinicians, 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. 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[46].

[46] Find that article with that big long discussion of HoB in vents

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. This is the onloading process and the focus of our discussion for now, we will talk about how that oxygen gets off or unloaded from the Hgb later on. But let's draw those onloading bits out for now:



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, for the most part, 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 (i.e. adequate MV) and comfort are addressed simultaneously (see below).

Ventilation (and $EtCO_2$)

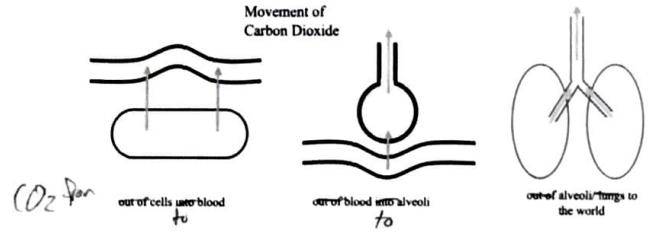
Next super duper important thing is ventilation. Ventilation refers to the movement of air in and out of the system as we deliver breaths and allow exhalation. As discussed before, this is vitally important for the movement out of carbon dioxide. Too much carbon dioxide hanging out in the lungs with 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:

$$MV = 100ml/kg\ (IBW)\ /min$$

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 appropriate as a minimum for most patients. Having a goal minute ventilation in mind and then assessing actual minute ventilation (typically calculated by the vent and readily available) is great way to ensure that the patient's minimum needs are met.

Concurrently, we can 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:



So the value we get on our quantitative EtCO2 reading is a function of all of these factors. It gets a bit complicated and has been deferred to the **Appendix**, but the standard approach to managing ventilation with $EtCO_2$ is to use a base range and then 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 this respiratory alkalosis) 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 ~~above~~ 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, ~~that's why they are both mentioned here~~. 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 in to 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.

Comfort

The third super duper important parameter that we need to consider with vent management is patient comfort. If your patient is not comfortable, he or she will be "fighting the vent" or "out of synch" and the therapeutic effects that we want to achieve will be more difficult to attain. Also, 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 this 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. 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[47].

[47] Cite this – that article about oversedation

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

Table 1.
Adult Nonverbal Pain **Scale** (NVPS)

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 the body	Splinting areas of the body, tense	Rigid, stiff
Physiology (vital signs)	Baseline vital signs unchanged	Change in any of the following • SBP > 20 mm Hg • HR > 20/min	Change in any of the following • SBP > 30 mm Hg • HR >25/min
Respiratory	Baseline RR/SpO_2 synchronous with ventilator	RR >10 above baseline, or 5% decrease SpO_2, or mild asynchrony with ventilator	RR >20 above baseline, or 10% decrease SpO_2, or severe asynchrony with ventilator

From Odhner, M., Wegner, S., Freeman, N., Stevenson, A., & Ingersoll, G. (2003). Assessing pain control in nonverbal critically ill adults. Dimensions in Critical Care Nursing, 22(6), 263

Richmond Agitation-Sedation Scale (RASS)

Score	Term	Description	
+4	Combative	Overtly combative, violent, immediate danger to staff	
+3	Very agitated	Pulls or removes tube(s) or catheter(s); aggressive	
+2	Agitated	Frequent nonpurposeful movement, fights ventilator	
+1	Restless	Anxious but movements not aggressively vigorous	
0	Alert and calm		
-1	Drowsy	Not fully alert but has sustained awakening (eye opening eye contact) to voice (≥10 seconds)	Verbal Stimulation
-2	Light sedation	Briefly awakens to voice with eye contact (<10 seconds)	
-3	Moderate sedation	Movement or eye opening to voice (but no eye contact)	
-4	Deep sedation	No response to voice but movement or eye opening to *physical* stimulation	Physical Stimulation
-5	Unarousable	No response to voice or *physical* stimulation	

Relevant story to put this in context: when some of us were first taught GCS, we might remember it being put more or less like so, "GCS is one of those things that nurses like to hear on radio reports and that supervisors like to see on charts, so even though we generally don't calculate a GCS in the field, it is important to sort out before you get to the hospital and before you submit your chart." Sedation scales are not those types of things, they actually help in real time are not simply another box to check to avoid a nit-picky peer review from a colleague. With that said, it's also OK to recognize that putting someone on the vent involves a lot of steps and other important things and so it is alright (opinion alert!) to have an initial, preplanned strategy for first round of sedation and analgesia, and then pull out a sedation scale reference card once we are sailing smoothly and work through it checklist-style with our partner.

Let's imagine a hypothetical scenario to get in to the details on this: we pick up a vented guy from a hospital, he's obviously uncomfortable and out of synch with the vent, we address ventilation and oxygenation (per ~~above~~ 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'll talk about this more later, but breaths are delivered differently in different modes ~~(depending on the specific machine)~~ 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 ~~trigger~~ a breath when he or she wants. Again, more on that to come.

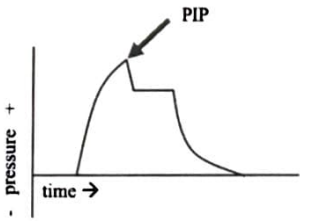
So last summary here and we'll include all three of these super duper important parameters that we need to address on all of our patients, hands down and no matter what. Comfort should be assessed using an actual scoring tool and can be fixed with both drugs and vent manipulations. Oxygenation is measured by SpO_2 and gets fixed by increasing FiO_2, adding PEEP and lengthening the I-time. Ventilation is evaluated by a comparison to expected MV and then $EtCO_2$, 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.

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 other 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 dialed in for volume control ventilation and then we'll talk about how these concepts carry over in to pressure control ventilation.

Peak Inspiratory Pressure[48]



Peak Inspiratory Pressure (PIP) is the highest point on this waveform. It represents the maximum pressure present in the airways as we deliver a breath into the system. 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 <35mmH_20. A PIP that is too high generally won't cause significant damage to the patient, but it likely

[48] Nickson, 2019b – Short article that provides another good review of both PIP (this subsection) and Pplat (next subsection)

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.

But let's get back, real quick, to what goes in to giving us this value of PIP. On the machine end, it is the result of flow. Flow is a term we haven't touched on, but it essentially describes how fast we push air to achieve a breath. In many transport ventilators, flow is calculated internally by the machine and we don't manipulate it directly. In these cases, the closest we have to adjusting this parameter is via I-time or what we call "rise profile." That said, let's hold off on discussing how to decrease PIP by adjusting vent settings, as it is more often the result of other airway stuff that we can address more directly.

This airway stuff would be the passageway from the vent circuit to the alveoli. If it inhibits adequate movement of air (such as with a small or kinked ETT), we will see a higher PIP. Another factor here is patient comfort and the idea of laminar flow. Without getting too far into the weeds on this, recognize that air can flow freely and efficiently through a uniform pipe or tube, but if we cause movement or disruptions to that tube airflow will be less uniform and more chaotic and will result in higher pressures. Morale here: make sure your vented patient is comfortable.

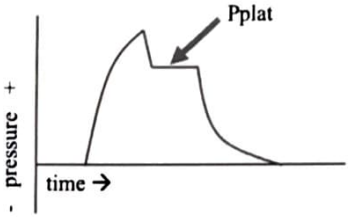
Other causes of an elevated PIP would be secretions in the airway and/ or ETT, tension pneumothorax, migration of the ETT to one of the mainstem bronchi, bronchospasm and decreased compliance (i.e. the lungs don't expand like we want them to). Any time we see a high PIP we ought to try and identify a cause. 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 pneumo 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 at the ETT and not the patient's anatomy (i.e. alveoli), so we may decide to leave it alone (especially is there is good reason for that small ETT, such as airway swelling).

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.

So in summary, PIP represents the maximum pressure present in the airways as a breath is delivered by the machine.[49] A normal value is <35mmH_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.

[49] Before moving on, it's also worth mentioning that you may sometimes see PIP noted as Ppeak or "peak pressure" – same game, different name

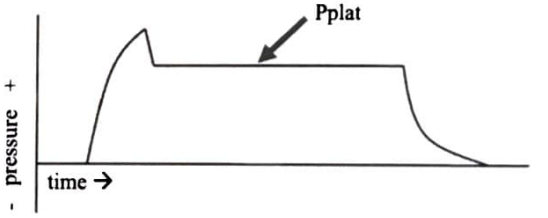
Plateau Pressure



Plateau pressure (Pplat) is the pressure in the system once the lungs fill with air and essentially holds its breath until the breath cycles off. It represents the pressure at the alveoli during gas exchange or pulmonary respiration. 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'll discuss this concept here in just a moment.

The primary cause of a high Pplat is too much tidal volume. That said, it can also be due to decreased compliance, pneumo, mainstem intubation and inhibition of chest wall expansion (such as in burns). 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 lung unnecessarily. We do, however, want to avoid a sustained high Pplat over many breaths, as that will likely lead to damage at the alveolar level.

