Rykerr Medical's

Vent Management Guide

for Invasive Mechanical Ventilation in Transport

Version 1
May 2020

RYKERR MEDICAL LLC

to do once book is done & site is up:

- update server to these pages
  - Wingfield's clips x2
  - Criticalthinkingwrd & pedistat ✓ ✓
  - Bacon's? never heard back...

/criticalthinkingwrd

/pedistat

- which pdfs are free online?
  - Nagler & Chiefetz, 2019

[pull the stars down - maxwell stern]

☒ inventory refs to Gas Laws
- Boyle p114, 36
- Charles p43,
- Fick p64
- Graham p67
- Dalton p6
- ✓ Henry p63

to do

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☒ look @ imgs for red squiggles

☒ review all footnotes

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☐ re-format all QR codes

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☐ update ToC

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☐ red squiggles

☐ read it all out loud

☐ list refs w/ RM page (see above)

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RMc

~~PEEP~~ ~~APRV~~ ~~ARDS~~ ~~TV~~ ~~Pplat~~ ~~HQ~~

~~FiO2~~ ~~I time~~ ~~ETT~~ 4P ~~PPV~~ ~~CO~~ ~~VTe~~

~~O2~~ ~~PO2~~ ~~mmHg~~ ~~PC~~ ~~cmH2O~~ ~~S~~

~~VC~~ ~~SpO2~~

+ ~~Cst~~

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ACRI Legend, Take 2 :)

triggers

~~cmH2O~~ PEEP LPM

overbreathing

~~EtCO2~~ ~~RR~~ ~~MV~~ ~~pH~~ ~~PaCO2~~ ~~TV~~ AC ~~SIMV~~

~~IMV~~ ~~PEEP~~ ~~VC~~ ~~Pplat~~ ~~FE~~ ~~I time~~

DPRC

~~4P~~ ~~Pplat~~ ~~PEEP~~ ~~cmH2O~~ ~~TV~~ ~~ARDS~~ ~~CO2~~

~~AV~~ ~~VTe~~ ~~Vt~~ PIP ~~LPM~~ ~~ETT~~ ~~PC~~

~~MVe~~ ~~VC~~

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EMT – emergency medical technician; **NM** – New Mexico; **OK** – alright; **QR** – quick reference; **SpO₂** – pulse oximetry;

A Personal Intro

There are a lot of good reasons why I thought it'd be good to put together a primer on vent management, but the main one is that my first vent experience was a near-disaster and I'd like to share what I've learned since then so that others can avoid what I had to go through. I also think there's some room for diversity in how we, as an industry, present material to each other and move forward with our understanding of complicated things. And lastly, I hope is that this interactive style of writing can be of help to some folks and maybe inspire others to do the same and build on the whole idea. Collaborating together to improve our skills and holding one another accountable to live that out through clinical practice means better care for more people.

But to start with the awful beginning story: I was brand new to an ambulance service in New Mexico, having moved from Pittsburgh about two years after I first got my medic. I was still green but felt like I had gotten a lot of experience back in the city and was maybe a tad over-confident. Anyway, I started at this rural service in mid-November and the call that prompted my journey down this path of ventilator self-education was the day after Thanksgiving. I had basically just arrived in NM, barely gotten settled into the second EMS service I had ever been given medical control at, and was turned loose to practice in the field.

Things were different for sure. Five- and ten-minute transport times had been replaced by ones that sometimes took hours. With a five thousand 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. Protocols and capabilities were a lot more lenient and included vents, surgical crics, hiking 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. And 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 – 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 stayed low, the alarms were yelling at me, the EMT was like "come on, 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.

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BLS – basic life support; **BVM** – bag valve mask; **DVD** – digital versatile disk; **$EtCO_2$** – end-tidal carbon dioxide; **EMS** emergency medical services

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. 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.... We did, however and finally, arrive to the big city in a presentable state – perfusion was good, sats weren't embarrassing, the patient appeared comfortable enough and was making some effort to breathe on his own, and that machine had stopped yelling alarms at me.

With the patient handoff complete, I returned back to small town New Mexico late on that day after Thanksgiving, year 2012, and decided then and there that I was never, ever, going to be in that situation again. My initial study list looked something like this:

<u>The Ventilator Book by William Owens</u>

<u>The LTV1200 Product Manual</u> (and the DVDs)

EMCrit Dominating the Vent Series
<u>Part 1</u>, <u>Part 2</u>

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 manual you can just scan the QR codes for any of the references to access them (if available for free) or to see where you can purchase them (if they want your money); if you have an electronic version, just click the links. And if you have a version where the links don't work because it isn't legit, that's cool too: go <u>here</u> to get it all free and official.[1]

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 :)

[1] To visit us at <u>rykerrmedical.com</u> or follow the QR code on the cover to link to the website

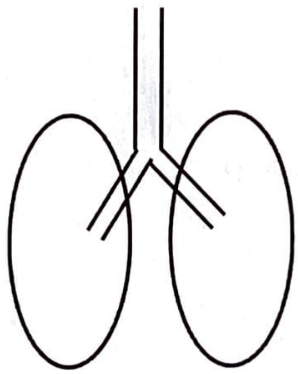
O_2 – oxygen; **OK** – alright; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PCO_2** – partial pressure of carbon dioxide; **PO_2** – partial pressure of oxygen

Some Very Basic Physiology

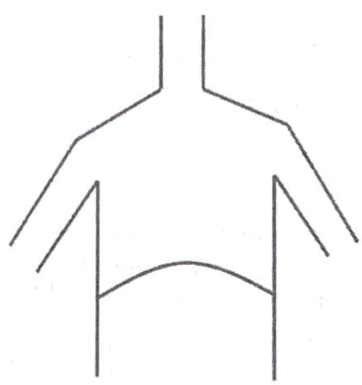
As a disclaimer, the stuff outlined here is intended only to give a foundation for the fundamental concepts of vent management. One recommendation for looking into the details beyond this (much of which comes up later when we talk about specific conditions) is a good, solid, heavy Anatomy and Physiology textbook or any of the references noted.[2]

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:



[2] Also see __Suggestions for Further Study__ at the end

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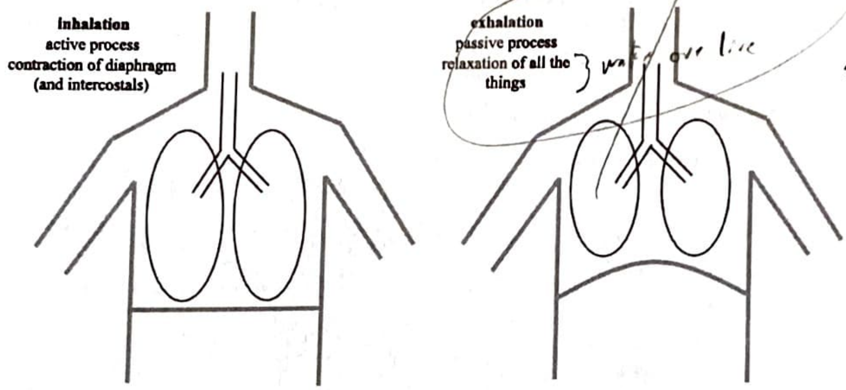
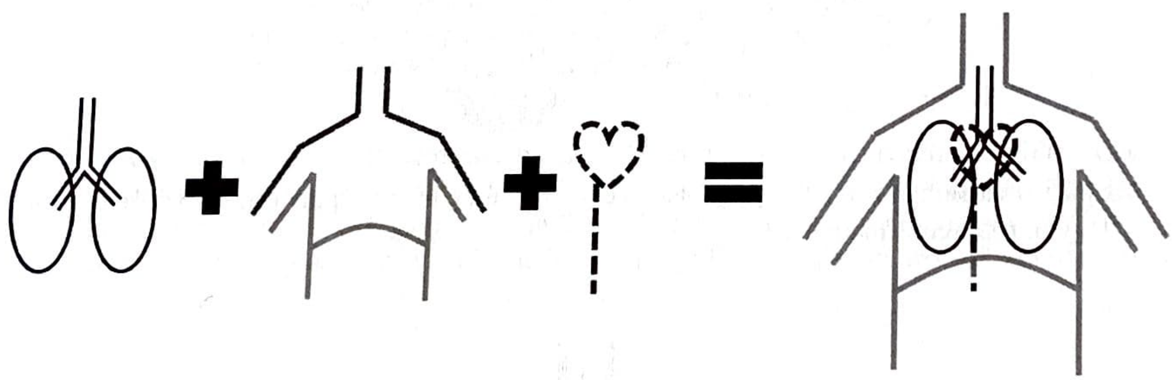
ARDS – acute respiratory distress syndrome; **AOK** – all good; **BVM** – bag valve mask; **cmH₂O** – centimeters of water; **CO** – cardiac output; **L** – liter; **mmHg** – millimeters of mercury

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:[3]



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.[4] Because this air movement occurs via negative pressure, blood return via the inferior vena cava is facilitated by normal ventilation – this will be important when we move on to talk about positive-pressure ventilation in just a minute.[5]

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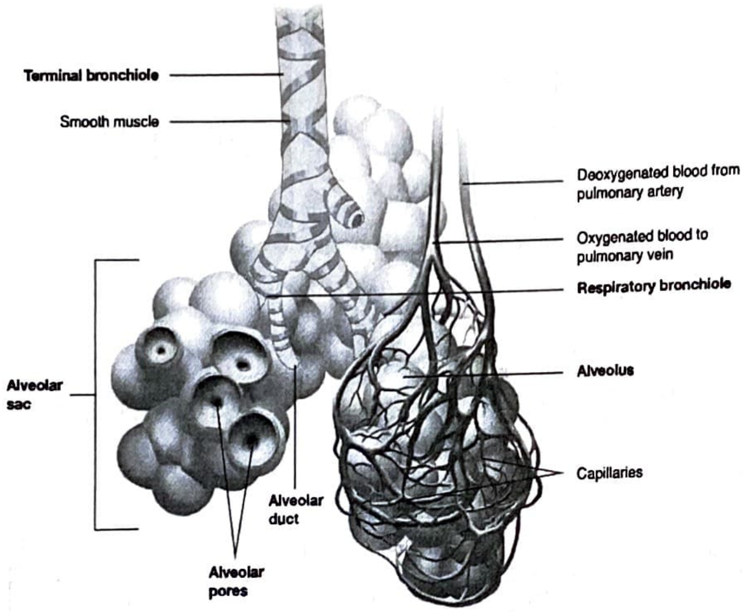
[3] ~~This assumption mostly holds true for our need in the transport setting, so we won't take it much further than that here~~

[4] Hasudungan, 2014 – To review the physiology in a bit more detail, refer to this video

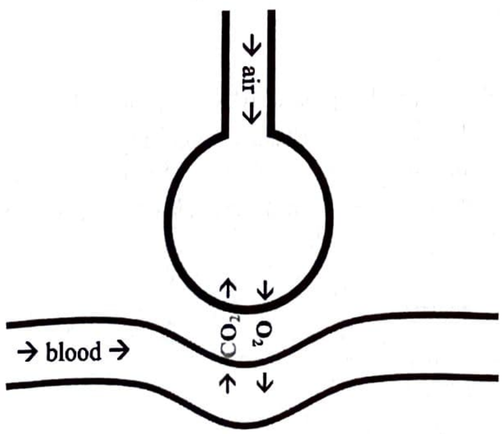
[5] Azizov, 2017 – Video that explains ~~how this~~ mechanism works

O_2 – oxygen; **OK** – alright; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PCO_2** – partial pressure of carbon dioxide; **PO_2** – partial pressure of oxygen

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 pulmonary gas exchange where oxygen (O_2) goes into the blood and carbon dioxide (CO_2) goes out:[6]



A simplified version of a single alveoli with a corresponding blood supply can help us understand the pathophysiology of different situations:



[6] Betts & friends, 2013 (image) – This image is from a free online textbook that we'll reference a few more times in the pages to come

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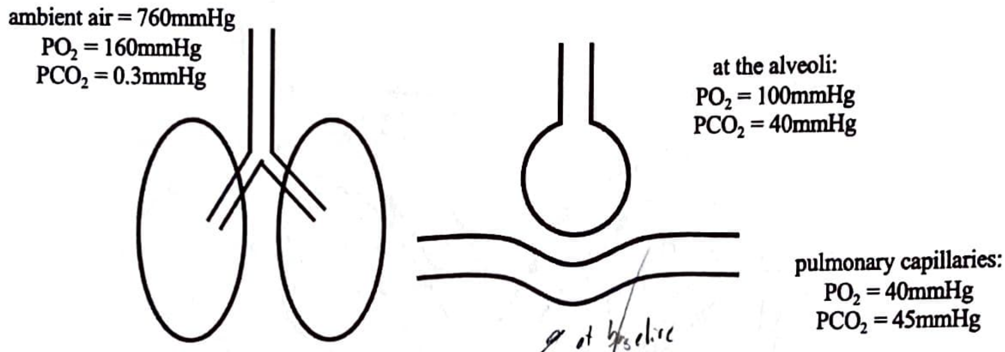
ARDS – acute respiratory distress syndrome; **AOK** – all good; **BVM** – bag valve mask; **cmH₂O** – centimeters of water; **CO** – cardiac output; **L** – liter; **mmHg** – millimeters of mercury

Next, let's add some numbers to that graphic of a single alveoli and its blood supply.[7] Note that in real life blood is continually moving past the alveoli and gases are constantly shifting to reach equilibrium, so that as CO_2 is offloaded and O_2 is onloaded, there is a new supply of blood and a reset of the gradients across that membrane. Plus the diffusion of gasses from alveoli to pulmonary capillaries happens very quickly. This means we generally aren't worried about this timeframe (i.e. how fast these gasses diffuse) being the limiting factor in this process:[8]



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 the two gasses: O_2 has a pressure difference of about 60mmHg, CO_2 has one of just 5mmHg. While this may seem, at first glance, to put the body at risk of some sort of imbalance, CO_2 moves more easily through liquids, and thus the membrane between capillary and alveoli, (roughly twenty times so) and the net result is that O_2 and CO_2 exchange at about the same rate.

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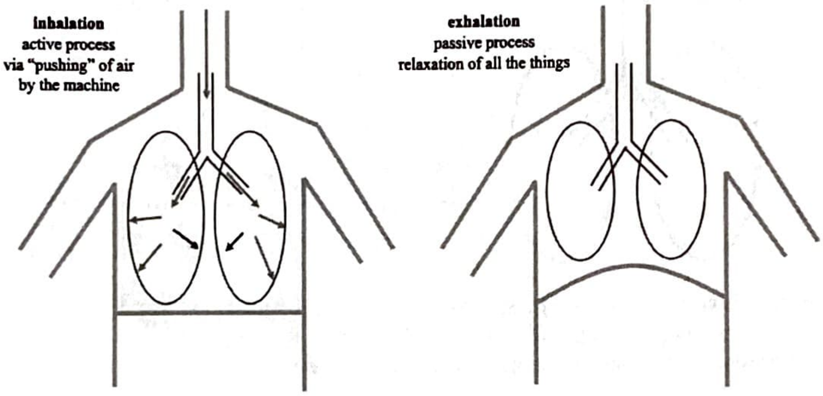
[7] Betts & friends, 2013 – They both discuss these numbers in the context of Dalton's Law and list all these values except for PO_2 at the alveoli; that one is cited as 104mmHg in their text, but we calculated it out in the **Appendix** and use our calculated value to maintain consistency throughout this text

[8] Speller, 2018 – Outlines how both O_2 and CO_2 diffuse in the pulmonary system in the context of gas laws; do note, however, that certain states can slow this process down (and we'll get to those later on!)

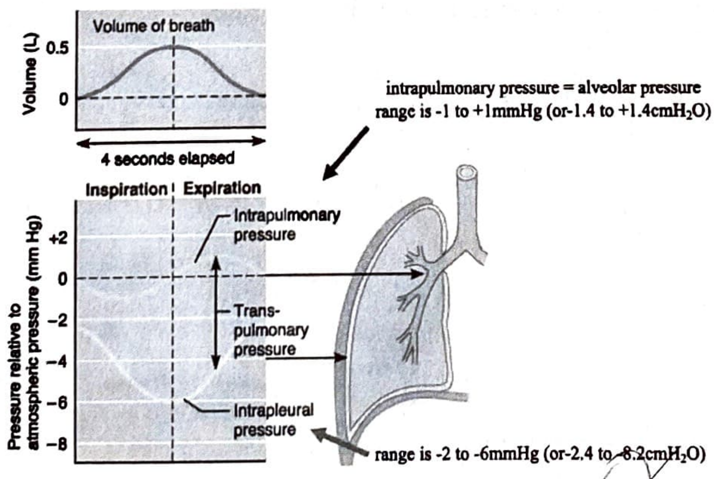
O_2 – oxygen; **OK** – alright; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PCO_2** – partial pressure of carbon dioxide; **PO_2** – partial pressure of oxygen

How is Positive-Pressure Ventilation Different?

Now we need to consider what happens when we bypass the whole negative-pressure mechanism for ventilation and instead opt for a positive-pressure approach.[9] Let's start at the top with the basic sketch of airways and lungs superimposed on the chest wall and diaphragm. When we ventilate by positive-pressure ventilation (PPV) we have to physically displace the diaphragm and chest wall while simultaneously pushing air into the system – this requires a lot more pressure than we needed for that negative-pressure, spontaneous mechanism:



We will get to airway pressures and limits for them later on, but a normal **Plateau Pressure** (which reflects the average alveolar pressure in positive-pressure ventilation) is in the range of 15-25cmH₂0; compare this to the pressures represented in the following illustration:[10]

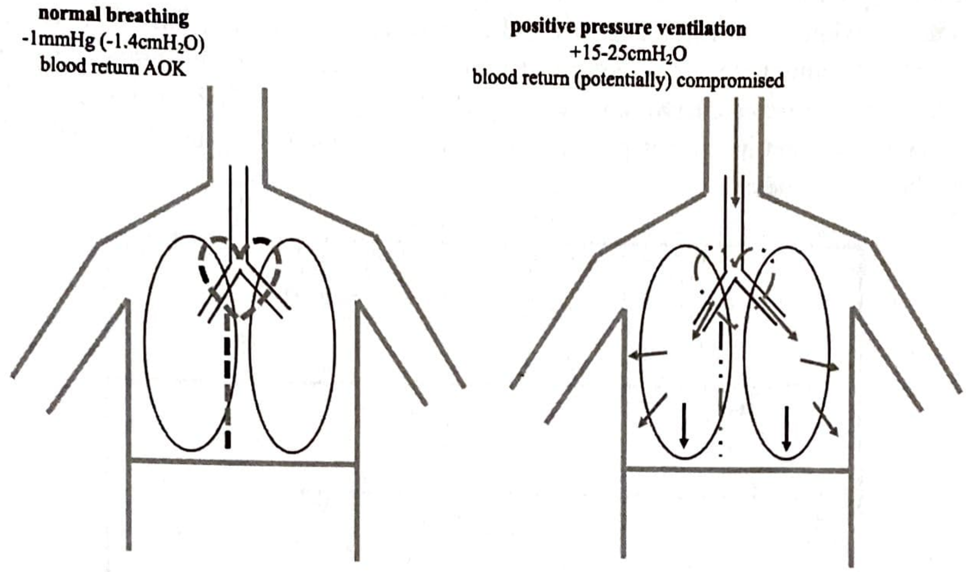


[9] This assumes that the patient is not contributing to this effort of breathing; to say it another way, this description is accurate for the patient who is not making any respiratory effort or is out of synch with mechanical efforts – in reality we can synch patient effort to machine effort to minimize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

[10] Kahathuduwa, 2013 (image) – Two things: we'll talk about the mmHg and cmH₂O conundrum at the end of the next section (in **Measuring Pressures**); alveolar pressure is the most relevant to our discussion for now, the concepts of transpulmonary pressure and intrapleural pressures are deferred here

ARDS – acute respiratory distress syndrome; **AOK** – all good; **BVM** – bag valve mask; **cmH$_2$O** – centimeters of water; **CO** – cardiac output; **L** – liter; **mmHg** – millimeters of mercury

The biggest impact of that increased intrathoracic pressure is the effect it can have on cardiac output (CO). Increased intrathoracic pressure can decrease blood return to the heart via pressure on the vena cava, resulting in decreased preload and, therefore, less output.[11] Let's represent it this way:



Other negative sequalae of PPV (which may still occur even if we have all the settings dialed in right!) would be patient discomfort, muscle fatigue and/ or weakening,[12] and physiologic changes to other body systems.[13] And then if we have things dialed in wrong on the machine or don't ventilate appropriately based on patient presentation, we can also cause things like direct injury to the 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.

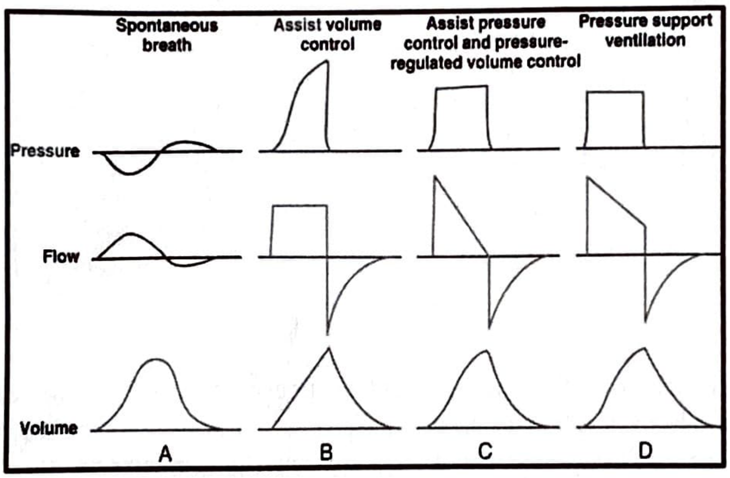
[11] Strong, 2013; Mahmood & Pinsky, 2018 – Both this video and the article explain in more detail on how PPV (and particularly **Positive End-Expiratory Pressure**, discussed later) can affect CO, especially with concurrent hypovolemia; while it isn't always true that PPV decreases CO (sometimes the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

[12] 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 breathe; 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

[13] Yartsev, 2019 – In fact, navigate to the 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

O_2 – oxygen; **OK** – alright; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PCO_2** – partial pressure of carbon dioxide; **PO_2** – partial pressure of oxygen

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 and 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 noteworthy point is that the graphic representations of the types of breaths (i.e. each column of the three towards the right) are each slightly different – sometimes one mode will be more comfortable for a certain patient in spite of trying to do all the other things we know how to do, simply because how that patient's body responds:[14]



In an effort not to discourage anyone from ever putting a patient on a vent, there are advantages to PPV and mechanical ventilation. Most obvious of these is that it allows us to breathe for a patient in a relatively simple way when that patient is unable to do so on his or her own. More specifically, mechanical ventilation allows us to control and direct recovery with specific conditions and diseases (such as acidosis, asthma, and ARDS – all of which we will discuss later on). Positive pressure can help move O_2 into the bloodstream more easily, managing ventilation can help that O_2 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 does have its place in the cosmos.[15]

[14] Fuller & friends, 2014 (image) – This assessment of what the body wants in terms of smooth waveforms and avoidance of harsh changes in amplitude is a subjective concept – it seems to make intuitive sense, but there may not be a good way to verify the idea
[15] Hill, 2019 – And if you need convincing that mechanical ventilation is preferred to simply using a BVM, take a look at this discussion of a recent paper

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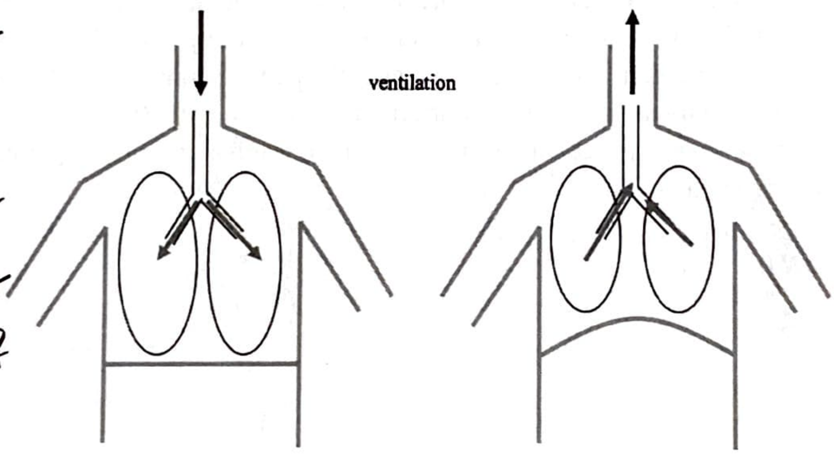
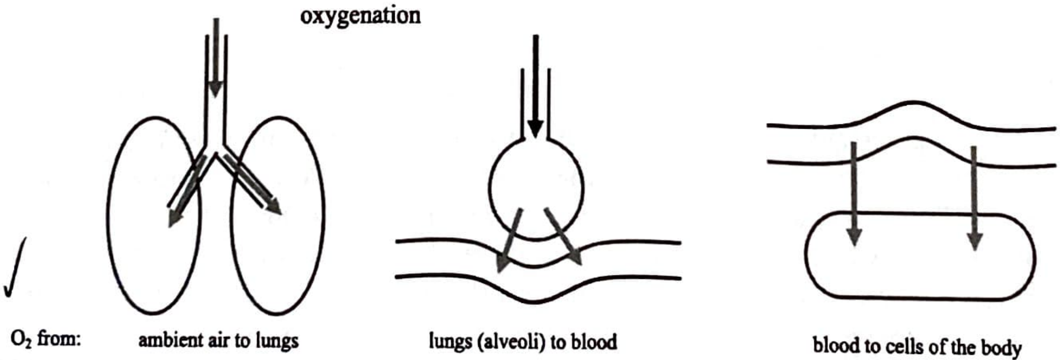
ARDS – acute respiratory distress syndrome; **ATM** – atmosphere; **cmH$_2$O** – centimeters of water; **CO_2** – carbon dioxide; **EtCO_2** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HME** – heat & moisture exchanger
HPV – hypoxic pulmonary vasoconstriction

Other Important Concepts

Terms to Describe Breathing

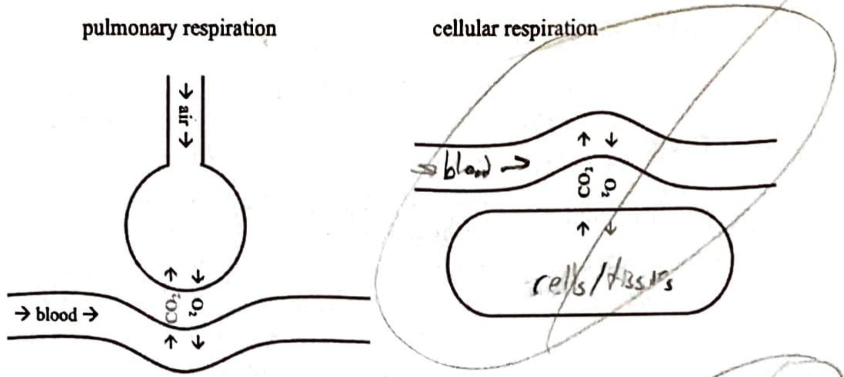
Just to differentiate the words that collectively describe breathing, let's chat about a few terms.[16] **Ventilation** refers to the gross movement of air as the body breathes in and out. **Oxygenation** refers to the transition of O_2 from the air outside of the body, through the respiratory and circulatory systems, and to the capillaries where it can be picked up by tissues for use. Lastly is respiration, which has two specific types. Pulmonary respiration refers to the exchange of CO_2 and O_2 in the alveoli of the lungs; cellular respiration refers to a comparable gas exchange at the tissues. To visualize it all, here are a few images:





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

IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; **O_2** – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; **V_D** – dead space; **V/Q** – ventilation/ perfusion



There is some overlap between oxygenation and pulmonary respiration in ~~this context~~, but it helps to separate these ideas.[17] When managing the vent, we are most focused on the processes of ventilation and oxygenation. While respiration (in both forms) is very important, our ability to manipulate it isn't as straightforward as it is with ventilation and oxygenation; also, the part of respiration that we can impact, the pulmonary part, is covered in a roundabout way by our actions to address oxygenation. We will come back around to this idea in a bit when we talk about how to control both ventilation and oxygenation by changing different parameters on the ventilator.

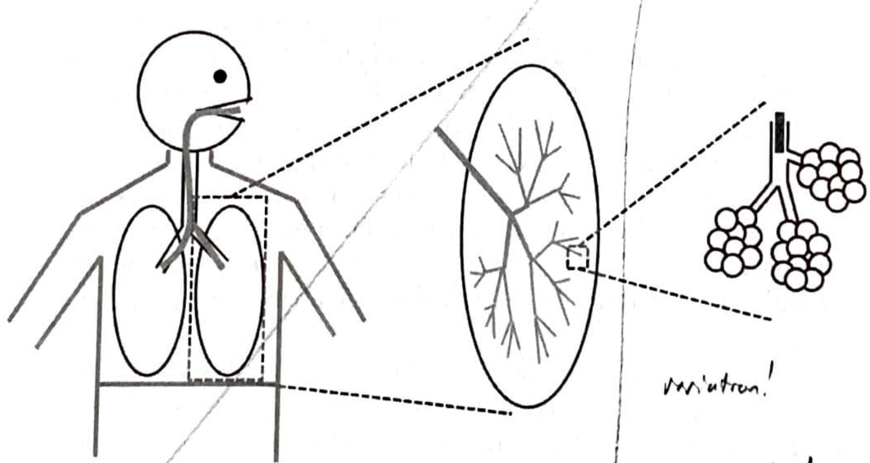
[17] Hasudungan, 2018 – This video reviews both of these concepts, but also includes a general recap of respiratory system physiology and the oxygen-hemoglobin dissociation curve; we will reference it again when we discuss **Oxygenation**

ARDS – acute respiratory distress syndrome; **ATM** – atmosphere; **cmH$_2$O** – centimeters of water; **CO$_2$** – carbon dioxide; **EtCO$_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HME** – heat & moisture exchanger **HPV** – hypoxic pulmonary vasoconstriction

Dead Space[18]

Dead space can be an intimidating concept when it comes to vent management and we are going to try to both simplify it and identify specific situations in which it matters in the context of patient management. To start with, there are four types of dead space that we will discuss: anatomic, alveolar, physiologic, and mechanical. We don't always see every one of these discussed in references, but we will include them all here to make sure that our understanding of dead space is complete. Dead space, as a term, can be used to describe any one of these subtypes, but it helps to recognize which type of dead space is of concern in a given situation. And know that we sometimes see dead space notated as V_D, but we spell it out in this manual just to keep consistent track of the various subtypes.[19]

First of all, 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 over a thousand terminal bronchioles in a single lung and hundreds of millions of alveoli total, so the majority of air ends up in the alveoli.[20] It's also worth noting that this process is dynamic and that anatomic dead space refers to the air outside of the alveoli and respiratory bronchioles when those alveoli are fully inflated at peak of inspiration.

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

[19] With one exception, but that will make sense when we get there…

[20] Betts & friends, 2013; Ochs & friends, 2003 – And just to clarify: the terminal bronchioles (marked by the thick blue line in the far right side of this graphic) are different then the respiratory bronchioles, which are the stems distal to that blue line that feed into each cluster of alveoli

IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; O_2 – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; V_D – dead space; **V/Q** – ventilation/ perfusion

Anatomic dead space is most relevant in our discussion of ventilated patients when we need to alter the amount of air that participates in alveolar gas exchange (i.e. **Ventilation**). We will talk about this more later, 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 get more of that additional volume at the alveoli because anatomic dead space has already been considered for each breath.[21]

The next type of dead space is alveolar dead space. Alveolar dead space refers to the air in the alveoli that doesn't participate in gas exchange. This can be due to a few different things: decreased capillary blood flow, fluid in the alveoli, damage to the alveolar surface, etc. Regardless of cause, any time that alveolar air is limited in its ability to participate in gas exchange, we get alveolar dead space. In the normal human body, alveolar dead space is close to zero and we assume it to be negligible. In the sick or injured human body, however, we assume some alveolar dead space and proactively take steps to accommodate that with our settings.

Interventions to address an assumed alveolar dead space would be ensuring adequate oxygenation,[22] applying end-expiratory pressure,[23] utilizing appropriate ventilator settings for patient size, and proper patient positioning. All of these things will be discussed in sections to come, so no need to remember them here. Just know that the takeaway in regard to alveolar dead space is that we always assume it exists to some degree and we do what we can to mitigate it. Worst case scenario is that the lungs were healthy and that there was no alveolar dead space to begin with and that's totally fine – none of the interventions we do here would cause damage to the healthy lung when used appropriately. On the other hand, if we forget to make this assumption in a patient that does have some degree of alveolar dead space, we can increase mortality, delay recovery, and decrease the patient's ability to compensate for other threats that might come up during the clinical course (i.e. an infection along the way).

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

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[21] There are some nuances to this idea, but we'll cover those later on

[22] This ties into the very next section on **Hypoxic Pulmonary Vasoconstriction**

[23] While this does facilitate oxygenation, it also helps address the alveolar dead space situation via recruitment of more alveoli; these two ideas are discussed, respectively, in **Oxygenation** and **Positive End-Expiratory Pressure**

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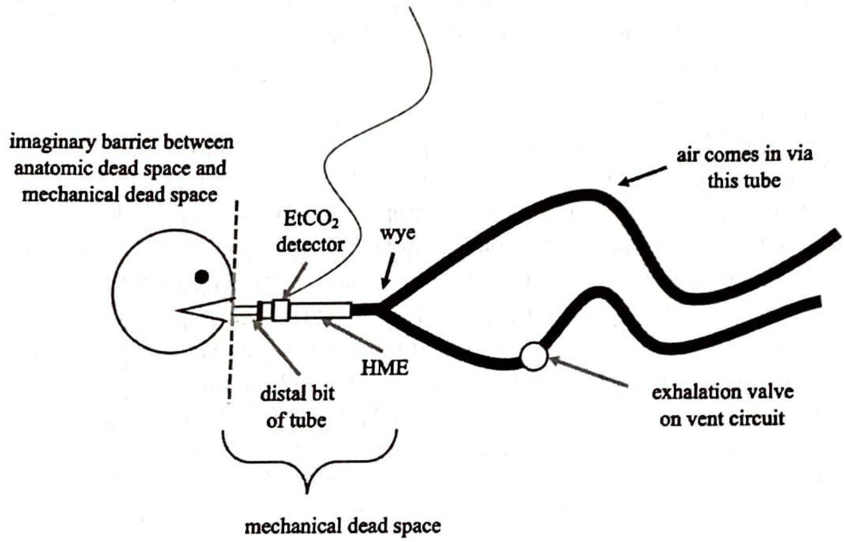
ARDS – acute respiratory distress syndrome; ATM – atmosphere; cmH_2O – centimeters of water; CO_2 – carbon dioxide; $EtCO_2$ – end-tidal carbon dioxide; ETT – endotracheal tube; HME – heat & moisture exchanger
HPV – hypoxic pulmonary vasoconstriction

Last type of dead space is what we will call mechanical dead space. Mechanical dead space, which may also be noted as equipment or apparatus dead space, is the dead space that we add on to the system with our equipment: vent circuits, end-tidal CO_2 ($EtCO_2$) detector, heat & moister exchanger (HME), etc.[24] 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 incoming air must be mixed before it gets to the alveoli. In the normal human being, fresh air is pulled into the airways starting right at that imaginary blue line in the above picture; in the ventilated patient, fresh air begins at that wye. We've discussed this effect in the **Appendix**, but suffice it to say that we should try to minimize mechanical dead space when possible (i.e. think about whether or not an in-line suction device or HME is needed rather than placing them blindly for all patients) and that the effect is more pronounced with smaller patients and higher respiratory rates (i.e. pediatrics).

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

There is another related concept to consider in this discussion of dead space that doesn't quite fit any of the types mentioned above. We like to think of all of these volumes as fixed quantities of air, but the truth is that the containers that hold this air are flexible or have stretch and therefore we sometimes see differences in expected versus actual values. One example of this is that the amount of air we put into the system doesn't always match up exactly with air out of the system.[25] 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 is lost to the tissues and airway structures, etc. While this isn't exactly dead space per se, it helps to recognize that it is a thing that can cloud our understanding of air volumes.

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[24] HMEs will be discussed in detail when we get to **Humidifiers**

[25] This difference between tidal volume and exhaled tidal volume will be addressed in **Volume Control** and then expanded on in **Tidal Volume**

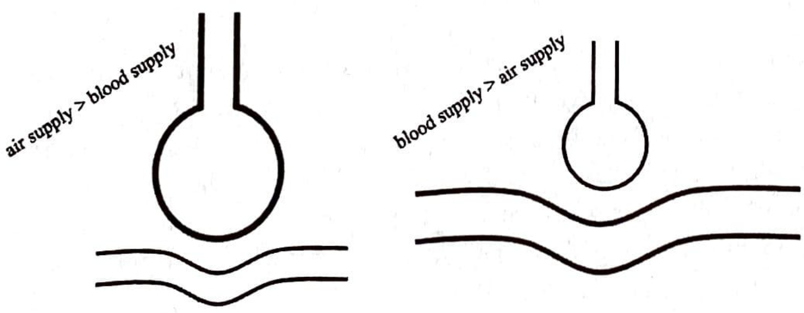
IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; O_2 – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; V_D – dead space; **V/Q** – ventilation/ perfusion

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 we look at the package of the tubing, there is a value that says how much volume of stretch a given circuit has per unit of pressure. We will revisit this idea in later sections (once we discuss a few of the concepts mentioned here) but know that in certain types of ventilation we may inadvertently overestimate the amount of air delivered if we ignore the stretch of the circuit. This is particularly relevant with little patients (i.e. infants), as the impact of this effect (ratio of misestimation to potential outcome) is more pronounced with smaller breaths.[26]

Hypoxic Pulmonary Vasoconstriction[27]

Hypoxia results in vasoconstriction of the pulmonary vascular bed (thus the term, hypoxic pulmonary vasoconstriction or HPV), which is opposite of what happens in systemic circulation. This mechanism helps the lungs to avoid wasting blood supply to part of the lung that isn't getting enough O_2 – it's a mechanism to conserve resources and maximize efficiency in the system. Just as with other vascular beds in the body, the pulmonary capillaries are in a constant state of flux and respond to the needs of the body and the availability of resources (O_2, in this case, being the driving force) by opening and closing.

Carrying on this conversation with a new term: HPV helps to avoid ventilation-perfusion (V/Q) mismatch, which could look like either of the following:[28]



[26] Bauer, 2018 – He discusses this idea in his book on vent management; we also mention it in our discussion of **Volume Control** and then demonstrate this impact in the context of managing a pediatric patient later on in the **Appendix**

[27] For more reading on the subject:

Dunham-Snary & friends, 2017 – They describe how this response can be inhibited by certain interventions and discuss the role of HPV in different pathologies

Lumb & Slinger, 2015 – This paper outlines the timelines discussed and also points out a number of relevant pharmacological agents that contribute to the effect

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

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ARDS – acute respiratory distress syndrome; **ATM** – atmosphere; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HME** – heat & moisture exchanger **HPV** – hypoxic pulmonary vasoconstriction

The left side type of V/Q mismatch demonstrates alveolar dead space. It shows that air supply (i.e. O_2) in the alveolus is in excess of blood flow and therefore some of that O_2 won't get utilized or move into the bloodstream. The right side state is what we call a shunt. In a shunt, blood ends up passing through the pulmonary vascular bed without getting its full complement of O_2. It isn't always the case that the mismatch is due to volume of air in the alveoli as shown, it can also be related to some kind of impediment that prevents the movement of air out of the alveoli examples of this would be pulmonary edema, ARDS, and pneumonia. In either of these cases, dead space or shunt, HPV is one of the body's mechanism for reversing or avoiding these types of mismatches.

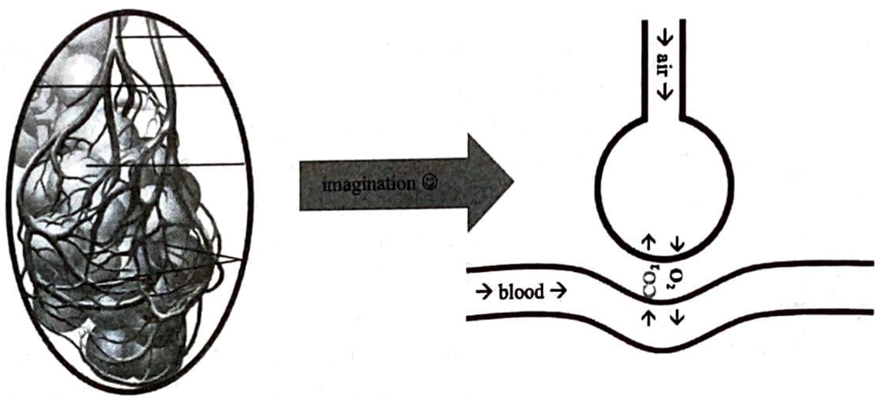
Now one thing to know about this whole process is that it goes both ways: vasoconstriction is the response to hypoxia and vasodilation occurs when oxygenation is adequate or that hypoxic state is resolved. We might consider these to be similar processes, just in opposite directions. There is a distinction,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

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

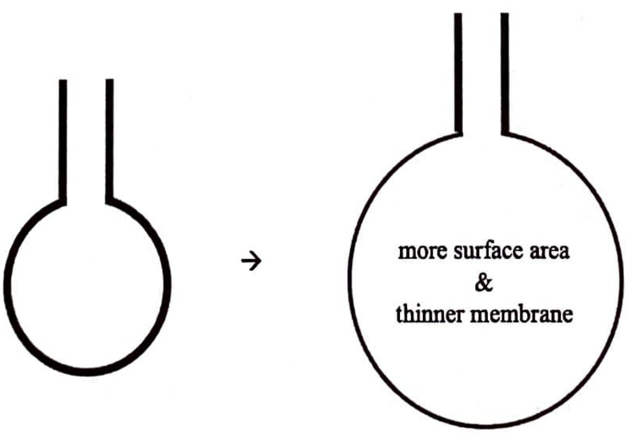
IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; **O_2** – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; **V_D** – dead space; **V/Q** – ventilation/ perfusion

Alveolar Surface Area

Even though we have been demonstrating the alveoli-capillary interface as a single blood vessel running past an air sac, it is important to recognize, again, that this is a simplification of how things are and that the surface of the alveoli are covered by a network of vessels:[29]



When we inflate the alveoli we get more surface area and that means more interface between air and blood. In addition, inflation of the alveoli causes the alveolar membrane to stretch and become thinner, allowing for easier diffusion of gasses:[30]



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[29] Betts & friends, 2013 (image)

[30] And we spell this out in much more detail in the section on **Oxygenation** when we talk about Fick's Law

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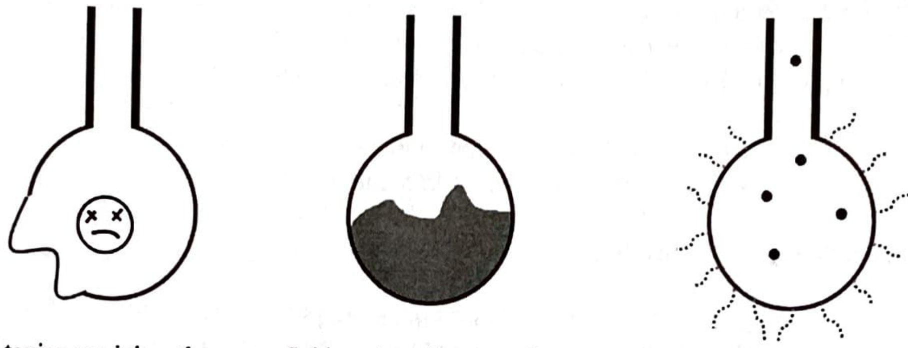
ARDS – acute respiratory distress syndrome; **ATM** – atmosphere; **cmH$_2$O** – centimeters of water; **CO_2** – carbon dioxide; **EtCO_2** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HME** – heat & moisture exchanger **HPV** – hypoxic pulmonary vasoconstriction

More surface area and a thinner membrane make it easier to move air from inside of the alveoli to the circulatory system, so lots of our interventions with the vent are focused on this idea.[31] That said, certain things can get in the way of this gas exchange even if we do get the surface area up and membrane thinned out. Think of these as things that impact access to usable alveolar surface area:[32]



toxins can injure the membrane directly | **fluid can impede gas exchange across the membrane** | **inflammation can damage the membrane and impair diffusion**

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

[31] Desai, 2012 – ~~We cite this video in **Oxygenation**, but here it is now if anyone is curious before then~~

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

IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; **O_2** – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; **V_D** – dead space; **V/Q** – ventilation/ perfusion

Ideal Body Weight

Second to last thing ~~related to underlaying physiology~~ before we move on to talking about the machine: lung size is most strongly correlated with patient height. Because of this, we use a patient's height to calculate an ideal body weight (IBW) when doing vent things.[33] The idea is that a six-foot human could weigh either 120lbs or 300lbs and the size of his or her lungs wouldn't change. There is a formula to calculate IBW for both males and females, often presented as a hybrid of metric and standard units:

$$IBW_{male}\,(kg) = (2.3(\text{height in inches}) - 60) + 50$$
$$IBW_{female}\,(kg) = (2.3(\text{height in inches}) - 60) + 45.5$$

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

$$IBW_{male}\,(kg) = (0.91(\text{height in cm}) - 152.4) + 50$$
$$IBW_{female}\,(kg) = (0.91(\text{height in cm}) - 152.4) + 45.5$$

Or we can use charts like this:[34]

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 Studie

[33] IBW may also be referred to as predicted body weight (PBW)
[34] NHLBI ARDS Network, 2005 (image)

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HPV – hypoxic pulmonary vasoconstriction

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

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

When dealing with pediatric patients, our go-to reference ought to be the Broselow Tape. If that isn't available, we do have some formulas we can refer to:[35]

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

And note that the Broselow overlaps with the equations and chart above, so if we have a small grownup or a big kid we should still be able to get an IBW just fine. So ~~no excuses!~~ And very last thing: there are some apps out there that can help with this sort of thing, both for adults and for pediatrics.[36]

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

[36] Critical-Medical Guide & Pedi STAT – Both are excellent resources to have on hand for quickly referencing relevant things

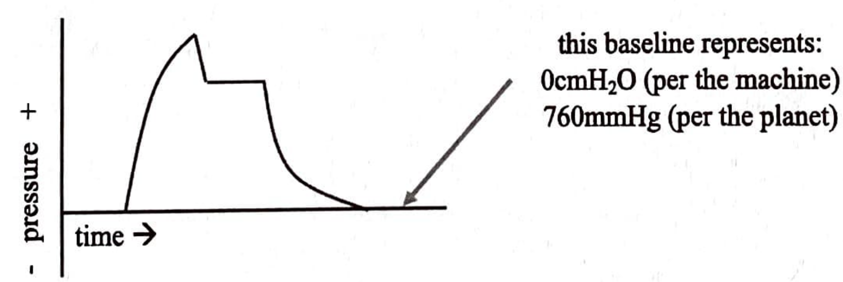
IBW – ideal body weight; **kg** – kilograms; **kPa** – kilopascal; **lbs** – pounds; **mmHg** – millimeters of mercury; O_2 – oxygen; **PBW** – predicted body weight; **PEEP** – positive end-expiratory pressure; **PSI** – pounds per square inch; V_D – dead space; **V/Q** – ventilation/ perfusion

Measuring Pressures

During mechanical ventilation we measure pressures in centimeters of water (cmH_2O). We 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 takes -1mmHg of pull 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 chart just to make things clear:[37]

	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 ambient pressure as it relates to airway and vent stuff is zero; so while true atmospheric pressure at sea level is 760mmHg (1 ATM), we call it 0cmH_2O to make things easier.[38] And then we have a way to represent breaths we give as waveforms showing pressure as a function of time with this new zero point (representing atmospheric pressure) as the baseline. For now we are going to ignore <u>PEEP</u> (since we haven't discussed it yet); we also don't have to worry about the specific components of the waveform – all those things will be discussed later on:



[37] We built this chart by Googling conversions for these values...

[38] <u>Yartsev, 2019</u> – Scroll down to the section called Airway Pressure for some interesting background on why we measure/label pressures the way we do

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legend

AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

Modes of Ventilation

This next section discusses how we organize the delivery of breaths to a patient. We've distinguished this concept of mode with that of control (see next section) in order to make things easier to conceptualize, but the terms sometimes get used with a bit of overlap. In reality the way we control a breath is one component of how we describe what mode we are in, but we will keep the two ideas separate to make things flow more smoothly. It helps to think of mode as the overall pattern or organization of breaths and control as the specific way we choose to deliver them.[39]

There is one concept that will be needed in order to understand things moving forward. **Triggers** are the thresholds by which the machine knows when to give a breath. We will talk about these in detail much later, but it is tough to explain the following ideas without a basic comprehension. In its simplest form, a trigger could simply be a quantity of time. An example of this would be one breath given every four seconds; we could then describe those instances as time-triggered breaths. There are also ways we can infer inspiratory effort made by the patient and have the machine give breaths based on those cues. We'll discuss how that happens later, but the general term for this type of thing is a patient trigger (i.e. a patient-triggered breath) and we will use that idea in the next few sections.

~~IMV~~
~~APV~~
~~CMV~~ ✓
~~AC~~
~~OK~~
~~PEEP~~
~~SIMV~~
~~PS~~

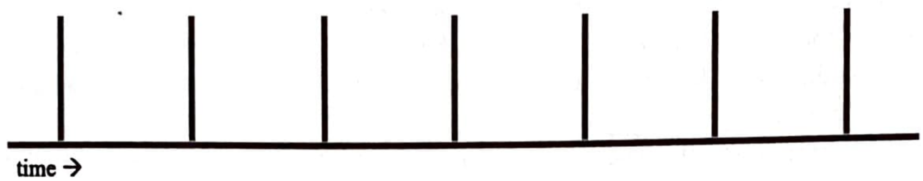
modes & concepts of ventilation

[39] Chatburn & friends, 2014 – For specifics on how all of these things ought to be labeled and described, this article outlines a taxonomy for vent concepts

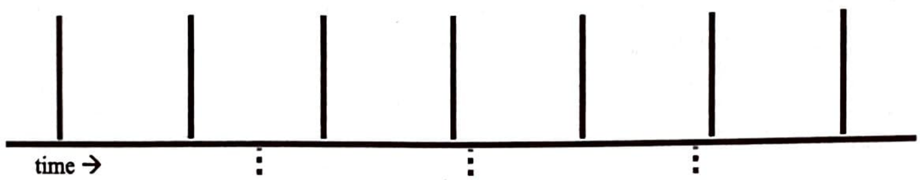
PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

Basic Modes of Ventilation

The simplest way to ventilate a patient with a machine is to give breaths on a schedule and to ignore what the patient does on his or her own. Let's assume a hypothetical timeline running left to right over an arbitrary amount of time with black hashes to represent time-triggered breaths:



Now if the patient tries to take breaths overtop of this timeline, that effort gets ignored. We'll show that effort like this:



In terms of triggers, we could say that ventilation via this mechanism utilizes time triggers only and does not have a mechanism for patient triggers. Now there are two versions of this type of ventilation, controlled mechanical ventilation and intermittent mandatory ventilation (IMV).[40] The difference here is subtle: in controlled mechanical ventilation (which precedes all the other modes), the patient is physically unable to draw a breath on his or her own with effort made; in IMV (a subsequent technological improvement) the patient is, in fact, able to draw a breath, it's just that the machine doesn't offer support and instead allows the patient to draw in air from the vent circuit independently and without help.

Controlled mechanical ventilation isn't routinely used, but it does get utilized in surgical settings when patients are paralyzed and for sure not making an effort to breathe spontaneously. IMV, on the other hand, is normally not available on its own – it typically comes with additional features which we will line out in just a bit. Now we may be able to manipulate our vent to approximate either of these modes in the event that we want to ignore patient triggers, but those would be specific cases and we'll get to them later on.

Moving on, subsequent modes build on this framework by introducing mechanisms to support the patient's effort to breathe. When we introduce patient triggers, however, there is the potential that harm can result and we will talk about how different modes work to mitigate that risk. That said, utilizing the machine to augment patient effort improves comfort, facilitates recovery, reduces the negative effects of PPV, and gives us more control over the management of the patient.[41] So now let's get on to the two modes commonly used in transport that take us beyond the basics and allow us to use patient triggers.

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[40] Frakes, 2007; Kacmarek & Branson, 2016 – We won't refer to controlled mechanical ventilation as CMV (even though you sometimes see it that way), because CMV more often refers to continuous mandatory ventilation which we describe in just a moment; for more on this refer to both articles referenced

[41] Mauri & friends, 2017; Goligher, 2017 – We will talk about these specific things later on (in **Comfort**), but the article and essay provide a bit of context for these claims

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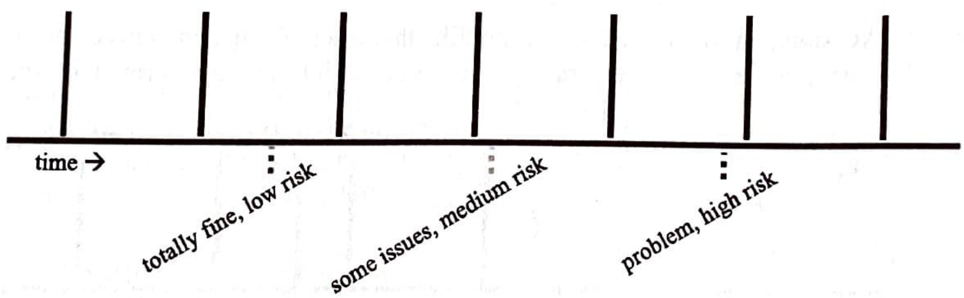
AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

Assist Control

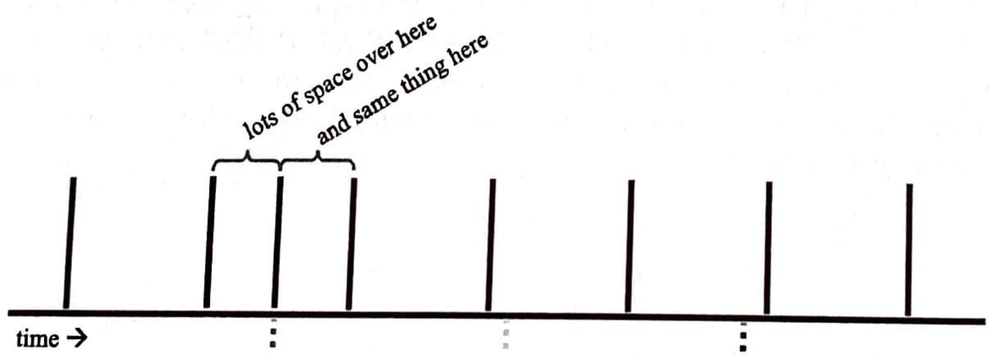
Assist control (AC) ventilation is a mode that augments a patient's spontaneous respiratory effort by delivering a preset amount of air when an inspiratory effort is detected. AC is sometimes known as continuous mandatory ventilation (CMV). In the case where the patient is trying to breathe in addition to an underlaying rate, the machine recognizes that and then responds by giving a full breath on each of those occasions. To phrase it differently: time-triggered breaths are still given as scheduled, but patient-triggered breaths can also occur. The obvious advantage here is that the patient's expressed need for more breaths per unit time is met. On the other hand, and as we just said, this method of air delivery has the potential to cause harm.

To start to make sense of this, let's return back to a series of time-triggered breaths with effort notated by dotted lines below the timeline. In AC mode, each instance of patient effort (whether true patient effort or simply perceived patient effort due to some other factor) has a varying potential for harm based on where it lines up in relation to other breaths. We'll draw it out first and then explain in detail:

color of the dotted lines indicates how subjectively dangerous these instances of patient effort are or how much potential there is for them to cause harm based on proximity to the time-triggered breaths

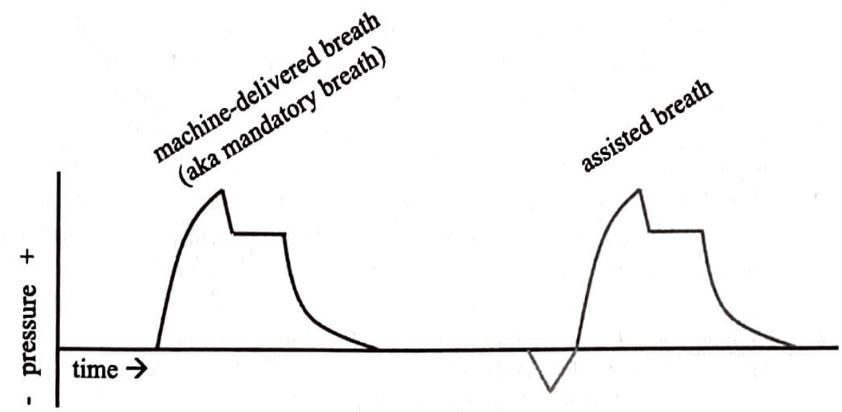


To expand on this: with the green effort, there is space (in time) on either side of the breath, so the machine can actualize that breath without affecting other breaths in proximity:



PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

The difference between that green, patient-triggered breath and the baseline, time-triggered ones can be represented via those pressure-over-time waveforms that we mentioned before. Note the dip at the start of the second waveform as the patient breathes in – this is the effort that gets sensed by the machine right before a full positive-pressure breath is then given:[42]



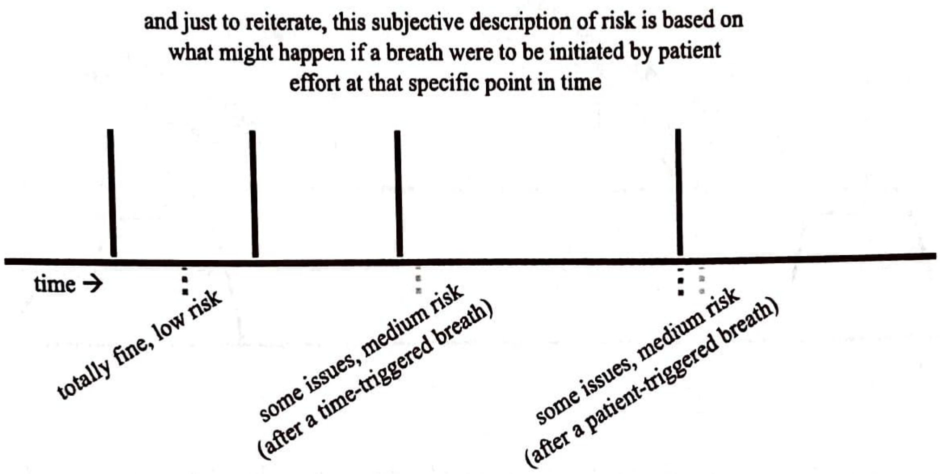
A pretty good AC situation might look something like this where the patient's need for more breaths are generally met and that need, in the form of inspiratory effort, doesn't interfere or overlap with the scheduled breaths:

Do note that not all patient effort will result in delivered breaths. That's where triggers come in and we can adjust that parameter to make it more or less likely that a breath will occur. Again, more on this later, just something to note for now. And the ideal AC situation (in contrast to the pretty good one shown above) would be when all patient effort results in a delivered breath, none of those patient-triggered breaths interfere with the time-triggered ones, and when no miscellaneous factors cause accidental triggers. No need to draw that one out, as it will be much clearer later on. For now we want to stay on track with describing AC and how we can potentially cause harm in this mode.

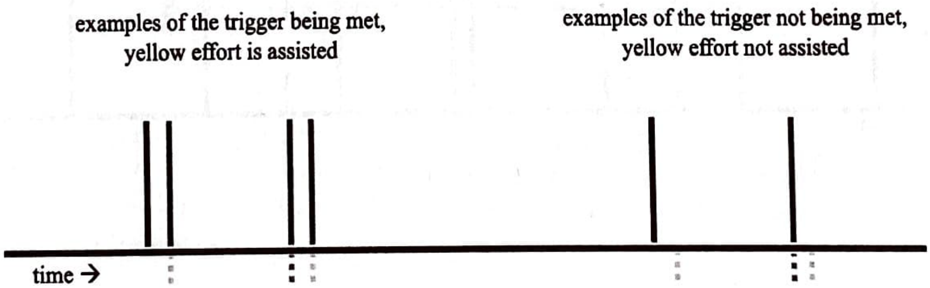
[42] Now this graphic makes it seem as if a pressure change detected by the machine leads to an assisted breath; while that could potentially be the case, the more common situation is a flow trigger; ~~regardless of the trigger, however, the drop is pressure as shown in the graphic would occur in either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

AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

In the case of the yellow effort (which we labeled as medium risk) there is a potential trigger that immediately follows another breath. It could be that the other breath in question is a machine-delivered one (as we had it in the first graphic) or it could be another patient-triggered one:



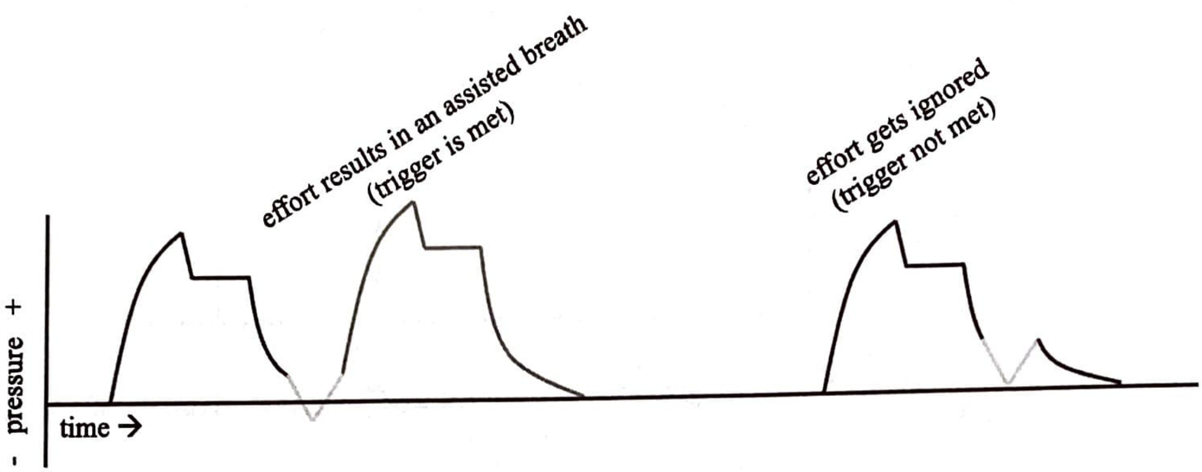
Now what happens next depends largely on how the trigger is set up, but we can generalize it by saying that the further along the first breath is or the closer the breath is to an end-exhalation baseline, the more likely that the effort will catch and result in a full breath. There are two possible outcomes: one in which the trigger results in an assisted breath and one in which the trigger does not result in a breath:[43]



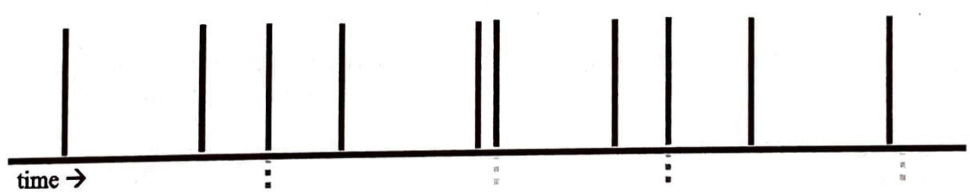
[43] And as already mentioned, we will discuss this idea of <u>**Triggers**</u> in much more detail later

PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

And then we can carry on with the idea to show either of those outcomes as a waveform of pressure over time:



These yellow-effort situations do have the potential to cause harm, but they are less likely to result in breaths being delivered because of the ongoing breath that precedes them.[44] That harm comes from the increased pressure as a full breath is given before the prior one was completely done (left side, note the drift of maximum height on waveform). If the yellow effort doesn't result in a breath being delivered, this may cause some discomfort (right side, due to an expressed need that goes unaddressed), but that's probably OK. That said, a combination of green and yellow effort is just fine for our patients in AC mode:



[44] Chatburn & friends, 2014 – There is also a refractory period with some machines that prevents a subsequent trigger from occurring too soon after a breath has been given

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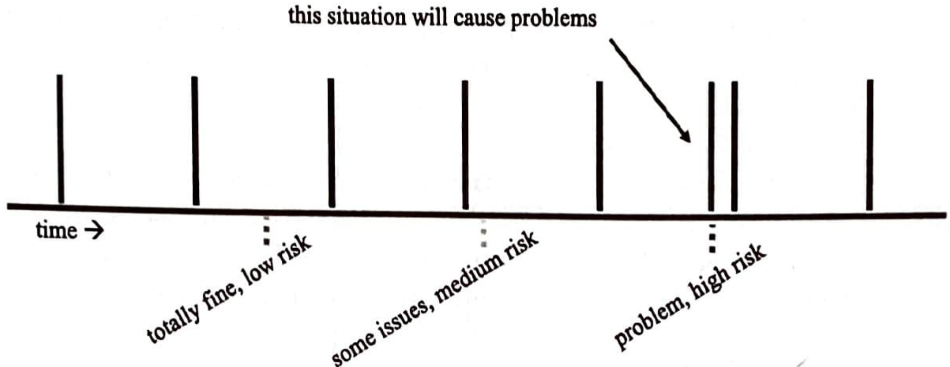
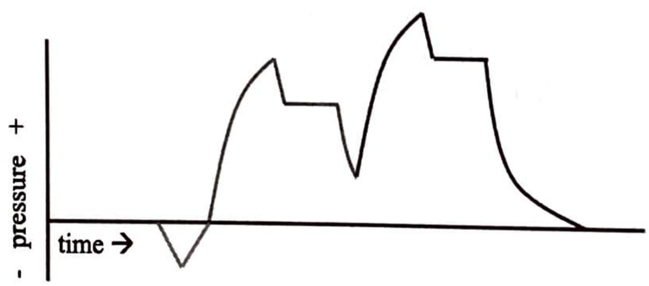
AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

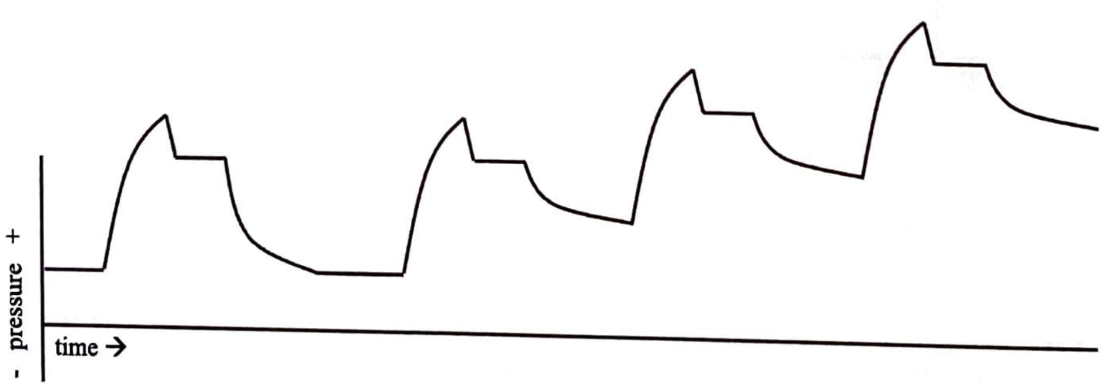
The issues with AC mode begin when we get those red-effort situations in which a patient-triggered breath immediately precedes a time-triggered one:



When this happens we get that same increase in pressure we described just above (the drift of maximum height on the pressure waveform), but consistently and to a greater effect. As a waveform it would look like this:



There is a complication known as **AutoPEEP** in which this happens with some regularity resulting in sustained high pressures. AutoPEEP can also occur in AC mode if we accidently trigger a number of breaths in sequence. We'll revisit this idea again later, but here's how that might look (and we've left out color and triggers just to make it clearer):[45]



[45] In reality these breaths will likely get cut short due to pressure limits we have set on the machine, but we'll explain that fully when we get to the section on **AutoPEEP**

PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

So we've established that AutoPEEP can occur in AC, but recognize that it doesn't always happen and that we can take proactive steps to make AC safe for our patients. The primary benefit of AC mode is that we can readily meet the patient's need for more volume using patient-triggered breaths. Another benefit is that it is predictable: patient effort that meets the trigger will get assisted to whatever parameter we have set into the machine. To say it another way, time-triggered and patient-triggered breaths will be the same. And then subjectively, AC mode is easy to use. While this may not be the best reason to advocate its use in the field, it is simple to set up, easy to conceptualize, and when the primary complication of the mode does arise (AutoPEEP) there are specific actions we can take to fix it.[46]

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

the specifics of how we avoid & fix breath stacking

[46] And all of that will be discussed later, both in **AutoPEEP** and **Triggers**

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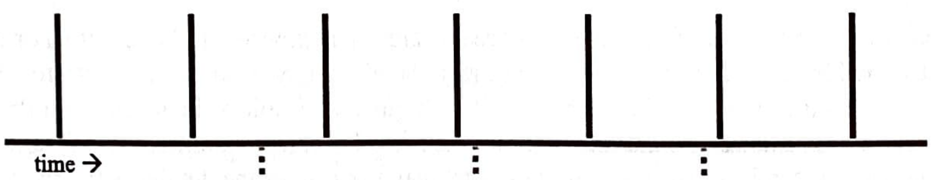
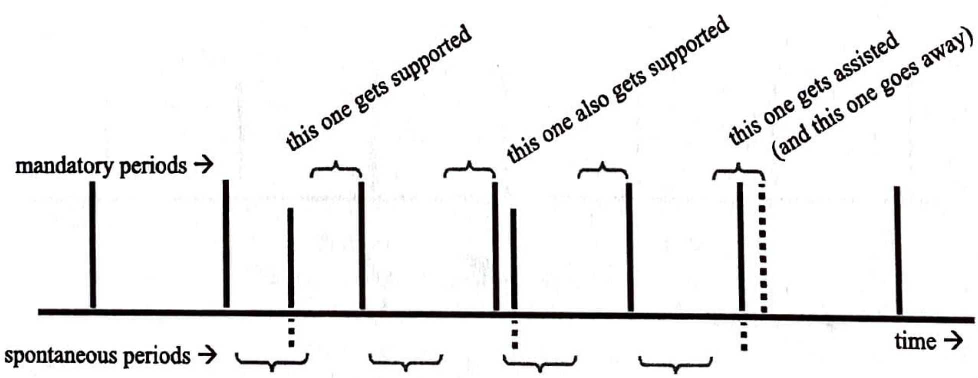
AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

Synchronized Intermittent Mandatory Ventilation

SIMV is an alternative mode of ventilation that also seeks to mitigate the shortcomings of the more basic modes by using patient triggers, but has a mechanism built in that helps avoid the **AutoPEEP** complication of **AC** mode. SIMV starts with the idea of mandatory breaths or a guaranteed number of time-triggered breaths to be given per minute. It then will support breaths taken in between these mandatory breaths. Furthermore, SIMV recognizes when patient effort is made in close proximity to a time-triggered, mandatory breath and assists that effort in a way similar to how breaths were assisted in AC mode (i.e. a full breath is given). Now there are more differences between these various **Types of Breaths** and we'll get back to that eventually, but let's focus on the timing aspect of SIMV first. Going back to our original idea:



SIMV's method for determining how to handle the instances of patient effort is to break the timeline into two alternating categories: mandatory and spontaneous periods. If a patient trigger happens within a spontaneous period, it gets supported and that effort is facilitated by the machine in a manner that we will discuss real soon;[47] if an effort occurs within a mandatory period it gets assisted, a full breath is delivered, and the breath that had been planned for that mandatory period gets skipped:[48]



[47] Ollie, 2015 - This video demonstrates the idea in another way by way of a discussion about SIMV ventilation (versus IMV – a distinction we will sort out in just a second)

[48] Wheeler & friends, 2008; Kumar, 2015 – The first explains this process as we've labeled it, the other is a brief overview that explains it using a different labeling system

PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

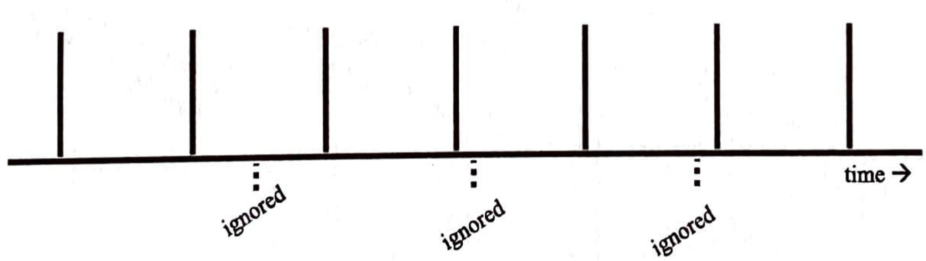
To make sense of this graphic, we'll point out a few things. We are assuming that each instance of patient effort meets the trigger threshold to result in a breath. The difference in height of the blue lines is to distinguish supported breaths (smaller, left two) from assisted breaths (taller, right one). And that time-triggered breath at the far right gets omitted to avoid the AutoPEEP complication we saw in AC mode.

As for the difference between supported breaths and assisted breaths, the idea is that supported breaths only get a little bit of help from the machine and the assisted breaths are fully facilitated by the machine to a target amount of air, just as in AC mode. Supported breaths are always supported via pressure, which helps the patient draw a breath a little bit easier; assisted breaths are carried out to meet specific goals by the machine based on settings we input and can be either volume-targeted or pressure-targeted (which we will expand on in the next section).[49] The practical difference is that pressure support (PS) breaths will give us a variable result that depends largely on the patient's contribution to that specific breath, while assisted breaths are more predictable.[50]

At the risk of getting ahead of ourselves, PS breaths are often expected to be less than or smaller than mandatory and assisted breaths (in terms of volume of air). While it may make sense to titrate PS up so that supported breaths match the other ones in this regard, it isn't quite as simple as increasing the PS value on the machine. That said, there is no reason that the volume of air in a PS breath should be less than the other ones, it's more an issue that it often just happens to turn out that way because of the details as to how these different **Types of Breaths** are brought into existence by the machine.[51]

And a few more things about SIMV mode: It originally came onto the scene as IMV, which we already discussed. The "S" for synchronization was added when the mode was adapted to consider patient-triggers in close proximity to time-triggered breaths (i.e. breaths initiated by the patient within that mandatory period). The next improvement was PS to breaths triggered in the spontaneous period, so we sometimes see SIMV as we described it notated as SIMV + PS. To draw these all out: [52]

IMV



[49] Lodeserto, 2018 – This series provides an alternative explanation to this concept (i.e. how PS and pressure control breaths differ) and we will touch on it again in **Types of Breaths**

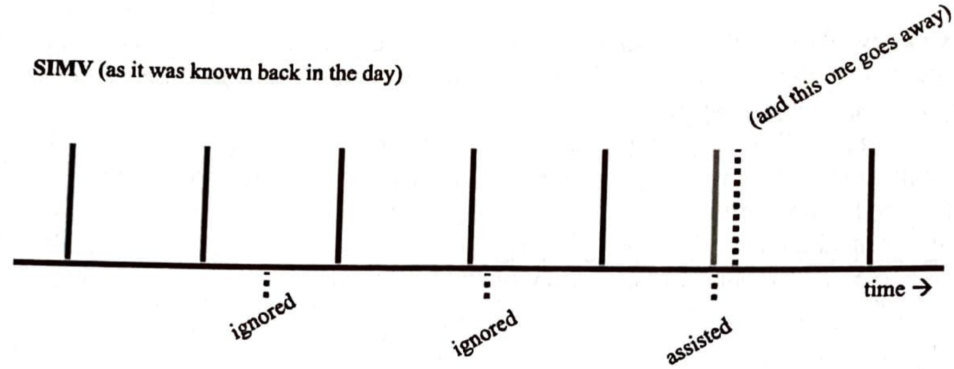
[50] Chatburn & friends, 2014 - And by convention both supported and assisted breaths could be labeled as assisted, it's just that PS breaths are a more specific type and we will discuss them that way just to remind readers about that difference

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

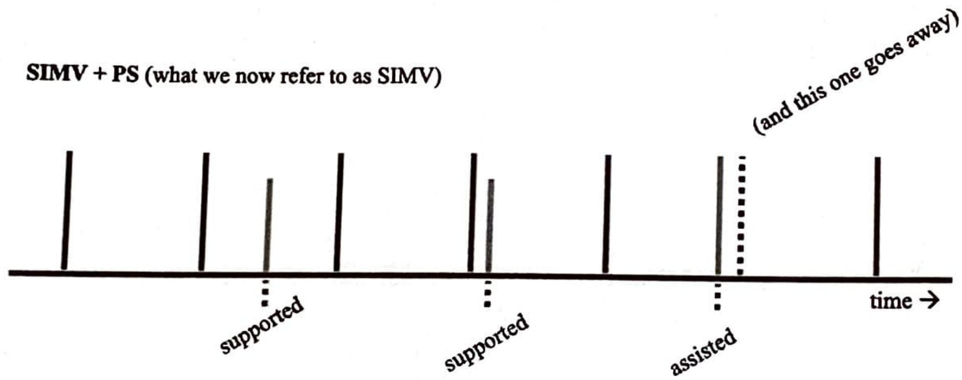
[52] Ghamloush & Hill, 2013 – We recognize that this is confusing, but navigate here for another explanation of how SIMV as we know it came to be

AC – assist control; **CMV** –mandatory ventilation; **IMV** – intermittent mandatory ventilation; **OK** – alright
PEEP – positive end-expiratory pressure

SIMV (as it was known back in the day)



SIMV + PS (what we now refer to as SIMV)



Another historical tidbit is that the mode was popularized as a method of weaning or getting someone transitioned from vent life to spontaneous breathing after an illness or intervention – the efficacy of SIMV for weaning has since been shown to be inferior to other methods. The result of all of this is that content on SIMV is often confounded by stuff that more accurately relates to IMV or SIMV without PS and that draws conclusions from a concept (weaning) that doesn't much matter in the transport setting.

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

Don't introduce new shit in conclusion, put downsides in its own ¶

Finish the thought: So even tho SIMV But used ... after... blah blah

PPV – positive pressure ventilation; **PS** – pressure support; **SIMV** – synchronized intermittent mandatory ventilation

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 at the very end when we talk about building out a 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 we can make it. All that said, there are cases in which one mode may be preferred over another and we will talk about those as they come up.

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AC – assist control; **cmH$_2$O** – centimeters of water; **ml** – milliliter; **OK** – alright; **PC** – pressure control; **PIP** – peak inspiratory pressure

Control of Ventilation

We already discussed the first big choice in vent management: which mode (AC or SIMV) to utilize for our patient. The next decision is to choose whether we want to control volume or pressure. If we choose to control volume, airway pressure will function as the dependent variable (i.e. we won't be able to directly control it); if we choose to control pressure, volume will function as the dependent variable. There is no right or wrong answer to this dilemma, but the general trend is that we use volume control in most cases and pressure control with pediatrics or when we are especially concerned about airway pressures.[53] Not saying this is the best decision, just saying that's how it's been done.