Measuring a Pplat is little less direct that measuring a PIP and involves what we term 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:



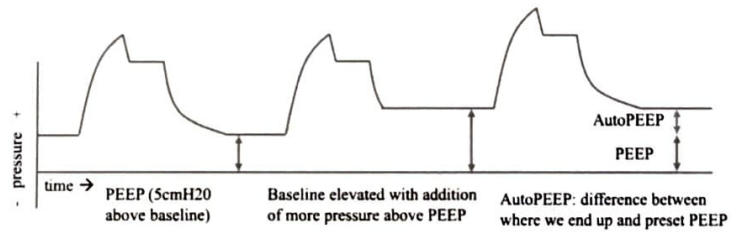
Basically we just perform the inspiratory hold maneuver (in whatever way is appropriate for our particular machine) and the Pplat either pops up on the screen for us or we have enough time to read the value from the barometer. Easy enough, but when and how often do we do this thing? There isn't a universally 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/ 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).

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, pneumo, decreased compliance, restriction to chest wall expansion and mainstem intubation.

AutoPEEP

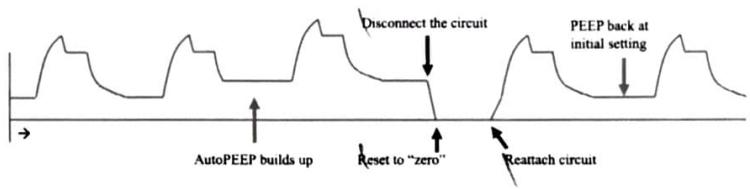
AutoPEEP is the idea of PEEP being cumulatively added into the system inadvertently. Remember how we said before that we assume atmospheric pressure to be 0cmH_20 as the starting point for our vent discussions and that PEEP is the addition of pressure on top of that (i.e. "adding 5cm of PEEP" to reset that baseline to 5cmH_20)? 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. To measure AutoPEEP or to check its presence, we have to perform another maneuver called an "expiratory hold." 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 my I:E ratio and allow more exhalation; otherwise we could consider more sedation/ pain control and make sure we aren't accidentally triggering.

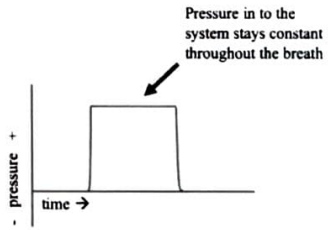
One other thing we can do to eliminate AutoPEEP and reestablish our baseline at actual PEEP is disconnect the patient from the vent circuit to allow a full and complete exhalation. This is one of those rare cases in which it is OK to disconnect the vent circuit from the patient during transport for therapeutic 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 or 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 amount of time spent in exhalation (shorter I-time, lower RR), treating discomfort and avoiding accidental triggers. In addition, we can reset AutoPEEP back to zero (~~and then actual PEEP~~) by disconnecting the vent circuit.

PIP & Pplat in Pressure Control?

Up to this point we've discussed PIP and Pplat only 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:



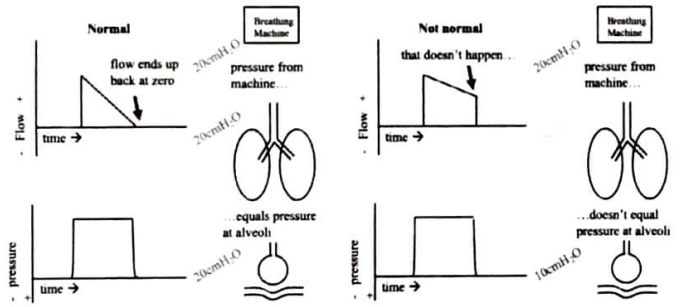
First thing to mention here is that PIP will only be above that flat line at the top of the square wave form (marked by the red arrow in the graphic) if something causes a disturbance in what the machine is doing – a hiccup, patient movement, speedbump, etc. The machine won't intentionally put more pressure than what we have dialed, but a PIP higher than 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.[50]

[50] In PC ventilation, we become aware of those obstruction issues by monitoring flow and VTe

Next thing: it generally happens that the 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 PC = Pplat. And because of this assumption that mostly holds true, 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[51]. 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 dialed in 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.

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. No need for us to get in to the specifics of flow at this point, but recognize that flow is a measure of how fast we are pushing air in to the system in order to give a breath and that pressure control breaths start with a higher flow and then dial down to zero flow throughout the breath. While it may be hard to see with quantitative values on your machine (unless you can view waveforms, which are discussed in the **Appendix**), 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:



[51] Hamilton Medical, 2018 – Has a nice explanation/ visual representation of how pressure equalizes throughout the respiratory system over time as a breath is delivered; also gives a brief outline of different methods to measure Pplat that we discuss in the following paragraph

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.[52]

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[53]). These are all cool things to work with, but it takes us in to realm of weeds and 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.

[52] Mojoli & friends, 2015 – Another article referenced in the above content; this short paper assesses the efficacy of these alternative methods of measuring Pplat (and also delta pressure)

[53] In the section **Other Random Things There May Be Questions About**

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A General Vent Strategy

In this section we are going to summarize some general parameters that we work with in each type of ventilation (i.e. each combination of mode and control). The idea here is to demonstrate what parameters and goals are shared among all methods and what parameters are specific to certain types of ventilation. We will also hash out a few of the differences in determines general settings for adult versus pediatric population. We will start with a discussion of things that apply to most vented patients, regardless of mode or control:

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

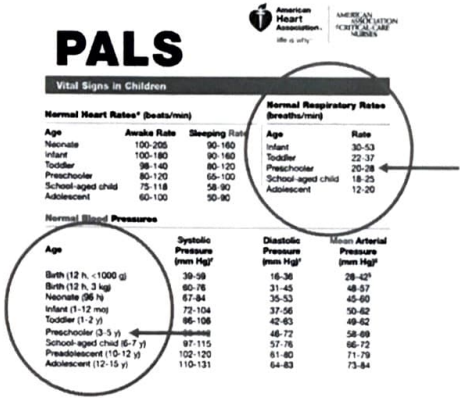
If we choose a TV of 6ml/kg and our goal is 100ml/kg/min, then our rate ought to be 17:

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

Likewise, if we go with 8ml/kg our initial rate (to match that MV goal) comes to 13/ min. Although it's not uncommon to see recommendations for an initial rate of 10 or 12 with adults, for the sake of using reason and math and not pulling things out of thin air, calculating a RR based on a MV is a solid strategy. There are often good reasons to use a lower RR, but we'll get to those later.

To expand on this idea, if we have a range of TVs to choose from, sometimes it just makes life easier to pick a nice, even number. For example, in an 80kg patient we end up with a TV goal range of 480-640ml and 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. Or a TV of 600 and a RR of 14. Either is cool for now and we'll dial in our settings once we see how the patient responds to it all.

As for kiddos, the preferred strategy 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, we can titrate values to address that later on. For example, let's assume a 4-year-old kid of 16kg. Based on this chart (again, from PALS) we want a RR in the 20-28/min range:



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[54]:

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

[54] 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:

$$TV = 6 - 8ml/kg\ IBW$$
$$TV = 6 - 8ml \times 16kg$$
$$TV = 96 - 128ml$$

$$MV_{goal} = 100ml/kg\ (IBW)\ /min$$
$$MV_{goal} = 1600ml/min$$
$$MV_{goal} = 1.6L/min$$

$$MV_{calculated} = RR \times TV$$
$$MV_{calculated} = (20 - 28)/min \times (96-128)ml$$
$$MV_{calculated} = (1920 - 3584)ml/min$$
$$MV_{calculated} \approx 2 - 3.5L/min$$

The result here is a MV goal that differs pretty significantly from a 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 a volume control mode. There is always some mechanical dead space that we introduce into the system that sneaks its way in to our calculated MV number and this overestimation will mitigate that.[55]

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

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 them in to a chart just to make it easier to visualize. And this chart is basically a summary of the section **Vent Parameters, Round One**– if you need to review the specifics of any of them, just refer back to that bit:

Parameter	Value	Pro Tips/~~Extra Info~~
TV	6-8ml/kg	Pick an easy number to work with that falls in 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	
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[56]
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

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 made and via any method of control, so long as we know what to monitor for depending on what we choose. Let's draw it all out in a quick chart:

	Additional Parameters[57]
AC Volume	None
SIMV Volume	***Pressure Support*** – start at 10-15mmH_2O and titrate as needed
AC Pressure	***Pressure Control*** – start at 15-20cmH_2O and titrate to TV goal
SIMV Pressure	***Pressure Control*** – start at 15-20cmH_2O and titrate to TV goal ***Pressure Support*** – start at 10-15mmH_2O and titrate as needed
AC PRVC	***"Pressure Cap"***[58] – 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 10-15mmH_2O and titrate as needed

[56] Reference EMCrit's drop to 40% asap

[57] Find sthg to support these starting #s for PC and PS!