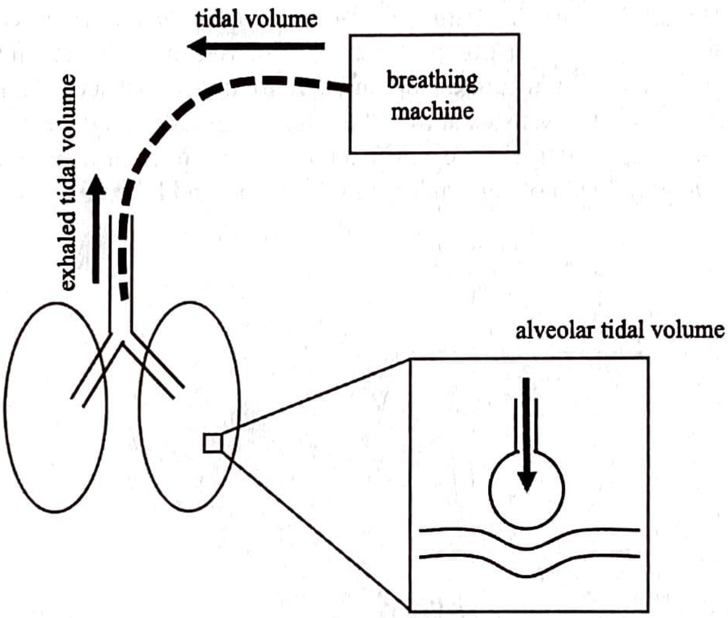
The reason for this is twofold. First (and arguably most relevant), the machines tend to default to volume control unless we do something to intentionally get out of it. Second, volume control is a bit easier for some folks to wrap their heads around – it's a little more intuitive to think about set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

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

Pplat – plateau pressure; **PRVC** – pressure-regulated volume control; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

Volume Control

In volume control (VC) ventilation we choose how much volume we want to push down the circuit with each breath delivered.[54] This tidal volume that we put in goes to the lungs, does its thing at the alveolar level, and then gets exhaled out of the circuit. When we say tidal volume we are referring to the air going into the system from the machine; those other two concepts (alveolar tidal volume and exhaled tidal volume) vary from that value due to a number of different factors. Let's see how this looks in a graphic and then we'll hash out the details:



[54] To say it another way, in VC we control **Tidal Volume** directly – a concept we mention here and then discuss again in much more detail in the near future

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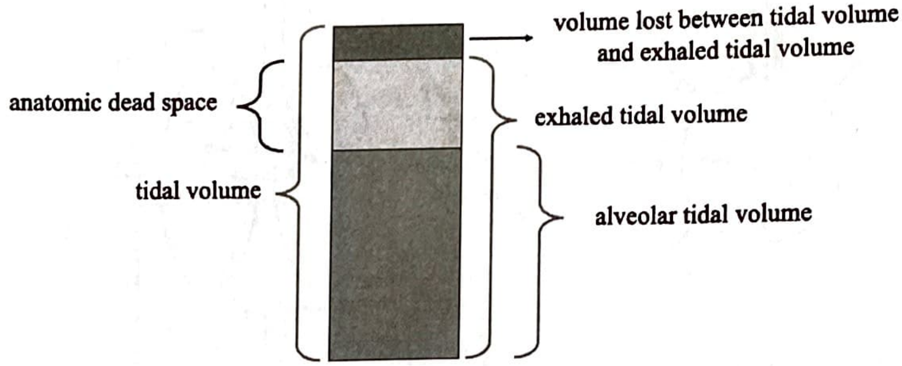
AC – assist control; cmH_2O – centimeters of water; **ml** – milliliter; **OK** – alright; **PC** – pressure control; **PIP** – peak inspiratory pressure

Exhaled tidal volume is generally about the same as tidal volume, but after some air is lost to the vent circuit and/ or to the tissues in the respiratory system. This results in the potential to overestimate volume delivered, which becomes particularly important with smaller volumes of air (i.e. pediatrics).[55] And alveolar tidal volume is exhaled tidal volume minus anatomic dead space. Recognizing the fact that not all of that alveolar tidal volume will always participate in gas exchange due to the idea of alveolar dead space, the volume of air that makes it to the alveoli is about two-thirds of what we push into the system.[56] Here's how it all looks:



So while alveolar tidal volume seems a few steps removed from the tidal volume we set on the machine, VC ventilation allows us to control alveolar tidal volume as directly as possible. The result of that, however, is an increase in pressure that is dependent on the amount of air we set and how that air moves through the respiratory system. 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.[57] While the alveoli do expand with added volume, at a certain point we can overinflate them. This results in what we call barotrauma and we for sure want to avoid that.

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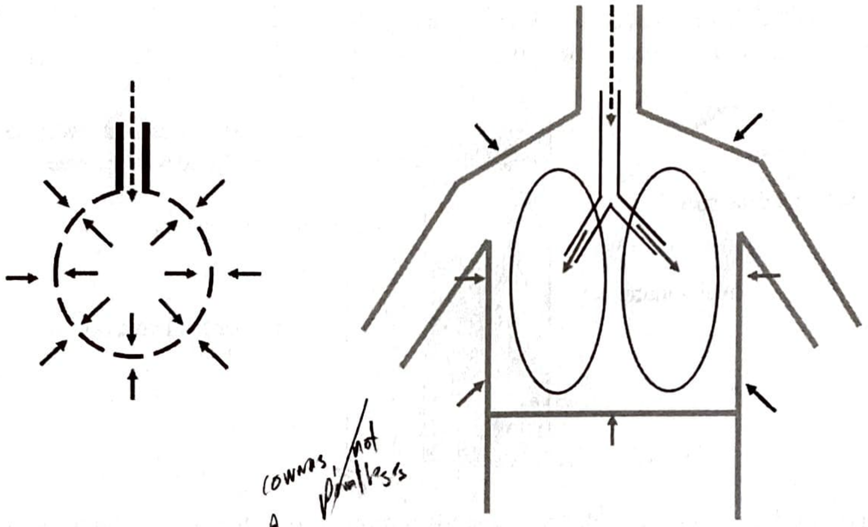
[55] We talked about this already in **Dead Space** and will address it again in the **Appendix**

[56] Yartsev, 2019 – And this fraction of overall volume does seem to fluctuate with changes in volume delivered (i.e. a weight-based estimate may not be accurate); that said, as we increase the volume of breath, anatomic dead space does not always increase to a comparable degree – we'll come back to this later in **Ventilation**

[57] Flowers & friends, 2019 – As another example of Gas Laws in action, this process is partly described by Boyle's Law (pressure change as a result of volume increase)

Pplat – plateau pressure; **PRVC** – pressure-regulated volume control; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

We will get more into all of these concepts later, but a high pressure in VC can be due to alveoli inflated beyond their capacity, some restriction to the expansion of the chest wall, or decreased air movement through the airways:



The risk we face in VC ventilation is that we can injure the alveoli or cause barotrauma when they are overinflated, as shown above on the left. To say this another way: there are three distinct reasons why we may see high pressures and one of those three (overdistended alveoli) is our area of concern in this discussion. We avoid this by monitoring airway pressures and adjusting the volume input to avoid causing damage.[58] We will get to the specifics as to how we do that eventually, for now it's OK to leave it as so: in VC ventilation we control the amount of air going into the circuit at the expense of control over resultant pressures; that said, we always need to monitor airway pressures during VC ventilation in order to avoid causing damage to the alveoli. In addition, VC ventilation lends itself to an overestimation of alveolar tidal volume if we forget to factor in dead space.

[58] While there are other parameters that we can adjust to avoid higher pressures (which we will get to later), it helps to simplify things this way: more volume = more pressure

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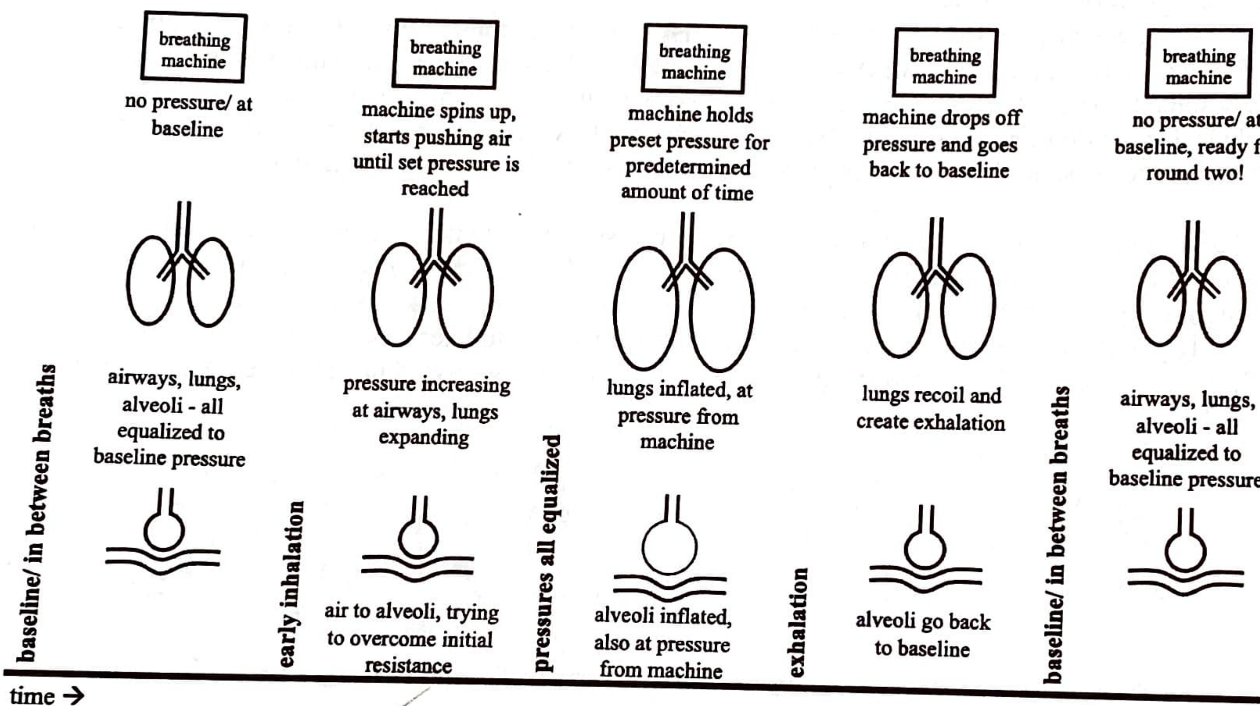
AC – assist control; **cmH₂O** – centimeters of water; **ml** – milliliter; **OK** – alright; **PC** – pressure control; **PIP** – peak inspiratory pressure

Pressure Control

In the other corner of the arena we have pressure control (PC) ventilation.[59] In this system, 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 an amount of time, then the breath cycles off and we go back to baseline.[60] Because our input here is pressure, volume becomes our dependent variable (exhaled tidal volume, to be exact and as we noted before).[61] Let's draw it out and see if we can make it a little clearer:



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

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

[60] Just to clarify: as we move forward from here we will use PC as an abbreviation to describe pressure control as a way to control ventilation, but when we refer to pressure control as the parameter we dial in on the machine we will spell it out

[61] And if a machine is capable of pressure control ventilation it will 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 VC ventilation only

Pplat – plateau pressure; **PRVC** – pressure-regulated volume control; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

One thing worth pointing out here is that in PC ventilation we don't have to bother with considering that flexibility or stretch that we discussed when we talked about dead space (i.e. the compliance of the vent circuit), as the only way we have to measure volume is via exhaled tidal volume or what the patient breathes out (which is downstream of all that flexing). We do still need to consider anatomic and alveolar dead space, just as we did with VC, but the stretch factor we introduce in our circuit is eliminated. This is a big advantage of PC ventilation with small patients: forgetting to factor in 10ml (arbitrary number) in an adult is no big deal, forgetting to do so for a baby with small tidal volumes is huge. We'll discuss more later, but just know that this is one advantage of PC.[62]

Another advantage of PC is that we avoid the risk of over-inflation or high pressures at the alveolar level. The highest pressure those alveoli will see is whatever value we program into the machine.[63] As long as we follow some basic guidelines as to what a safe pressure is, there's not much risk of harm or barotrauma. The downside is that we don't have as good of control (compared to VC) over the amount or volume of air that we are putting into the system; instead we have to continually monitor exhaled tidal volumes and adjust to our goals.[64]

To summarize: in PC ventilation we control the pressure put into the system at the expense of control over resultant volumes. That said, we always need to monitor those volumes when we have a patient in PC in order to avoid hyper- or hypoventilation. In addition, PC ventilation makes it a little more difficult to control ventilation (as opposed to oxygenation – again, concepts we will get to later on), due to the breath to breath variability in volumes.[65] The big advantage of PC ventilation is that we avoid the high pressures that can result from VC.[66]

[62] This will happen in the **Appendix**

[63] 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?**

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

[65] And to skip ahead and look at these sections, link to them here: **Ventilation** and **Oxygenation**

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

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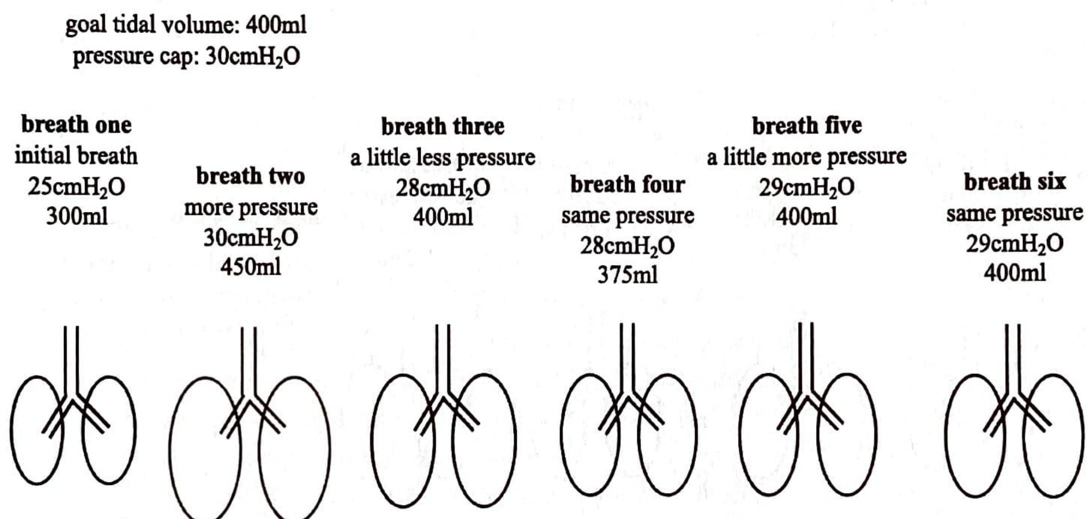
AC – assist control; **cmH_2O** – centimeters of water; **ml** – milliliter; **OK** – alright; **PC** – pressure control; **PIP** – peak inspiratory pressure

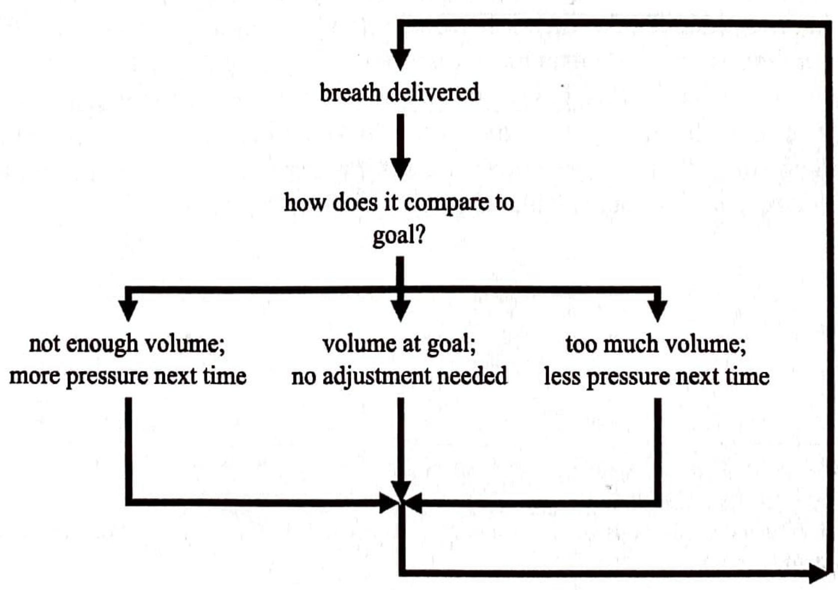
Pressure-Regulated Volume Control

Pressure-regulated volume control (PRVC) is one attempt to get at the best of both worlds when it comes to this volume versus pressure conundrum. In this type of ventilation we dial in a goal for tidal volume and put an upper limit on pressure, then the machine tries to give breaths to the goal volume using the lowest possible pressure and without exceeding the limit we have set.[67] The machine makes adjustments to how it delivers each breath by looking at previous breaths and adjusting delivery to add or take away volume working towards the preset tidal volume 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 algorithmic fashion where we decide where each breath ends up in relation to our goal and then adjust the subsequent breath in a cyclical manner:

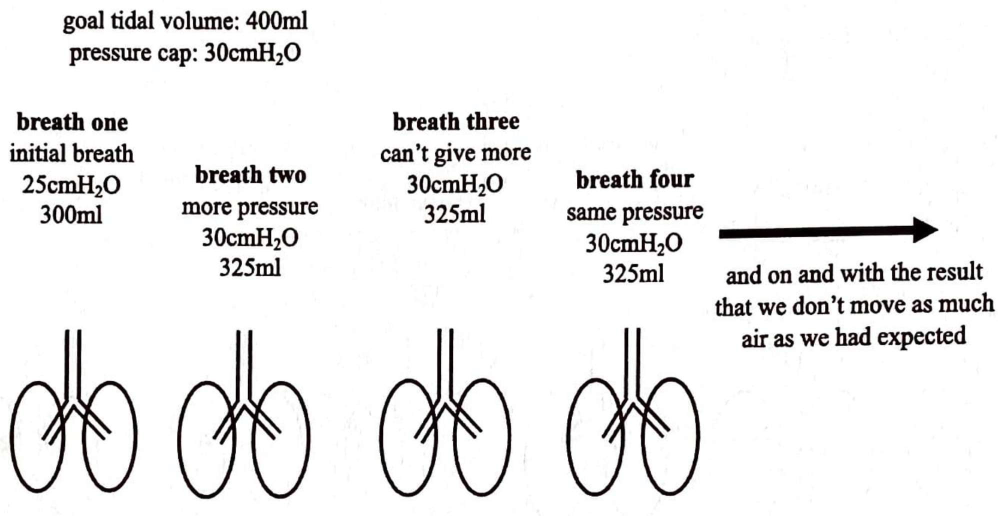


[67] In the graphic below and moving forward we call this limit the pressure cap for lack of a better term, but we will discuss it more in just a moment

Pplat – plateau pressure; **PRVC** – pressure-regulated volume control; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

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

To flush out a few more details on this PRVC concept, let's look at another example of a few consecutive breaths. In this example something is causing an increase in pressure, therefore breaths fall under goal (in terms of volume). The result of this would be a drop in minute volume or air moved per unit time.[68] It's important to keep this in mind with PRVC, as we can inadvertently lose some minute volume in an effort to avoid high pressures:



A few more things about PRVC: pressure cap is a make-believe term – the machine most often uses 5cmH20 less than the set high-pressure limit for this value.[69] There are also restrictions on how much variation occurs from one breath to the next, to say it another way, the machine won't make 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.[70] Along that same idea, the machine doesn't know how much air (i.e. **Tidal Volume**) it gives with each breath until after the fact when it sees the exhaled tidal volume, that's why it can overshoot the goal. Last thing: PRVC is good when we are worried about barotrauma or giving too much pressure, but it is important to make sure we keep an eye on volumes delivered and compare it to our calculated goal.

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

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cmH₂O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO$_2$** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

Vent Parameters, Round One

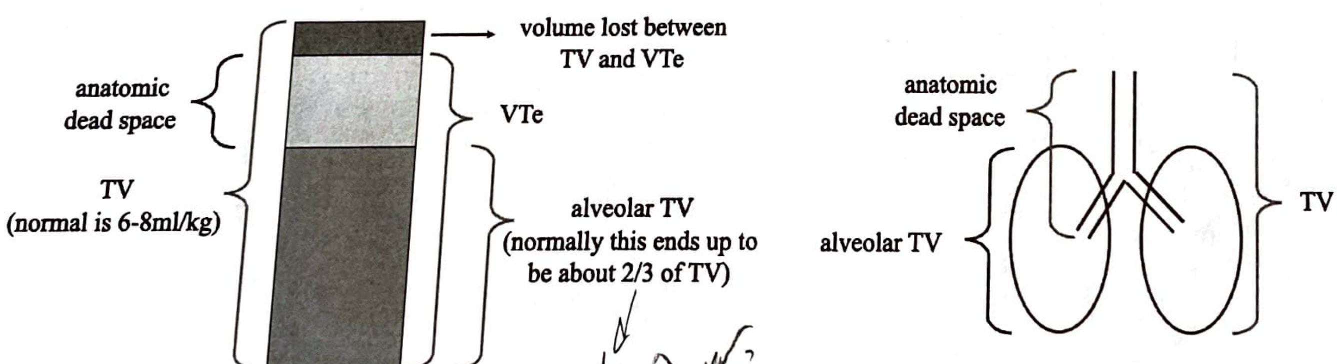
The next step on our journey is to explain ~~fully~~ the ins and outs of some of the terms we use to describe different aspects of ventilation. Some of these have been mentioned already (and a few discussed in detail), but most of the complete explanations have been left out up until this point in an 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 and more will come later.

Tidal Volume

Tidal volume is the amount of air moved in a given breath. As previously discussed, it ~~sometimes~~ helps to break this concept up into two distinct terms: tidal volume and exhaled tidal volume.[71] 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.[72]

TV varies with the size of the patient and the normal range is 6-8ml/kg IBW. Recall the discussion we already had about **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 TV can range from 4-12ml/kg IBW, depending on the specific situation that we are up against. ~~Enough on that for now~~, we will talk further on that when we get into ventilator strategies.[73]

And just to recap what we already discussed, add in some values, and then demonstrate it again in a slightly different way:



[71] This was back in **Volume Control**

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

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

PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; **s** – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

One more thing to mention here. We have a quantity of air in that last graphic labeled "volume lost between TV and VTe" and this is generally an OK assumption to make (as we discussed before in **Volume Control**), but it isn't always the case because VTe can sometimes exceed TV for various reasons. For example, there is some breath to breath variation with the spontaneously breathing patient or (s)he may forcibly exhale after a breath is given. There is also the idea that cold air from the supply or machine end of the system will warm and expand as it enters the airways and lungs.[74] These are just a few examples, we just wanted to point out that VTe doesn't always have to be less that TV and that reasons for this vary widely.

[74] Flowers & friends, 2019 – And this would be an example of Charles' Law

cmH$_2$O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO$_2$** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

Respiratory Rate

Respiratory rate (RR) describes how many breaths are delivered and/ or taken in one minute. While rate is often considered the value we put into the machine and frequency (f) is the total number of breaths per minute after patient-triggered breaths are considered, we will use RR to describe this concept as a whole moving forward. With that said, know that if we want to decrease RR and the patient is triggering breaths spontaneously, decreasing rate on the vent won't have the intended effect. No need to dwell on that idea now, we'll come back to it later on.

Normal parameters vary by age, but the typical adult rate is 12-20 and pediatric rates are as outlined on our Broselow Tape or by this chart from the PALS Manual:[75]

American Heart Association. life is why | AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

PALS

Vital Signs in Children

Normal Heart Rates* (beats/min)

Age	Awake Rate	Sleeping Rat
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

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

PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; **s** – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

For the detail-oriented people out there, there are some data points missing from this PALS chart. One strategy would be to guess based on available data (i.e. no listed rate for a 9-year-old, but we could assume a value that falls in between the school-aged child range and that for adolescents). Another option is to use this chart we've put together based on the existing data in the PALS chart:[76]

age description	age (years)	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 kid	8 – 9	17 – 25
preadolescent	10 – 12	14 – 23
adolescent	12 – 15	12 – 20
adult	16 and up	12 – 20

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

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

cmH_2O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO_2** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

Minute Volume

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

$$MV = RR \times TV$$

Minute volume or minute ventilation can be abbreviated as MV or VE and is the primary mechanism by which we control **Ventilation**. We will discuss soon how to manipulate both TV and RR to address ventilation in just a bit, so don't worry about that for the moment. A normal MV for the adult patient is often cited at 4 – 8 liters per minute (LPM), but we prefer to use a weight-based calculation so that it applies to all patient sizes:[77]

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

We've chosen to represent that MV is roughly (≈) 100ml/kg IBW/min because that goal is less of a hard-set requirement than a guideline by which we initiate ventilation in most cases. For the majority of patients this calculated value will be adequate, but there are times in which we ought to aim above or below for various reasons. For example, with both pediatric patients and those with **Acidosis**, we will aim higher than that; with others we may tolerate a MV below that value by way of a concept known as permissive hypercapnia.[78] We will eventually get into the details on how we go about making that decision for different patient types, but for now we'll leave it at that.

Last thing: there can be different types of minute volume, just as there were with TV. Minute volume or minute ventilation typically describes what we dial into the machine, then we tag exhaled on to either term (abbreviated MVe) to describe feedback the machine gives us about what the patient breathes out. Lastly there is alveolar minute ventilation (VA) which takes out anatomic dead space from the equation. While alveolar minute volume (another way of describing VA) is an important concept to consider, we base initial goals and calculations on MV or MVe and not on alveolar ventilation.[79]

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

[78] Pruitt, 2007 – We cite this again later when we discuss both the **Obstruction** and **Acute Lung Injury/ Acute Respiratory Distress Syndrome** strategies, but it outlines the idea of this permissive hypercapnia approach to certain patient populations

[79] We do, however, make subsequent changes to address ventilation with these alveolar volumes in mind and we will get to that in **Ventilation**

PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; s – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

Fraction of Inspired Oxygen

Fraction of inspired oxygen (FiO_2) describes the amount of O_2 in the mix of gasses that we push into the patient's vent circuit when we give a breath. 100% O_2 would be an FiO_2 of 1.0; 21% O_2 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. FiO_2 is typically a parameter we dial in directly to the machine, but it can also be calculated based on how much O_2 we put into the machine and how much total air the machine puts out:[80]

$$FiO_2 = \text{total amount of } O_2 \div \text{total amount of air}$$

and then we can use the concept of flow to quantify these this equation:[81]

assume 10LPM of O_2 going in
and 60LPM of total flow

$$FiO_2 = [(10LPM \times 100\%) + (50LPM \times 21\%)] \div 60LPM$$
$$FiO_2 = 34\% \text{ or } 0.34$$

Now there is never really a need to do this sort of calculation, as the machine will allow us to bypass the math and directly provide a chosen FiO_2 as long as our O_2 source is adequate (such as one of those 50PSI adapters like we see on the wall of the ambulance or hospital). And if we do bypass that mechanism by using a low-flow O_2 source (i.e. normal O_2 tubing), each manufacturer has different recommendations as to how we should estimate an FiO_2 based on the settings we have dialed in and the flow of O_2 into the system.

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[80] Reading, 2016; Flowers & friends, 2019 – For more detail on this type of calculation, take a look at this article; also note that this is an application of Dalton's Law

[81] And we won't talk about flow in depth until **Types of Breaths**

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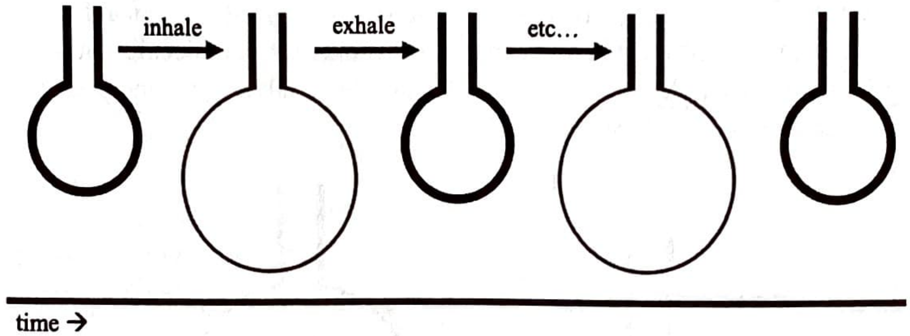
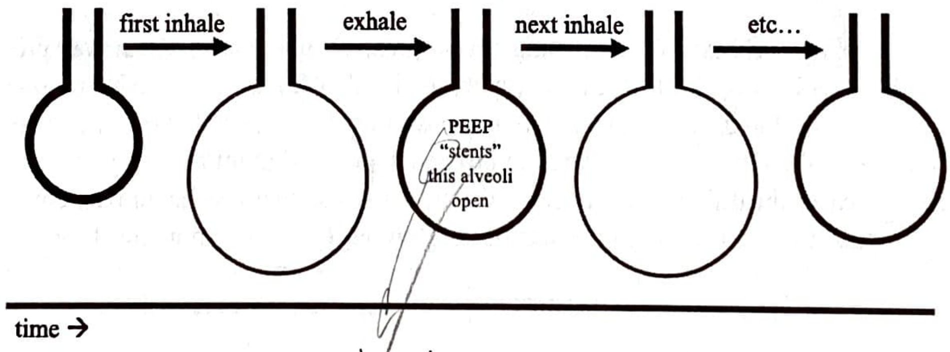
cmH$_2$O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO$_2$** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

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 as a net-zero movement of air where the alveoli go from deflated to inflated and then back to deflated, as so:



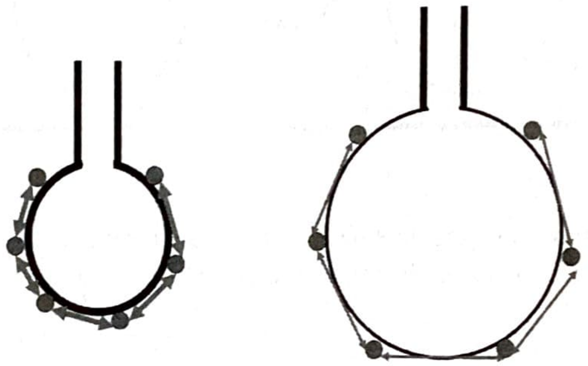
The truth is that we can put pressure into the alveoli and then leave it there to hang out throughout exhalation (in the form of PEEP). So rather than the alveolar air sac deflating all the way back to its original size, it deflates only part way:



PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; **s** – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

Recall from our previous discussion of **Alveolar Surface Area** that the more inflated the alveoli are, the more they can participate in gas exchange. This is due to both more surface area and a thinner membrane across which gas must diffuse.[82] Next, add to that the idea that blood flow though the pulmonary capillary bed is continuous, it doesn't stop when inhalation stops. This means that pulmonary respiration or gas exchange across the alveolar membrane occurs throughout the respiratory cycle, both on inhale and exhale. PEEP helps facilitate this gas process on the exhalation side and then makes it easier to further maximize the effect during inhalation (i.e. a better starting point from which inhalation begins).

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



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

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[82] Desai, 2012 – We cite this again in the section on **Oxygenation** when we return to the idea

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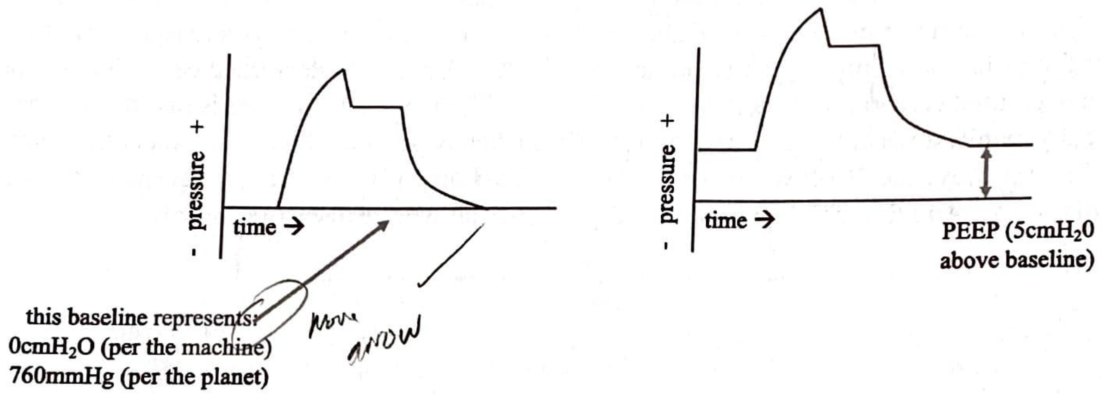
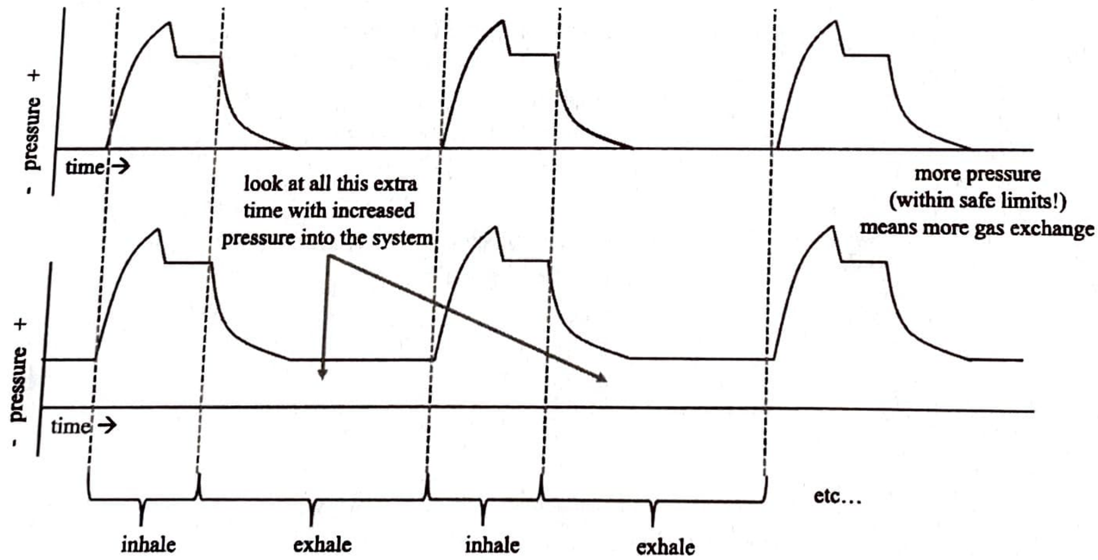
cmH$_2$O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO$_2$** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

To summarize so far: PEEP is a residual pressure that we leave in the alveoli during exhalation to both maximize pulmonary respiration and maintain recruitment of alveoli.[83] So now that we have that clarified, let's look at a waveform representing pressure into the system as we deliver a breath. We've seen this image previously, but now we are going to add to it. The first breath is with no PEEP or zero PEEP or "ZEEP," the second one (right) is with 5cmH20 worth of PEEP added in:



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



[83] Kallet & Branson, 2016 – They explain that PEEP doesn't necessarily open the alveoli as we often hear it described, rather PEEP keeps the alveoli open after inspiratory pressure changes (or **Recruitment Maneuvers**) open them up; also, to review the idea of pulmonary respiration look back to the section on **Terms to Describe Breathing**

PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; s – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

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

Let's next take a look at the downsides of PEEP. The most relevant one to mention is that PEEP can decrease CO.[84] Recall from a previous discussion that any increase in intrathoracic pressure can impede blood flow back to the heart. Because of this, normal PEEP is less than 10cm$H_2$0. That said, we sometimes use PEEP up to 20cm$H_2$0 in specific cases and we will talk about those later. Other negative consequences of PEEP vary widely from things like worsening hypoxia and increased V/Q mismatch to decreased extra-thoracic organ function and decreased cerebral perfusion pressure.[85] That said, the important thing is that these negative effects typically manifest when the application of PEEP is taken beyond the level of therapeutic benefit. To phrase it a different way: use PEEP when needed, but don't assume it is without consequences, and be sure to utilize it judiciously. And the specifics for how we go about that will be discussed shortly.

[84] Clinical Analysis Management, 2019; Strong, 2013 – And this effect of decreased CO due to PEEP isn't so much a thing with a well-hydrated patient, so we can mitigate that somewhat by giving fluids if our patient will tolerate it (and just to clarify, the first reference says euvolemia mitigates this effect, the second says that hypervolemia is needed – that distinction is difficult to make in the field, but the takeaway is that volume fixes the issue)

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

cmH_2O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO_2** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

Inspiratory Time (and I:E Ratio)

The next term to consider is inspiratory time (I-time), which is the amount of time over which we deliver a breath. It varies by age as so:[86]

age description	age (years)	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 kid	8 – 9	0.6 – 1.2
preadolescent	10 – 12	0.7 – 1.4
adolescent	12 – 15	0.8 – 1.7
adult	16 and up	0.8 – 1.7

One idea related to PPV is that the more time we spend pushing air into system, the more O_2 gets moved into the bloodstream. This means that more time spent on the inspiration side of the breath cycle (versus exhalation) equals better **Oxygenation**.[87] With that in mind, one 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 **RR**. Consider seventeen breaths over one minute:

$$60s \div 17 \text{ breaths} \approx 3.5s \text{ per breath}$$

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

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

86 See **Appendix** for how we got all these numbers
87 Discussed again later when we get to **Mean Airway Pressure**

PC – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PSI** – pounds per square inch; **RR** – respiratory rate; **s** – second(s); **TV** – tidal volume; **VA** – alveolar minute volume; **VE** – minute ventilation; **V/Q** – ventilation/ perfusion; **TV** – tidal volume; **VTe** – exhaled tidal volume; **ZEEP** – zero end-expiratory pressure

We often represent this ratio between I-time and expiration time as an I:E ratio to describe the amount of time spent at inspiration in comparison to the amount of time spent at exhalation. A normal I:E ratio is anywhere from 1:2 – 1:3.[88] 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

now we need to simplify the ratio so that one of the numbers is 1:
simply divide both sides by the first number: $\frac{1.5}{1.5} : \frac{2.0}{1.5}$
and solve for our new I:E ratio of 1:1.33

To bring it back home: we had a rate of 17 and an I-time of 1.0s 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 1.5s 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 RR instead of I-time:

per above: RR 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 = 3s per breath

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

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 = 0.8s, then out or exhalation = 3.0s – 0.8s
therefore out or exhalation = 2.2s

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

---

[88] Yartsev, 2019 - To clarify this idea: a normal I:E for the spontaneously breathing patient is in the neighborhood of 1:2, but often times we see something more like 1:3 with vented patients because we leave I-time alone at a default of 1.0s – because of this convention and the facts that it is both common practice and generally well-tolerated, we've stated things as we did and will carry on with this assumption

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cmH₂O – centimeters of water; **CO** – cardiac output; **f** – frequency; **FiO₂** – fraction of inspired oxygen; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **kg** – kilogram; **LPM** – liters per minute; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **MVe** – exhaled tidal volume; **OK** – alright; **PALS** – pediatric advanced life support

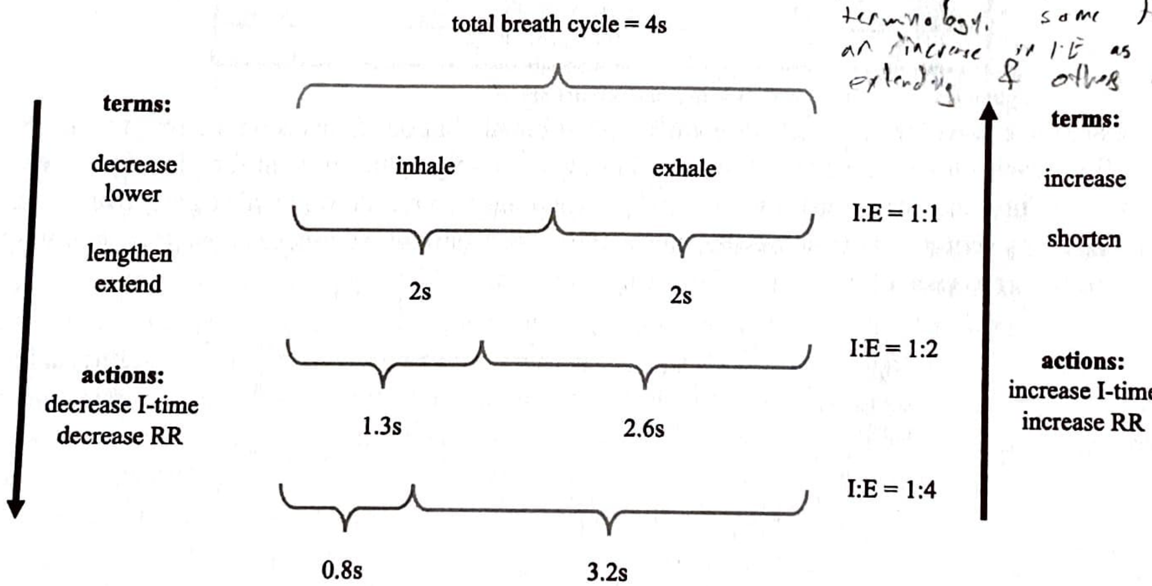
Last thing to touch on is how we describe these different I:E ratios. To make sense of this, consider the I:E ratios as fractions or decimal numbers:

$$1{:}1 = \frac{1}{1} = 1$$

$$1{:}2 = \frac{1}{2} = 0.5$$

$$1{:}4 = \frac{1}{4} = 0.25$$

In this sense, an I:E ratio of 1:2 can be decreased to 1:4 or increased to 1:1. We can decrease I:E ratio by either decreasing I-time or decreasing RR. Likewise, we can increase the I:E via a longer I-time or higher RR. T~~his can be a bit confusing, so sometimes~~ we describe an increase in I:E as a shortening and a decrease as a lengthening – this convention essentially describes the relative length of exhalation as compared to the overall time of the breath cycle.[89]



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 RR; we can lengthen our I:E ratio by decreasing I-time or decreasing RR. A shorter I:E ratio means less time (in relation to the whole in/out cycle) spent on exhalation, a longer or lengthened I:E ratio means more time for exhalation. We will talk about this later when we get to ventilator strategies, but know that some patients can benefit from a shorter I:E ratio and others can benefit from a longer I:E ratio, so it is important to know which changes affect the I:E ratio in which direction.

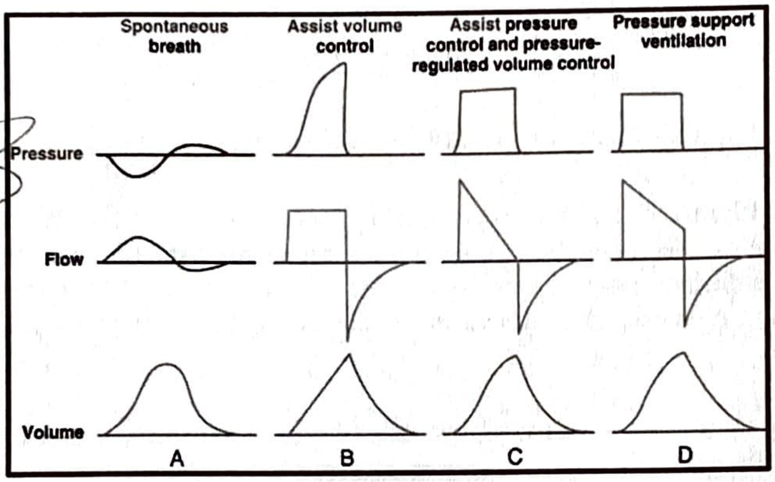
---

[89] ~~We occasionally see this lengthening and shortening concept described in the opposite way, but the manner in which we've shown it here is by far more common.~~

OK – alright; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PS** – pressure support; **RR** – respiratory rate; **s** – second; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

Types of Breaths

Let's take a few minutes to discuss an image we presented towards the beginning of this manual. We want to explain in a little more detail each of the following types of breaths depicted below:[90]



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

90 Fuller & friends, 2014 (image)

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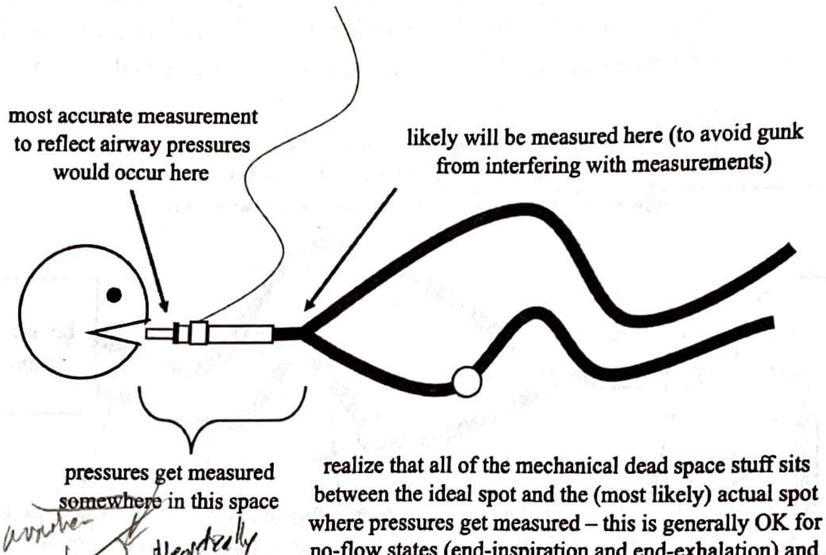
AC – assist control; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **I-time** – inspiratory time; **LPM** – liters per minute; **MV** – minute volume

Pressure is measured by the machine somewhere between the ETT and the wye where the inhalation side of the circuit splits off from the exhalation side of the circuit:[91]

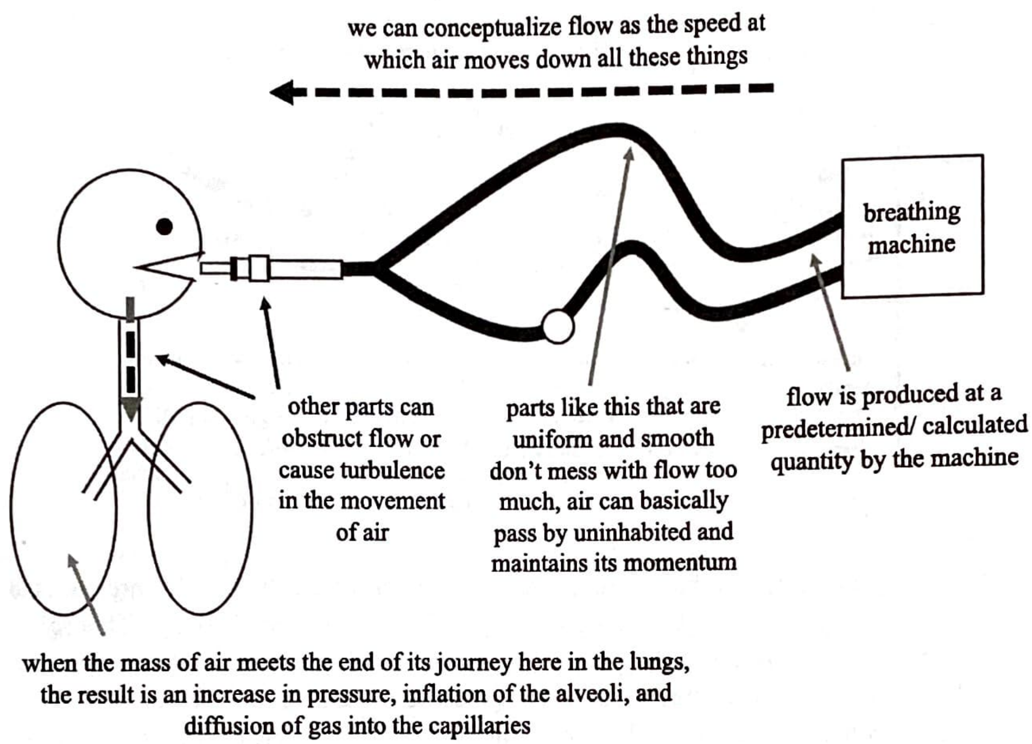


Another thing to mention is that the pressures we measure don't directly describe pressures at the alveoli or terminal ends of the airway, they reflect what's going on outside of the patient's body. That said, we can manipulate the system to approximate alveolar pressures (and we will discuss how to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

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

OK – alright; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PS** – pressure support; **RR** – respiratory rate; s – second; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

Next concept to discuss is flow. Flow is a description of how fast we move air through the system and is quantified in LPM.[92] When we describe flow, we do so at the machine side of the system. As air moves away from the machine, however, different things can interfere with the speed at which the body of air is moving. We create flow and send it out into the universe via the machine, then we see all of this interference indirectly via other parameters (such as pressures and volumes). Here's how it looks mapped out on top of the system:



[92] And sometimes notated by the symbol V̇, but we also use that same symbol in Fick's Law stuff in the next section and don't want to get things confused...

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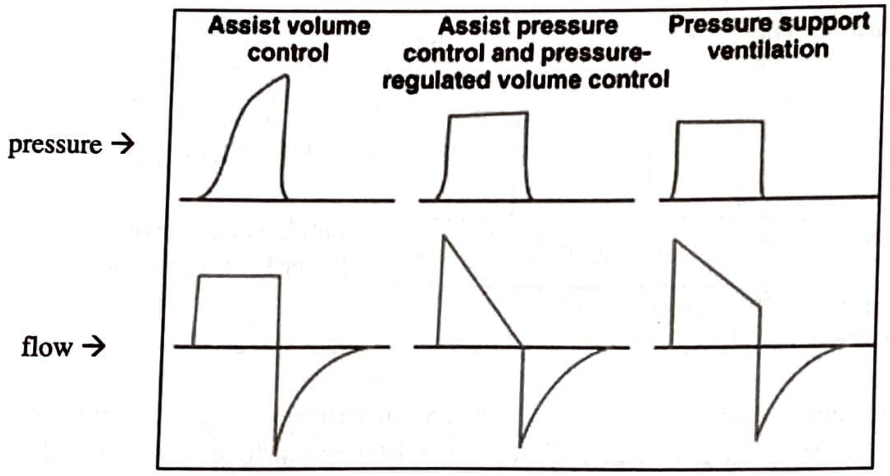
AC – assist control; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **I-time** – inspiratory time; **LPM** – liters per minute; **MV** – minute volume

Now that we are set on the basics of pressure (as measured in the system) and flow (as produced by the machine), let's look at a few of these waveforms again and see how we can deliver breaths in different ways:[93]



The first thing to note is that there are three general categories: VC breaths (left), PC breaths (middle), and PS breaths (right).[94] In VC a breath is most commonly delivered via a square-waveform flow pattern in which the machine spins up to a set flow, holds it for a predetermined amount of time, then cycles off. With PC and PS breaths, however, flow is delivered via a decelerating-waveform flow pattern in which the machine starts a breath by spinning up to a max pressure and then slowly maintaining that pressure by delivering less and less flow until the breath cycles off. To say this all another way: VC gives constant flow for variable pressure, PC and PS give constant pressure at variable flow.

Let's follow this up with a series of sequential facts: There are some machines nowadays that can give VC breaths via a decelerating pattern, but those aren't available in the transport setting.[95] That means we can lump these three types of breaths into two groups: volume/ square flow and pressure/ decelerating flow. Unless we are in VC and SIMV, we ventilate patients with one type of breath at a time. In very general terms: the pressure/ decelerating-waveform breaths are more comfortable for patients but take longer to deliver (i.e. may not be ideal when we need to give breaths fast or allow lots of time for exhalation).[96]

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93 Fuller & friends, 2014 (image)

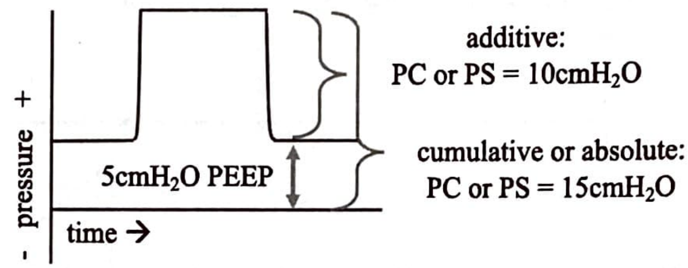
94 Our labels differ slightly from those in the image, but we'll hash all of this out soon; and to review these concepts look back to **Modes** and **Control of Ventilation**

95 Gonzales & friends, 2012 – At least we are pretty sure the option doesn't exist in any transport ventilators, but new products come up all the time; amongst many other fun things, the article explains how pressure/ decelerating-waveform breaths may be best for ARDS patients and volume/ square-waveform breaths may be best for obstruction related to COPD

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

OK – alright; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PS** – pressure support; **RR** – respiratory rate; s – second; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

As for the two types of pressure/ decelerating-pattern breaths (PC and PS), there are a few things to mention. First is that the pressure used to describe these breaths can either be referred to in addition to PEEP or inclusive of PEEP. We describe the value as cumulative or absolute to include PEEP or additive to say it is added on top of PEEP.[97] This varies by machine, so just be aware of it:



Another concept to discuss is rise time. This term describes how fast we get from zero to our set inspiratory pressure (either in PC or with a PS breath). Different machines describe and label this parameter differently, but the general idea is that a shorter rise time (which may be also referred to as rise profile) the faster pressure gets up to what we have set.[98] This isn't something we mess around with too often in transport, but it is good to know if we are troubleshooting issues. Just keep in mind that a higher or longer rise time may mean less TV if I-time is not adjusted (i.e. lengthened) to accommodate that change. And then if I-time changes, we may end up with less time for expiration (i.e. we will have a shorter I:E ratio). Again, not something we routinely mess with in invasive ventilation, but it is good to know.

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

[98] Yartsev, 2019 – For a more detailed discussion of that and how things differ between machines, navigate here

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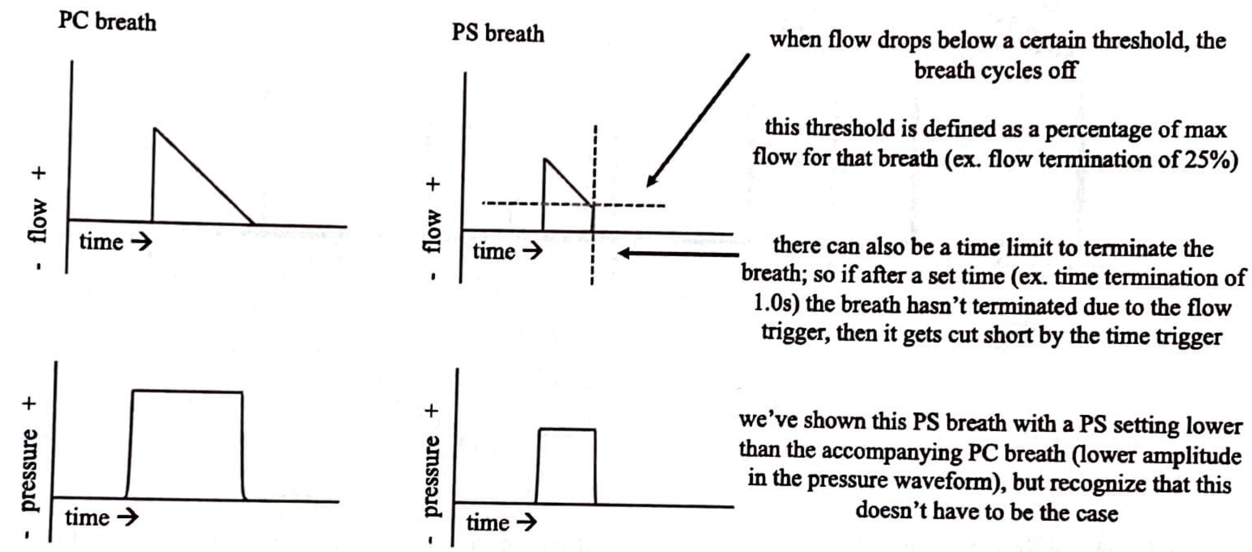
AC – assist control; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **I-time** – inspiratory time; **LPM** – liters per minute; **MV** – minute volume

The next thing to mention here is how PC and PS breaths differ. While both are given via a decelerating-waveform pattern, the mechanism by which the breath cycles off changes things. A PC breath is designed to deliver a full breath even with no patient effort, whereas a PS breath is designed to simply relieve some effort of breathing on the front end of a breath. Because of this difference, a comparable titration of pressure (i.e. a change of 5cmH_2O for both PC and PS) may result in different changes of volume in the very same patient. Now the mechanism by which this works is known as termination, the parameter by which the machine decides to stop supporting a breath and begin exhalation:[99]



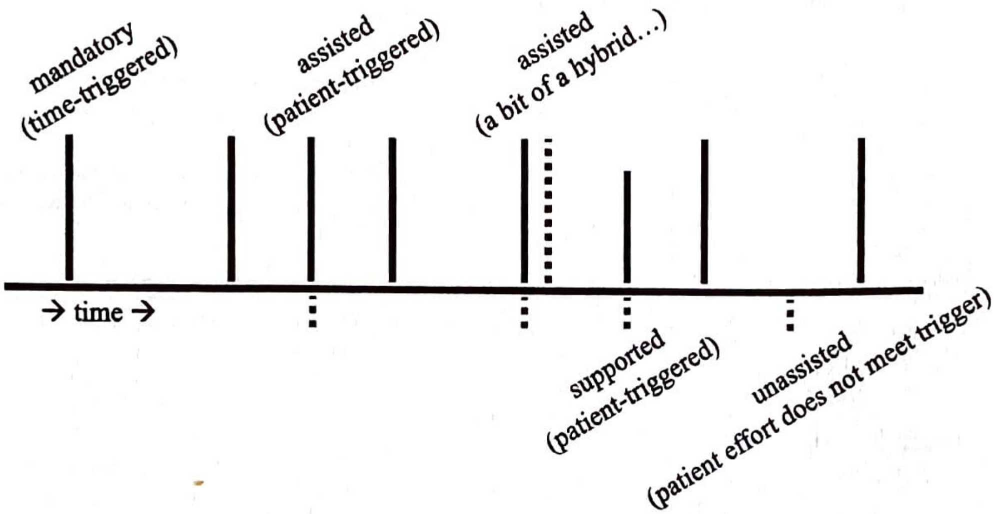
So to get more volume in a PS breath (represented by the area under the flow-time waveform), we either need more patient effort (i.e. don't take away a patient's respiratory drive with too much sedation) or we need to maximize our termination triggers (i.e. lower value for flow termination, longer time termination).[100] We don't typically get that far into the weeds with invasive ventilation and PS, but we will often see this idea discussed in terms of non-invasive ventilation (which, again, we don't cover in this manual).

[99] To expand on this, the term cycle refers to how the machine decides to stop giving a breath in a general sense, termination as we've drawn it out is specific to PS breaths

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

OK – alright; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PPV** – positive pressure ventilation; **PS** – pressure support; **RR** – respiratory rate; s – second; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

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



Mandatory or time-triggered breaths are the ones that we deliver via our set RR on the vent and to a specific goal, whether that be volume or pressure. Assisted breaths are triggered by patient effort and then the machine delivers a full breath to match the same goal as for the machine-delivered ones.[101] Moving right, supported breaths are also patient-triggered, but get delivered via pressure support and not to a set goal. Supported breaths are often smaller than mandatory or assisted ones (in terms of volume), that's why they have been shown with a shorter green line.[102] And lastly is spontaneous effort that doesn't get supported or assisted - these efforts get ignored by the machine and function solely via patient effort.

[101] Chatburn & friends, 2014 – For more on that hybrid situation, take a look here; these guys would identify it as a machine-triggered breath, but since we started our discussion with a differentiation between time- and patient-triggers we will just refer you to their article – the labeling doesn't change our treatment

[102] But again, this doesn't necessarily have to be the case – see section on **Synchronized Intermittent Mandatory Ventilation** for more on this

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AC – assist control; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **I-time** – inspiratory time; **LPM** – liters per minute; **MV** – minute volume

To take this discussion one step further, let's consider which types of breaths apply to which types of ventilation. In AC mode we have time-triggered or mandatory breaths and assisted breaths. In SIMV mode we have time-triggered or mandatory breaths, assisted breaths (when a trigger is sensed within the mandatory period), and supported breaths (when a trigger is sensed in the spontaneous period). In neither mode do we see spontaneous effort that meets the trigger threshold and does not get assisted in some way. While there may be spontaneous effort that doesn't meet the trigger (and this theoretically could contribute some to MV), all noteworthy patient effort (defined by meeting whatever trigger threshold we have set) will get facilitated by the machine in some way in either mode.[103]

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

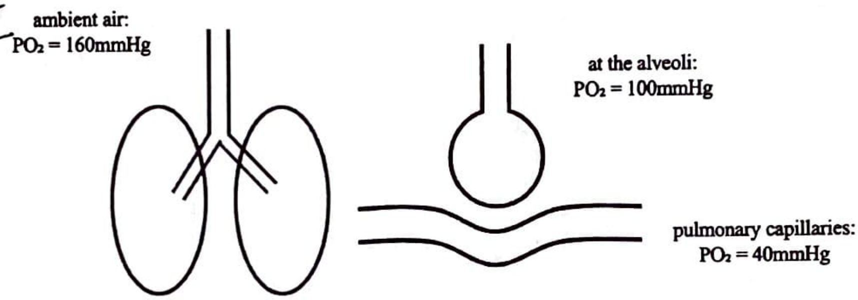
O_2 – oxygen; **PaO_2** – partial pressure of arterial oxygen; **PEEP** – positive end-expiratory pressure; **PO_2** – partial pressure of oxygen; **PvO_2** – partial pressure of venous oxygen; **RBC** – red blood cell; **SpO_2** – pulse oximetry

Three Big Things

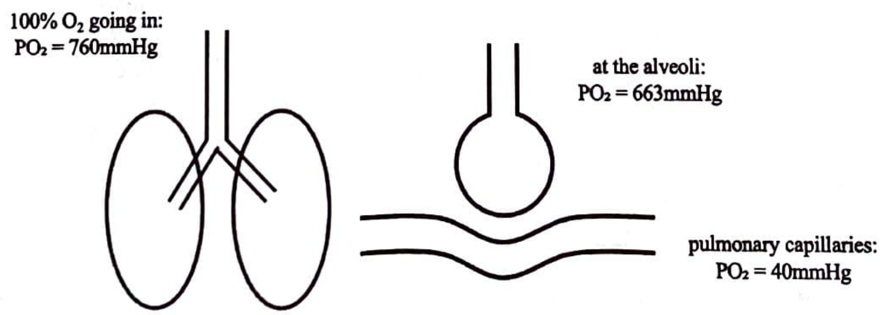
There are three very important things that need to be monitored and addressed for all ventilated patients, hands down and no matter what. The order in which we discuss them here is totally arbitrary as they all hold equal weight and are interrelated. The discussions that follow are in general terms and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

Oxygenation

It may have come up once or twice before, but O_2 is pretty important stuff. O_2 gets to tissues via a few steps, some of those we can affect directly with the ventilator. Let's start with a version of a graphic we used earlier that shows partial pressures at a few steps along the way in the spontaneously breathing patient: [104]



Recognize that gasses will diffuse from areas of high concentration to areas of lower concentration. In this baseline example, O_2 will move from the ambient air to the alveoli, then into the pulmonary capillaries. The first way that we can speed this process up is by changing the partial pressure of O_2 at the start of the system. Instead of 21% of the gas mix or 160mmHg of O_2, we can titrate that all the way up to 100% (**FiO_2** 1.0) or 760mmHg. This will increase the rate at which O_2 diffuses to the alveoli, resulting in a higher partial pressure of O_2 downstream and, subsequently, faster diffusion into the bloodstream:



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[104] Bauer, 2016c – Take a look at this short article for a review of Henry's Law and the concept of PO_2 at the pulmonary capillaries, offers a preview of how we can improve oxygenation in the ventilated patient (the steps of which we will lay out in this section)

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ABG – arterial blood gas; **CO** – cardiac output; **CO_2** – carbon dioxide; **FiO_2** – fraction of inspired oxygen; **Hgb** – hemoglobin; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **mmHg** – millimeters of mercury

Let's recap this bit and do some math: PO_2 at the alveoli on ambient air is 160mmHg, PO_2 at 100% FiO_2 is 663mmHg. To quantify the result of this difference let's apply Fick's Law:[105]

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

$\dot{V}$ = rate of gas diffusion across a membrane (i.e. at the alveolar membrane)
P_1 = ingoing pressure (i.e. to the alveoli)
P_2 = pressure at other side (i.e. in the blood)
area = self-explanatory…
D = diffusion constant for a particular gas (O_2 in this case)
thickness = also self-explanatory…

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

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

and let's add in some numbers for the ambient air and 100% FiO_2 situations:

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

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

That means that O_2 diffusion occurs ten times faster at 100% (or a FiO_2 of 1.0) than at room air. Which is both nuts and a clinically significant thing to be aware of. The takeaway here is that whenever we need to increase the diffusion of gas across the alveolar membrane, FiO_2 is a heck of a way to get that done. The holdup is when other factors in the equation (area and thickness) are also issues or if the problem is with O_2 transport after the point at which it diffuses into the blood. In those cases we may need to augment this strategy with other techniques, as we will discuss real soon.[106]

Another thing to mention here is that O_2 can cause damage when given in excess of physiologic need for a sustained amount of time. Now the timeline at which the bad stuff can occur is likely longer than our transport, but that doesn't mean we need to get reckless and ventilate everyone with an FiO_2 of 100%. We'll talk in just a moment about how we evaluate oxygenation and the idea is to make sure a need is met while being conscious that all interventions, even something as seemingly benign as O_2, have consequences.[107]

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[105] Desai, 2012 – Best ever explanation of this concept courtesy of Khan Academy

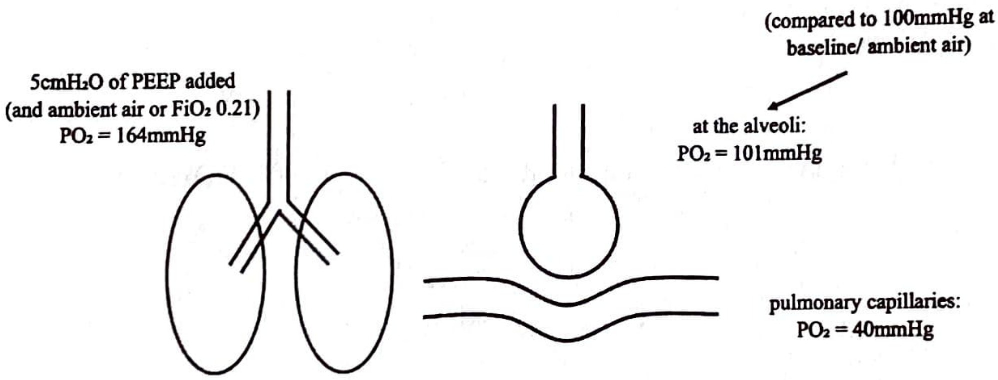
[106] Murphy, 2017b; Macintyre, 2014 – And to review the different types of hypoxia, take a look at this video (lots of detail, reviews the four types as we often label them in critical care transport) and that article (different system of considering the various causes, but equally informative)

[107] Kallet & Branson, 2016 – This article looks at both why it may make sense to limit oxygenation and how the negative consequences of O_2 may be exaggerated

O_2 – oxygen; PaO_2 – partial pressure of arterial oxygen; **PEEP** – positive end-expiratory pressure; PO_2 – partial pressure of oxygen; PvO_2 – partial pressure of venous oxygen; **RBC** – red blood cell; SpO_2 – pulse oximetry

To expand on this idea just a bit before we move on, one specific argument against a high FiO_2 is the idea of absorption atelectasis – the closing of alveoli related to nitrogen washout and the fact that O_2 quickly diffuses into the bloodstream leaving less gas in the alveoli.[108] While the clinical impact of this theoretical consequence is questioned by some, it is worth keeping in mind.[109] And if we do give credence to the idea, ways to mitigate this effect would be maintaining a patient's spontaneous effort to breath (discussed shortly in **Comfort**) and performing **Recruitment Maneuvers** (discussed much later).[110]

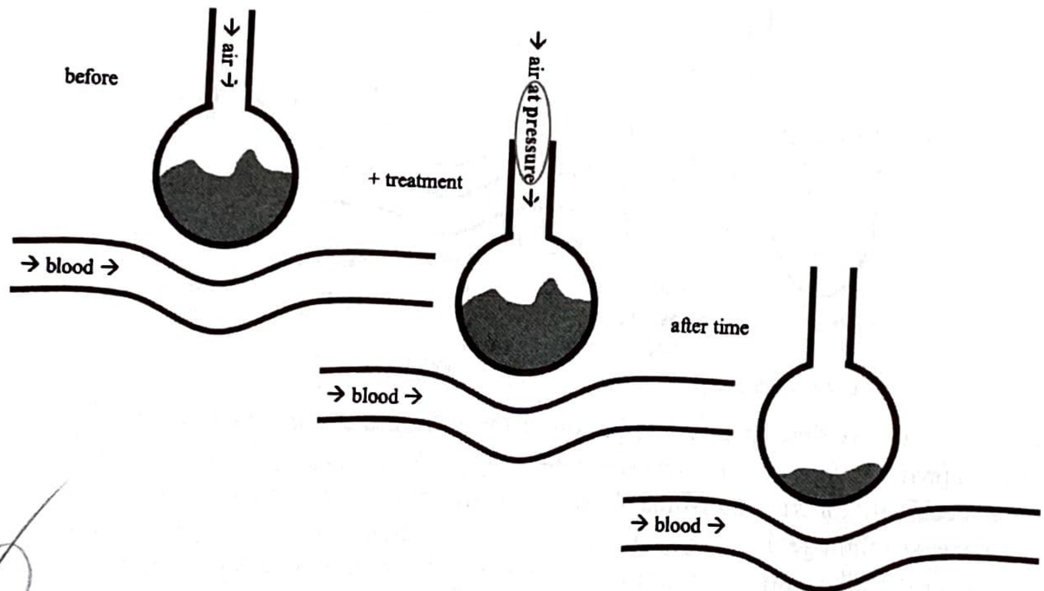
Increasing FiO_2 is one way to get more oxygen into the bloodstream. While this isn't the fix for all types of hypoxia and there are some potential negatives, we generally start here when looking to address oxygenation issues. The next way we can increase oxygenation is via **PEEP**. Now PEEP doesn't quite work by the same mechanism, as the addition of PEEP doesn't much change the partial pressure situation as we saw with an increase in FiO_2:



[108] Dunphy, 2012 – Short video that explains this mechanism and how patient effort can mitigate the effect
[109] Yartsev, 2019 – Also describes some of the other mechanisms by which O_2 can adversely affect our patients
[110] Hartland & friends, 2015 – The article outlines an argument for the use of these maneuvers in certain patients (which seems reasonable to extrapolate to some of the patients we see in the transport setting)

ABG – arterial blood gas; **CO** – cardiac output; **CO_2** – carbon dioxide; **FiO_2** – fraction of inspired oxygen; **Hgb** – hemoglobin; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **mmHg** – millimeters of mercury

Instead, PEEP facilitates oxygenation primarily by increasing and thinning out the alveolar surface throughout the respiratory cycle. We discussed this concept way back in the section on **Alveolar Surface Area** and again just a moment ago in the section on **PEEP**, so no need to redo all of that here. One more mechanism by which PEEP helps oxygenation is that it cleans up the alveolar membrane, in a sense, by pushing out or displacing fluid that accumulates there. Think of it this way:



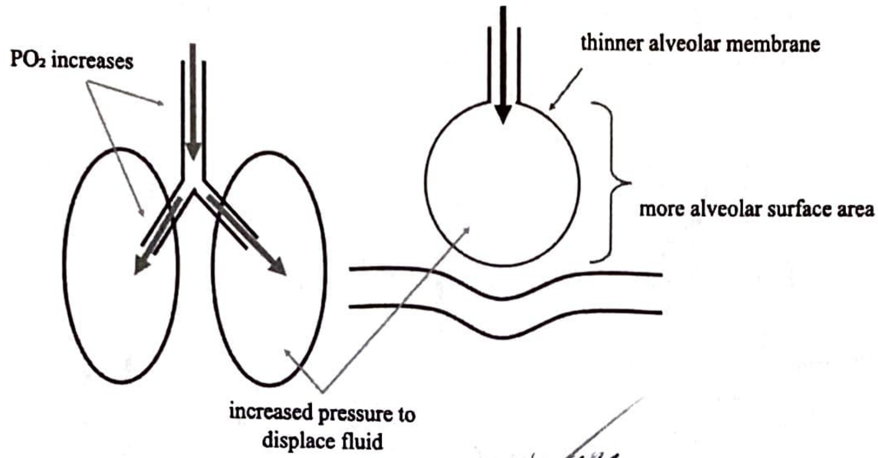
So assuming **Ventilation** and **Comfort** are adequate (see next sections), initial steps to fix oxygenation are increasing FiO_2 and then adding PEEP. While it's totally OK to use a stepwise approach that titrates both FiO_2 and PEEP in line with one another, recognize that FiO_2 is our most direct fix for improving partial pressure of O_2 at the alveoli and has few consequences in the acute (i.e. short term) setting.[111] PEEP, on the other hand, is especially helpful in facilitating gas exchange across the alveolar membrane and driving fluid out of the lungs, but may decrease CO by way of a drop in preload to the heart (especially if our patient is down on fluids).[112] Lastly, both of these techniques (FiO_2 and PEEP) improve oxygenation throughout the respiratory cycle.

[111] We'll touch a bit more on this subject in the section on **Acute Lung Injury/ Acute Respiratory Distress Syndrome** later on

[112] This was discussed in both **How is PPV Different?** and **Positive End-Expiratory Pressure**

O_2 – oxygen; PaO_2 – partial pressure of arterial oxygen; **PEEP** – positive end-expiratory pressure; PO_2 – partial pressure of oxygen; PvO_2 – partial pressure of venous oxygen; **RBC** – red blood cell; SpO_2 – pulse oximetry

The next point to make is that all of the benefits of FiO_2 and PEEP (in the context of oxygenation) are further maximized during inspiration:



This means that more time spent at inspiration further maximizes oxygenation, therefore strategy number three to improve oxygenation is to increase the **I-time**. And changing I-time consequently changes our I:E ratio. More specifically, increasing I-time shortens our I:E ratio. For example, if we have an I:E of 1:2 and then increase I-time we might get an I:E of 1:1. And then 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. The primary drawback of a long I-time (and of an inverted I:E ratio) is that it can be uncomfortable for our patients and we will need to get aggressive to maintain patient **Comfort**. An inverted I:E may also make it tough for the patient to exhale fully, predisposing us to **AutoPEEP**.

Summary up to this point is that there are three ways to improve oxygenation by adjusting settings on the vent: increase FiO_2, add PEEP, and extend I-time. Now why do we not just fill the lungs up with 100% O_2 and keep them inflated – we'd have a forever-long maximum diffusion of oxygen into the bloodstream, right? There are two reasons for this. One is that we don't want to affect hemodynamics indefinitely (as discussed above and previously). Two is that it isn't all about O_2. We also have to consider its partner in crime, CO_2, which doesn't diffuse so well in gas (as compared to O_2) because it is a bigger, heavier molecule.[113] The movement of CO_2, therefore, is partially dependent on the 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.

Other things we can do to improve oxygenation include sitting our patient upright or elevating the head of bed,[114] ensuring adequate perfusion, utilizing more lung volume via **Recruitment Maneuvers**, and considering **Mean Airway Pressure**.[115] We won't get into the details of all of these things here, as the focus for now is on how to manipulate the machine.

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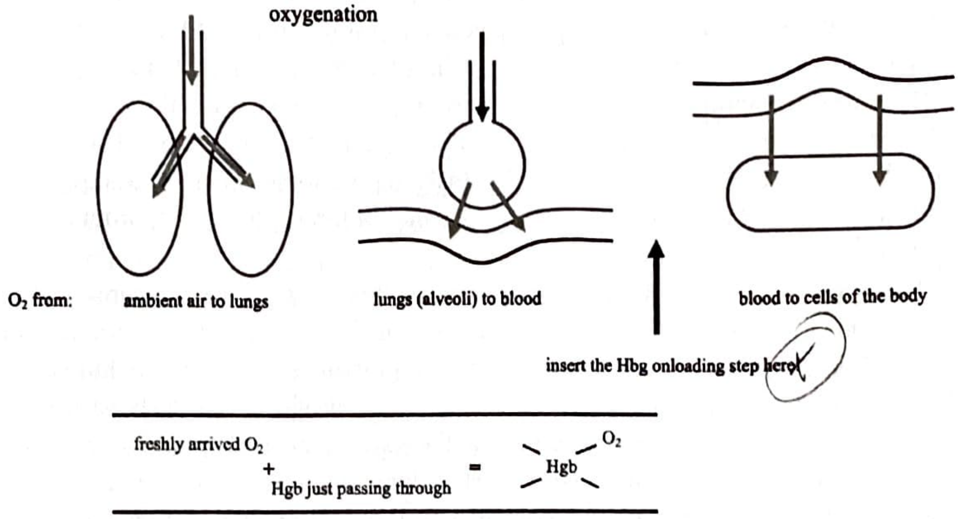
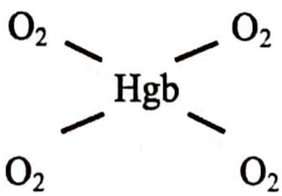
[113] Flowers & friends, 2019 – Another application of Graham's Law in the clinical setting
[114] Spooner & friends, 2014 – While this article mainly focuses on lung volumes (which is a step or two removed from oxygenation), it goes into detail on the physiologic effects of elevating the vented patient's head
[115] While we could also make the argument that going up on RR increases ~~both~~ the amount of time spent on inspiration, doing so also impacts ventilation (next section) so we generally don't consider RR one of the variables by which we control oxygenation

ABG – arterial blood gas; **CO** – cardiac output; **CO$_2$** – carbon dioxide; **FiO$_2$** – fraction of inspired oxygen; **Hgb** – hemoglobin; **I:E** – inspiratory to expiratory: **I-time** – inspiratory time: **mmHg** – millimeters of mercury

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 O$_2$ (or oxygen-like things, but we won't worry about the tricky parts here).[116] The process here goes like so: O$_2$ gets to the alveoli, it crosses into the bloodstream via diffusion gradients, then once in the bloodstream it gets picked up by hemoglobin (Hgb) for a ride down the blood vessel. Let's draw out the onloading process:



So we have a Hgb with four seats free, 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:



Drawing it out this way is a bit of a simplification, but it does help us to understand what it is that SpO$_2$ is looking at. Do note that Hgb doesn't cruise freely through the vessels, it comes attached to red blood cells (lots and lots of Hgb per each RBC), but the four seats per Hgb is a fair description. Also consider that we measure this saturation peripherally (hence the p in SpO$_2$). This means that if blood isn't getting to the periphery where we have our little probe attached, numbers may not be accurate. And lastly, recall that as O$_2$ binds to that first seat on the Hgb train the physical shape of the Hgb molecule changes to attract subsequent O$_2$ molecules to the remaining vacant seats. This is why we aim for higher SpO$_2$ values over 93% - once we get those Hgb molecules mostly filled up, it makes it way easier to fill up the remaining ones.

116 Silverston, 2016 – Short article that describes both the technology and the limitations of SpO$_2$

O_2 – oxygen; **PaO_2** – partial pressure of arterial oxygen; **PEEP** – positive end-expiratory pressure; **PO_2** – partial pressure of oxygen; **PvO_2** – partial pressure of venous oxygen; **RBC** – red blood cell; **SpO_2** – pulse oximetry

To carry on with this idea: when we get to 100% saturation, all further oxygen we put into the system will remain as dissolved O_2 molecules in the blood. This has the potential to cause damage (as we've discussed before), so we tend to titrate SpO_2 to an upper limit of 99%. We don't normally take it much further than this in transport, but there are some programs that have the ability to measure blood gasses, so let's just touch on that for a moment. Partial pressure of arterial oxygen (PaO_2) allows us to see how much O_2 is dissolved in the bloodstream on the arterial side of the circulatory system. If we have an SpO_2 of 100% and a normal PaO_2, then our potential for causing damage is less than if we had a saturation of 100% and a markedly elevated PaO_2.

There are, however, many things that impact this relationship. Different factors can change Hgb's affinity for O_2 (and CO_2) and we can better understand values for SpO_2 and its relationship to PaO_2 by considering this affect. For example, we may see a low SpO_2 paired with a normal PaO_2 to indicate that Hgb isn't holding on to oxygen as well as normal. This gets a bit beyond the scope of our discussion, but we'll return back to this idea later on when we talk about **Acidosis** to give some concrete examples and clinical application.[117] Just keep in mind that PaO_2 provide as snapshot in time, while SpO_2 provides a continuous stream of information.[118]

One very last thing about this and then we'll get on to other things: There is some potential for utilizing partial pressure of venous oxygen (PvO_2) in the transport setting. PvO_2 samples are normally mixed-venous samples from a central line, but you could measure the partial pressure of O_2 from any blood source, to include venous blood from a normal venipuncture. Now blood on the venous side has already passed the capillary beds and therefore is dependent on delivery, metabolic need at the tissues, and offloading, so we would need to keep those things in mind. And in fact, there have been investigations into how we could use a peripheral blood gas to direct treatment if we do keep these other components in mind.[119] It's not that common at this point, but neither are arterial sticks in transport – using a PvO_2 could be a bridge to fill that gap.