[58] 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 a ~~decision tree of sorts~~ to describe how we determine vent settings, in general and for the "normal" patient:

Step One: Set These Guys		Step Two: Make a Choice and Dial in Extra Stuff[59]	
TV[60]	6-8ml/kg	**AC Volume**	None
MV	100ml/kg/min	**SIMV Volume**	***Pressure Support*** – 10-15mmH_2O
RR	Adults: 13-17/min Kiddos: use a chart	**AC Pressure**	***Pressure Control*** –15-20cmH_2O
FiO_2	1.0, then titrate down	**SIMV Pressure**	***Pressure Control*** –15-20cmH_2O ***Pressure Support*** – 10-15mmH_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*** – 10-15mmH_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.

[59] PS/PC variation with kiddos?

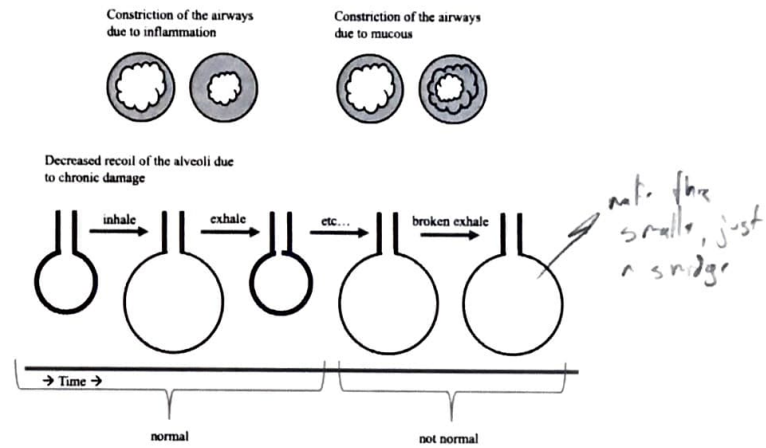
[60] In pressure control we don't actually set this guy, but we do need to have this value in mind and calculated out so that we can use it as a goal

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. Let's look at a few situations/ etiologies in turn to see how it all looks.

Bronchospasm

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 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 dialing 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, to a lesser extent) 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 ≈ 3.53s/breath
3.53s – 1.0s (I-time) = 2.53s
∴I:E ratio = 1:2.53

With I-time 1.0s and RR 13:
60 ÷ 13 breaths ≈ 4.62s/breath
4.62s – 1.0s (I-time) = 3.62s
∴I:E ratio = 1:3.62

With I-time 0.8s and RR 13:
60 ÷ 13 breaths ≈ 4.62s/breath
4.62s – 0.8s (I-time) = 3.64s
∴I:E ratio = $\frac{0.8}{0.8}$: $\frac{3.64}{0.8}$
I:E ratio = 1:4.55

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 need for these bronchoconstricted patients. Let's keep up with some of these calculations and put them in to a small chart: [61]

I-time 1.0s		I-time 0.8s		I-time 0.5s	
RR	*I:E*	*RR*	*I:E*	*RR*	*I:E*
17	1:2.5	17	1:3.4	17	1:3
13	1:3.6	13	1:3.8 4.8	13	1:4.1
10	1:5	10	1:5.2 6.5	10	1:5.5
8	1:6.5	8	1:6.7 8.4	8	1:7

While an I-time of 0.5s may be uncomfortable for some patients, we've included it here just as a point of reference. We typically leave I-time alone at "normal" range, but it's worth keeping in mind and giving it a shot to see how the patient does. Shorter I-time may lead to increased PIPs with volume control ventilation, but so long as our Pplat is within range we are AOK with that. Doing the same in PC would lead to decreased VTe.

[61] Redo these cales, I think some are wrong...

Now assume we choose an I-time of 0.8s and a RR of 8 (for a calculated I:E of 1:6.7), 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
$MV_{calculated}$ ≈ 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
$MV_{calculated}$ ≈ 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. We will simultaneously be doing pharmacological interventions (Albuterol, Ipratropium, MagSulfate, Ketamine, Epi – 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) 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 15-20cmH_2O, consider going straight to the top and starting at 25-30cmH_2O (the upper limit for a safe Pplat) to see what out VTe values look like. If we happen to overshoot our TV goal of 12ml/kg, we can always titrate back down. In addition, recognize that 30cmH_2O as the upper limit is a generalization that may not be OK 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 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 evidence that applied PEEP can help fix

AutoPEEP; but we do want to keep applied PEEP lower than AutoPEEP[62]. Just know that we'd prefer 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 5, but there isn't much content on that and we'll leave it as a "maybe" in the overall scheme of things.

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 bronchospasm 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 bronchospasm strategy: utilize a lower rate (and consider a lower I-time also) to a goal I:E of 1:≥5. Consequently, we need to titrate TV (or PC[63]) 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 considering dropping PEEP if no oxygenation issues are noted. Also, be sure to check for AutoPEEP periodically and consider disconnecting the vent circuit to reset it back to zero if need be.

[62] Sagana & Hyzy, 2019 – These guys offer more than you ever wanted to know about PEEP; more relevant to our discussion, they note that applied PEEP can facilitate exhalation and help to fix an AutoPEEP problem

[63] Medina & friends (2016) - As a side note on PC ventilation for bronchospasm with kiddos: these guys cite a set of four cases in which PC ventilation failed to adequately ventilate pediatric patients with airway obstruction; while this may be an isolated set of cases and could potentially be mitigated by a strategy focused on ensuring adequate exhalation (via a long I:E, as described above), it is worth considering VC ventilation for pediatrics with bronchospasm in spite of the fact that we often advocate PC ventilation for pediatrics

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, 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 in inspiration during the overall respiratory cycle. Think of it this way: preload drops 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)[64]:

$$\%\,TaDP = (RR \times I\text{-time}) \div 60\ seconds$$
$$\%TaDP = (17 \times 1.0s) \div 60s$$
$$\%TaDP = 17s \div 60s$$
$$\%TaDP = 28\%$$

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

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

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

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 ~~adequate~~ 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:

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

$$TV = 8ml/kg \times 65kg$$
$$TV = 520ml$$

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

$$MV_{calculated} = TV \times RR$$
$$MV_{calculated} = 520ml \times 10/min$$
$$MV_{calculated} = 5200ml/min$$
$$MV_{calculated} = 5.2L/min$$

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 bronchospasm 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:

$$TV = 10ml/kg \times 65kg$$
$$TV = 650ml$$

$$MV_{calculated} = TV \times RR$$
$$MV_{calculated} = 650ml \times 10/min$$
$$MV_{calculated} = 6500ml/min$$
$$MV_{calculated} = 6.5L/min$$

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 patient with 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 ~~work~~ in that direction while ensuring adequate ventilation[65].

To summarize: in the hypotensive patient we want to decrease the amount of time spend 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 20-25cmH_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 leave PEEP alone.

[65] 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…

Acidosis

With acidosis one of our primary vent goals is to facilitate the 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 ~~quantify~~ the MV difference between a rate of 12 and 30 with an assumed TV of 500ml:

$$MV_{calculated} = TV \times RR$$
$$MV_{calculated} = 500ml \times 30/min$$
$$MV_{caclualted} = 150000ml/min$$
$$MV_{caclualted} = 15L/min$$

$$MV_{calculated} = TV \times RR$$
$$MV_{calculated} = 500ml \times 12/min$$
$$MV_{caclualted} = 6000ml/min$$
$$MV_{caclualted} = 6L/min$$

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 aim for a goal double that of the normal patient: 200ml/kg/min. To achieve that goal, we may need to increase both RR and TV. We said before that to increase MV (i.e. 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:

$$MV_{goal} = 200ml/kg/min$$
$$MV_{goal} = 200ml/kg/min \times 65kg$$
$$MV_{goal} = 13000ml/min$$
$$MV_{goal} = 13L/min$$

$$TV = 8ml/kg \times 65kg$$
$$TV = 520ml$$

$$MV_{goal} = TV \times RR$$
$$13L = 520ml \times RR$$
$$13L / 520ml = RR$$
$$25 = RR$$

This means that a TV at 8ml/kg and a RR of 25 will get us the theoretical MV of 200ml/kg/min. But what is the consequence of a MV that high? 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. 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 a 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) prior to taking the airway. We can then match the patient's RR (as above) or set RR to 25 (as calculated) 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 PCO2 is compared to a calculated PCO2 to determine if a mixed disorder is present), but we can modify its use to the transport setting to guide our titration of EtCO2 (via MV):

$$EtCO_2 \text{ should} = (1.5 \times HCO_3^-) + 8 \pm 2$$

Assuming compensation was adequate when HCO_3^- was measured
(and HCO_3^- from either BMP or ABG)

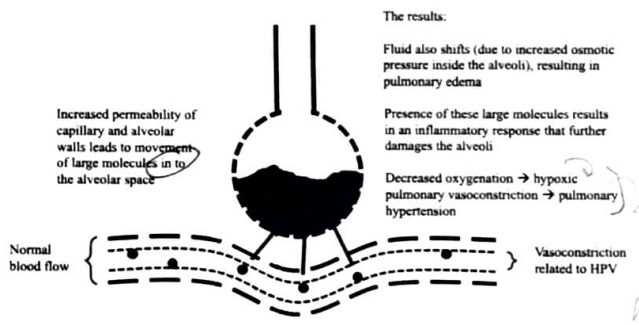
And in fact we can do all of these strategies together: try to match the patient's RR and $EtCO_2$ as measured before we intervene, then compare MV to our calculated goal of 200ml/kg/min and compare $EtCO_2$ (both the patient's pre-intervention one and our subsequently-measured one) to EtCO2 goal from 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 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).