One last summary before moving on from oxygenation. Oxygenation is one of the three big things in mechanical ventilation. We measure it via SpO_2, which tells us how filled up with O_2 the Hgb (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 things around on the vent interface, we have three options and we typically do them in this order: increase FiO_2, add PEEP, lengthen I-time. All that said, let's not forget the basics: position the patient appropriately, verify that perfusion is adequate, and make sure ventilation and comfort are addressed simultaneously (see next sections).[120]

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117 Hasudungan, 2018 – And to dig more into this idea, refer to this video (it also applies to the following section)
118 Farkas, 2016 – In fact, this piece outlines a number of situations in which SpO_2 might be preferred to the ABG
119 Chemtob & Moller-Sorenson, 2018 – These guys looked at the use of a peripheral value in comparison to a central-venous sample and found some correlation to indicate that a peripheral value might be used to guide and direct treatment
120 And to link to these other ideas: **FiO_2**, **PEEP**, **I-time**; **Ventilation**, **Comfort**

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%TaDP – percentage of time at decreased preload; CO_2 – carbon dioxide; $EtCO_2$ – end-tidal carbon dioxide; IBW – ideal body weight; kg – kilogram; min – minute; ml – milliliter

Ventilation

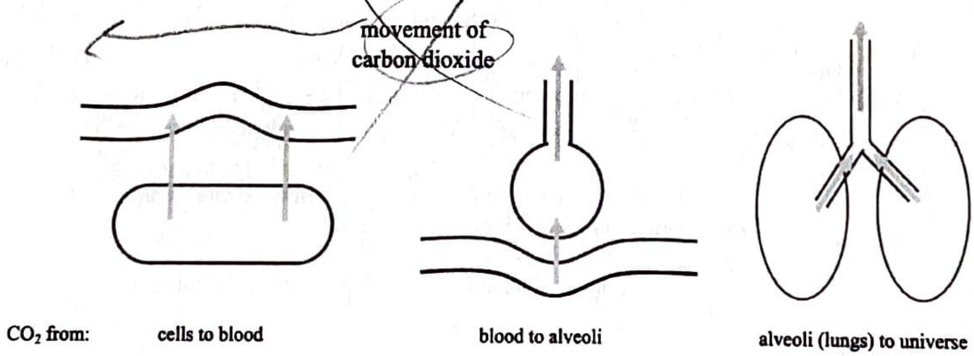
The next very important, big thing is ventilation. Ventilation refers to the movement of air in and out of the system as we both deliver breaths and allow exhalation. As discussed before, this is vitally important for the movement of CO_2. Too much CO_2 hanging out in the lungs with no escape is bad news, so we can't just focus on getting O_2 in. And while we typically think of CO_2 in the context of acid-base analysis, recognize that there are a great many reasons to keep it well-controlled.[121] 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 MV and $EtCO_2$.

If we math it out, our minute volume goal for the typical patient should be:[122]

$$MV \approx 100 ml/kg\ IBW/min$$

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

Concurrently, we also use $EtCO_2$ to monitor ventilation. When the body uses up O_2 at the tissue level, it kicks back CO_2 into the bloodstream. That CO_2 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 O_2 moves through the system:



The value we get on our quantitative $EtCO_2$ reading is a function of all of these factors. The standard approach to managing ventilation with $EtCO_2$ is to use a base range and adjust MV 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 CO_2, values below range require us to read the next paragraph carefully.

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[121] Yartsev, 2019 – And why we won't take the time to get distracted by them all here, take a look at this page for more details on this idea

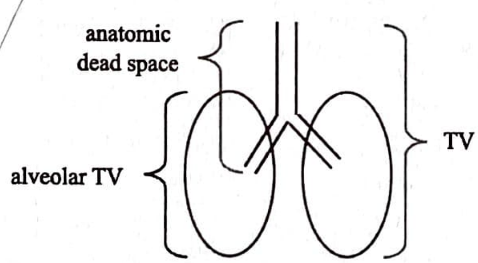
[122] And we discussed where this number comes from previously in the section titled **Minute Volume**; and see section on **IBW** to review ~~that~~

mmHg – millimeters of mercury; **MV** – minute volume; O_2 – oxygen; **PEEP** – positive end-expiratory pressure; **RR** – respiratory rate; **TV** – tidal volume; **VA** – alveolar minute ventilation

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

We can navigate this whole situation by managing ventilation by looking at both ~~minute volume~~ MV and $EtCO_2$ instead of just $EtCO_2$. 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 involved. ~~So to~~ summarize: we measure ventilation using both a calculated MV goal and $EtCO_2$. The MV goal, which is considered a minimum in most cases, is around 100ml/kg IBW/min; normal $EtCO_2$ is 35-45mmHg.

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



TV 450ml		MV = 6750ml/min
- anatomic dead space 150ml	x 15/min	
alveolar TV 300ml		VA = 4500ml/min

lets do it this way:

- to address ventilation, Δ MV (& MV = RR x TV)
- our thought on this
  ↑ MV w/ TV } as outlined
  ↓ MV w/ RR
- f/u on that: V_D A's and TV; Pav & O_2 idea
- summary: do whatever :)

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%TaDP – percentage of time at decreased preload; CO_2 – carbon dioxide; $EtCO_2$ – end-tidal carbon dioxide; **IBW** – ideal body weight; **kg** – kilogram; **min** – minute; **ml** – milliliter

Now assume we need to increase VA by an arbitrary value of 300ml. We could do this by either of two ways: increasing RR to 16 or increasing the TV of each breath by 20ml.[123] While either method is just fine mathematically, adding in an extra breath is a bit less efficient and puts more stress into the system. That stress comes in a few different forms, but we'll get to all of those later.[124] And here's how the math would look in either case:

increase in RR → more time spent during inspiration (which may theoretically be OK for oxygenation issues, but we prefer to keep ventilation and oxygenation independent in our vent changes)

case one: increase RR

TV 450ml
- anatomic dead space 150ml
----
alveolar TV 300ml **x 16/min**

MV = 7200ml/min
VA = 4800ml/min

case two: increase TV

TV 480ml
- anatomic dead space 150ml
----
alveolar TV 320ml **x 15/min**

MV = 7050ml/min
VA = 4800ml/min

MV is different (i.e. more dead space in case one)

VA is the same

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

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[123] Yartsev, 2019 – A few things about this argument (which are addressed in the linked page and discussed even further in the references mentioned there): while anatomic dead space is correlated with TV delivered and not a weight-based value, there is some credence to the idea that increasing TV as we've shown doesn't much affect anatomic dead space; lots of factors are involved in this process and the best way to know for sure would be to use volumetric capnography to quantify dead space for a given patient and then again after changes are made, but this isn't something most of us have access to in transport; we'll work on this assumption by convention and because it simplifies things for us, just know that there is more to it if we dig deeper

[124] More breaths means more %TaDP (a made-up term discussed in the **Hypotension** strategy) and an extra inflation/ deflation cycle which can put stress on the alveoli (discussed already in **PEEP** and again later on in **Driving Pressure**)

PS – pressure support; **RASS** – Richmond agitation-sedation scale; **SIMV** – synchronized intermittent mandatory ventilation; **VC** – volume control

Comfort

The third very important parameter that we need to consider with vent management is patient comfort. On one hand, if our patient is not comfortable (i.e. fighting the vent or out of synch), then the therapeutic effects that we want will be more difficult to attain. This asynchrony can also lead to increased airway pressures (due to the development of **AutoPEEP**), which then leads to more problems downstream.[125] And one more thing: it's kind of rude to shove a 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.

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

TABLE 3. RICHMOND AGITATION-SEDATION SCALE

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

Procedure

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

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[125] And we first mentioned this idea in **Modes of Ventilation**, specifically when we talked about **Assist Control**

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I:E – inspiratory to expiratory; **I-time** – inspiratory time; **NVPS** – nonverbal pain scale: **PC** – pressure control; **PPV** – positive pressure ventilation

TABLE 1. NONVERBAL PAIN SCALE

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

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

Reprinted by permission from Reference 15.

On the other hand, however, a completely sedated patient making no effort to breathe on his or her own suffers 100% of the negative consequences of PPV.[126] Maintaining patient effort and supporting it appropriately with the machine decreases the degree of all those bad things we previously discussed, shortens clinical course,[127] and helps improve both **Ventilation** and **Oxygenation**.[128] Furthermore, having access to subjective feedback from the patient (effort to breath, movement, response to stimuli, etc.) allows us to better monitor whatever else is going on. Because of this, sedation to the point of no spontaneous effort to breathe (or even paralysis, for that matter) should be a last resort for nearly all ventilated patients; instead, we should attempt to maintain comfort to a controlled level by both analgesia and sedation (which, just to clarify, are two distinct concepts).[129]

[126] Which we discussed in **How is PPV Different?**

[127] Ghamloush & Hill, 2013 – While this article focuses on SIMV and how we maybe ought to get over our love for the mode and move on to better things, it touches on the idea of synchrony ~~in a general sense~~ along the way

[128] Mauri & friends, 2017; Macintyre, 2014 – The first discusses how to navigate the benefits of spontaneous breathing in the vented patient with potential consequences; the second outlines how comfort can decrease oxygen consumption in the vented patient

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

PS – pressure support; RASS – Richmond agitation-sedation scale; SIMV – synchronized intermittent mandatory ventilation; VC – volume control

Just to be clear, there are some patients who will need to be completely sedated and/ or paralyzed. In the transport setting, this often comes up in the context of safety concerns. A patient who has previously been difficult to restrain or who has already self-extubated may need to be paralyzed to ensure safe transport. There may also be the argument that breathing spontaneously or making the effort to do so increases oxygen consumption and could exacerbate certain conditions. So while we prefer not to paralyze anyone, it may very well be in his or her best interest for us to do so. Just remember that we will still need to consider discomfort (and pain in particular) and address it appropriately. In those cases, we can make a subjective impression about patient comfort by looking at physiologic changes as outlined on the bottom two lines of the NVPS score.

Now let's move on to differentiate between the ideas of synchrony and compliance. Synchrony is when the ventilator's efforts are in line with the patient's respiratory effort.[130] Asynchrony, therefore, would be when the patient wants a breath in a given instant, but the machine decides to give a breath some other time. Compliance, on the other hand, is often used to describe how well the patient follows the lead of the ventilator. A paralyzed patient is for sure compliant, but that doesn't mean it should be the goal we aim for. Rather we should strive for synchrony and let the patient take the lead on things, adjusting settings along the way to match mechanical support to patient cues.

The strategy to address comfort for the vented patient is to treat the extreme end of discomfort using drugs (both analgesia and sedation) and then do what we can to optimize synchrony on the vent itself once the patient is comfortable enough to respond to more fine-tuned settings. To begin this fine-tuning, we first want to make sure that patient effort to breathe is supported by the machine. We'll talk about **Triggers** later, but the general idea is that we don't want to ignore patient effort, and we also don't want to send breaths down the circuit accidentally. We may have to trial different thresholds and types of triggers until we find what best suits the patient and that's completely OK.

Another thing we can do is adjust **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. One thing to keep in mind is that a normal I:E is 1:2 and that a ratio closer to 1:1 is common at exercise. While the link from exercise to acute illness may or may not be valid, this could mean that the higher ratios we commonly end up with on the vent due to how settings get auto-calculated may predispose our patient to discomfort.[131]

Switching mode or control may also help in the discomfort situation. We mentioned this already, but different **Types of Breaths** are delivered differently in each mode or method of control, and sometimes one may work better for the patient. Discomfort is a completely valid reason to switch from VC to PC (or vice versa) or to move from AC to SIMV (again, or vice versa).[132] We will outline a few cases in which one style of ventilation may be preferred over another, but barring a specific reason not to do the swap this is one way we can attempt to address comfort by changing settings on the vent. And specifically to **SIMV**, if we have a patient triggering breaths we can vary PS and see how (s)he responds. We mentioned already that it is the custom to have PS breaths be smaller than mandatory or assisted ones, but that doesn't have to be the rule.

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[130] Goligher, 2017 – This article (which we referenced before) is brief, but gets into the weeds on some of the more subtle concepts in this discussion of synchrony

[131] Johnston, 2017 – This article looks at this idea in terms of a fraction of I-time over expiratory time and is geared to pulmonary function tests and exercise physiology

[132] And refer back to **Modes of Ventilation** and **Control of Ventilation** to brush up on these ideas

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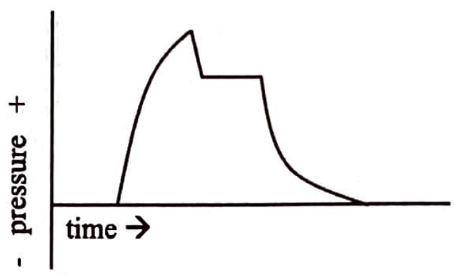
I:E – inspiratory to expiratory; **I-time** – inspiratory time; **NVPS** – nonverbal pain scale; **PC** – pressure control; **PPV** – positive pressure ventilation

Now let's summarize our approach to patient comfort, the third of the three big things to be addressed for all vented patients. Our goal in addressing comfort is to maximize synchrony and this includes both matching ventilator effort with patient need and maintaining the patient's spontaneous effort. To help gauge our efforts, a scoring system should be utilized. Extremes of discomfort get treated pharmacologically with both analgesia and sedation. After that, however, we can fine-tune ventilator settings to further maximize efficacy. Specific strategies include adjusting triggers, changing I-time to yield to a shorter I:E ratio, trialing a different mode or method of control, and trialing higher values of PS if in SIMV mode.

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

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 from the values previously discussed because they are dependent on other things. We don't typically dial them into the machine, but rather we measure them to assess how things are coming along with the values we are able to control. To help clarify these ideas, 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 VC ventilation:



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

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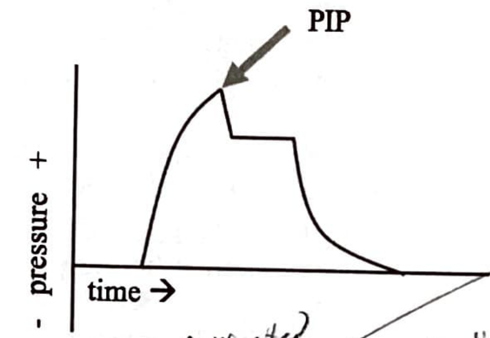
ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

Peak Inspiratory Pressure[133]



Peak inspiratory pressure (PIP) is the highest point on this waveform. It represents the maximum pressure as we deliver a breath into the system. It is also known as peak pressure (Ppeak). PIP is a function of both how we deliver a breath via the machine and how easily that breath can get from the machine down to the lungs. A normal PIP is <35cmH20. An isolated PIP that is too high generally won't cause damage to the patient, rather it likely indicates something gone wrong in the system. This is particularly relevant when we have a normal PIP that then becomes elevated. In these cases it is important to seek out the cause and fix the underlying issue.

On the machine end, PIP is the result of flow, which (recall from our section on **Types of Breaths**) essentially describes how fast we push air to achieve a breath. We sometimes can't manipulate flow directly on transport ventilators, so to decrease PIP by pushing buttons on the machine we have to make things happen in a roundabout way. Which isn't ideal and the truth of it all is that most of the PIP issues we face are due to pathophysiology or equipment issues, so let's just skip right on ahead to how we can decrease PIP via other mechanisms outside of the vent itself.[134]

Causes of an elevated PIP include things like secretions in the ETT tube, bronchospasm, patient discomfort, mainstem intubation, pneumothorax, pulmonary edema, etc. Any time we see a high PIP we ought to try and identify a cause.[135] Once that cause is identified, then we can decide whether or not an action is needed. For example, a high PIP due to secretions should get suction and a high PIP due to a pneumothorax should lead to decompression; on the other hand, a high PIP due to a small ETT may be acceptable. The PIP in this last case represents an impediment to airflow due to the ETT and not the patient's anatomy, so we may decide to leave it alone (especially is there is good reason for that small ETT, such as airway swelling).

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133 Nickson, 2019a – Short article that provides another good review of both PIP (this section) and Pplat (next section)
134 But for the curious folks out there: in VC flow is determined from TV and I-time; in PC (and with PS breaths) it is a function of pressure and rise time
135 And one part of how we do that is by assessing **Plateau Pressure** (next section) – and we have this all drawn out in a flowchart later, but first we need discuss all the terms and concepts first (see **Watching Pressures**)

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

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

To measure PIP we simply need to look at the vent display. Most machines will either give us the value of PIP or show a barometer 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 us when a certain pressure is reached. That said, there is one critical thing to know about this: yes it will tell us 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 our patient and we will get into that a bit more later on.[136]

So in summary, PIP represents the maximum pressure as a breath is delivered by the machine. A normal value is <35cm$H_2$0 and we measure it by looking at the feedback on the vent interface. Potential causes include too much air, too much flow, small or kinked ETT, patient discomfort, secretions, pneumothorax, mainstem ETT placement, and bronchospasm. While there are subtle ways to address a high PIP that develops after placing a patient on the vent, interventions should focus instead on airway issues and comfort.

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

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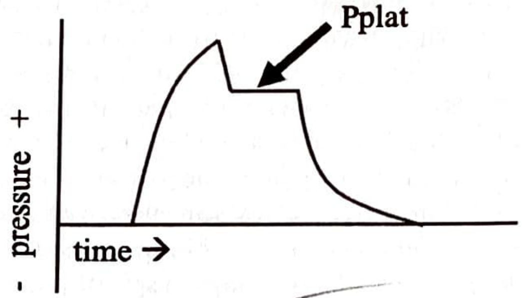
ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

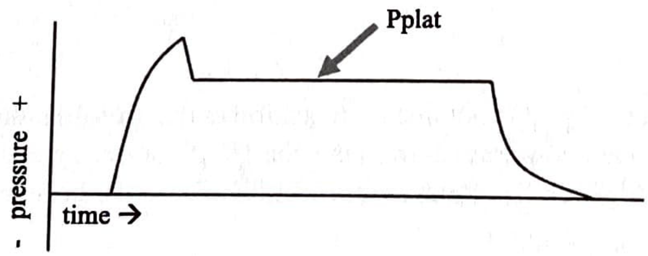
Plateau Pressure



Plateau pressure (Pplat) is the pressure in the system once the lungs are filled with air and before the breath cycles off. It represents the average pressure at the alveoli as they are at maximum inflation during inhalation. A normal Pplat is less than $30cmH_20$. Values higher than that can lead to direct damage to the alveoli, which can subsequently cause issues with pulmonary respiration.[137] There is no too low for Pplat, but recognize that lungs that aren't being filled all the way (i.e. a low Pplat) may not be maximizing the surface area of alveoli and therefore **Oxygenation** may not be at its best. And we will discuss this concept later on.[138]

The primary cause of a high Pplat at the start of ventilation is too much **TV**. That said, it can also be present or develop over time due to patient discomfort, mainstem migration of the ETT, atelectasis, and pulmonary edema. If we get a high Pplat, consider these other causes (and address them appropriately) before dialing down TV, as we don't want to give up ventilation unnecessarily.[139] We do, however, want to avoid a sustained high Pplat over many breaths, as that can lead to damage to the alveoli.

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



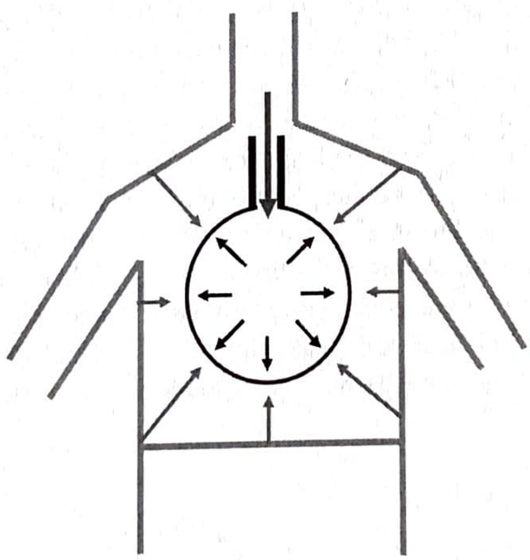
[137] We defined this concept back in **Terms to Describe Breathing**
[138] See **Titrating Up on TV?**
[139] And we will revisit this idea in an algorithmic fashion in the section called **Watching Pressures**

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

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

Another thing about Pplat is that the value we get is an average of alveolar pressures across the lung - some regions will experience higher pressures and others will experience lower pressures. The lung is not uniform throughout, but we can't measure alveolar pressures in specific lung regions or see to what degree this value would vary across the different parts. The safe limit of <30cmH$_2$O is a good guideline by which to limit our vent settings, but recognize that this doesn't mean that a pressure higher than that to one alveolus or a region of the lung will always cause harm. Likewise, a Pplat <30cmH$_2$O is not a guarantee that damage will not be caused ~~to a particular region of the lung~~.

One more subtlety here is that an elevated Pplat doesn't always reflect stress on the alveoli – there may be something external to the alveoli that prevents them from opening:



In these cases the elevated Pplat is not due to distention at the alveoli, rather it is due to something else. Examples would be a tension pneumothorax, burn to the chest wall, or even physical compression as in an entrapped patient. So while we generalize Pplat as a reflection of alveolar pressure, know that this isn't always the case.

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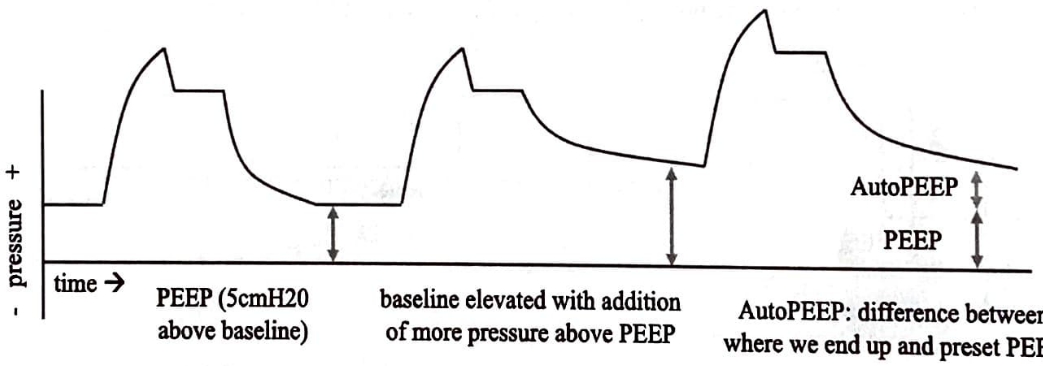
ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **CO$_2$** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO$_2$** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

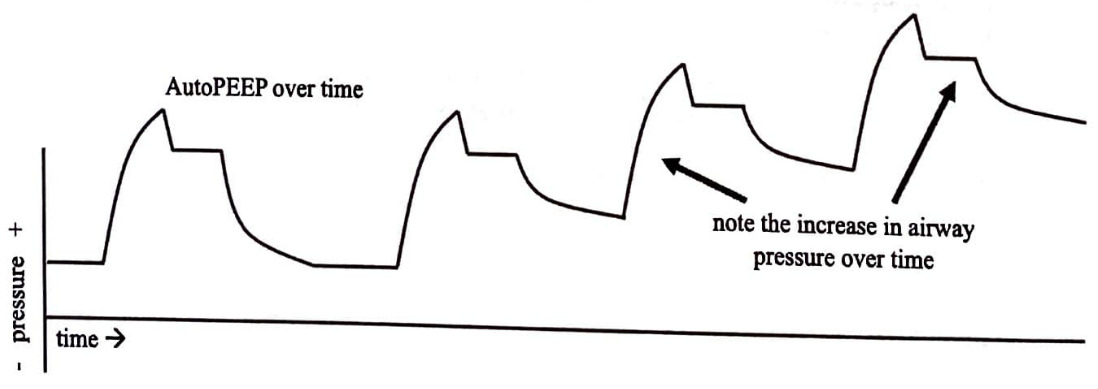
In summary, Pplat is typically the pressure seen by the alveoli when we deliver a breath in VC ventilation. A normal value is <30cmH$_2$0 and we measure it by performing an inspiratory hold maneuver. While there is no bottom limit to Pplat, it is important to recognize that we want to fill the lung and alveoli up with each breath delivered, so be wary of a super low Pplat and consider inadequate TV (and subsequently MV). High Pplat can be caused by too much TV, pneumothorax, restriction to chest wall expansion, mainstem intubation, and a few other things that we'll spell out later on.[140]

AutoPEEP

AutoPEEP is the idea of **PEEP** being cumulatively added into the system inadvertently. Remember how we said that we assume atmospheric pressure to be 0cmH$_2$0 as the starting point for our vent discussions and that PEEP is the addition of pressure on top of that (i.e. "adding 5cm of PEEP" to reset that baseline to 5cmH$_2$0)? Well, AutoPEEP is when that baseline starts to creep up from whatever we have set as PEEP to higher values because the patient isn't able to exhale all the way back to baseline before the next breath comes around. This idea is commonly referred to as breath stacking and can be represented like this:



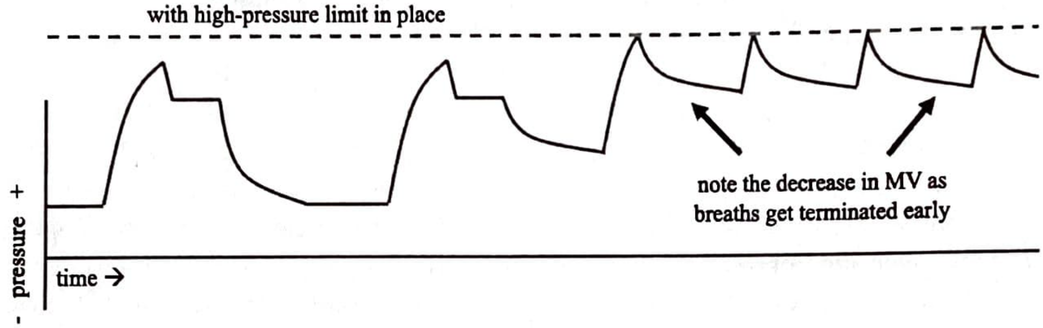
Normal AutoPEEP is zero, which means we shouldn't have any AutoPEEP in the system at all. That said, we may be OK with a few cmH$_2$O worth of AutoPEEP before we take action. Presence of AutoPEEP in **VC** can lead to an increase in other airway pressures, most importantly of which is **Pplat**:



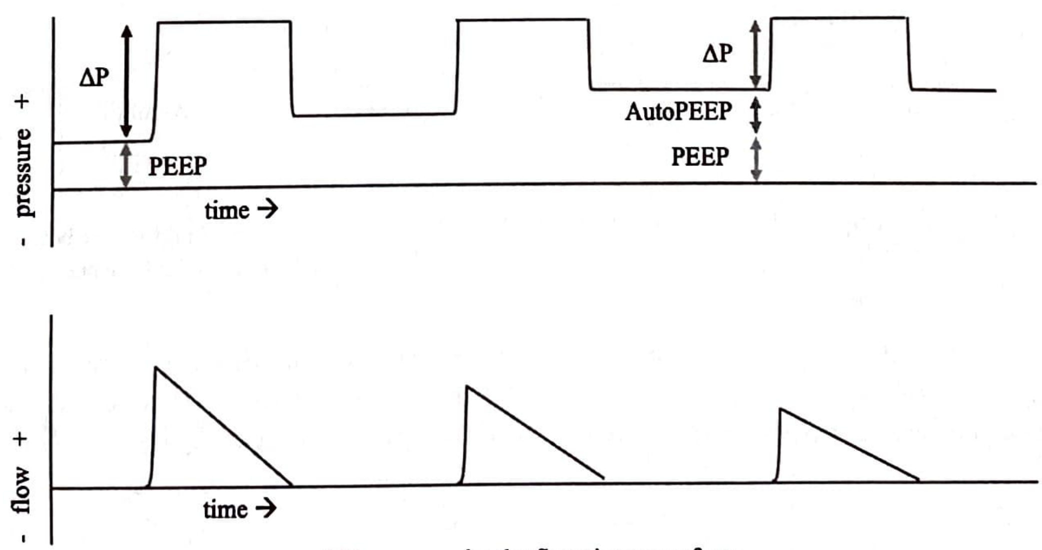
[140] See section on Watching Pressures

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

Another thing to realize is that if we have a normally-set high-pressure limit in place, then MV will suffer as breaths get terminated early:[141]



AutoPEEP in PC will also result in decreased less MV (due to less VTe per breath), but by a slightly different mechanism. Breaths don't get cut short as they do in VC, rather the flow to get to that set pressure is less. And since volume delivered is the product of flow and time, we get less volume:[142]



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

[141] And we talk about this idea more in **Alarms**
[142] And we used the symbol ΔP to represent the difference between PC and (Auto)PEEP, this is also the notation for a concept known as **Driving Pressure** which we will get to in just a bit

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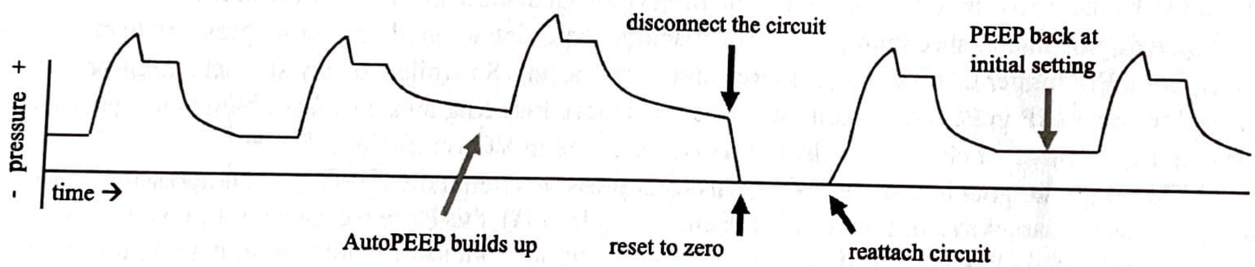
ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

To measure AutoPEEP or to check its presence, we have to perform another maneuver called an expiratory hold.[143] Doing an expiratory hold allows us to accurately see what the 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 measures 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 lengthen the I:E ratio and allow more exhalation; otherwise we could consider more sedation/ pain control and make sure we aren't accidentally triggering.[144]

One other thing we can do to eliminate AutoPEEP and reestablish our baseline at actual PEEP is to 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 canceling 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 summarize, AutoPEEP is a movement of the pressure baseline above whatever we have dialed in for PEEP. While a small amount of AutoPEEP may be tolerable, its presence is always an abnormal finding. Issues with this are increased pressures (VC) or decreased volumes (both VC and PC). Causes would be the inability to exhale fully, discomfort, and inadvertent triggering. Fixes include lengthening the I:E ratio, treating discomfort, and avoiding accidental triggers. In addition, we can reset AutoPEEP back to zero by temporarily disconnecting the vent circuit.

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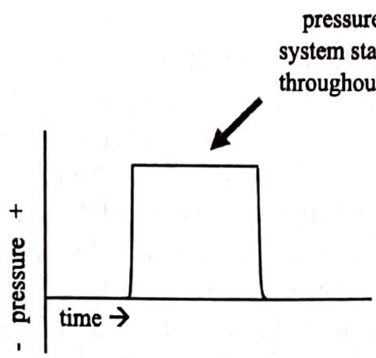
[143] There are other ways to check for AutoPEEP, but they aren't typically available in transport unless we have access to scalars or waveforms

[144] See sections on **Comfort**, **Triggers**, **Inspiratory Time (and I:E Ratio)**, **Types of Breaths**, and **Obstruction** for more on these things

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

PIP & Pplat in Pressure Control?

Up to this point we've discussed **PIP** and **Pplat** mostly in the context of **VC** ventilation, but things are a bit different in **PC**. Let's start with what a PC breath looks like mapped out as pressure over time:[145]



First thing to mention here is that PIP will only be above that flat line at the top of the square waveform (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 set, but a PIP higher than the set pressure control can occur. So while we may still set a high-pressure alarm and monitor PIP in PC ventilation, our concern is more for being aware of disturbances to the system rather than being aware of changes to airflow, as was the case in VC ventilation.[146]

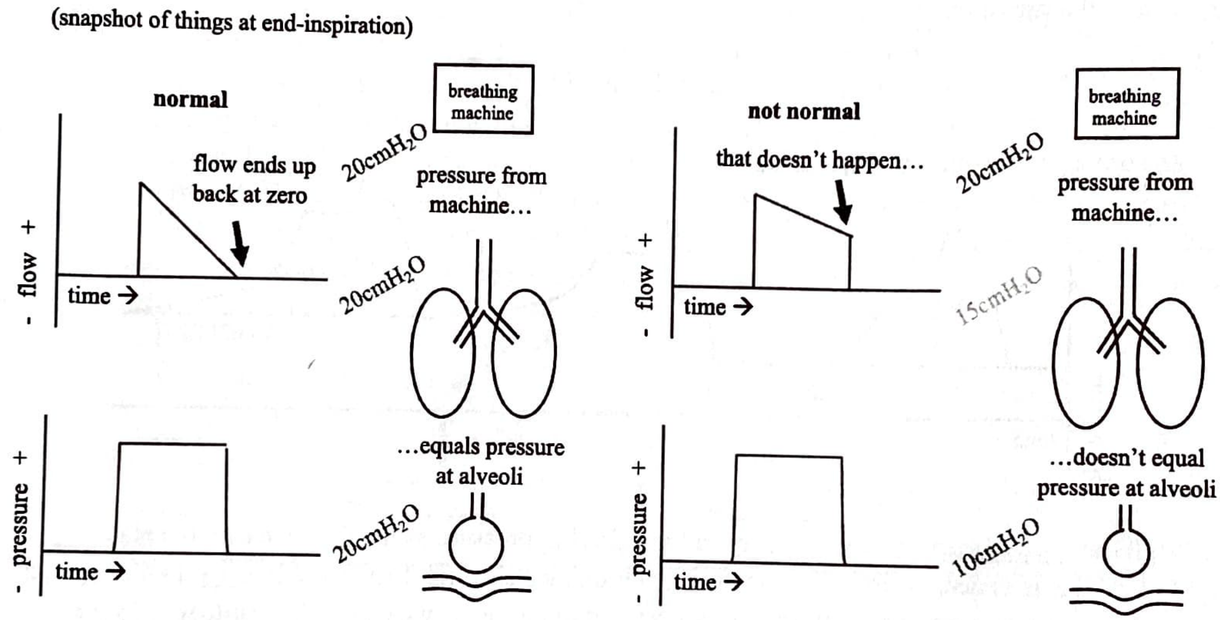
Next thing: the goal is that the average alveolar pressure eventually does equal that pressure represented by the top of the square waveform (towards the end of expiration), therefore we assume it to be true that pressure control equals Pplat.[147] And because of this assumption that mostly holds true, it's OK that some machines don't let us do inspiratory holds in PC 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 VC) is to rule out high alveolar pressures (to ensure the wellbeing of the alveoli); in PC if Pplat doesn't match pressure control it's because true Pplat is less than the pressure control (which is a bummer, but not a safety concern for the alveoli).

Now the way it works is that it takes time for the alveolar pressure to rise up to match the pressure going into the system. Even though we start with a high pressure at the machine end of the system, it may take some time for that pressure to equalize down to the alveoli. If our **I-time** isn't long enough to allow that to happen, the alveolar pressure may not ever get up to the level we have set for pressure control. We work around that in VC 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 PC because, as we said just a moment ago, the **Pplat** won't be above our pressure control value and so there isn't so much of a concern.

[145] The was first covered in **Types of Breaths**
[146] In PC ventilation, we become aware of those obstruction issues by monitoring VTe and maybe flow (if available on our particular machine)
[147] Hess, 2014 – Another way to say this is that if flow gets to zero during the inspiratory phase, then PIP = Pplat

ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

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



All that said, this isn't a great method unless we 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.[148]

So what utility is there in knowing Pplat in PC anyways? We said already that the usefulness of this information in VC is to prevent damage to the alveoli, but that isn't as much of an issue in PC. Potential uses of knowing a Pplat in PC 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).[149] These are all cool things to work with, but it takes both time and effort and, therefore, may not be the best use of one's cognitive capacity when managing a sick patient in the transport setting. We will discuss this stuff, but know that Pplat is primarily a tool for ensuring alveolar safety in VC ventilation.

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

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

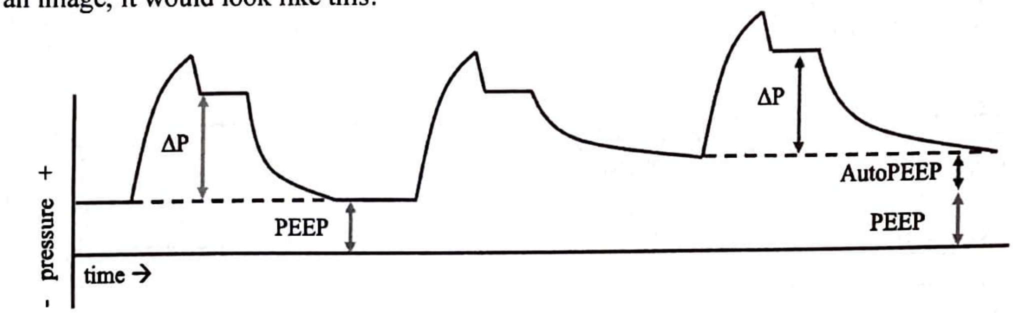
MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; **P_{aw}** – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

Driving Pressure[150]

Driving pressure is a term to describe how much we inflate and deflate the alveoli with each inhale and exhale on the ventilator. The idea is that too much opening and closing (inflation and deflation, up and down – however we want to term it) can put stress on the alveolar walls and cause damage.[151] This damage, in turn, leads to decreased diffusion of gasses across the alveolar membrane. Driving pressure is the difference between **Pplat** and end-expiratory pressure (normally **PEEP**, but sometimes affected by **AutoPEEP**) and is sometimes referred to as delta pressure:

$$\Delta P = Pplat - PEEP$$

As an image, it would look like this:



With our **Lung Injury** patients, we try to limit driving pressure as much as we can to a max of $15 cmH_2O$.[152] This is generally pretty reasonable, given that we use high PEEP and low **TV** in these patients anyways. And if driving pressure is close to or above that upper limit, we can do **Recruitment Maneuvers** to try and utilize more lung, increase compliance, and drop driving pressure. This approach may sound familiar and is often referred to as open-lung ventilation.[153] The basic idea is that we keep the lungs as filled as possible (i.e. alveoli inflated) throughout as much of the respiratory cycle as we can. This concept of limiting driving pressure and an open-lung strategy is specific to the 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 or open-lung approach is that it can be tough to blow off CO_2 as much as we'd want.[154] We said way back when that permissive hypercapnia is often a thing with ARDS, but that may not be the case with other patient groups. Another consideration here is PEEP – it is not a benign thing and we for sure need to consider the negative consequences of this approach before applying it to all patients. For now we have pretty good evidence that limiting driving pressure and utilizing high PEEP is a good thing with ARDS, but such a strategy may not be best for everyone.

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150 Bugedo & friends, 2017 – Succinct overview of the concept of driving pressure and research done to date (as of a few years ago, at least)
151 Grune & friends, 2019 - While this is commonly accepted idea and we will assume it to be valid in our discussion, know that there is ongoing research on all of this (as shown in this article)
152 Weingart, 2016a; Bauer, 2016b – Both podcasts look at a 2015 study on the subject
153 Nickson, 2019b – Concise overview of the idea with many more resources cited
154 To say it another way: **Ventilation** is affected and **Minute Volume** is less than we might want

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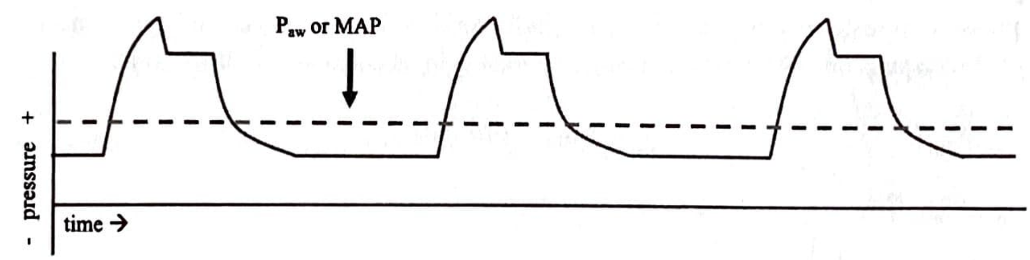
ΔP – driving pressure; ΔV – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

Mean Airway Pressure

Last pressure to talk about is mean airway pressure. It's typically represented as P_{aw} (stands for airway pressure) and less often as MAP (mean airway pressure). P_{aw} is the average pressure in the system throughout the respiratory cycle. There are formulas to estimate P_{aw}, but it's probably easiest to just read off of our machine (assuming it's there).[155] We don't often use this pressure to guide treatment, but if we notice changes in P_{aw} we can then look in to details as to what changed in the system. For example, a high P_{aw} can result from all sorts of things, each of which is a totally different issue: an increase in either **PIP** or **Pplat**, the presence of **AutoPEEP**, and increased **RR**. And same thing on the opposite end, lots of things can cause P_{aw} to drop and we then must work to identify a specific cause.

One other thing about P_{aw} is that it is strongly correlated with **Oxygenation**, particularly due to the variables of **PEEP** and **I-time**.[156] More of either of these things leads to a higher P_{aw}, so it can help to think of oxygenation in terms of this pressure and **FiO_2**. Just recognize that too much of this good thing can turn bad (i.e. too much pressure can have bad outcomes, as previously discussed). And while we commonly separate oxygenation into multiple concepts (as we did previously), it may be worth keeping this in mind as we look for trends in patient presentation:



[155] Mentioned in passing in the section on **Hypotension**, then demonstrated via calculations to justify that strategy in the **Appendix**

[156] Lodeserto, 2018 – Provides an explanation of this relationship between P_{aw} and oxygenation

MV – minute volume; **MVe** – exhaled minute volume; **OK** – alright; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Ppeak** – peak pressure; **Pplat** – plateau pressure; **PS** – pressure support; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

Compliance (and Resistance)[157]

Compliance is a measure of how much the lungs fill per unit of pressure put into the system. In math terms it looks like this:[158]

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

While normal compliance (healthy and breathing spontaneously) is somewhere in the neighborhood of 100ml/cmH_2O, we often see values much smaller than that in our ventilated patients. The best way to utilize compliance during transport is to keep track of trends: increasing compliance is good, decreasing compliance is bad. If we do something that results in poorer compliance, maybe second guess whatever that change was; if we do something that results in better compliance, high fives are warranted. Acute causes of decreased compliance would be a worsening pneumothorax, inhibition of chest wall expansion, chest wall rigidity caused by certain medications, increasing <u>**TV**</u> beyond the capacity of the lungs at that given time, etc.[159]

A related term is resistance. Resistance and compliance are often discussed together under the umbrella terms of respiratory mechanics or pulmonary mechanics – that's why we talk about it here.[160] Now the algebraic expression of resistance isn't quite as straight forward as for compliance and we often simplify it by assuming that flow equals 60LPM, so we're just going to skip on ahead and note it like this:

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

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

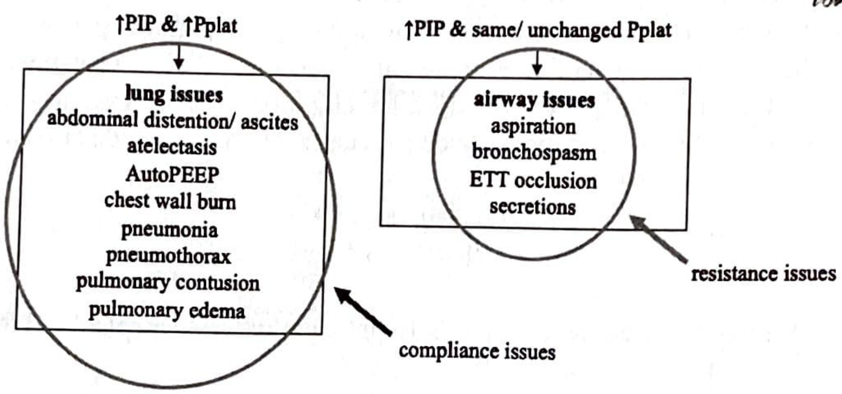
[158] And to be more specific, this is what we would call static compliance; we won't get into dynamic compliance here

[159] And all of these high **Peak Inspiratory Pressure**, high **Plateau Pressure** situations will be discussed in the section on **Watching Pressures**

[160] Hess, 2014 – And take a look here for even more on the idea

ΔP –driving pressure; **ΔV** – change in volume; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **CO_2** – carbon dioxide; **COPD** – chronic obstructive pulmonary disease; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **MAP** – mean airway pressure

Resistance, in this simplified manner, is the limitation to air movement that must be overcome for us to arrive at a state in which air in from the machine gets to the alveoli. Assuming Pplat remains constant, resistance is represented by PIP. This means that we can approximate changes to PIP to signify changes to resistance. So things like kinks in the tubing, biting on the tube, excessive secretions, etc. that are causes of increased PIP and unchanged Pplat correlate with an increase in resistance:[161]



And we mentioned already that the alternative strategy in **PC** ventilation when we don't have PIP or Pplat to guide us is to look at VTe and MVe to gauge when these things are happening (a drop in volume will indicate an increase in resistance or decrease in compliance). We can also look at a quantitative value for compliance (if available to us on our machine) or see how flow is changing from breath to breath.[162]

[161] Cassone & friends, 2019 – We will expand on this in **Watching Pressures**, but know for now that this graphic is a piece of an algorithm that we lay out when we get there – it just made sense to include it here to differentiate these two concepts
[162] Again, this in PC only; in VC flow will be the same with each breath delivered

MVe – exhaled minute volume; **PALS** – pediatric advances life support; **PC** – pressure control; **PEEP** – positive end-expiratory pressure ; **PRVC** – pressure-regulated volume control; **PS** – pressure support; **RR** – respiratory rate; s – second; **SIMV** – synchronized intermittent mandatory ventilation; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

A General Vent Strategy

In this section we are going to summarize general parameters in each type of ventilation (i.e. each combination of mode and control) in order to demonstrate what settings and goals are shared among all methods and which are specific to certain types of ventilation. This general strategy is similar to what is often described as a lung-protective strategy that first came on the scene in regard to management of patients with ARDS; we've opted to present the two as distinct strategies and we'll come back to this idea when we get there.[163] We will also hash out a few of the differences in determining general settings for adults versus pediatrics. Let's start with a discussion of things that apply to most vented patients, regardless of mode or control:[164]

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

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

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

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

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

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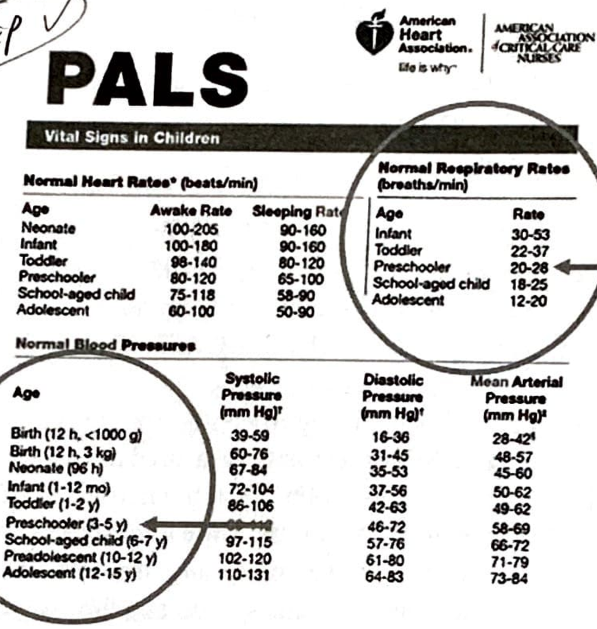
[163] That will happen in the section on **Acute Lung Injury/ Acute Respiratory Distress Syndrome**

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

ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH₂O** – centimeters of water; **EtCO₂** – end-tidal carbon dioxide; **FiO₂** – fraction of inspired oxygen; **IBW** – ideal body weight; **I-time** – inspiratory time; **kg** – kilogram; **L** – liter; **ml** – milliliter; **min** – minute; **MV** – minute volume

As for kids, the approach is to choose a rate in line with a reference card and disregard the above suggestion of 13-17/min. While this will result in an overestimation of MV,[165] we can titrate values to address that later on. For example, let's assume a 4-year-old child of 18kg. Based on this chart (again, from PALS) we want a RR in the 20-28/min range:[166]



PALS

American Heart Association. Life is why | American Association of Critical-Care Nurses

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

We can also use this chart based on the PALS data:[167]

age description	age (years)	RR	I-time (s)
infant	.083 (1 month)-1	30-53	0.3-0.6
toddler	1-2	22-37	0.4-0.9
preschooler	3-5	20-28	0.5-0.9
school-aged child	6-7	18-25	0.6-1.1
big kid	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

[165] Because a weight-based TV (or TV goal in PC) stays the same

[166] American Heart Association, 2016 (image)

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

MVe – exhaled minute volume; **PALS** – pediatric advances life support; **PC** – pressure control; **PEEP** – positive end-expiratory pressure ; **PRVC** – pressure-regulated volume control; **PS** – pressure support; **RR** – respiratory rate; s – second; **SIMV** – synchronized intermittent mandatory ventilation; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

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

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

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

$$MV\ calculated = RR \times TV$$
$$MV\ calculated = (20 - 28)/min \times (108 - 144)ml$$
$$MV\ calculated = 2160 - 4032ml/min$$
$$MV\ calculated \approx 2 - 4L/min$$

The result here is a MV goal that differs pretty significantly from the calculated MV, but what to do with this information? We will eventually want a MV (preferably measured as MVe) that matches or exceeds 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 we decide to ventilate a child in VC. There is always some dead space that we introduce into the system and this overestimation will help to mitigate that. Along those same lines, it may also be worth using a TV on the higher end of the range (again, this is only for kids in VC ventilation) to further mitigate this effect.[168]

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

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ΔP –driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **$EtCO_2$** – end-tidal carbon dioxide; **FiO_2** – fraction of inspired oxygen; **IBW** – ideal body weight; **I-time** – inspiratory time; **kg** – kilogram; **L** – liter; **ml** – milliliter; **min** – minute; **MV** – minute volume

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

parameter	value	pro tips
TV	6-8ml/kg	pick an easy number to work with that falls in that range
MV	100ml/kg/min	just take IBW in kg and move the decimal over (75kg IBW = 7.5L MV goal)
RR	adult: 13-17/min kids: use a chart	carry a reference card or have an app on a device to quickly reference the pediatric values[169]
FiO_2	1.0, then titrate down	we can titrate down in big jumps also, no need to go in small increments unless we have good reason to do so[170]
PEEP	5-6cmH_2O	for most vents this will be whatever the machine defaults to
I-time	adult: 0.8-1.7s kids: use a chart	normal for the adult is 1.0s

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

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

MVe – exhaled minute volume; **PALS** – pediatric advances life support; **PC** – pressure control;
PEEP – positive end-expiratory pressure ; **PRVC** – pressure-regulated volume control; **PS** – pressure support; **RR** – respiratory rate;
s – second; **SIMV** – synchronized intermittent mandatory ventilation; **TV** – tidal volume; **VC** – volume control;
VTe – exhaled tidal volume

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

	additional parameters[171]
AC VC	None
SIMV VC	***pressure support*** – start at 5-10mmH_2O and titrate as needed
AC PC	***pressure control*** – start at 10-15cmH_2O and titrate to TV goal
SIMV PC	***pressure control*** – start at 10-15cmH_2O and titrate to TV goal ***pressure support*** – start at 5-10mmH_2O and titrate as needed
AC PRVC	***pressure cap***[172] – set to 25-30cm$H_2$0 (often by setting high-pressure limit to 5cmH_2O above what we want this to be)
SIMV PRVC	***pressure cap*** – set to 25-30cm$H_2$0 (often by setting high-pressure limit to 5cmH_2O above what we want this to be) ***pressure support*** – start at 5-10mmH_2O and titrate as needed

[171] It's a bit tough to identify specific starting points for both PC and PS in the literature and recommendations vary a lot, but these are points to start off at and then we should always titrate towards VTe and MVe goals as soon as possible; as for more insight into these initial settings:
Ashworth & friends, 2018 – They say start with PC at 5-10cmH_2O and limit ΔP (Pplat or PC – PEEP, which we will discuss later on **Driving Pressure**) to 16cmH_2O (which correlates with an additive PC of that amount – 16cmH_2O)
Kneyber & friends, 2017 – These guys recommend limiting a ΔP to 10cmH_2O for all (pediatric) patient types
Nagler & Chiefetz, 2019 – This duo suggests a starting PS of 5-10cmH_2O for kids
And just to be clear, all the pressures listed here (for PC and PS) are additive, not cumulative (and for a refresher on what that means, head back to **Types of Breaths**)
[172] Recall that this is a made-up term and is typically represented by 5cm less that what we set as the high-pressure limit

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ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **$EtCO_2$** – end-tidal carbon dioxide; **FiO_2** – fraction of inspired oxygen; **IBW** – ideal body weight; **I-time** – inspiratory time; **kg** – kilogram; **L** – liter; **ml** – milliliter; **min** – minute; **MV** – minute volume

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

step one: set &/ or calculate		**step two: make a choice and dial in extra stuff**	
TV[173]	6-8ml/kg	**AC VC**	None
MV	100ml/kg/min	**SIMV VC**	***pressure support*** – $10mmH_2O$
RR	adults: 13-17/min kids: use a chart	**AC PC**	***pressure control*** –$10\text{-}15cmH_2O$
FiO_2	1.0, then titrate down	**SIMV PC**	***pressure control*** –$10\text{-}15cmH_2O$ ***pressure support*** – $10mmH_2O$
PEEP	$5cmH_2O$	**AC PRVC**	***pressure cap*** – set to $25\text{-}30cmH_20$ (normally: set high-pressure limit to $5cmH_2O$ above what we want this to be)
I-time	adult: 0.8-1.7s kids: use a chart	**SIMV PRVC**	***pressure cap*** – set to $25\text{-}30cmH_20$ (normally: set high-pressure limit to $5cmH_2O$ above what we want this to be) ***pressure support*** – $10mmH_2O$

In the ideal world, that's how we get vent settings for a specific patient. In the actual world we have a few things to consider (and we'll frame them as questions): What pathophysiologic 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 our 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 the vent in any scenario, and then we will consider 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.

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

Specific Vent Strategies

We have a chart from the last section that 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 his or her breaths delivered in a specific way (different to what we identified as normal) due to intricacies of a given illness or disease. We sometimes take those normal parameters and alter them to meet specific needs and issues. It's totally OK to break the rules we've established so far, as long as we know when and how to do it and can justify a good reason. We will look at a few situations, in turn, to see how it all looks.

Vent strategies are often presented as a choice of two distinct categories: the injured or sick lung approach and an obstructive strategy. We've opted to present this decision-making process as a set of five possible strategies from which providers can choose. First of those is the general strategy discussed just now, the other four include obstruction, hypotension, acidosis, and lung injury. There is no right or wrong in this process, we just think it makes sense to take things a bit further as we have outlined in the following sections.[174]

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

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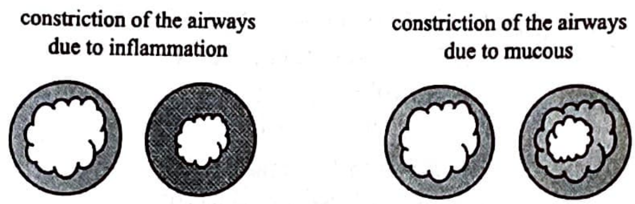
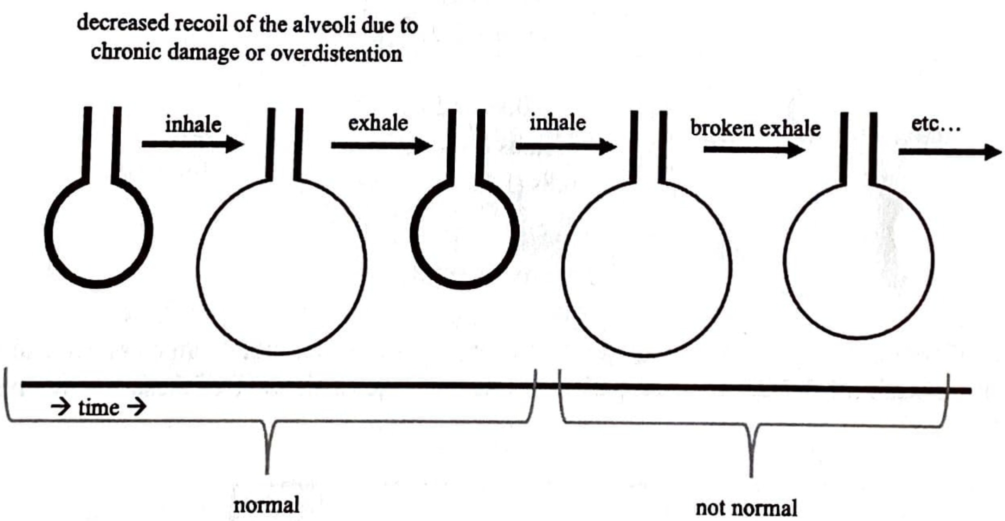
cmH_2O – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **IBW** – ideal body weight; **I-time** – inspiratory time; **I:E** – inspiratory to expiratory; **kg** – kilogram; **min** – minute; **ml** – milliliter; **MV** – minute volume

Obstruction

In patients with asthma, COPD, and/ or allergic reaction, we tend to run into 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 underlaying cause, but can be summarized as some combination of the following:





OK – alright; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; s – second; **TV** – tidal volume; **VTe** – exhaled tidal volume; **VC** – volume control

The fix is to adjust vent parameters to allow for more time at exhalation. We do this by extending or lengthening the I:E ratio. A normal I:E ratio is 1:2-3 and we can adjust that by decreasing either the **I-time** or **RR**.[175] A good starting point in this population is an I:E ratio of around 1:5. The typical way to get here is to decrease RR (and also I-time) until we see an I:E ratio in that range that we want. The machine normally does this calculation for us, but just an example we'll show it all here:

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

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

With I-time 0.8s and RR 13:
60 ÷ 13 breaths ≈ 4.6s/breath
4.6s – 0.8s (I-time) = 3.8s

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

I:E ratio = 1:4.8

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

I-time 1.0s		I-time 0.8s	
RR	***I:E***	***RR***	***I:E***
17	1:2.5	17	1:3.4
13	1:3.6	13	1:4.8
10	1:5.0	10	1:6.5
8	1:6.5	8	1:8.4

[175] And as noted back in **Inspiratory Time (and I:E Ratio)**, normal is 1:2 and the 1:3 is more due to convention that what the patient would breathe at if left alone to nature

cmH₂O – centimeters of water; **COPD** – chronic obstructive pulmonary disease; **IBW** – ideal body weight; **I-time** – inspiratory time; **I:E** – inspiratory to expiratory; **kg** – kilogram; **min** – minute; **ml** – milliliter; **MV** – minute volume

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

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

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

$$\text{MV calculated} = \text{TV} \times \text{RR}$$
$$\text{MV calculated} = 520\text{ml} \times 8\text{/min}$$
$$\text{MV calculated} = 4160\text{ml/min}$$
$$\text{MV calculated} \approx 4.2\text{L/min}$$

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

$$\text{TV} = 12\text{ml/kg} \times 65\text{kg}$$
$$\text{TV} = 780\text{ml}$$

$$\text{MV calculated} = \text{TV} \times \text{RR}$$
$$\text{MV calculated} = 780\text{ml} \times 8\text{/min}$$
$$\text{MV calculated} = 6240\text{ml/min}$$
$$\text{MV calculated} \approx 6.2\text{L/min}$$

And at this point we run the risk of barotrauma or over-inflation injury (assuming VC ventilation). 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 30cmH₂O). 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.[176] We will simultaneously be doing pharmacological interventions (Albuterol, Magnesium Sulfate, Ketamine, Epinephrine – whatever our 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.

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

OK – alright; PC – pressure control; PEEP – positive end-expiratory pressure; Pplat – plateau pressure; RR – respiratory rate; s – second; TV – tidal volume; VTe – exhaled tidal volume; VC – volume control

In **PC**, we still drop the rate (and maybe I-time too) to lengthen I:E, but we also want as much volume per breath to try and get as close to our MV goal as possible. Instead of a pressure control of 10-15cmH_2O, consider going straight to the top and starting at 20-25cmH_20 to see what our VTe values look like.[177] In addition, recognize that this Pplat upper limit (30cmH_2O) is a generalization that may not be necessary for all patients.

Second to last thing to mention: it may be tempting to drop **PEEP** to zero in these cases to better allow the patient to exhale. The thought process goes like so: if they are breathing out while we are pushing air in, this has the potential to be problematic. That said, there is some thought that applied PEEP can help fix AutoPEEP, but we do want to keep applied PEEP lower than AutoPEEP. Just know that we may want to maintain PEEP at our minimum of 5cmH_2O to maximize **Oxygenation** and help recruit more alveoli, but sometimes we let that go in order to avoid AutoPEEP. There may be a happy middle ground with a PEEP somewhere between zero and a normal value, but there isn't much content on that and we'll leave it as a maybe in the overall scheme of things.[178]

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

To summarize our obstruction strategy: utilize a lower RR (and consider a shorter I-time also) to a goal I:E of 1:≥5. Consequently, we need to titrate TV (or pressure control) up as far as the patient's lungs will allow.[179] 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 ventilation goals. Maybe consider dropping PEEP, but know that there isn't yet a good consensus on that. Also, be sure to check for AutoPEEP periodically and consider disconnecting the vent circuit to reset it back to zero if need be.[180]

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

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

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

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

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%TaDP – percentage of time at decreased preload; **CO** – cardiac output; **FiO$_2$** – fraction of inspired oxygen; **IBW** – ideal body weight; **I-time** – inspiratory time; **kg** – kilogram; **L** – liter; **min** – minute

Hypotension

In patients with hypotension (or the potential for hypotension) the primary concern is that mechanical ventilation can decrease CO and further contribute to the problem. We discussed this already in reference to both PPV generally and **PEEP** specifically. We mentioned then that volume seems to mitigate this effect, but that takes time.[181] So first strategy here (since we are committed to PPV) is to restrict PEEP to whatever minimum value we need to maintain adequate oxygenation. Beyond that, however, we can limit the time spent at inspiration during the overall respiratory cycle. Think of it this way: preload drops further when we increase intrathoracic pressure, so if we decrease the amount of time spent pushing air into the system we can limit this effect.

Now to quantify the idea of how blood return and CO are affected due to breaths given by the machine, 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):[182]

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

with RR of 17

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

with RR of 10

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

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

with RR of 10 and I-time 0.8s

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

[181] See **How is Positive Pressure Different?** to review the discussion on the negative effects of PPV
[182] This is another one of those made up terms which we identify as %TaDP or percentage of time at decreased preload

ml – milliliter; **OK** – alright; **P_{aw}** – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **PPV** – positive pressure ventilation; **RR** – respiratory rate; s – second

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

MV goal = 100ml/kg/min
MV goal = 100/ml/kg/min x 65kg
MV goal = 6500ml
MV goal = 6.5L/min

TV = 8ml/kg x 65kg
TV = 520ml
MV calculated = TV x RR
MV calculated = 520ml x 10/min
MV calculated = 5200ml/min
MV calculated = 5.2L/min

Now 5.2L/min isn't super far off from 6.5L/min, but we need to remember that a hypotensive patient is likely at risk of shock and, therefore, we need to make sure that **Ventilation** is adequate by delivering at least what our calculated MV minimum is.[183] This idea is in stark contrast to the obstruction strategy in which we decided it was OK to let MV fall below goal; in hypotension we need to maintain (or even exceed, especially with **Acidosis** – discussion on that to follow) our MV goal. So let's titrate TV up to 10ml/kg and see where we end up:

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

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

[183] Mannarino, 2014 – Refer to this video for a review of what shock is and how it is related to O_2 delivery

%TaDP – percentage of time at decreased preload; **CO** – cardiac output; **FiO_2** – fraction of inspired oxygen; **IBW** – ideal body weight; **I-time** – inspiratory time; **kg** – kilogram; **L** – liter; **min** – minute

If we drop RR to 10 (and I-time to low of normal by age) to minimize the percentage of time spent at decreased preload (i.e. inspiration) and increase TV to 10ml/kg, then we maintain our MV goal of 100ml/kg/min. Now that we've logically arrived at a strategy of decreased RR and increased TV, let's rewrite the order of the steps as so: increase TV first, then decrease RR to match MV goal. The reason for this is that we don't want to arbitrarily drop RR and then wind up in a situation where we can't titrate TV up to goal – that would result in a decreased MV (which we said is an important thing in the patient at risk for shock). So let's go up on TV as much as we can (even beyond 10ml/kg if we can maintain a safe **Pplat**) and then drop RR afterward. Even if we aren't able to drop %TaDP by half as in the example shown, we can at least move in that direction while ensuring adequate ventilation.[184]

Now there are other justifications for using a high TV and low RR strategy that don't include this %TaDP concept, we just find that this concept makes it easy to appreciate. An alternative justification would be that the strategy decreases dead space.[185] We talked about this idea back when we discussed making changes to address MV needs and the idea is that dead space gets introduced with each breath given, so fewer breaths (with more volume each) means less dead space overall.[186] Another rationale would be **P_{aw}** – this high TV, low RR approach decreases average pressure into the system, especially when we consider lowering PEEP towards zero (i.e. using the bare minimum necessary to maintain oxygenation). While lowering P_{aw} could negatively impact **Oxygenation**, we may be able to counteract that with higher FiO_2 to meet our goals. The point here is that there are multiple justifications for this strategy; one has been spelled out here and the other two are deferred until the **Appendix**.

To summarize: in the hypotensive patient we want to decrease the amount of time spent at decreased preload while maintaining MV at our weight-based minimum. To do this, we drop I-time to low of normal, increase TV as much as we safely can manage (in PC this may mean starting at 15-25cmH_2O), and then decrease RR to maintain our MV goal. We also want to be cautious of high PEEP while recognizing that oxygenation (facilitated by PEEP) is important in these patients with potential low perfusion 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 maintain MV goal), and keep PEEP to a minimum.

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[184] 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 does help to keep processes simple and applicable across the board…

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

[186] This was in **Ventilation**

min – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; **O_2** – oxygen; **PCO_2** – partial pressure of carbon dioxide; **pH** – power of hydrogen; **PS** – pressure support; **RR** – respiratory rate; **RSI** – rapid sequence intubation; **SIMV** – synchronized intermittent mandatory ventilation; **TV** – tidal volume

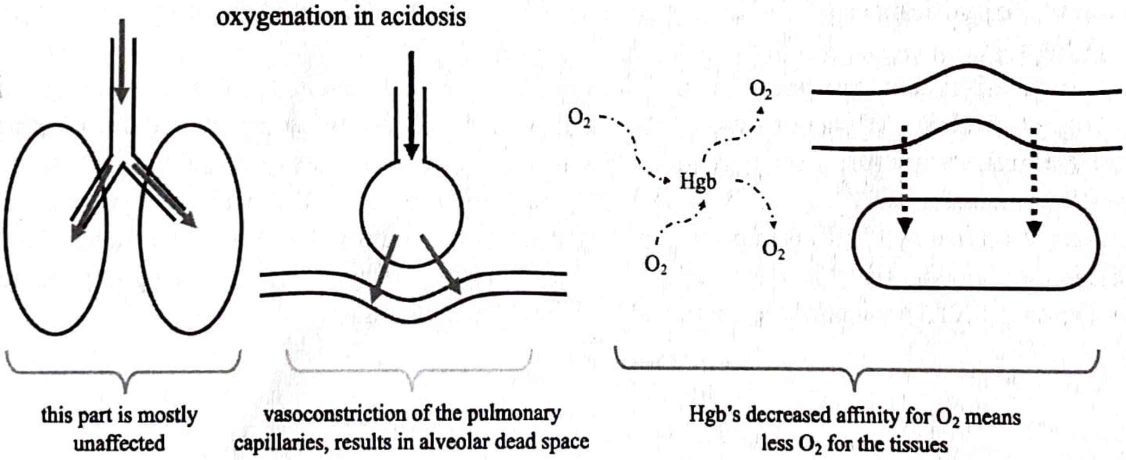
legend!

Acidosis

One of our primary ventilation goals with acidosis is to facilitate respiratory compensation against the underlying acidosis. We mentioned already that certain factors can impact Hgb's affinity for O_2 and pH is one of those things. More acid and lower pH means that O_2 is held less tightly by Hgb as it moves through the blood. Going back to our train analogy from before, we could say that this right shift would be comparable to taking all the seatbelts off and leaving the cabin doors open as Hgb moves through the blood.[187] And then as O_2 leaves Hgb we get that physical restructuring of the Hgb molecule that makes it easier for further O_2 molecules to do the same – O_2 in an acidotic state is more susceptible to peer pressure and wants to both follow its friends as they jump off the train and avoid getting on the train in the first place if less of its friends are on board.[188]

The practical takeaway is that it may be difficult to get oxygen onto Hgb for delivery to the tissues in the acidotic patient. While we can increase the amount of O_2 to the alveoli and expect it to diffuse into the blood, getting it from that dissolved state to the Hgb itself is the challenge. And then when we do get it loaded onto Hgb, it may offload early before getting to the tissues where we intended it to go. To summarize these ideas and a few more, here's how we would draw it all out:[189]

oxygenation in acidosis



While we still do all the things we already know how to do in regard to **Oxygenation**, the focus of the acidosis strategy we've outlined here is to work towards correcting these shifts so that O_2 delivery returns to normal. The primary mechanism is to increase **MV** in an effort to blow off more CO_2. This helps offset the impact of acidosis to some degree. Failing to do so will exacerbate the problem and worsen O_2 delivery with potentially catastrophic consequences.

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[187] This was in the section on **Oxygenation**

[188] Hasudungan, 2018; Smith, 2014 – The first is a video that reviews this concept of a right shift and also covers some basics we discussed before; the second is a quick video to review acid-base analysis (and for more resources on this, look ~~forward~~ to the section **Patient Already on the Vent**)

[189] Lumb & Slinger, 2015 – Refer back to this article for a review of **Hypoxic Pulmonary Vasoconstriction**

ABG – arterial blood gas; **AC** – assist control; **BMP** – basic metabolic panel; **CO_2** – carbon dioxide; **DKA** – diabetic ketoacidosis; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FDA** – Federal Drug Administration; **Hgb** – hemoglobin; **HCO_3^-** - bicarbonate ion; **kg** – kilogram; **L** – liter

To expand on this, the classic example here is a DKA patient breathing at a **RR** of 30: 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, but that compensation gets taken away suddenly. As a result, the patient quickly decompensates, crashes, and suffers a less-than-ideal outcome.[190] We've already reviewed the idea of oxygenation as it relates to acidosis, but the other factor here is that a rapid increase in PCO_2 can lead to hemodynamic issues such as decreased cardiac contractility, lessened response to catecholamines, and systemic vasodilation.[191] Given all of this, we need a strategy that maintains MV both to prevent these hemodynamic effects and to maintain oxygen delivery to the tissues.

While a bit tricky to pinpoint exactly what our MV goal ought to be, let's start with a minimum goal double that of the normal patient: 200ml/kg/min.[192] To achieve that goal, we may need to increase both RR and TV. In order to increase MV and get our $EtCO_2$ within a normal range we typically start by changing TV first and then RR.[193] The reason for this is that adding a breath also adds in dead space to the equation. In the acidosis situation, however, the patient is likely already breathing fast, so let's just use a high of normal TV (i.e. 8ml/kg) and see what kind of RR we'd need to get to this increased MV goal of 200ml/kg/min:

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

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

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

[190] Weingart, 2009 – And while we don't focus on the RSI process in this manual, look to this throwback episode of EmCrit for a strategy on how to do so while simultaneously avoiding the problems we discuss here
[191] Carter & friends, 2010 – This article reviews how oxygen delivery is affected by acid-base imbalances
[192] Weingart, 2010 – Our suggestion vaguely resembles the one recommended here (double MV to drop CO_2 from 40 to 30, that's with a starting MV of 120ml/kg/min); that said, this is a minimum starting point and we may need to take it further than that – the idea is that we initiate ventilation to prevent immediate deterioration and then go from there to work towards goals (as outlined later in this section)
[193] This was discussed in **Ventilation**

min – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume; O_2 – oxygen; PCO_2 – partial pressure of carbon dioxide; **pH** – power of hydrogen; **PS** – pressure support; **RR** – respiratory rate; **RSI** – rapid sequence intubation; **SIMV** – synchronized intermittent mandatory ventilation; **TV** – tidal volume

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

First thing we can do is to match our 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 beforehand and that we are the ones intubating or taking that airway away. 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 with our settings, complete the transport, and then have the receiving facility check ABGs when we arrive to see how things have improved (or gotten worse, for that matter). Or if we can do gasses en route, we can always start this strategy and then evaluate progress along the way.

Another strategy is to measure the patient's $EtCO_2$ (perhaps via a nasal canula device or by cutting the ETT connector off a regular in-line attachment and sticking in the patient's mouth[194] or by using a side-stream $EtCO_2$ device with non-invasive ventilation[195]) prior to taking the airway. We can then match the patient's RR (as above) or set RR to twice normal and then adjust to this $EtCO_2$ that the patient was at before we messed with things. Again, this strategy is similar to the above strategy in that it requires that the patient was compensating adequately on his or her own before we intervened.

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

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

The formula is designed to measure the respiratory component with a known metabolic acidosis (i.e. measured PCO_2 is compared to a calculated PCO_2 to determine if the patient is compensating adequately or if a mixed disorder is present),[196] but we can modify its use in the transport setting to guide our titration of EtCO2 (via MV).[197] This strategy is of use if we are taking over care of an acidotic patient who is already on the ventilator:

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

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

[195] Weingart, 2009 – We referenced this podcast already in the previous page, but refer here for a step-by-step as to how this would work

[196] Foster & Grasso, 2014 – Short video to explain the formula and its use in a clinical setting

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

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ABG – arterial blood gas; **AC** – assist control; **BMP** – basic metabolic panel; CO_2 – carbon dioxide; **DKA** – diabetic ketoacidosis; $EtCO_2$ – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FDA** – Federal Drug Administration; **Hgb** – hemoglobin; HCO_3^- - bicarbonate ion; **kg** – kilogram; **L** – liter

A few notes about all of this. $EtCO_2$ generally correlates with PCO_2 fairly well, with $EtCO_2$ normally 2-5mmHg below PCO_2. That normal difference is due to anatomic dead space and will increase with additional dead space (i.e. alveolar dead space). That said, even with more dead space in play, $EtCO_2$ and PCO_2 will move in stepwise fashion at the same rate.[198] If we use this modified formula, adjust MV to that goal, and get our $EtCO_2$ right at the calculated value based on HCO_3^- from labs, we still may be a bit shy of our MV goal. Just keep that in mind and know that's why we wrote it out as we did without the "±" and with the "≤." And the HCO_3^- can be from either the BMP or ABG for our use in the transport settings, but know that there are varying opinions on that.[199]

To bring it all home, we can do all of these strategies together: try to match the patient's RR and $EtCO_2$ as measured before we intervened, then compare both MV to our calculated minimum goal of 200ml/kg/min and $EtCO_2$ to a goal derived from Winter's Formula. And if we aren't the ones taking the airway and intubating, the Winter's Formula approach is likely the best strategy. The only next best thing here would be to remeasure gasses en route to see how the patient is responding to treatment, but most of us don't have that capability in the field and we'll withhold a discussion of it here.

We went on a bit of a tangent here, but let's get back to our vent strategy for the acidotic patient: use a TV goal high of normal (8ml/kg) and increase RR (either to match patient's intrinsic rate or even just double normal for patient's age), then aim for a goal MV of 200ml/kg/min and an $EtCO_2$ of patient's baseline prior to intervention or as determined by Winter's Formula. Because we are shooting for a high MV in the acidotic patient, AC mode may be the best for these patients if they are triggering breaths. 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).[200]

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198 Siobal, 2016 – And look here for more information on CO_2 monitoring in general

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

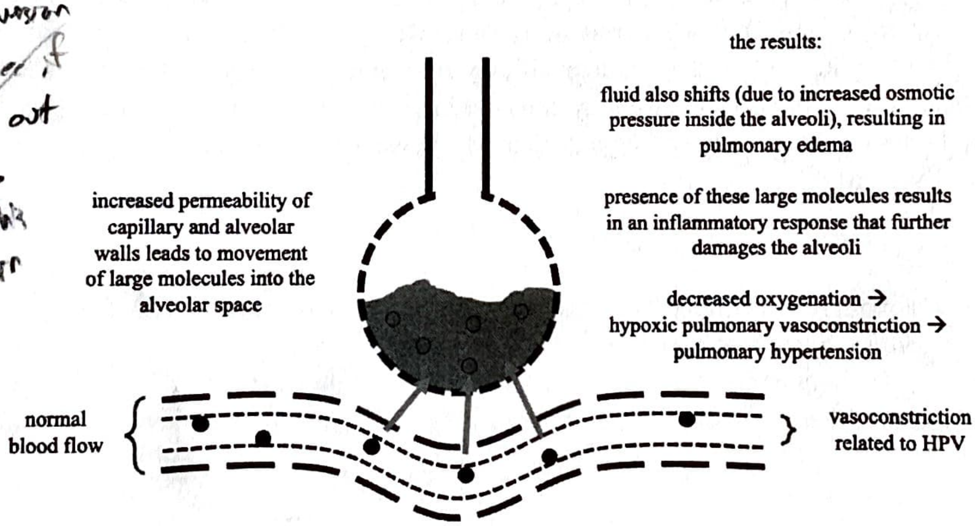
200 We talked about this idea way back in the section on **Synchronized Intermittent Mandatory Ventilation**; also review **Assist Control** mode

min – minute; **ml** – milliliter; **MV** – minute volume; **OK** – alright; **PaO_2** – partial pressure of arterial oxygen; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **SpO_2** – pulse oximetry; **TV** – tidal volume

Lung Injury

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

Acute lung injury (ALI) refers to a number of pathologies that inhibit normal pulmonary gas exchange.[201] Specific causes include sepsis, pneumonia, bleeding from a traumatic injury, inhalation of toxins or smoke, and aspiration. ALI is a concept that lives on a spectrum with acute respiratory distress syndrome (ARDS) being the 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 underlying pathophysiology is the same. The main component of the disease process is that the alveolar and capillary walls become permeable to stuff that normal is normally sequestered in the blood:



[201] Ragaller & Richter, 2010 – Not only do they provide an overview of the disease process, they also discuss this whole vent strategy and summarize research to date (at least as of 2010)

4x4 – four-by-four dressing; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HPV** – hypoxic pulmonary vasoconstriction; **IBW** – ideal body weight; **kg** – kilogram; **L** – liter

There are quantitative criteria for ALI and/ or ARDS (depending on how we choose to define it), but that isn't necessary for our field treatment. Given our capabilities in the transport setting, we generally identify a patient who needs this vent strategy from a report per sending facility or suspicion based on clinical progression of the illness. There are also many recommendations to use this strategy for all patients who don't fit any other category.[202] The strategy includes low TV, higher than normal 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 6ml/kg IBW, but we may get as low at 4ml/kg eventually. This recommendation is from the ARDSNet studies which compared TVs of 6ml/kg against 12ml/kg and determined that lower TVs resulted in significantly better outcomes for these patients.[203] While it may seem that 6ml/kg and 12ml/kg represent two extremes and it could be tempting to rationalize that 8 or 10ml/kg probably isn't all that bad, we do know that 6ml/kg is OK and the rest is still up in the air at this point.[204]

In addition to low TV, we go up on PEEP to improve oxygenation. Consider doing so in a stepwise fashion as recommended in these charts:[205]

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

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

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

[204] Sahetya & friends, 2017; Burrell, 2018 – And for a more detailed discussion of this idea, take a look at both this article and a review of another paper that sought to investigate this idea

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

min – minute; **ml** – milliliter; **MV** – minute volume; **OK** – alright; **PaO$_2$** – partial pressure of arterial oxygen; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **SpO$_2$** – pulse oximetry; **TV** – tidal volume

Another really important component of our lung injury strategy is alveolar recruitment. This is a concept that we've talked about some, but we'll get into it more here.[206] Recruitment is the idea that we can actively re-inflate collapsed or underinflated alveoli as we drew out in our previous discussion of **PEEP**. One component of the ALI/ARDS disease process is that the alveoli are particularly susceptible to both barotrauma and stress due to repetitive expansion and collapse. By slowly filling the alveoli with air and then using small volumes of air with each breath, we maximize usable space within the lungs and avoid causing damage. PEEP allows us to make this happen (i.e. it maintains recruitment through this process) and in turn we sacrifice net movement of air per a permissive hypercapnic approach (i.e. we sacrifice **Ventilation** to maximize **Oxygenation**).

Carrying on with this idea, if we have a partially inflated alveolus stented open with PEEP and then disconnect the vent circuit, that alveolus goes back to where it was before we started. In a normal lung there are forces that maintain recruitment to prevent this loss and we can also re-recruit these 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 on the vent again, add a bit of PEEP, and we are back where we want to be with no real negative outcome. With the lung injury patient, however, it can take hours to recruit alveoli. ~~This means that~~ if we lose recruitment, we lose all of that progress towards better oxygenation and our patient can deteriorate very quickly.