Acute Lung Injury/ ARDS[66]

Another well-known and established strategy in vent management is the "injured" or "sick" lung strategy. 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 EtCO2. Another component of this strategy is higher than normal PEEPs to improve oxygenation, recruit 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 ARDS, 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. 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 syndromes (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 pathophysiology 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. We can identify a patient who needs this vent strategy from a report per sending facility, suspicion based on clinical progression of the illness or the simple fact that we can't get to a point of adequate oxygenation by other methods. The strategy includes low volumes, PEEP, maintaining recruitment and permissive hypercapnia. Let's discuss each of these in turn and give some specific guidance.

Starting TV for these patients should be 4ml/kg IBW. This recommendation is from the ARDSNet studies which compared TVs of 4ml/kg against 12ml/kg and determined that lower TVs resulted in significantly better outcomes for these patients. While it may seem that 4ml/kg and 12ml/kg represent two extremes and it could be tempting to rationalize that 6ml/kg (to stay within norms) probably isn't all that bad, we do know that 4ml/kg is OK and we don't know much about 6ml/kg for these patients, so let's just stick with the data and ventilate at 4ml/kg until the science people tell us otherwise.

[66] Sajjad & friends, 2018 – A very heady article that looks deep into the pathophysiology of ARDS to investigate why a Pplat was measured to be higher that a PIP during the treatment of a ventilated ARDS patient; provides good insight on transpulmonary pressures, which is something we skimmed past way back at the beginning of this manual when we assumed that the lung (alveoli) and chest wall always move in synch with one another – more on this in the **Appendix**, but a good read at this point along the way!

In addition to low TV, we go up on PEEP to improve oxygenation. ~~Recall this chart from an earlier~~ discussion about titrating FiO_2 and PEEP ~~in a stepwise fashion~~ to achieve our oxygenation goals. ~~We included in our discussion of FiO_2 and deferred a chat about it then, but it actually is specific to~~ this ALI/ARDS vent strategy:

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

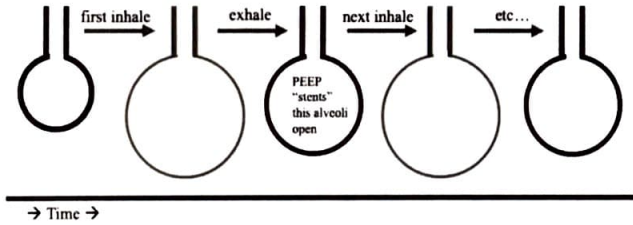
FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

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

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. Recruitment is the idea that we can actively re-inflate collapsed or underinflated alveoli as we depicted in our previous discussion of PEEP:



In all lungs we lose recruitment more or less immediately. Which means that 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 we can 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 takes 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, just to be safe let's always 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 what the technique looks like:

Get a photo and label it in ppt

Last thing to mention with this ALI/ARDS strategy is MV. We mentioned already that dropping our TV to 4ml/kg will reduce MV and increase $EtCO_2$, but let's quantify that difference in MV:

$MV_{goal} = 6.5L$

$TV = 4ml/kg \times 65kg$
$TV = 250ml$

$MV_{calculated} = TV \times RR$
$MV_{calculated} = 250ml \times 17/min$
$MV_{calculated} = 4250ml$
$MV_{calculated} \approx 4.3L$

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

$MV_{goal} = TV \times RR$
$6.5L = 250ml \times RR$
$6.5L / 250ml = RR$
$26 = RR$

So to maintain our MV goal with a TV of 4ml/kg we need a RR of 26. 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 of benefit to 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, that rate of 26 ought to be more or less adequate (unless we have a very young patient), so we should aim to meet (or exceed if in volume control) our MV goal. (And again, more on this pediatric stuff in the **Appendix**)

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 at 4ml/kg to avoid barotrauma, high PEEP to both recruit alveoli and displace fluid, maintenance of recruitment at all times in order to avoid rapid deterioration, and either an increase in RR to maintain MV or a strategy of permissive hypercapnia (often times somewhere in the middle of those last two).

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 work 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 expect 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.

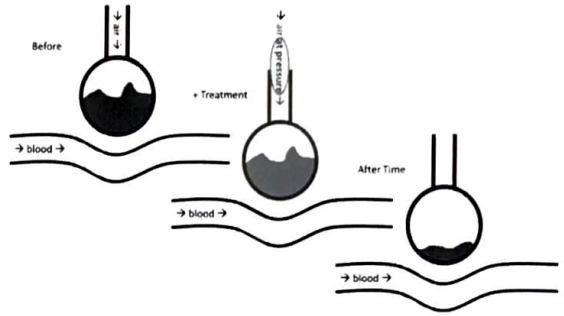
In the patient with a head injury/ traumatic brain injury (TBI), we may choose to aim for an $EtCO_2$ below what we'd typically use for a standard patient. While we don't necessarily "hyperventilate" these patients anymore, we could adjust MV to a goal $EtCO_2$ of 30-35mmHg by going up on either TV (preferred) or RR. We also want to maximize oxygenation and, therefore, may be OK with an SpO_2 of 100% for a little while (whereas we would normally titrate FiO2 down in response). We may also make small adjustments to our settings in an effort to maximize patient comfort, 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 baby. We also need to consider an increased MV goal for the patient, 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.

Significant chest trauma is another one. We'd like to treat these patients via the acute lung injury strategy, but we also are concerned with hypotension and may 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. 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, 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 may warrant more PEEP and increased pressure (either via PC or TVs to a maximum acceptable Pplat). This would facilitate the movement of fluid out of the alveoli:



In this case we could increase PEEP, then increase PC (or TV to the max before Pplat reaches its limit), then titrate RR down to maintain our MV goal. And if the patient is hypertensive along with this pulmonary edema, aggressively maximizing oxygenation to alleviate any pulmonary vasoconstriction (which leads to pulmonary hypertension) may be the best approach.[67]

It quickly becomes evident that there are a number of situations that don't quite fit the cookie cutter mold by which we try to simplify vent strategies. And that's totally OK. The templates are there as a framework 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 he or she presents in a given situation. There are lots of cases in which there isn't a straightforward answer[68], but as long we don't make things worse by dialing the buttons the wrong way, we are still facilitating transfer to a higher level of care where these things can be sorted out.

[67] I had a note: "also PPV/ PEEP to increase CO (maybe w/ MI too?)" on the last edit; not sure what that was about, but look in to it ☺

[68] Lodha & friends, 2006 – As an excellent example of this, this paper describes a case study on the vent management of a pediatric patient with tracheal stenosis; their approach was similar to the bronchospasm strategy, but required normal I-times to ensure adequate ventilation

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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 TVs for good reason, etc. Because of this, we need a strategy 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 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 patients has responded to or will respond to strategies we might have in mind. We then decide on a strategy based on how we think that patient ought to be ventilated (i.e. hypotensive strategy, bronchospasm 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 ~~a bit more~~ straightforward: we just 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. 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 or 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

Now 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 strategy 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[69]. Again, no need to get in to specifics here ~~(that will come later in case reviews)~~, 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.