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

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

[206] And then again in **Recruitment Maneuvers**

4x4 – four-by-four dressing; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **HPV** – hypoxic pulmonary vasoconstriction; **IBW** – ideal body weight; **kg** – kilogram; **L** – liter

Last thing to mention with this lung injury strategy is MV. We mentioned already that we start at a TV of 6ml/kg and may need to go down to 4ml/kg. With higher PEEP we increase overall airway pressures and therefore that 6ml/kg TV on top of a higher PEEP (up to 20 in some cases!) means we might run into a high **Pplat**. If we notice Pplat encroaching on our safe limit of $30cmH_2O$, then we can dial the TV down to 5ml/kg and then to 4ml/kg (or if we are in PC we can just go up on PEEP and look at VTe). Dropping our TV to 4ml/kg will reduce MV and increase $EtCO_2$, but let's quantify that difference in MV with an assumed patient of 65kg IBW:

$$\text{MV goal} = 6.5\text{L}$$

$$\text{TV} = 4\text{ml/kg} \times 65\text{kg}$$
$$\text{TV} = 260\text{ml}$$

$$\text{MV calculated} = \text{TV} \times \text{RR}$$
$$\text{MV calculated} = 256\text{ml} \times 17/\text{min}$$
$$\text{MV calculated} = 4420\text{ml}$$
$$\text{MV calculated} \approx 4.4\text{L}$$

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

$$\text{MV goal} = \text{TV} \times \text{RR}$$
$$6.5\text{L} = 250\text{ml} \times \text{RR}$$
$$6.5\text{L} / 250\text{ml} = \text{RR}$$
$$25 = \text{RR}$$

So to maintain our MV goal with a TV of 4ml/kg we need a RR of 25 for the adult patient. Which is OK if we can comfortably get the patient there. If not, that's also OK. In fact, there is some evidence that hypercapnia (i.e. a high $EtCO_2$ related to a lower MV) is alright for these lung injury patients.[207] The data isn't super clear at this point, but rest easy knowing that if we can't attain our MV goal there may be a silver lining in this case. With pediatrics (when a RR of 25 is too slow), we just go up on RR as much as we can to meet (or exceed if in VC) our MV goal. Consider doubling RR or using the high end of normal for a given age range or just titrate up from a normal rate – the limiting factor will be comfort and exhalation (i.e. monitor for AutoPEEP to ensure full exhalation).[208]

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

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[207] Just to clarify: the idea here is that permissive hypercapnia is allowable, not that it provides an extra benefit

[208] A few notes on this: we talked about this overshooting of MV in the section on **Volume Control** and will do so again in the **Appendix**; also, the likelihood of transporting a pediatric patient with ARDS is slim and low-frequency, so ask for help and/ or consult with a specialist on this

min – minute; **ml** – milliliter; **MV** – minute volume; **OK** – alright; **PaO_2** – partial pressure of arterial oxygen; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **SpO_2** – pulse oximetry; **TV** – tidal volume

And one last thing to mention about this strategy. We said just a moment ago that lots of folks recommend a two-strategy approach to ventilation in which we use either this lung injury approach or an obstruction approach.[209] We have a general vent strategy for routine ventilation and then specific strategies for certain patient types. The differences between our general strategy (which is similar to a general lung-protective one) and this lung injury strategy is related to recruitment of alveoli (and being super careful to not lose it) and the idea that we may need to go down on TV to 4ml/kg. Both of these things are totally OK in the normal patient that we ventilate using the general strategy, it's primarily a matter of emphasis. If it makes things easier to default to this lung injury strategy in all cases that don't warrant one of the others, that's completely acceptable.

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

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ALI – acute lung injury; **ARDS** – acute respiratory distress syndrome; **CHF** – congestive heart failure; **CO** – cardiac output; **COPD** – chronic obstructive pulmonary disease; **cmH₂O** – centimeters of water; **EtCO₂** – end-tidal carbon dioxide; **FiO₂** – fraction of inspired oxygen

Other Potential Strategies

This list of vent strategies addresses four markedly different situations that we often come across in the transport setting, but there are other injuries or disease processes that might also warrant specific adjustments to the normal list of settings. 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 that might be difficult to navigate through when time is of the essence. The idea is to work towards an understanding of how the body responds and how the vent does its thing so that we can make changes on the fly and anticipate the results that will come of any adjustment away from normal. But just to mention a few examples without going into the same level of detail as we did above, consider the following situations.

In the patient with a head injury or TBI, we often choose to aim for an $EtCO_2$ low-of-normal to what we'd typically use for a standard patient.[210] While we don't necessarily hyperventilate these patients anymore, we could adjust **MV** to a tighter $EtCO_2$ goal of 35-40mmHg by going up on either **TV** (preferred) or **RR**. Carrying on with this idea: we want to simultaneously avoid hypoxic events and minimize the effects of too much O_2. We also want to find a balance between maintaining comfort to avoid increases in ICP and being able to perform a neuro assessments to track patient progress. All that said, consider using tighter parameters and/ or reassessing these patients more often.[211]

In the pregnant patient we might utilize a **FiO_2** of 100% to maximize O_2 delivery to the fetus. Since many services don't have the capability of fetal monitoring during transport, this is a way to ensure that we don't have a hypoxic injury or put any undue stress on the fetus. We also need to consider an increased MV goal for the patient (which may mean an $EtCO_2$ goal low of normal, somewhere in the 30-35mmHg range), as we have a baby to consider as well.[212] Another consideration is patient positioning – in the vented pregnant patient we not only have decreased preload due to PPV, we could see that drop in CO compounded by pressure of the fetus on the inferior vena cava. So either turn the patient to a lateral recumbent position or displace the gravid uterus over to the side.

Significant chest trauma is another one. We'd like to treat these patients via the **Lung Injury** strategy, but we may also be concerned with hemodynamics and want to use the **Hypotension** strategy. Those two are at odds with one another (low TV and high RR for lung injury, high TV and low RR for hypotension). In this case we have to get creative. Maybe we forgo the hypotensive strategy and choose the lung injury one, but get aggressive early on with vasopressors and fluids and/ or 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 with lower **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.

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[210] Godoy & friends, 2017 – Detailed overview of this concept and research that has been done to date
[211] Refer back to **Oxygenation** for a discussion of the negative effects of O_2 and then to **Comfort** for a discussion of all that
[212] Wingfield, 2012a; LoMauro & Aliverti, 2015 – The idea was suggested in a video by the first guy; the physiology behind it is discussed in an article by the other two

mmHg – millimeters of mercury; **MV** – minute volume; **O_2** – oxygen; **OK** – alright; **PEEP** – positive end-expiratory pressure; **Pplat** – plateau pressure; **PPV** – positive pressure ventilation; **RR** – respiratory rate; **SpO_2** – pulse oximetry; **TBI** – traumatic brain injury; **TV** – tidal volume

On a tangent to this chest trauma idea: if a patient develops a tension pneumothorax en route, the best thing we can do is to take the patient off the vent.[213] Not take them off the vent and bag them, but take them off the vent and don't breathe at all for them until we fix that problem. PPV can tension a pneumothorax very quickly and we want to avoid making things worse.[214] So disconnect the vent, decompress (or place a chest tube/ perform a finger thoracotomy), and then get the patient back on the vent. Because of this, we may consider keeping all patients with the potential for pneumothorax on a FiO_2 of 100% – that allows us more time to perform the procedure, if a pneumothorax develops, before the patient desaturates.

A patient with CHF or pulmonary edema may warrant more PEEP to facilitate the movement of fluid out of the alveoli.[215] In addition, PEEP might help drop afterload to facilitate both perfusion and clearing of fluid from the pulmonary side of circulation. And while it may make sense that a high FiO_2 could mitigate the effects of an **HPV** effect in these patients, there is some risk to that strategy and treatment focused on adequate MV and PEEP are preferred with CHF.[216] Folks with COPD may ought to have oxygenation tightly controlled due to the potential effects of O_2.[217] We could even argue the case for a specific toxic-exposure strategy – some combo of lung injury plus or minus acidosis, depending on the agent and route of exposure.

It quickly becomes evident that there are a number of cases that don't quite fit the mold by which we try to simplify vent strategies. And that's totally OK. The templates are there as frameworks from which we then consider the specifics of each patient, one at a time. The important thing is to know what impact any vent change will have on the patient depending on how (s)he presents in a given situation. There are lots of cases in which there isn't a straightforward answer, but as long we don't make things worse by titrating things the wrong way, all is good.

[213] Flowers & friends, 2019 – And we may cause this tension by taking a stable pneumothorax to elevation, per Boyle's Law
[214] Wingfield, 2012b – Haven't seen this idea discussed elsewhere, but it seems appropriate to discuss for all of us transport folks
[215] Perlman & friends, 2010 – While a Pplat up to $30cmH_2O$ is likely still just fine with these patients, just know that pulmonary edema can make the patient more susceptible to injury (and this article discusses why that might be via a unique experiment)
[216] Kuhn & friends, 2016 – See discussion of these ideas here
[217] Swaminathan, 2015 – Short and sweet discussion of whether or not these are even valid claims

%TaDP – percentage of time at decreased preload; $EtCO_2$ – end-tidal carbon dioxide ; IBW – ideal body weight; I:E – inspiratory to expiratory; MV – minute volume; OK – alright

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 when 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 different patient types, the truth is that there is a lot of variation in how patients respond to the vent. Sometimes an asthmatic patient is happy with an I:E of 1:2, other times a hypotensive patient has a high RR and low TV for good reason, etc. Because of this, we need a method to determine when changes are needed and when we can leave things alone as we find them.

Getting the Intel Ready

The first thing we do for any patient who needs to be or is already ventilated is to listen. We listen to a report from whoever was hanging out with the patient before we got there. This is very important for all patients, as it can tell us how the patient has responded to or will respond to strategies we might have in mind. We then (as in *after* listening) decide on a strategy based on how we think that patient ought to be ventilated. 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**.

Following that 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. Our mental construct of a strategy based on the report we received should match what we see in the exam. If not, we need to clarify that amongst ourselves before moving forward. No need to elaborate on that here, we all know the importance of a good assessment. So once we have a report, have done an assessment, and are decided on a strategy, we move forward.

From Scratch

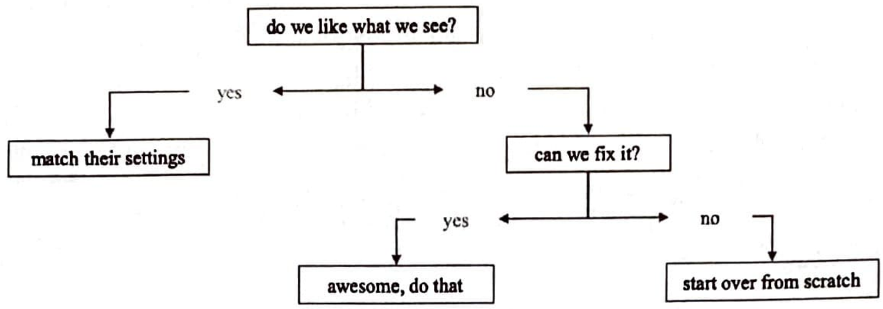
When we are the ones initiating the vent, it's fairly straightforward: we take the settings we've come up with based on presentation and pathophysiology, then plug them into whatever mode and method of control we decide to use. We've already talked about the different strategies and why we may choose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

Just to reiterate: the settings we conceptualize prior to initiating ventilation (and as discussed in the previous sections) 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 OK. But the starting point ought to be based on an understanding of what is going on with the patient and calculated goals. And if we have no idea which strategy to choose or if the patient fits too many categories all at once, just start with those basic settings we discussed in **A General Vent Strategy** and go from there.

PC – pressure control; **PEEP** – positive end-expiratory pressure ; **Pplat** – plateau pressure;
PRVC – pressure-regulated volume control; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control

Patient Already on the Vent

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



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 with what we had in mind? In the case of a hypovolemic patient with a high RR, for example, we may say, "yes, this strategy may be detrimental to the patient." In the case of an asthmatic patient with an I:E of 1:3 we may decide, "this isn't what I would've set up from scratch, but let's see if it is working for the patient or not before deciding to change things." The idea here is to see what puts our patient at risk and what doesn't: a high %TaDP and hypotension does put a patient at risk, while an I:E of 1:3 in an asthmatic with no AutoPEEP doesn't.[218]

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. The idea is to ensure that both perfusion and acid-base balance are all good, in the context of our vent strategy, and that we don't identify a life-threatening value or pattern of values with whatever information we have available. No need to get into specifics here, but if all is well in each of those general three subject areas, then there is no reason for us to go messing with settings and we should match what they are using.[219] The only exception here is if our machine can't do the settings they have. For example, if the patient is on **PRVC** and we don't have that choice, then we match their settings as best we can in either **VC** or **PC** and go from there.

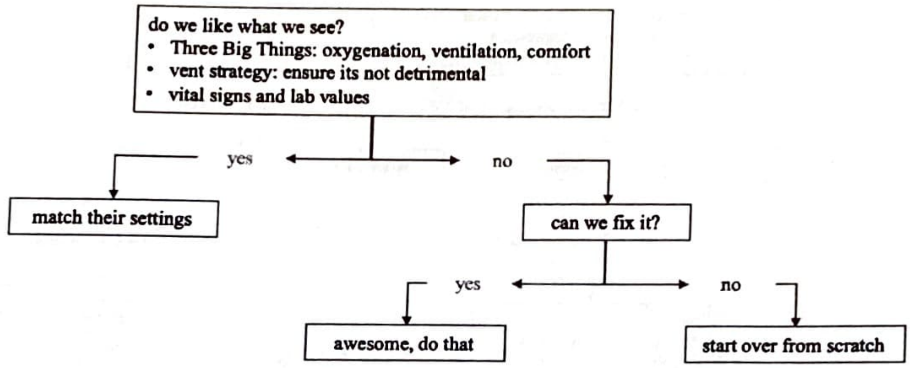
But what about checking a **Pplat** and **AutoPEEP**? If our patient is alive and well and passes an assessment in all three categories we just discussed (the Three Big Things, vent strategy, vitals and labs), then those things can wait until we get them on to our vent. Some reasons for this: the delay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

[218] To review these concepts: I:E ratio was in **Inspiratory Time (and I:E Ratio)**, %TaDP was in **Hypotension**, and **AutoPEEP** in this context was in **Obstruction**

[219] And to brush up on ABG interpretation, navigate to any of the following resources:
Woodruff, 2007 – Article that outlines a six-step approach
Smith, 2014 – Short video to review the basics; while he uses kPa versus mmHg to outline normal values, it's the best of this type we've been able to find
Strong, 2014 – Much more detailed video series on the subject

%TaDP – percentage of time at decreased preload; **EtCO₂** – end-tidal carbon dioxide ; **IBW** – ideal body weight; **I:E** – inspiratory to expiratory; **MV** – minute volume; **OK** – alright

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



Next question to discuss further is, "can we fix it?" We'd like to address 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.[220] 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.[221] If, however, we are getting into a situation where it will take lots of changes to set things right, it may make the most sense to start from scratch with a whole new set of parameters. And in that case we may as well change a bunch of things and go with our preferred strategy.

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

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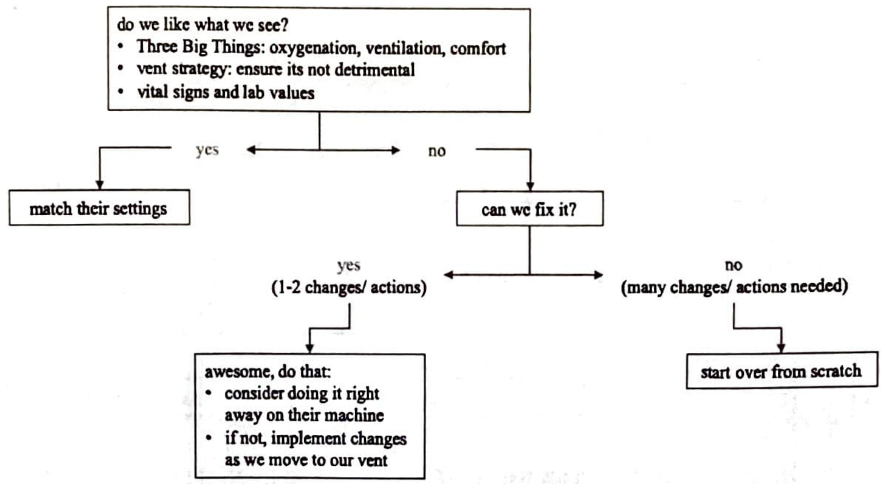
[220] And for help in deciding this, consider using Critical-Medical Guide – it's an app that's got a nifty feature in which we simply enter in current vent settings and an $EtCO_2$ goal and it spits out suggested vent changes

[221] We discussed these things in the sections on **Comfort**, **Ventilation**, and **Hypotension**

PC – pressure control; **PEEP** – positive end-expiratory pressure : **Pplat** – plateau pressure;
PRVC – pressure-regulated volume control; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control

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 weird, we can always skip ahead to the "start over from scratch" end of things. Just recognize that the more changes we make, the less able we are to evaluate the efficacy of a single intervention. Like a science experiment, it helps to isolate variables and know that the observed result can be attributed to a specific adjustment. And even though we mentioned it already, interpersonal dynamics also come into play here: make changes based on necessity, not on personal preference – that will help maintain positive relationships with referring staff and crews.

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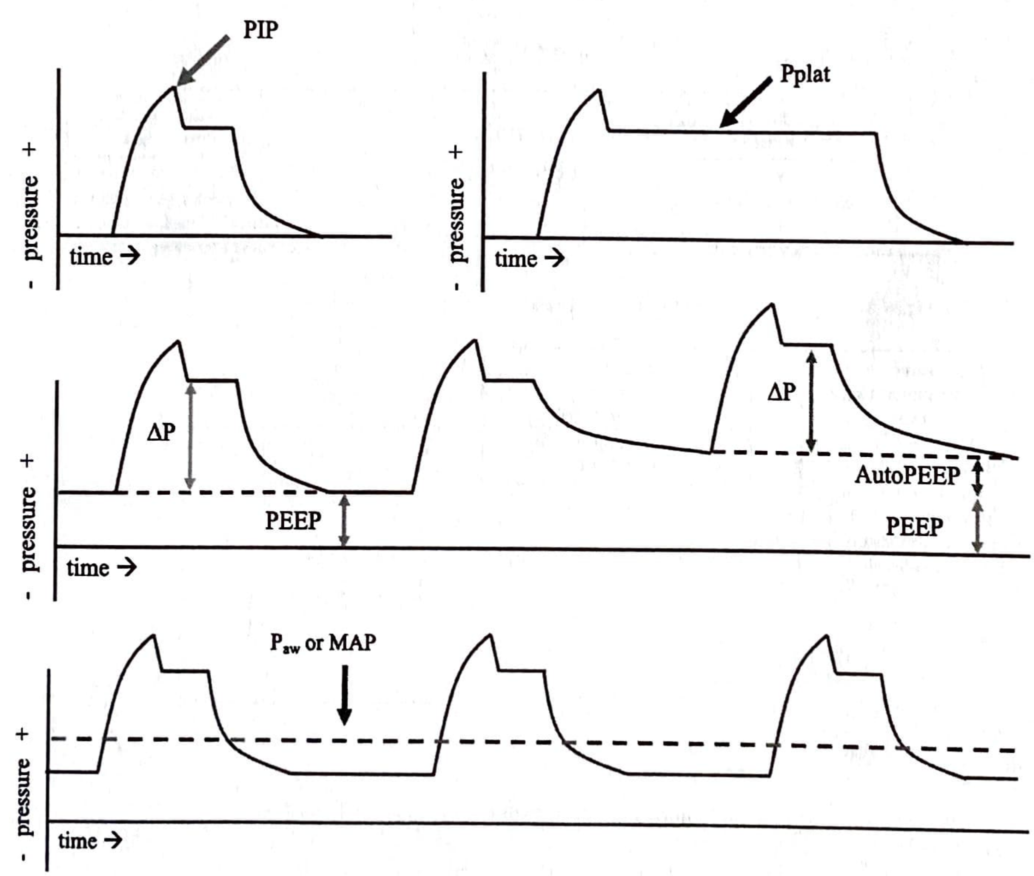
%TaDP – percentage of time at decreased preload; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **ETT** – endotracheal tube; **ICU** – intensive care unit; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **JEMS** – Journal of Emergency Medical Services; **kg** – kilograms; **MAP** – mean airway pressure; **FiO$_2$** – fraction of inspired oxygen

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 already talked about how we sometimes vary from the settings we start out at and this section explains how that happens. We want to both avoid injury and optimize air delivery, so we make adjustments to work towards those goals.

Watching Pressures

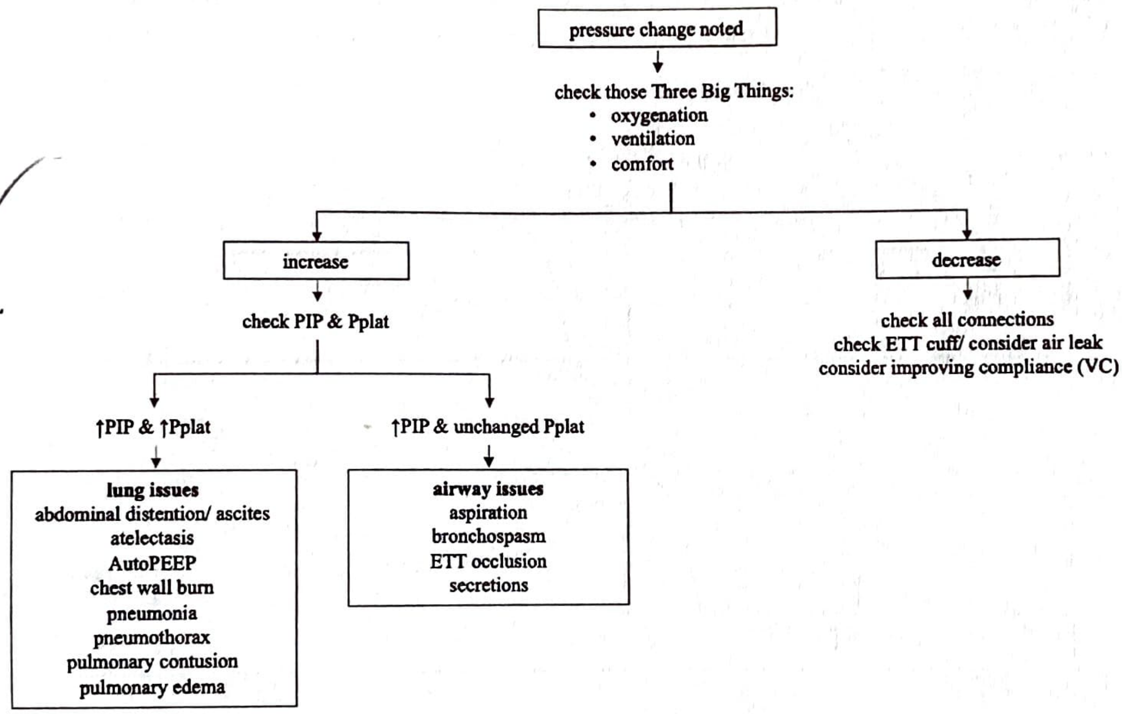
We discussed these things in the section titled **Vent Parameters, Round Two**, but here they are again: PIP, Pplat, AutoPEEP, ΔP, and P_{aw}. And for visualization, in case we forgot, here's what they look like on a pressure waveform in **VC** ventilation:



ml – milliliters; **MV** – minute volume; **MVe** – exhaled minute volume; **O_2** – oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{aw}** – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

High for PIP is 35cm$H_2$0, although we may go beyond that in certain situations (such as a small ETT). Pplat max is normally 30cmH_2O and we do try to stick by that one whenever possible except in those cases where Pplat may not reflect alveolar pressure.[222] AutoPEEP is normally zero; we generally take actions to address AutoPEEP when we see evidence of it, but may tolerate a small amount before addressing it. As for P_{aw}, we don't generally cite a normal range, but know that a change in this value can be the first indicator of an alteration somewhere in the system. 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.[223] It may help to simply add these pressures on to a mental list of vital signs to reassess as we go.

As far as what to do with this information once we have it, here's a flowchart to help sift through the information and take action to address potential problems:[224]



[222] We talked about these situations in **Plateau Pressure**

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

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

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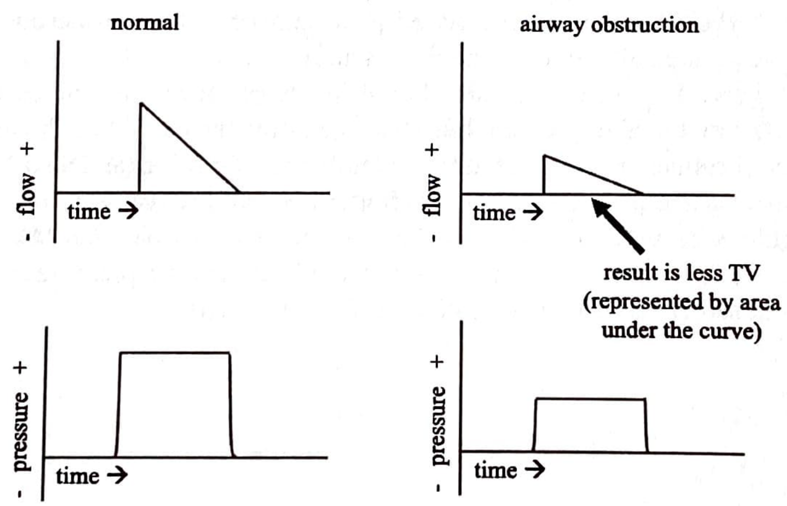
%TaDP – percentage of time at decreased preload; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **ETT** – endotracheal tube; **ICU** – intensive care unit; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **JEMS** – Journal of Emergency Medical Services; **kg** – kilograms; **MAP** – mean airway pressure; **FiO$_2$** – fraction of inspired oxygen

And then let's look at potential solutions for each of these cases:[225] [226]

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

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

In **PC** 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).[227] In the case of airway obstruction, oftentimes we won't notice initially because the machine essentially accommodates for this increased airway resistance by using less flow:



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

[226] And to link back to sections listed in this graphic: **Positive End-Expiratory Pressure**, **Inspiratory Time (and I:E Ratio)**, **Volume Control**, **Acute Lung Injury/ Acute Respiratory Distress Syndrome**, **Comfort**, and **Obstruction**

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

ml – milliliters; **MV** – minute volume; **MVe** – exhaled minute volume; **O_2** – oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{aw}** – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

Since we don't typically monitor waveforms with transport ventilators, an airway obstruction may not get noticed in PC ventilation until it is severe enough to impact MVe.[228] The best way to catch these sort of things before they have an impact on patient outcome is by setting alarms appropriately so that we are notified right away as things change (see following section).

Alarms[229]

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 into two general categories: ones that are default on the machine and ones that we set. Those default ones may be different between machines, but deliver similar messages like, "hey friend, our circuit got disconnected" and "oh snap, we ran out of O_2." 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 ones we'll focus on here.

One important alarm we set on the machine is the high-pressure alarm (which goes off when our high-pressure limit is reached). The reason this alarm is so important is that if it gets triggered, inspiration cycles off. 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 **VC** 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.[230] 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 VC 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 to avoid dropping our MV. On the flip side, in **PC** we need to vigilantly monitor MVe (and also VTe) to avoid the same issue (of decreased MV). This leads us to the next most important alarm we can set: low minute volume. We set this limit at a reasonable value below our MV goal so that if things get weird and MV starts to drop, we get notified right away before our patient suffers. In this way, we utilize the high-pressure and low MV alarms to simultaneously ensure both safety and adequate ventilation for our patients.

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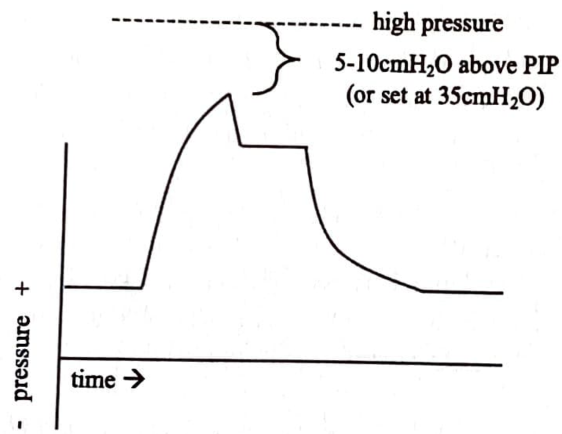
[228] We can also (again, this is in PC) look at flow as calculated and delivered automatically by the vent – higher flows mean less resistance, so even if we don't know ranges or normal values we can still use this concept to trend changes

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

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

%TaDP – percentage of time at decreased preload; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **ETT** – endotracheal tube; **ICU** – intensive care unit; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **JEMS** – Journal of Emergency Medical Services; **kg** – kilograms; **MAP** – mean airway pressure; **FiO_2** – fraction of inspired oxygen

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



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

As for the low MV alarm: set that within 25% of the MV goal that we calculated when we first started into this process of getting the patient on the vent.[231] If we have a patient breathing in excess of that goal and we want to know if that changes, we just set the low MV goal 25% below what they are currently at. In any case, the low MV alarm is just a catch to alert us when we've missed a change. Typically we will be on top of these trends and notice things before the alarm even gets sounded, but sometimes we get distracted by other interventions and this backup system can keep us informed.

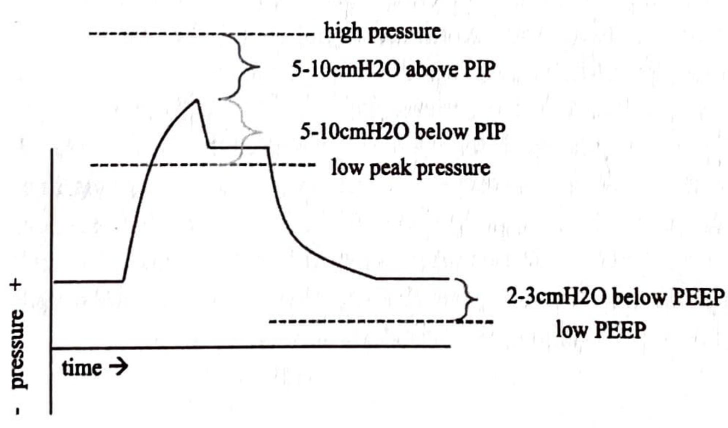
[231] And this 25% figure is an arbitrary number that we feel is appropriate, there aren't too many specific recommendations for this type of thing

ml – milliliters; **MV** – minute volume; **MVe** – exhaled minute volume; **O_2** – oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{aw}** – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

Other alarms that we can set to help us better keep track of what's going on with the vent and our patient are low peak-pressure, low frequency, high frequency, and low PEEP. Low peak-pressure alerts us when the PIP is lower than we would expect; this could indicate a cuff leak, increase in patient's respiratory effort (i.e. negative pressure produced with patient effort), 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).[232] Low frequency can let us know if the patient's RR starts to decrease – this is good if the patient is consistently breathing above a set RR and we want to be aware if that intrinsic effort changes. And reasonably enough, the high-frequency alarm advises us when the patient starts to breathe faster or if some mishap is causing the machine to think that (s)he is.[233] Lastly, low PEEP lets us know if the end-expiratory pressure drops below our set PEEP – this could indicate a leak, cuff deflation, or even an uncuffed tube (with pediatrics) that is too small.

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 we 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 us fine-tune the feedback from the vent so that we can better monitor what's going with the patient.

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



[232] Weingart, 2019 – This podcast proposes the idea that vent alarms ought to be addressed in the same way as a "code blue" in the hospital setting

[233] Which we call auto-triggering and will discuss again shortly in **Triggers**

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%TaDP – percentage of time at decreased preload; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **ETT** – endotracheal tube; **ICU** – intensive care unit; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **JEMS** – Journal of Emergency Medical Services; **kg** – kilograms; **MAP** – mean airway pressure; **FiO_2** – fraction of inspired oxygen

Titrating Up on TV?

Up to this point we've recommended considering **TV** above that 6-8ml/kg range in just a few circumstances: to increase MV (in the **Ventilation** section), with airway **Obstruction**, and as part of the **Hypotension** strategy. We also said that we want to limit our Pplat to a safe level <$30cmH_2O$ whenever possible, which includes when we decide to go up on TV.[234] The idea here is that more TV is OK, but only to a certain limit. And the best tool we have to establish that safe limit in the transport settings is Pplat, so that's what we use. All that said, it is worth discussing this idea further to see what we know about increasing TV and some of the intricacies of the whole idea.

One underlying idea here is that TV is a component of **P_{aw}** and that this is a determinant of oxygenation.[235] This means that it might make sense to go up on TV as much as we can (and within safe limits) to maximize oxygenation.[236] Increasing TV could also allow us to go down on **RR** (to keep MV constant). While this could, in rare cases, take away from P_{aw}, it could help in other ways (i.e. by decreasing that %TaDP value we made up previously).[237] Now regardless of motive, this strategy of increasing TV is a bit at odds with the lower TV, lung-protective approach pioneered by the ARDSNet studies.[238] That said, those studies looked at TVs of 6ml/kg versus 12ml/kg, so there may be some middle ground we just don't know much about.[239]

In light of this conversation, let's just say that we want to go up on TV for whatever reason. We've already said that our upper limit for Pplat is $30cmH_2O$, so that's one limiting factor in the game. Another concept here is that we'd prefer to make changes slowly; rather than jumping from 6ml/kg to 10ml/kg (or whatever other arbitrary amount), we get there in a stepwise fashion in small increments.[240] And lastly, we can utilize **Compliance** to help guide us towards our goal.

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234 Way back in the section on **Plateau Pressure**

235 Lodeserto, 2018 – We cited this once already in **Mean Airway Pressure**

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

237 And while this normally won't happen, it could possibly in the case where compliance is awesome at a low TV and awful at a higher TV – we explore this idea more in the **Appendix**

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

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

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

ml – milliliters; **MV** – minute volume; **MVe** – exhaled minute volume; O_2 – oxygen; PaO_2 – partial pressure of arterial oxygen; P_{aw} – mean airway pressure; **PC** – pressure control; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

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

volume control example

step #	TV (ml)	Pplat (cmH_2O)	compliance (ml/cmH_2O)	action
1	500	15	50	increase TV
2	525	16	48	increase TV
3	550	16	50	increase TV
4	575	21	36	decrease TV
5	550	16	50	no change, monitor
6	550	14	61	increase TV

Note that even though Pplat doesn't get up to our previously established limit of 30cmH_2O, we recognize that an increase beyond a TV of 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).

And as for a how it looks in PC:

pressure control example

step #	PC (cmH_2O)	VTe (ml)	compliance (ml/cmH_2O)	action
1	10	500	50	increase PC
2	11	550	50	increase PC
3	12	550	46	increase PC (or stay)
4	13	550	42	decrease PC
5	12	550	46	no change, monitor
6	12	600	50	increase PC

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 holds true. Also, this whole concept can be considered as an icing-on-the-cake sort of thing – we may not get to this point in our vent management and that's just fine.

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

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ABC – airway, breathing, circulation; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **BLS** – basic life support; **BVM** – bag valve mask; **DOPE** – displaced tube, obstruction, pneumothorax, equipment failure; **DOTTS** – disconnect the vent circuit, ~~O_2 100% via BVM~~, tube position or function, tweak vent, sonography; **EMS** – emergency medical services; **$EtCO_2$** – end-tidal carbon dioxide

Acute Deterioration

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

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

There are also some variations of this ~~guy,~~ so we may see it out there with an "S" at the end for stacking (i.e. **AutoPEEP**),[241] an "R" at the end for rigidity of the chest wall (a rare complication of Fentanyl administration),[242] or even with the "P" to represent pain and/ or (Auto)PEEP.[243] It is also sometimes accompanied by another mnemonic called DOTTS which outlines actions that can be taken to fix issues identified by DOPE. Now DOTTS includes a step where we bag the patient with a BVM and we've crossed that step out – we don't recommend routinely taking someone off the vent unless we have good reason to and we'll get back to this idea in just a little bit. But just so we can see it in its true representation, here it is:[244]

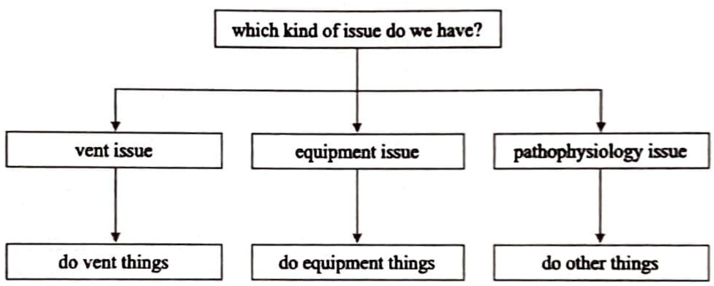
the DOTTS mnemonic		
	action	**explanation**
D	disconnect the vent circuit	to fix AutoPEEP or decreased preload (i.e. pneumothorax or hypotension)
~~**O**~~	~~O_2 100% via BVM~~	~~to manually assess for issues (i.e. look, listen, feel)~~
T	tube position or function	includes assessing placement and suctioning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

[241] Rezaie, 2018 – Also gives on overview of the DOTTS idea discussed below
[242] Thomas & Abraham, 2018 – While not all that common, it may be worth keeping in mind
[243] Wright, 2014 – A great read in general, but specific to this cause he's got a nice DOPE graphic that he got from another source
[244] To link back to sections mentioned in this chart: **Hypotension**, **Respiratory Rate**, **Tidal Volume**, and **Inspiratory Time (and I:E Ratio)**

ETT – endotracheal tube; FiO_2 – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; MV – minute volume; O_2 – oxygen; PaO_2 – partial pressure of arterial oxygen; P_{aw} – mean airway pressure; PEEP – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; SpO_2 pulse oximetry; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

The DOPE mnemonic (with or without DOTTS) is easy to remember and can be used to guide the initial troubleshooting process when the patient starts to tank due to some unknown.[245] Many of these occurrences can be tied to **Alarms** or other assessment parameters, but that depends on which type of machine we are working on and what tools we have available. For example, a tube displaced too deep will give a high-pressure alarm (and eventually a low MV alarm) and a tube displaced out of the airway will likely result in a low-pressure alarm. In regard to other assessments: a tube displaced too deep will lead to a high **P_{aw}** or **PIP**, low VTe, patient discomfort, etc. and a tube displaced out of the airway causes a low P_{aw}, drop in $EtCO_2$ with change in waveform, hypoxia, etc.

Because there are so many things to consider, building an algorithm to troubleshoot each possibility gets a bit difficult. We'll go ahead and do it anyway, we just need to consider a few more things in preparation. First of all is that acute deterioration of the vented patient doesn't always mean that there is an issue with the vent – it could be some other issue beyond the vent (i.e. ETT displaced or pathophysiologic process). If it's a vent thing, then we mess around with the vent; but if it's another issue, our interventions should focus on drugs and procedures and that sort of thing. Think of it this way:



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

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[245] Weingart, 2011 – For some useless trivia on where this mnemonic came from, take a look here

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ABC – airway, breathing, circulation; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **BLS** – basic life support; **BVM** – bag valve mask; **DOPE** – displaced tube, obstruction, pneumothorax, equipment failure; **DOTTS** – disconnect the vent circuit, ~~O_2 100% via BVM~~, tube position or function, tweak vent, sonography; **EMS** – emergency medical services; **$EtCO_2$** – end-tidal carbon dioxide

In light of the fact that there are so many variables involved, here's the stepwise approach we suggest for troubleshooting acute deterioration of a ventilated patient. This approach takes advantage of feedback that we may have available to us from vent alarms and assessment parameters:[246]

acute deterioration: what to do!?

↓
start with the basics: ABCs
↓
use alarms to guide treatment
↓
consider the Three Big Things
↓
address airway pressures

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

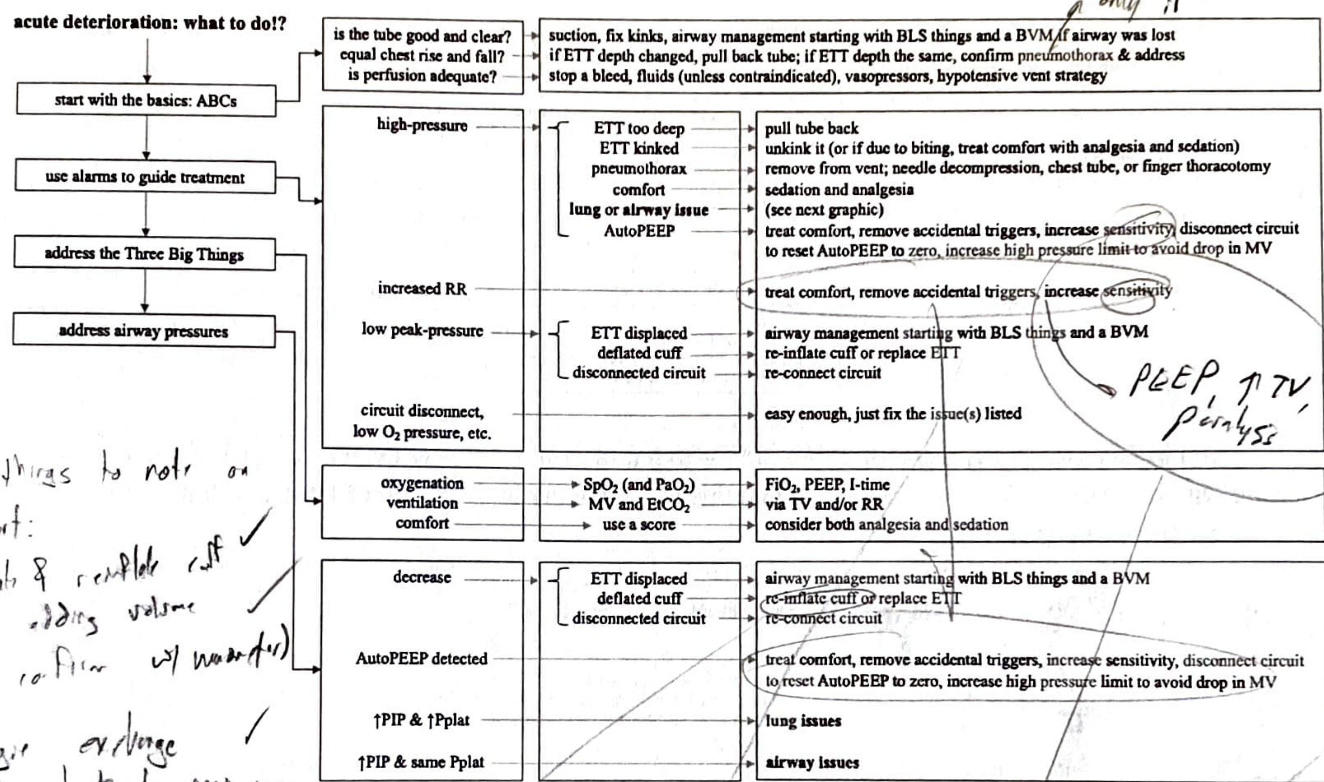
acute deterioration: what to do!?