[69] Since we don't talk at all about labs, refer them somewhere (also do the same for pharmacology) – maybe Bauer and Swearingen books? Take a look

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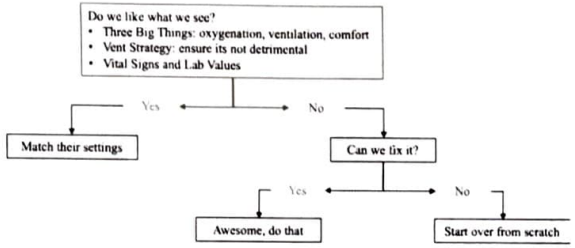
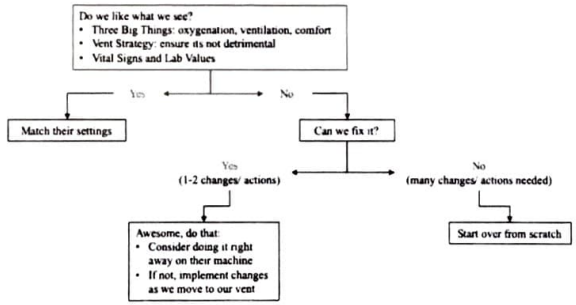
Let's redraw that simple algorithm we started with and add in just a little bit of detail to include all of these ideas and then we'll move on to the next question and talk about it in detail:



Next question to discuss further is, "Can we fix it?" The idea here is that 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. 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 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 changes 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. And last thing and probably already obvious, there is some middle ground here: we may make some changes/ do some things right away and then defer other things until transfer, all part of the same strategy. Example: give sedation now, adjust TV or RR during the transition.

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



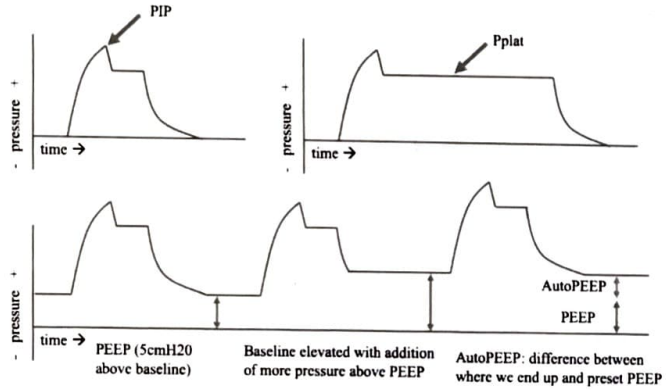
If at any time during this whole process things get too complicated, we can always skip ahead to the "Start over from scratch" end of things, but just recognize that the more changes we make, the less able we are to evaluate the efficacy of a single intervention. Just like a science experiment, it helps to isolate variables and know that the observed result can be attributed to a specific change we made. 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.

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. The general idea is that 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), and AutoPEEP. And for visualization, in case we forgot, here's what they look like on a pressure waveform in volume control ventilation:

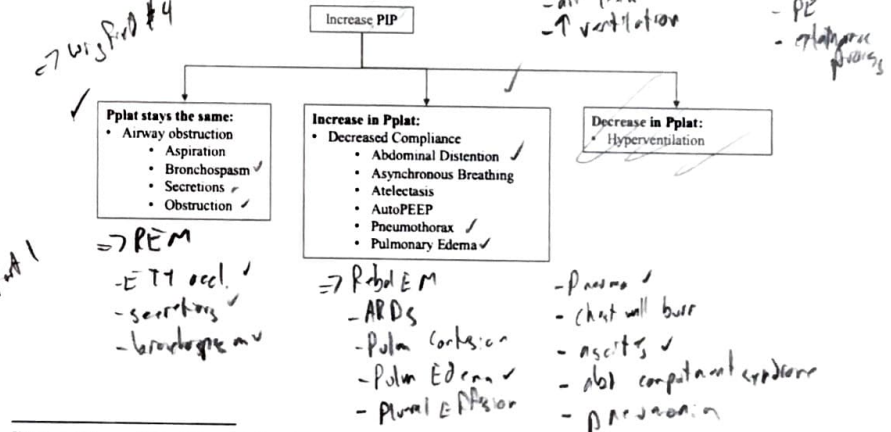


High for PIP is 35mmH$_2$0, although we may go beyond that in certain situations (such as a small small ETT). Pplat max is normally 30mmHg and we do try to stick by that one whenever possible. AutoPEEP normal is zero and we always take actions to address AutoPEEP when we see evidence of it. All of these parameters should be checked (when possible, depending on control and patient's respiratory effort) 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 guiding treatment based on these parameters, let's summarize the basics in a quick chart:

	Normal	Corrective Actions
PIP	<35mmH$_2$O	• Suction, check for kinks or obstructions in circuit/ ETT[70], reposition patient's head • Decrease TV, decrease flow/ rise profile • Consider bronchodilators
Pplat	<30mmH$_2$O	• Decrease TV
AutoPEEP	zero	• Increase I:E ratio • Consider disconnecting vent circuit to allow for exhalation

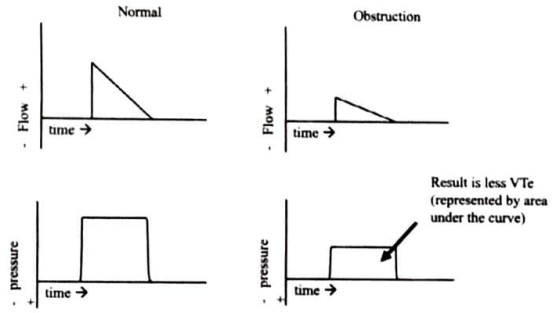
The solutions represented in this chart for both PIP and Pplat refer, in general, to actions to be taken if abnormal values are noticed in conjunction with initiation of ventilation or new changes to settings. There are other times when abnormal PIPs and Pplats develop during the course of ventilation due to pathophysiologic changes. For example, if PIP goes up and Pplat stays the same, this is likely due to some flavor of airway obstruction (aspiration, secretions, biting of the ETT, bronchospasm, etc.). If PIP goes up and Pplat also goes up, this generally indicates decreased compliance. We haven't talked much about compliance, but the idea here is that the lungs expand less per unit of air (volume or pressure) that we put in. Causes of decreased compliance (evidenced by increased PIP and Pplat) would be pneumothorax, pulmonary edema, mainstem migration, asynchrony, abdominal distention, and even AutoPEEP. Consider an algorithmic approach to troubleshooting like this[71]:



[70] Briggs & Freese, 2018 – This article from JEMS outlines a weird case of high airway pressures due to an ETT positioned so that the beveled end was up against the patient's trachea; the fix here was simply to rotate the tube 90 degrees to relieve the obstruction, but the solution took quite some time to sort out

[71] Put some work in to this, maybe also mention DOPE? Cite Wingfield's video here too, this concept is from him

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, 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:



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 VTe. 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.

Alarms[72]

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 we set and ones that are default on the machine. 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.

Most 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 – 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 bottoming out our MV. On the flip side, in pressure control we need to vigilantly monitor MVe (and also VTe, but to a lesser extent) to avoid the same issue. 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 ensure that we maintain an adequate ventilation for our patients.

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 about 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. In the event of one of those situations which may lead to repeated triggering of the high pressure alarm and sudden drop in MV, go up more on PIP (even beyond 35cmH2O 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.

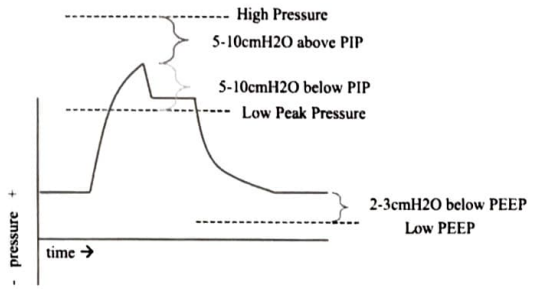
As for the low MV alarm: set that within 10-20% of the MV goal that we calculated when we first started into this process of getting the patient on the vent. If we have a patient breathing in excess of that goal and we want to know if that changes, we just set the low MV goal 10-20% below what they are 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, and low PEEP. Low peak pressure alerts us when the expected peak pressure is lower than we would expect; this could indicate a cuff leak 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. Lastly, low PEEP lets us know if the end expiratory pressure drops below our set PEEP. This could indicate a slow leak or cuff deflation.

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.

[72] Do a solid review of this section, language isn't super precise…

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:[73]



Titrating Up on VTe[74]

As a general rule, we always try to get VTe as high as possible (without causing damage) unless we have a good reason not to. The reason is that by recruiting as much lung as possible, we improve the patient's capacity to compensate for challenges and we can also titrate RR down while maintaining MV (which decreases that %TaDP concept). In addition, this will allow the patient to more easily maintain ventilation after extubation and can improve recovery times. So even though it may not seem like a necessary strategy in the acute settings, if all is well and we can make changes towards this end of higher VTe, we ought to do so for the sake of the patient.

As we work towards higher VTe, either by increasing TV in volume control or by increasing PC in pressure, we need to ensure that we don't cause barotrauma. The simplest way would be to limit Pplat ~~(or PC)~~ to 30cmH20 – the value which we identified earlier as the upper limit to safe ventilation. That strategy, however, ignores the fact that there is a significant amount of variation among individuals. Instead we can titrate up towards (and maybe even beyond) that limit and see how the patient's lungs do in response. If they seem to accommodate that change in pressure without problem, all is well; if they don't, we can dial back.

Before we get into the details on how to make that determination as to whether or not an increase in pressure is safe or not, it is worth mentioning that this strategy doesn't apply to patients with ALI/ARDS (i.e. this is one of those "good reasons not to"). As noted before, there is some evidence now that we want to limit the pressure difference between PEEP and Pplat (termed "driving pressure") in these patients. While we still want to maximize use of the lung in those patients, the approach is different and involves higher PEEP and smaller TVs to accomplish the same thing. There may eventually be comparable recommendations for patients other than those with ALI/ARDS, but for now the data is scarce and only focuses on this particular patient group.

[73] Keep looking for references for these arbitrary numbers…
[74] Review all this also, esp the chart bit at the end

Back to how we go about making sure our increased pressure doesn't get taken too far: in VC we 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 lines 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
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.

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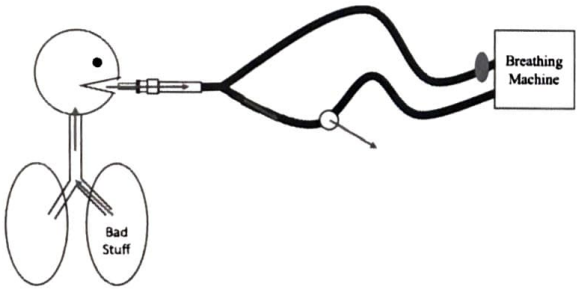
Other Random Things There May Be Questions About

Filters[75]

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:



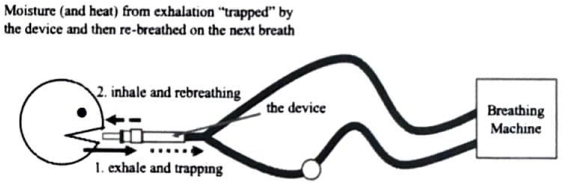
Now we could work around that by placing the filter at the patient's face/ ETT or even on the exhalation side of things, but the face option will increase mechanical dead space (discussed in **Appendix**) and the exhalation side option may not be available with our transport vent. That said, placing 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!

[75] Wilkes, 2011a & 2011b – He gives the most in-depth discussion of both filters and humidifiers (next section)

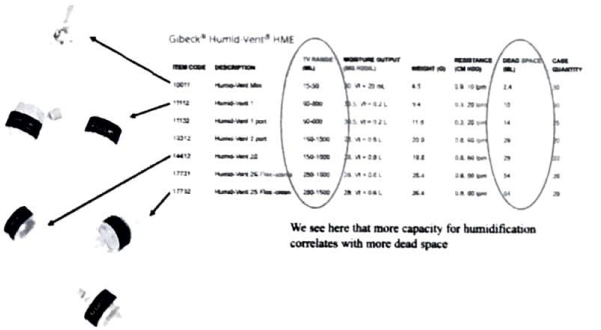
10.1002/14651858.CD004711.pub3

Humidifiers[76]

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 thus heat) from exhaled air and allowing it to be blown back into the patient's airways:



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 reason not to would be small TVs, such as kiddos or ALI/ARDS patients. 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:[77]



[76] Yartsev, 2018b – Excellent discussion of the passive style devices used in the transport setting

[77] Teleflex, 2019 – 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 ☺

Gillis et al, 2017
=> Cochrane
HMEs good w/ pt outcomes, b/c
more research needed

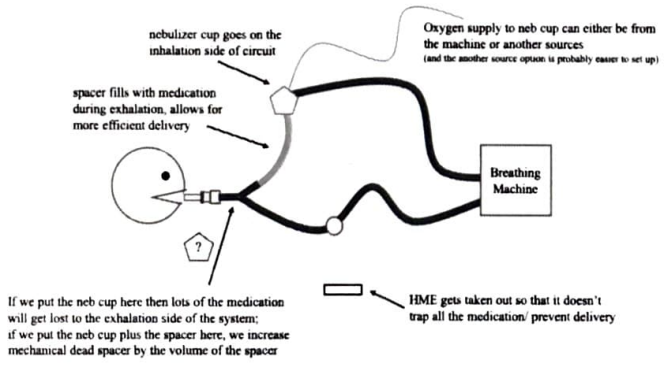
Second good reason not to use an HME would be the concurrent use of nebulized medications.[78] We want those drugs going into the patient, not getting absorbed by the HME. While we could theoretically place the in-line nebulizer between the ETT and the HME, that could also result in decreased medication 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.

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. 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 the HME does provide some filtration of exhaled air and certain devices may even be classified as both filters and HMEs, the filtration here generally isn't quite at the same level of efficacy as an actual filter.

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 demonstrate the important things:



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

That should be clear enough, but just to expand on a few things: 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.

Compliance (and Resistance)[79]

As we mentioned before, 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:

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

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, and increasing VT or PC beyond the capacity of the lungs at that given time.[80]

A related term that we haven't mentioned yet in this manual 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 assumptions, so we're just going to skip on ahead and note it like this:

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

Resistance, therefore, is the limitation to air movement that must be overcome in order for us to arrive at a state in which pressure in (from the machine) equals pressure at the alveoli (plateau pressure). Assuming Pplat remains constant, resistance is basically a function of 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 we previously mentioned were potential causes of increased PIPs also cause an increase in resistance. The one tricky part here is that in PC ventilation we don't have a PIP, so we either need to

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

[80] Look in to this a bit more and refine the list

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Driving Pressure[81]

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[82] alveolar walls and cause damage. 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 as delta pressure.

$$P_{driving} = P_{plat} - PEEP$$

With our ALI/ ARDS patients, we try to limit driving pressure to $10cmH_2O$. Which is generally pretty reasonable, given that we use high PEEPSs and low TVs in these patients anyways. All these concepts combined describe another strategy that may sound familiar – open lung ventilation.[83] The idea here is that we keep the lungs as open 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. For now we have pretty good evidence that limiting driving pressure is a good thing in the ALI/ ARDS population and that such a strategy should probably be avoided in those populations that need better control of ventilation (i.e. $EtCO_2$), but as for the rest it's all up in the air for the time being.

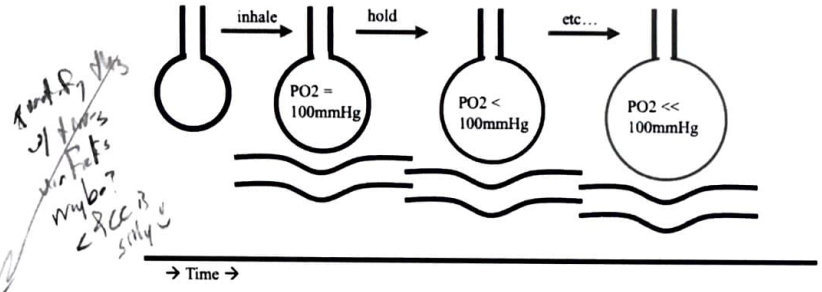
[81] Bugedo & friends, 2017 – Succinct overview of the concept of driving pressure and research done to date (as of a few years ago, at least!)

[82] Delta pressure, as a term, can get confusing though, as "delta" is a generic math term that we can use in lots of settings and to describe many different pressure changes

[83] Review those articles I have bookmarked and cite them

Recruitment Maneuvers[84]

A recruitment maneuver is an intentionally prolonged inspiratory hold that we perform in order to inflate alveoli. 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 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:[85]



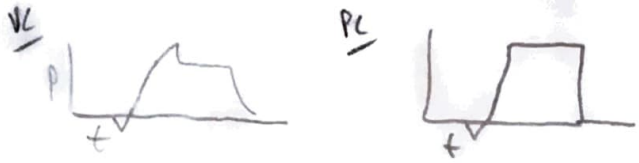
A recruitment maneuver can be used to gain recruitment in any patient group but has been most studied with ARDS patients. As far as the specifics about how long to do the maneuver for, how often to do it, if we should augment the pressure above what inspiratory pressure we are already at, etc. – answers to all those questions vary significantly and we will simply point everyone in the direction of some good sources of information and let fate take it from there. Just know that in the hypoxic patient, performing a recruitment maneuver or a series of them may help overcome alveolar inflation pressure which results in better diffusion of oxygen.

[84] See if I can get some concrete suggestions here, seems silly to even mention it and then not given guidance

[85] If we were going for a continuous-supply-of-fresh-oxygen strategy, we'd basically have a HFNC situation on our hands – but that's a whole different idea and we'll leave it alone for another day

Triggers

Triggers are the thresholds by which the machine knows when a patient is trying to breath on his or her own. Most commonly these are dialed in a pressure changes that must be overcome.



Prone Ventilation

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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 impact 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 and that is also generic enough to apply to different machines. 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 cognitive demand when things get busy on scene or in transport.

A few other things: there is a word version of this two-page algorithm on the website if you want to make changes to it in order to match a specific machine or set of guidelines/ protocols. And then there's an annotated version (just after this blank one) to reiterate some of the concepts and to link readers back to sections of this manual where things were discussed the first time around[86]. There's also a bit of extra information about a few straggler ideas that we didn't discuss in previous sections.

[86] Do this after everything else is done

1. **Prep Stuff**
   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

2. **Determine Specific Settings to Dial in to Machine**
   a. *Patient Already on Vent*
      i. Assess the Three Big Things: oxygenation, ventilation, & comfort
      ii. If acceptable, mirror setting
      iii. If unacceptable, either:
         1. Adjust to fix it
         2. Start from scratch (below)

   b. *Patient Not Yet on Vent*
      i. Determine Mode & Control (leave at default settings/ choose appropriate profile unless there is good reason to do otherwise)
      ii. Dial in desired TV (or PC at 15-20cmH2O)
      iii. Adjust rate to 17 for adults, within suggested range for pediatrics
      iv. Leave all other settings at whatever the machine defaults to, unless:

Consideration	Evidence (plus clinical impression/ diagnosis)	Intervention
Bronchospasm	Wheezes on auscultation "Shark fin" EtCO2 waveform	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	Adult: SBP <90 Pedi: SBP < 70 + 2 x years (or even a potential for these values)	Limit PEEP Increase TV to 10ml/kg IBW (or max Pplat) and decrease RR to maintain MV
Acidosis	Low pH Kussmaul's Respirations EtCO2 >45	Use high end of TV (goal): 8ml/kg IBW Increase RR: pre-intubation rate, to get prior/goal $EtCO_2$, or double normal value
*with concurrent Hypotension and Acidosis, defer to Acidosis initial settings		
Acute Lung Injury/ ARDS	Bilateral infiltrates on CXR PaO_2/FiO_2 < 300	Decrease TV to 4ml/kg IBW and increase RR towards MV goal (maybe consider permissive hypercapnia) Higher PEEP

3. **Initiate Ventilation**
   *consider clamping ETT on transfer to vent if concerned with recruitment

4. **Initial Parameters to Consider**

Parameter	Normal	Intervention
SpO_2	93-99%	*Low*: consider position & suction, increase FiO_2, then increase PEEP (1-2cm incrementally); consider pathophysiology/ medications; increase I-time/ invert I:E *High*: decrease FiO_2 (unless contraindicated, i.e. pregnancy, anemia, severe hemorrhage, etc.)
$EtCO_2$	35-45mmHg (30-35 with TBI)	*Any abnormal value*: consider etiology/ patient compensation for acid-base imbalance *High*: increase TV (max 10ml/kg IBW, monitor Pplat), then consider increase in RR *Low*: consider perfusion status, decrease RR (monitor MV), then consider decrease in TV
Comfort	Ramsay ≤5 or ANPS at provider discretion	Analgesia and sedation (Pain, Agitation & Nausea) Consider settings: MV, I-time Also consider tachypnea/ overbreathing
PIP[87]	<35mmH_2O	Consider secretions and/ or Bronchospasm Check circuit for kinks, consider patient position Decrease TV
Pplat	<30mmH_2O	Consider pneumothorax and/ or pulmonary edema Decrease TV
AutoPEEP	none	Increase I:E Consider disconnecting circuit to allow exhalation Consider triggers: accidental? if not, increase/ change
MV	100ml/kg/min (200 with acidosis)	*Low*: increase TV and/ or RR *High*: consider patient comfort, monitor $EtCO_2$, decrease TV and/ or RR, consider SIMV

5. **Ongoing Management of Specific Considerations**

Consideration	Strategy
Bronchospasm	Set up in-line neb treatment (away from wye on inhalation side, remove HME) Consider Ketamine for analgesia/ sedation $EtCO_2$ may be elevated at baseline
Hypotension	Use caution with PEEP Consider fluids and/ or pressors early
Acidosis	Consider and adjust to increased MV goal of 200ml/kg/min Realize that $EtCO_2$ may be out of reference range
Acute Lung Injury/ ARDS	Consider recruitment maneuvers Higher PEEP may be needed Inverted I:E may help, but will likely lead to discomfort

[87] Match this and Pplat sxn to that alhorithm thing we had a few sxns back

Suggestions for Further Reading

Just some suggestions for further study 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.

Audio
EmCrit Dominating the Vent Series
FlightbridgeED Vent Series

Video, Vent Specific
Strong Medicine Series on Mechanical Ventilation
Thoracic.org videos

Video, Physiology
Ninja Nerd Science, section on Respiratory
Kahn?

Text, Web-Based
Deranged Physiology, section on Respiratory
Life in the Fast Lane – get a list of specifics here

Text, Books to Buy
Look into this (Swearingen and Bauer maybe?)

Others: Airway Jedi?

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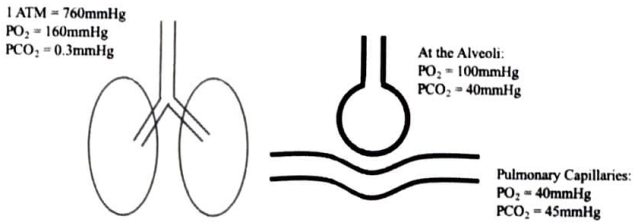
Appendix

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:[88]

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

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[89]

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

But back to our original equation:
$$PAO_2 = FiO_2(P_{atm} - P_{H2O}) - (PaCO_2/RespQ)$$
$$PAO_2 = 0.21(760 - 47) - (40/0.8)$$
$$PAO_2 \approx 100mmHg$$

[88] Yartsev, 2018a – 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
[89] 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

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

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[90] (room air):
$$PAO_2 = FiO_2(760 (+4) - 47) - 50$$
$$PAO_2 \approx 101mmHg$$

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

We can also use this equation to manage patients across elevation changes. This gets pretty nerdy, but it's kind of cool to see. Imagine we have a patient going from

maybe leave out

[90] Just a friendly reminder that 5cmH_2O is roughly 4mmHg

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A-a Gradient[91]

The primary utility in knowing PAO_2 is that we can compare it to PaO_2 (partial pressure of arterial oxygen) to see how well they match. The theory is that if the system is working well, the PaO_2 should equal what we calculate to be the PAO_2. If we have a gap between the two, we can assume some sort of issue, such as alveolar dead space (V/Q mismatch and shunt), diffusion problems, or increased oxygen extraction. Just know that sometimes these concepts overlap and more than one may be present at a given time.

We calculate an A-a Gradient (also referred to as "A-a Gap") as so:

$$\text{A-a Gradient} = PAO_2 - PaO_2$$

We said already that normal healthy folks are assumed to have no alveolar dead space, but that's not entirely accurate – our bodies aren't perfect and vascular beds are always in a state of flux, so a small degree of gap is baseline and this value actually increases somewhat by age via this rough estimate:[92]

$$\text{A-a Gradient should be} < (\text{age in years} \div 4) \div 4$$

Now we would typically utilize this gap to help direct treatment in a hypoxic state – if our patient is oxygenating well, there's probably not much value in working all of this out. Hypoxia with a normal A-a Gap (in the vented patient) would mean that we just dialed in or calculated something wrong: FiO_2 or MV are too low, so the moral here is to start simple and address the basics before getting the barometer and calculator out! Moving forward, hypoxia with a true A-a Gap can be caused by different etiologies like we said above. We've already talked about V/Q mismatch, shunt and diffusion issues (across the alveolar membrane) previously and mentioned that fixes for these include keeping the head of bed up, oxygenating well to address HPV, ensuring perfusion, etc. – all the things we do already.[93]

Another reason why we might see a larger than normal A-a Gradient is increased oxygen extraction.[94] This is a fancy way of saying that the body is using up oxygen (at the tissues and organs) faster than we can onload it into the blood at the pulmonary capillaries. Conditions in which this might occur would be sepsis, burns, thyroid storm, shivering, etc. – all the things that increase metabolic activity a whole bunch. Fix for these things (on the vent) would be to increase oxygenation via higher FiO_2 and then PEEP. There may also specific interventions for whatever pathology is going on: antipyretics to control fever, warming or paralysis to prevent shivering, etc.

So to sum up all the things on A-a Gradient: this gap tells us when we have alveolar dead space, diffusion problems, or increased us of oxygen. While it may be fun to calculate an A-a Gap in transport, the utility is knowing the actual number doesn't help us much in our setting and it may be best to make assumptions about the nature of a potential gap based on patient presentation. We should already be verifying that settings are at calculated goals, that oxygenation and ventilation are adequate, and that steps are taken to mitigate potential alveolar dead space. If we've done all of those things, still have an issue, and then have extra time on

[91] Doesn't Ninja Nerd Science have a video on this? Or the Strong Medicine one – maybe one of things cites that equation, not sure where he came from

[92] Nickson, 2019a – Has a very brief outline of A-a Gradient that both provides us with this formula and leads us down our discussion of oxygen extraction

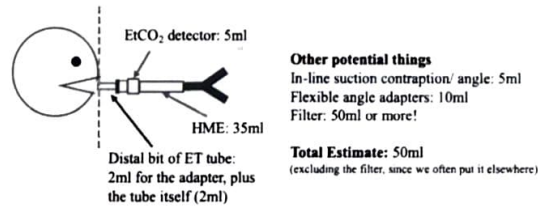
[93] Sarkar & friends, 2017 – much more detail in to how we identify causes of hypoxia using not only the A-a Gap, but also other concepts and calculations; provides a lot of detail that also re-explains some of the concepts we discussed way back at the beginning in the section on **Other Important Concepts**

[94] Nickson, 2019c – You can see a trend here that we really like LitFL and the way he presents things in a short, sweet sort of way – definitely worth looking at his page for answers to any other questions that come up

our hands, then it may be worth looking in to. Barring all of those factors, just keep it simple and manage the basics.

Mechanical Dead Space Math

In order to quantify the effect of mechanical dead space, we first need to know how much volume each of the extra components takes up. Now 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:



Now that we have a volume, let's see how this mechanical dead space impacts both oxygen and carbon dioxide. The effect on oxygenation is essentially negligible, but we want to demonstrate why that is instead of just telling you so:

1. We already know that the normal PAO_2 at baseline is about 100mmHg - this is a function of a TV going in at an FiO_2 of 0.21, which reflects the percentage of oxygen in ambient air. Now let's see what happens if 50ml (mechanical dead space) of that TV is exhaled air and not ambient air.

~~The effect on carbon dioxide, however, is potentially problematic~~.

see p49

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More on $EtCO_2$

Also use this space to describe EtCO2 waveform, esp w/ bronchospasm

see p49

- start @ how CO_2 ⇒ cells & factors that ⇅ this
- unloading to Hgb & off (Bohr & OxyHgb curve) → b/c no vent discussion is complete w/o it

- waveform
  - normal
  - broncho
  - ↓ resp. effort
- value
  - ↑
  - ↓

& then refer to someone else for full cardio-bronc dive

CO_2 dead space

assume 70kg

70kg × 6 ml / 1kg = 420 ml

420 × 0.33 ≈ 140 ml anatomic ds

420 × 0.33 ≈ 140 ml anatomic ds / 420 x 0.33 = 140 ml dead space

+ 50 ml

∴ $\dot{V}CO_2$ = 1.22 L/min @ 70kg

or

↗ reverse bags too (str or SIMV)

Vent Waveforms

ATS, 2016 (flowmic.org)

things to look for

- high exp. flow rates (lung overdistention r/t ↑TV, mainstem, etc)
- stress index <1 / downward convexity → potential recruitment
- auto-peep in real time - exp. flow doesn't return to baseline (& ↑ pressures)
- dystrxn r/t low flow state + autopeep
- inspiratory effort that doesn't catch → fatigue, ↑ agitation
- 2x triggering
- secretions

#2 r/t MDS (CO_2)

5% CO_2 in expired air

5% $\left(\frac{1}{10}\ 5\%\right) + \left(\frac{9}{10}\ 0\%\right)$

$\frac{Vd}{Vt}$ ratio, more so w/ kids

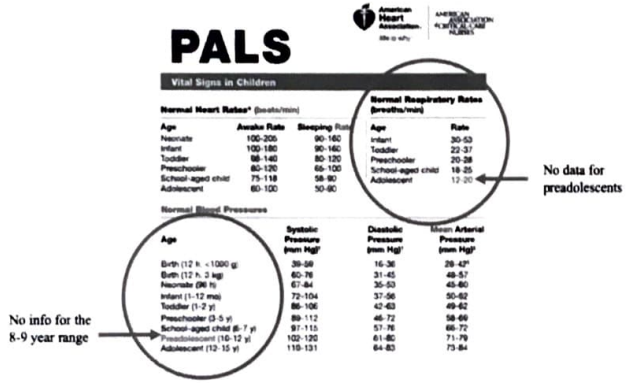
∴ more MV to maintain normal CO_2

More on 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:

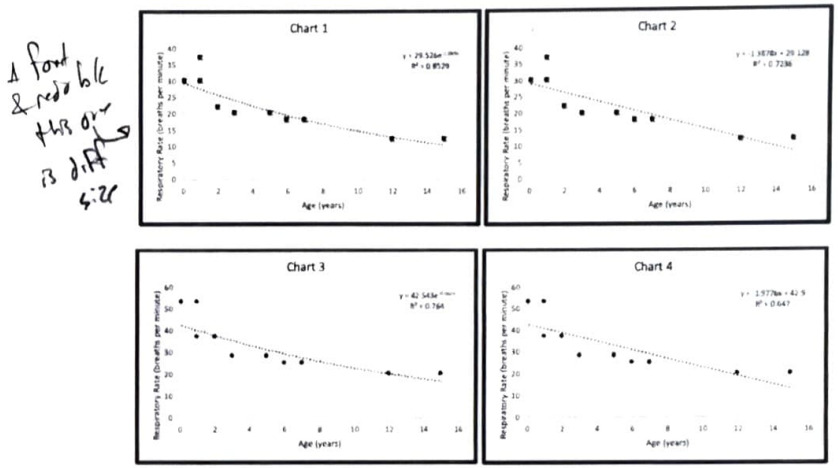
1. Make assumptions:
   a. "Normal Respiratory Rates" as outlined by PALS are good enough to work with[95]
   b. Normal RR range for an adult is 12-20 (cited in many, many sources)
      c. A normal I:E at rest/ spontaneous respiration is 1:2[96], but we often work with a ratio of 1:3 for vented patients

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



[95] And while there are gaps in their data, we can fill that in - so no worries!
[96] Cite this, maybe made an assumption

b. Plot the existing data using both high and low ends of RR by age, make charts, then add lines of best fit[97]



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

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[98]
Preadolescent	10 – 12	14 – 23
Adolescent	12 – 15	12 – 20
Adult	16 and up	12 – 20

[97] Redo these charts so that the sizes match, also change to TNR font
[98] Range here was calculated to be 17-26 (see Excel Spreadsheet), 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

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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

5. Compare the final chart to literature:

- Sept detail: Loring +, 2015
- find a video?
- open stax 22.3
- Grizzo +, 2017
  ↳ app. to clinical practice & techniques

Transpulmonary Pressure

(redo that image from p10 to start)

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A Personal Reflection

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Pearsall +, 2014
When does apparatus dead space matter?

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$VCO_2 = VE \times FeCO_2 - VI \times FiCO_2$

$= MV\ (FeCO_2 - FiCO_2)$

↳ assume same ↳ basically zero

$= MV \times 4\%$

$= 100 ml/kg/min \times .04$

$= 4 ml/kg/min$

$\times$ ~~70~~ 65 kg pt = ~~280~~ 260

great above; & still figure out how much ↑ w/ exertion / disease

⇒ Best, 2014
How to calculate VO_2 and VCO_2

Summarize it

1. $FaCO_2 = VCO_2 / VA$
2. $VCO_2 = 4 ml/kg/min$ — since we used that earlier, don't Δ to 200
3. solve for 65 kg pt
   a. w/ apparatus dead space
   b. w/o it
4. how we'd compensate for it (i.e. how ↑ in RR)
5. discuss consequences of that

$FaCO_2 = VCO_2 / VA$

$VA = RR (V_t - V_d)$

$PaCO_2 = FaCO_2 (760 - 47)$ (ø ambient, ø H_2O)

$FaCO_2 = PaCO_2 / 713$

$\frac{PaCO_2}{713} = \frac{VCO_2}{RR(V_t - V_d)}$

$(RR (V_t - V_d))\ PaCO_2 = VCO_2 \times 713$

$RR (V_t - V_d) = \frac{VCO_2 \times 713}{PaCO_2}$

$RR = \frac{VCO_2 \times 713}{PaCO_2 (V_t - V_d)}$

VCO_2 @ rest is 200 ml/min (assumes prdn & output NOK)*

$RR = \frac{200 \times 713}{40 (420 - 190)}$ + appdts

≈ 16

$RR = \frac{200 \times 713}{40 \times (420 - 140)}$ ø appdts

≈ 13

this shows a higher RR needed to maintain CO_2 w/ apparatus dead space, w/c still want to show effect on $EtCO_2$ ($PaCO_2$)

$\frac{PaCO_2}{713} = VCO_2 / VA$ @ 17/min

$PaCO_2 = \frac{VCO_2 \times 713}{VA}$

$= \frac{200 \times 713}{17 (420 - 190)}$

≈ 36

@ 280 ≈ 51

$= \frac{200 \times 713}{17 (420 - 140)}$

≈ 30

@ 280 ≈ 42

Maybe this # is an underestimate? more needed in mechanical ventilation?