↓
use alarms to guide treatment
↓
then move on to the ABCs
↓
consider the Three Big Things
↓
address airway pressures

[246] And to refer back to these things: <u>Three Big Things</u>, <u>Keeping Things Going</u> (and specifically, <u>Alarms</u>)

ETT – endotracheal tube; **FiO₂** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **MV** – minute volume; **O_2** – oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{aw}** – mean airway pressure; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **SpO_2** pulse oximetry; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

By working through each of these steps systematically, we hit all of the DOPE things and identify where in the system the issue lies (vent, equipment, physiology). Now, it gets a bit more complicated when we add in specifics for each step along the way, but remember that the basic idea is a simple set of four steps:[247]



[247] And refer back to **Vent Parameters, Round Two** to review the pressures

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

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

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

Now let's summarize what actions to take in the event of an acutely deteriorating patient on the vent. While there is a well-known memory tool (the DOPE mnemonic) to guide us through troubleshooting potential issues, that tool doesn't consider feedback from the machine (i.e. alarms) and, therefore, we suggest a simple sequence of four steps to work through it all: check our ABCs, look at and address any alarms, review the **Three Big Things**, then check pressures. If by then we haven't figured out our problem, we can consider taking the patient off the vent and bagging by hand (still not a great strategy though…) or getting out the ultrasound machine to try and identify an issue (if available).[248]

[248] Mojoli, 2017 – And for those of us who do have ultrasound, here's a short article that discusses application in mechanical ventilation

ETT – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **MV** – minute volume; **O_2** – oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{aw}** – mean airway pressure; **PEEP** – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **Pplat** – plateau pressure; **RR** – respiratory rate; **SpO_2** pulse oximetry; **TV** – tidal volume; **VC** – volume control; **VTe** – exhaled tidal volume

The final idea here is what to do if the patient goes into cardiac arrest while on the vent. Standard practice in this situation is to take the patient off the vent and have someone ventilate with a BVM. That may be a valid option if we have extra hands, but in transport with only two clinicians that may not be possible. If our particular machine allows us to cancel out patient-triggered breaths, we may ought to do that and free up those hands. The reason for turning off patient-triggered breaths is that the machine will likely be triggered to deliver a breath with each chest compression given. The only caveat here is that we will likely need to increase our high-pressure limit all the way so that breaths don't get cut short early during this time. Now most people are using BVMs with pop-off valves anyways, but the difference in a human giving breaths via a bag and a machine doing it by a timer is that the clinician can adjust the timing of breaths to give them in between compressions while the machine cannot.

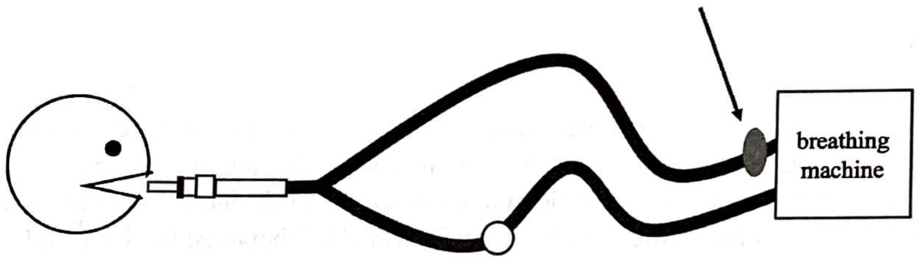
One last recap and then we'll move on. With a deteriorating patient on the vent, try to keep it simple and work through four steps: ABCs, alarms, Three Big Things, and pressures. If after that you can't figure out the issue, consider BVM ventilation or other assessment techniques. And in the ~~vent~~ that the patient arrests while on the vent, cancel out patient-triggers and increase your high-pressure limit.

ΔP –driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH₂O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO₂** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO₂** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O₂** – oxygen; **OK** – alright

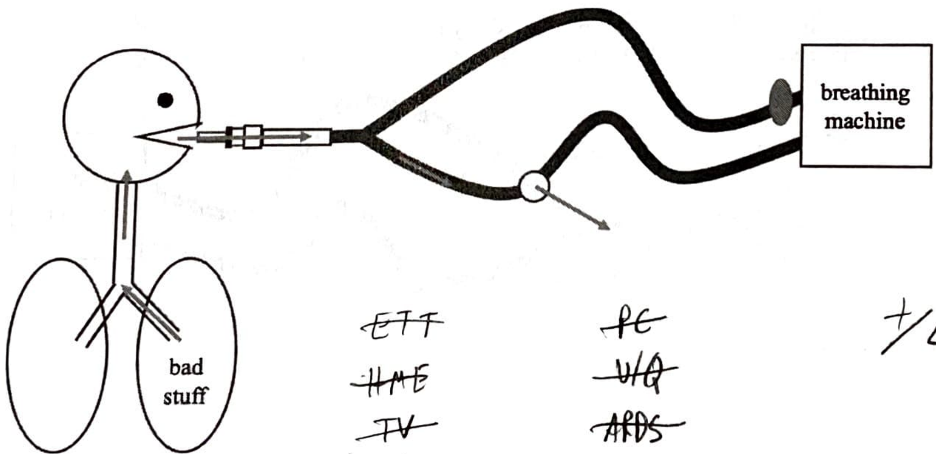
Additional Concepts

Filters[249]

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 into the vent circuit. While there are a few possible options as to where we place the filter, it is 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 at the machine from getting to the patient. Which is fine, just recognize that it doesn't keep bad stuff at the patient from getting to us and our coworkers:



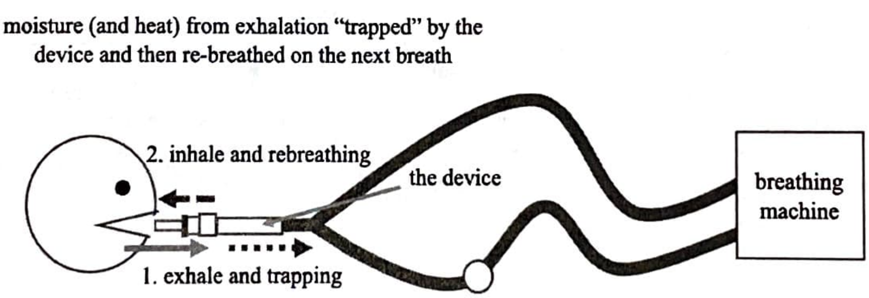
[249] Wilkes, 2011a & 2011b – He gives the most in-depth discussion of both filters (this section) and humidifiers (next section)

PC – pressure control; **PCO_2** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO_2** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO_2** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO_2** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/ perfusion; **VTe** – exhaled tidal volume

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 and the exhalation side option may not be available with our transport vent.[250] 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 (i.e. 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 exhaled volumes. But if we have a patient with some type of bad stuff that we don't want to breathe in and neither of these strategies is appropriate or possible, be sure to mask up!

Humidifiers[251 252]

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 between the ETT and wye of the vent circuit. Placing the device further up on the inhalation side of the circuit would not work, as the device functions by trapping moisture (and also heat) from exhaled air and allowing it to be blown back into the patient's airways on the subsequent breath:



[250] The impact of adding the HME (and other devices) to the circuit is discussed in both **Dead Space** and the **Appendix**
[251] Yartsev, 2019 – Excellent discussion of the passive style devices used in the transport setting
[252] Gillies & friends, 2017 – This Cochrane Review has determined that HMEs are comparable to actual humidifiers in providing therapeutic benefit and avoiding primary complications (airway obstruction, pneumonia, mortality) – while they admit that more research is needed, it's good to know that HMEs do have demonstrated value

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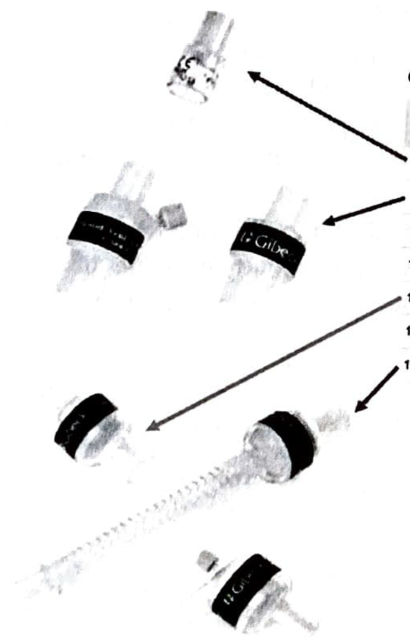
ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O_2** – oxygen; **OK** – alright

The HME is often the biggest contributor to mechanical dead space (as outlined in the **Appendix**), but it ought to be used unless we have good reason not to. One of these good reasons not to would be a small **TV**, such as with kids or **Lung Injury** patients.[253] In these situations, we want to minimize mechanical dead space as much as possible. Now there are smaller HMEs designed for pediatrics 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.[254] To make this clear, let's look at info from one particular product line:[255]



Gibeck® Humid-Vent® HME

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

we see here that more capacity for humidification means more dead space

[253] Hinkson & friends, 2006 – And we'll get back to this idea in the **Appendix** also
[254] Which means we could theoretically use a smaller-sized HME for an adult patient with some low-volume strategy
[255] Teleflex, 2019 (images) – Just to be clear, no relationship or conflict of interest here; it's just really nice how they lay out all the product info like this for us to talk about ☺

PC – pressure control; **PCO_2** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO_2** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO_2** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO_2** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/ perfusion; **VTe** – exhaled tidal volume

Second good reason not to use an HME would be the concurrent use of nebulized medications.[256] 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 would 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. 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 airflow. This isn't a case 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.[257] If this happens and we are worried about an HME getting clogged up, we can either remove the device or replace it with a fresh one.

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

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

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

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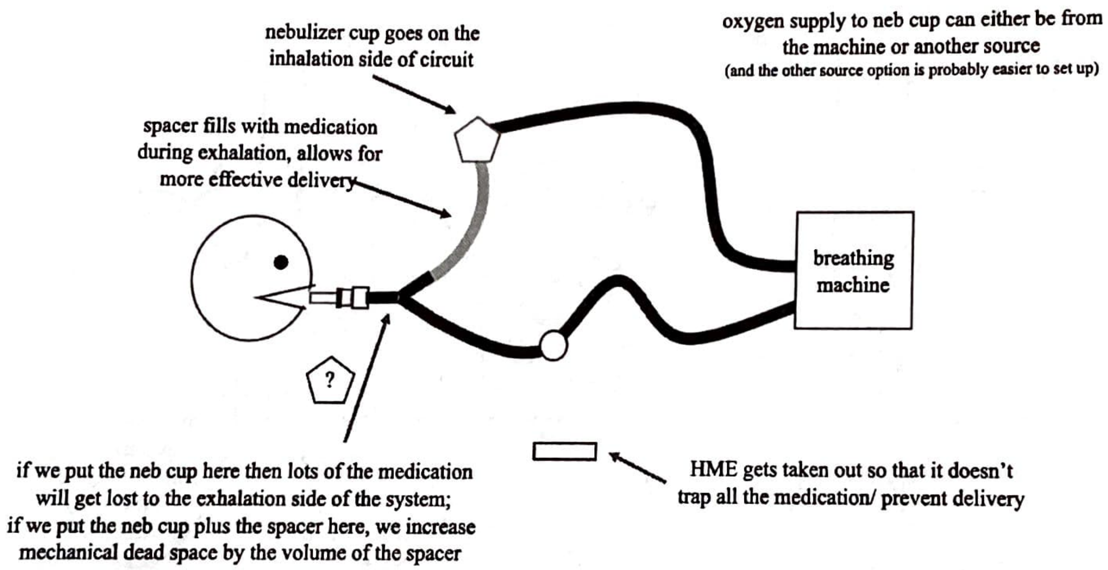
ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO$_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO$_2$** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O$_2$** – oxygen; **OK** – alright

In-line Nebulization

Just to demonstrate a few things about why we do nebs the way we do, let's look at a setup of how the system looks when we nebulize a medication through the vent circuit. Recognize that there may be some variation between models, this is just the setup with which we are most familiar with and serves to outline the important stuff:[258]



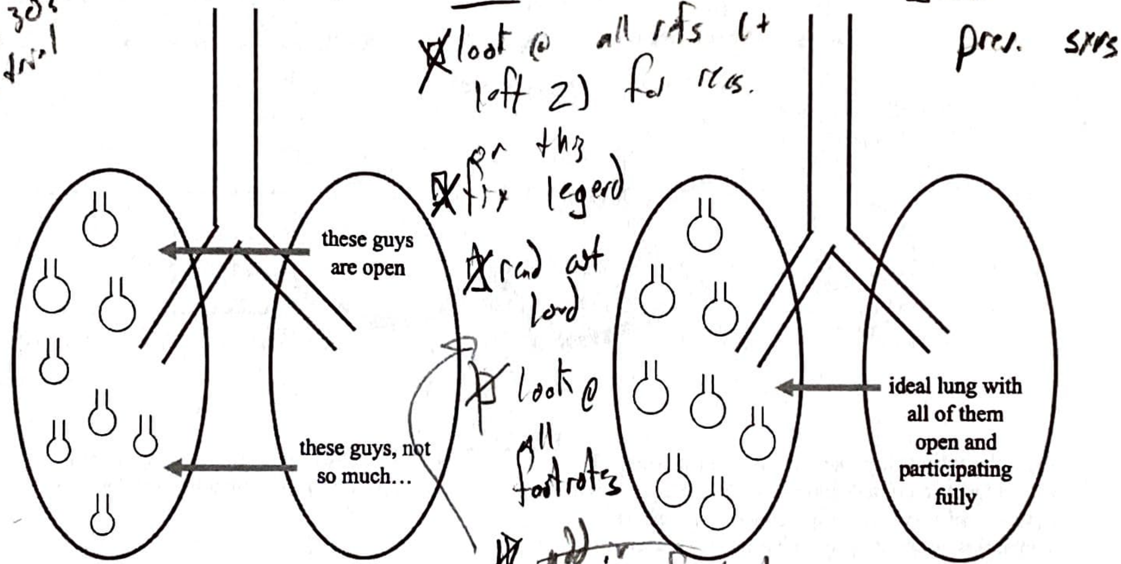
That should be clear enough, but just to expand on a few points: we may need adapters and extra vent tubing to make this work, so we should plan ahead and have that stuff available in pre-built kits. The spacer is important, as medication will be lost to the exhalation side of the circuit if it isn't there. Some machines recommend specific changes to settings to facilitate this process, read up on that or have a chat with the manufacturer's rep for details about a particular machine.

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

PC – pressure control; **PCO$_2$** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO$_2$** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO$_2$** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO$_2$** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/perfusion; **VTe** – exhaled tidal volume

Recruitment Maneuvers

A recruitment maneuver is a component of the open-lung strategy that seeks to get more alveoli involved in the ventilation process.[259] During ventilation, and even in the healthy lung, there are portions of the lung that are open or participatory and others that are closed down or non-participatory (or maybe just less-than-optimally-participatory), and we can do things to gain access to those clamped-down alveoli to improve both ventilation and oxygenation:



In a general sense, lots of things could qualify as recruitment maneuvers: prolonged inspiratory holds, higher **PEEP**, high-frequency oscillation ventilation,[260] prone ventilation, spontaneous breathing, etc. Basically anything that can help open those non-participatory alveoli falls into this category.[261] In the transport setting (and, in fact, for most vent people), we tend to consider recruitment maneuvers to be either the prolonged inspiratory hold or the stepwise approach, so we will stick with those two ideas moving forward.[262]

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259 Ragaller & Richter, 2010 – We talked about the advantages of this both in **Alveolar Surface Area** and **Oxygenation**; the article is an overview of ARDS management with reference to this idea of an open-lung approach and a section on the idea of recruitment
260 Prost, 2011 – This is the only mention we have of this mode, as it isn't routinely available in transport; the referenced video is an overview of it
261 Naik & friends, 2015 – This is an article that also discusses recruitment, but particularly the idea that breaths of various sizes (whether intentional via vent management or spontaneous via patient effort) further contribute to recruitment
262 Hartland & friends, 2015 – This paper both discusses this idea and describes the use of recruitment maneuvers in non-ARDS patients under anesthesia

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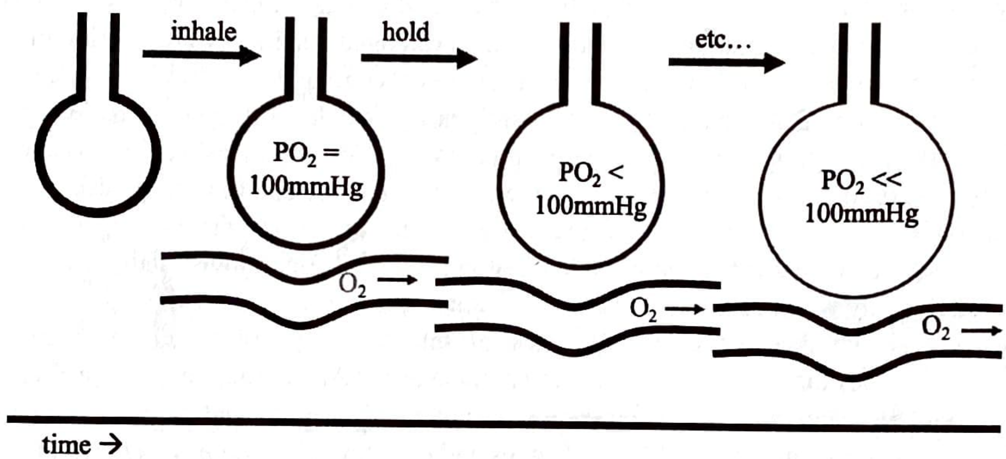
ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO$_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO$_2$** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O$_2$** – oxygen; **OK** – alright

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 O_2 and let it sit like that for a while. We said then that we still have to consider the ventilation side of things, but the idea itself does have some merit. The value of a recruitment maneuver (again, this is as a prolonged inspiratory hold) is more in the ability to keep alveoli open than in the inflow of O_2 for a sustained amount of time, as the amount of O_2 in that air quickly begins to drop as O_2 diffuses into the bloodstream and we don't replenish the supply:



A recruitment maneuver in this sense can be used to gain recruitment in any patient group, but has been most studied with ARDS patients. And while it has been shown to increase oxygenation, outcomes in terms of mortality and days on the vent seems to be unaffected or even worse.[263] To further complicate things, when we do try and get into the weeds as to how we should perform a recruitment maneuver, techniques vary significantly, and there are potential adverse effects. So here's where we stand on this: more data is clearly needed, but there is low-quality evidence that some benefit exists from performing recruitment maneuvers in ARDS patients; particularly as part of an overall open-lung strategy.[264] Translating that to the non-ARDS patients who are simply hypoxic is a bit tough, as there isn't much data out there and we can often fix the issue by way of things we've already talked about (**FiO$_2$**, **PEEP**, and **I-time**) and ensuring adequate perfusion.[265]

[263] van der Zee & Gommers, 2019 – This article describes lots of the research that has gone into understanding ~~this whole concept~~

[264] Hodgson & friends, 2016 – Cochrane Review that gives way more detail on this

[265] This was discussed in **Oxygenation**

But let's say we do want to do a recruitment maneuver anyways. Maybe we are struggling to oxygenate a patient, or we forgot to clamp the ETT on transfer of an ARDS patient to our vent, or we want to try for better **Compliance** and/ or decreased **Driving Pressure**, etc. First thing to know is that the maneuver can cause hemodynamic problems and we ought to be on the lookout for those to avoid decompensation. Just as we discussed back when we first got into **How is Positive Pressure Different?** and **PEEP**, an increase in intrathoracic pressure can drop preload and subsequently impact CO. So monitor all the things and have hard limits in place for abandoning the maneuver.[266] Also, recognize the risk for causing a tension pneumothorax and consider that a floppy ETT cuff or uncuffed pediatric tube will render the maneuver less effective.[267]

The simplest way to do a recruitment maneuver is the prolonged inspiratory hold option that we mentioned above.[268] While this was often taught in the past, it is becoming less common in deference to more gentle and stepwise strategies. But to make it happen, here's how it would work: put our patient in **PC**, set pressure control to get a goal **Pplat**, then perform an inspiratory hold for as long as we think is appropriate. As far as specific on pressures and time, the data varies widely on that and we can't make specific recommendations on how that might look. Same goes for how often to perform the maneuver – most of the data out there discusses vented patients in an in-patient setting, so it is difficult to translate that to the transport setting in which we are only with the patient for a short amount of time.[269]

We mentioned already that whenever we put more air into the lungs it seems advantageous to do so incrementally.[270] Same goes for performing a recruitment maneuver. An alternative to the prolonged inspiratory hold would be a stepwise approach in which we put a patient in PC and establish a **ΔP** that yields our goal TV, then slowly titrate up on PEEP in small steps and over time.[271] There is a rendition of this approach called the Staircase Recruitment Maneuver that titrates PEEP back down to a maximally beneficial level as determined by SaO_2 monitoring – perhaps a modified version with SpO_2 monitoring and longer times between titrations (to accommodate a potential lag in SpO_2 readings) would be appropriate in transport.[272]

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266 Claire & friends, 2019 – And for suggestions on these limits and an explanation of the next technique (the stepwise recruitment maneuver), take a look at this short guide
267 Chambers & friends, 2017 – This study primarily examined how VTe differed from delivered TV with cuffed and uncuffed tubes, but it also looked at the effect recruitment maneuvers have on this difference
268 Metz, 2016a – Video that shows this type of recruitment maneuver in a set of lungs attached to a vent circuit
269 And we recognize that the lack of concrete suggestion here might be frustrating, but this is one of those things better answered by the agency or medical director that we work for…
270 In the section, **Titrating Up on TV?**
271 Metz, 2016b – Another video by the same guy as above, this one is a version of the stepwise recruitment maneuver
272 Hess, 2015 – Take a look here for a discussion of this technique and others

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ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH₂O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO₂** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO₂** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O₂** – oxygen; **OK** – alright

In any event, the utility of recruitment maneuvers is to get more alveoli involved in ventilation. This improves compliance and allows us to ventilate to our TV goal with lower ΔP while working to correct V/Q mismatch across the lung.[273] While there are risks involved and the data is a bit vague when it comes to long-term benefits, it seems fair to conclude that if we mitigate those risks by using a stepwise approach and monitoring for patient decompensation along the way there is likely some use in the transport setting.

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

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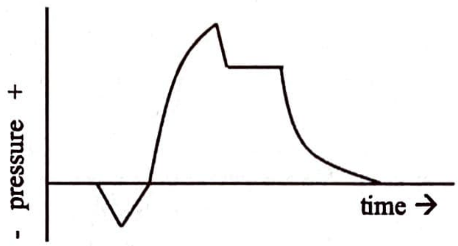
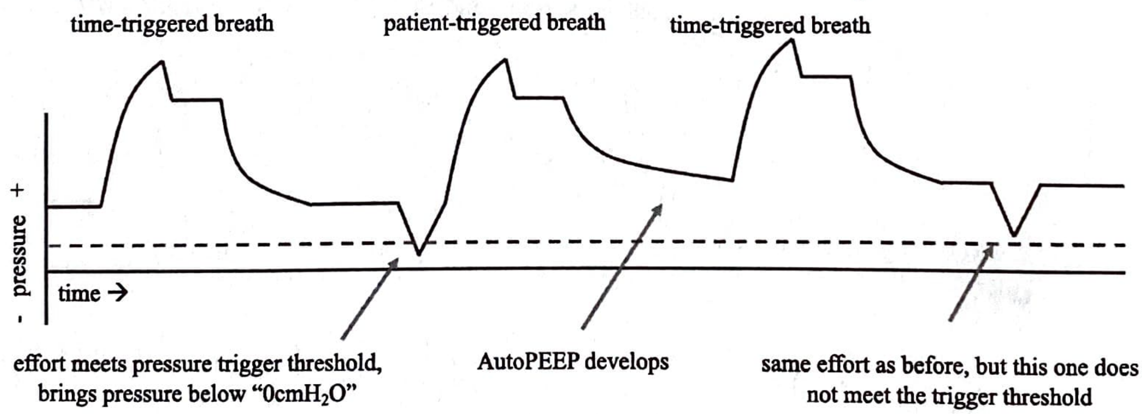
Triggers

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



And then we footnoted the idea that that downward dip in pressure at the start of the waveform is more a sketch of convenience than an accurate representation of how things normally occur.[274] In most cases the trigger that makes the machine recognize patient effort is based on flow rather than pressure. While some machines will allow us to use pressure triggers (normally around -1cmH_2O), this isn't commonly used. Pressure triggers have been shown to be more difficult for patients to overcome (at least with older model ventilators). In addition, the pressure trigger is relative to what we have dialed in for **PEEP** – this means that in the event of **AutoPEEP** there is an extra threshold that must be overcome:[275]



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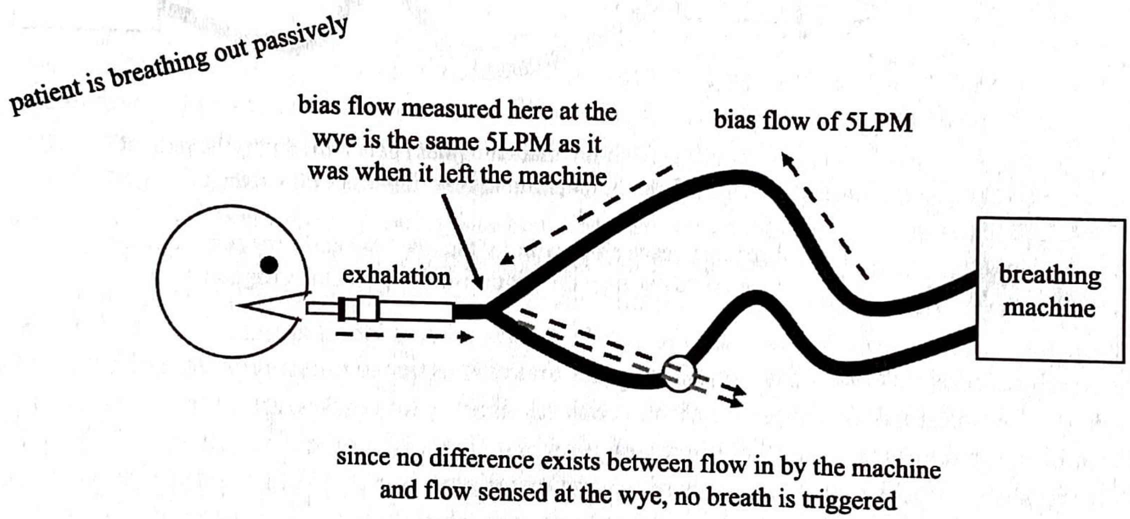
274 This was when we were talking about modes, in the section on **Assist Control**

275 Hess, 2005 – This explains how switching to a pressure trigger may mitigate breath stacking or AutoPEEP

ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH_2O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO_2** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O_2** – oxygen; **OK** – alright

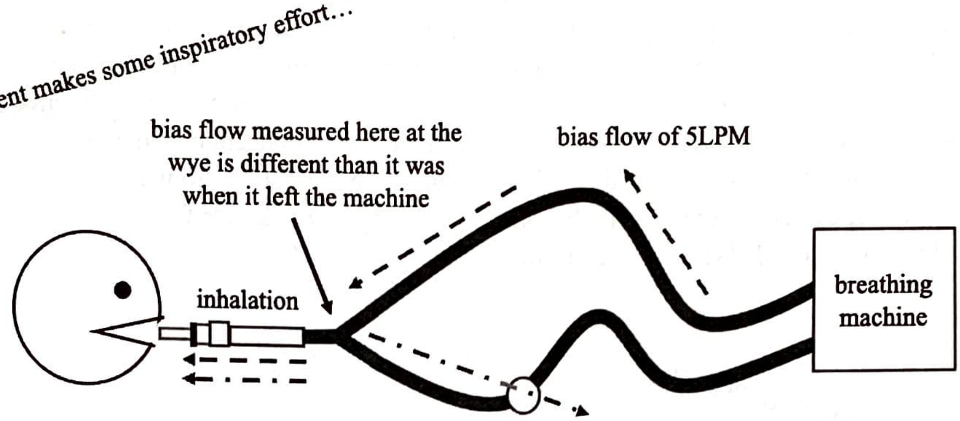
So pressure triggers are a thing as we initially drew it out, but not the most common thing. We sometimes do use pressure triggers in cases of auto-triggering (i.e. when we see too many triggered breaths due to things other than patient effort, such as bumpy roads in an ambulance or turbulence in an aircraft), but for the most part we stick with flow triggers.[276] To measure flow changes against a zero reference (i.e. we assume the pause between breaths to be a zero-flow state) the machine uses a concept called bias flow. Bias flow is a baseline flow of air into the system against which changes are measured. So when the machine says there is no flow going into the system, there is actually some flow going in, but it gets factored out. Let's draw it out with an assumed bias flow of 5LPM just to see how it works:[277]



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

[277] Yartsev, 2019 – For more information on these triggers, others, and some of the stuff discussed in the rest of this section, take a look at this article; it also cites the normal value of a flow trigger we mention on the next page

PC – pressure control; **PCO_2** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO_2** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO_2** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO_2** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/ perfusion; **VTe** – exhaled tidal volume



The machine does this bias flow thing because it makes it easier to measure patient effort. It also allows for things like PEEP and the delivery of nebulized medications.[278] The point worth knowing is that a flow trigger cannot be set to a value greater than the machine's bias flow. So in the case where we have lots of accidental triggers (i.e. auto-triggering is happening) and our trigger is set at 5LPM and we know our machine has a bias flow of 5LPM, we can do one of two things on the machine: switch to a pressure trigger or change (increase) bias flow to accommodate a higher trigger threshold.[279]

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

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

[279] That said, it is unlikely that we would utilize a pressure trigger this high unless we are experiencing some kind of extrinsic auto-triggering and want to prevent breaths from stacking (i.e. severe turbulence or a very rough ambulance ride)

ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO$_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO$_2$** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O$_2$** – oxygen; **OK** – alright

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

Prone Ventilation

Prone ventilation is when we lay our ventilated patient face down on the bed or stretcher. Arguments and evidence in favor of prone ventilation include things like better V/Q match, decreased shunt, improved **Oxygenation**, better **Ventilation**, etc.[280] That said, prone ventilation isn't for everyone, studies are shrouded a bit by bias, and efficacy seems to be related to early implementation, time of application each day (16 hours per day!), and severity of hypoxemia (i.e. proning has benefit when oxygenation is a major issue).[281] When we are called to transport a pronated patient, there are some logistical limitations to the process. Much of what we do requires access to the patient's front side and many of the tools we use in medicine are designed with the supine patient in mind. All that said, it is likely that we will see more of this in years to come so it makes sense to do a quick survey as to where things are at in regard to prone ventilation in the field.

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

We mentioned before in our section on **Lung Injury** that recruitment of alveoli and maintenance of this recruitment is very important. While it may be tempting to simply flip a pronated patient over for transport and then let the receiving facility re-pronate them, this could potentially set progress back quite a bit, so we want to do what we can to keep our actions in line with overall clinical course. That said, many treatment guidelines or algorithms for this sort of thing include cyclical proning on some sort of schedule – it may be worth timing these transfers in line with transport capabilities (i.e. with no capacity to transport a prone patient, simply wait until it's supine time and make it happen then).[282]

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

281 Bloomfield & friends, 2015 – Refer here for insight on research that has been done to date and recommendations for what ought to be investigated moving forward

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

PC – pressure control; **PCO_2** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO_2** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO_2** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO_2** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/ perfusion; **VTe** – exhaled tidal volume

When it comes to the physical process of flipping someone over, there are a number of techniques and tools than run the gamut from a RotoProne bed[283] to simply using a flat sheet or slider.[284] Proning can also be performed at the time of transfer from one bed or stretcher to another (for example, let's say we are going from a hospital that doesn't do this to one that does – we could facilitate this at either end of the transfer).[285] This means that even if we don't transport a patient in a prone position in our vehicle, we may still get caught up in the process at some point.

A few considerations about transporting a pronated patient: access to the airway may be difficult or impossible, access to the anterior chest wall (for EKGs, assessment of heart and lung sounds, needle thoracostomy, etc.) will be limited, and stretcher or sled configuration may dictate that the patient be horizontal. For all of these reasons (and probably a great many others), it may be quite some time (~~or eternity…~~) until certain programs and crews decide to attempt this, but rest assured that it has been done already and will likely become more common in years to come.[286]

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

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

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

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

ΔP – driving pressure; **AC** – assist control; **ALI** – acute lung injury; **ARDS** – acute respiratory distress syndrome; **cmH$_2$O** – centimeters of water; **EKG** – electrocardiogram; **EMS** – emergency medical services; **EtCO$_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **FiO$_2$** – fraction of inspired oxygen; **HME** – heat & moisture exchanger; **I:E** – inspiratory to expiratory; **I-time** – inspiratory time; **LPM** – liters per minute; **ml** – milliliter; **MV** – minute volume; **O$_2$** – oxygen; **OK** – alright

Overbreathing

Just to close the loop on one idea that we mentioned back in the section on **Respiratory Rate**, let's consider what to do if we think the patient is breathing too fast and/ or in a way that goes contrary to how we want to manage **Ventilation**. Say we have a patient breathing faster than we'd like and, as a result, our EtCO$_2$ is low. Normally we'd decrease **RR** to drop **MV** and get that EtCO$_2$ back in range, but now we have to consider these patient-triggered breaths and the fact that decreasing the parameter on the vent won't decrease the overall rate. First thing to think about is that an increased rate of breathing is the body's normal response to lots of things: decreased pH or increased PCO$_2$ (as we discussed in **Acidosis**), pain, fluid in the lungs, irritants in the airways, anemia, cardiac ischemia, etc.[287] So before we label this overbreathing as an anomaly and decide that something needs to be done, consider that it may actually be an appropriate response.

On a tangent to this idea, if the concern is MV in a general sense and not necessarily RR, we can simply decrease TV or consider switching from **AC** to **SIMV** to try and bring MV down. It's important to remember that MV is the product of both RR an TV. Even though we routinely modify RR to address an excessive MV, decreasing TV is also an appropriate strategy. But if we don't have an identifiable cause and RR is the thing we want to fix, the next considerations to address too many patient-triggered breaths are **Comfort** and **Triggers**. We talked already about both comfort and triggers, so we won't get into too much detail here. The general idea is that if patient-triggered breaths are the issue (i.e. too many of them happening), we can fix that by either reducing discomfort, manipulating the trigger threshold itself, and/ or avoiding accidental triggers.

Now if we've ruled out all of these situations and the patient is still breathing too fast and we've already considered both comfort and triggers, what else is there to do? First thing is to verify that the increased RR (the cumulative total of our set RR plus patient-triggered breaths) is, in fact, a problem. It may be the case that the patient is breathing fast, but it may be just fine. Reassess the patient with a focus on the **Three Big Things** and then decide if it's still a problem that needs to be addressed. If so, we have a few options: PEEP, increasing TV, and paralytics.

Adding PEEP can be more comfortable for a patient (which may lead to fewer triggered breaths) and will also reduce MV while keeping the lungs open and participatory in ventilation. Just recognize that in VC ventilation we will need to reevaluate pressures to ensure that the alveoli remain safe (i.e. that our Pplat is still acceptable). Moving forward, there is also the idea that increasing TV can lead to less dyspnea or air hunger due to an effect on chemoreceptors in the lungs.[288] And then if we increase TV and exceed the MV needs of the patient, they will respond will a slower rate of breathing. While this is a bit counterintuitive, it could be worth trialing. And at the extreme end of things we could administer paralytics, but we prefer to reserve that strategy for life-threatening situations.

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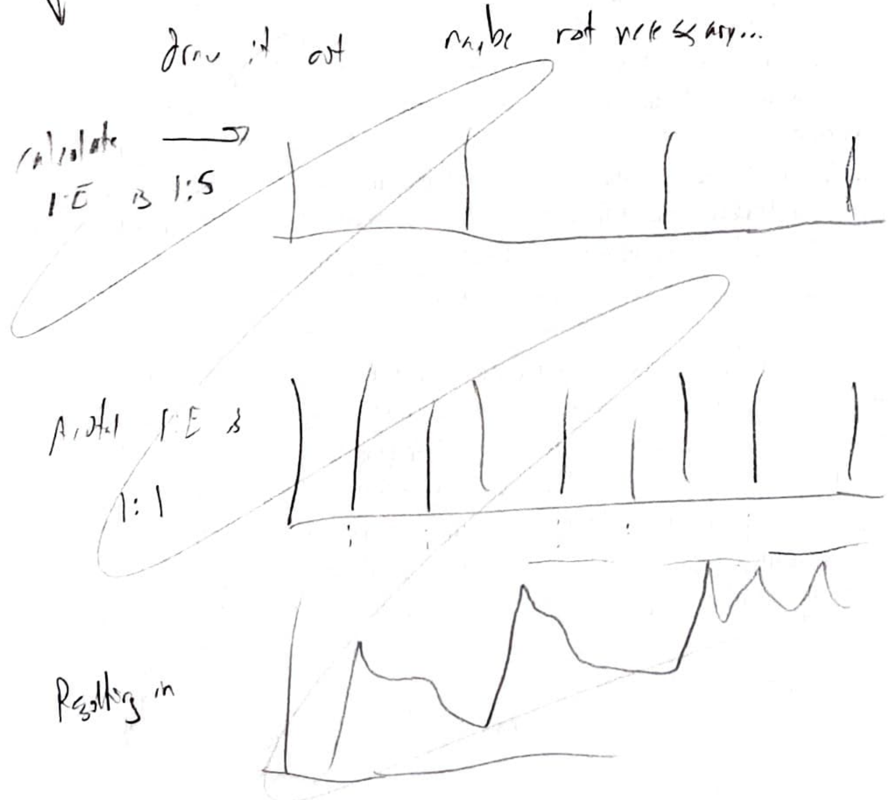
[287] Murphy, 2017a; Alexander, 2016 – The first is one video of three part series on control of breathing, feel free to check out the others for more; the second is an article about tachypnea in general, provides some insight on overlooked causes of tachypnea

[288] Banzett & friends, 2013 – For a very detailed discussed of this idea, take a look at this chapter in Tobin's textbook on vent management

PC – pressure control; **PCO_2** – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **pH** – power of hydrogen; **PIP** – peak inspiratory pressure; **PO_2** – partial pressure of oxygen; **Pplat** – plateau pressure; **RR** – respiratory rate; **SaO_2** – saturation of arterial oxygen; **SIMV** – synchronized intermittent mandatory ventilation; **SpO_2** – pulse oximetry; **TV** – tidal volume; **VC** – volume control; **V/Q** – ventilation/ perfusion; **VTe** – exhaled tidal volume

As a related example, let's say we have a ventilated patient who suddenly experiences acute bronchospasm or has an anaphylactic reaction. Per our **Obstruction** strategy we would like to lengthen our I:E ratio by decreasing both **I-time** and **RR**. So we do that on the vent, but the patient is still triggering breaths for an actual or calculated I:E ratio higher than we'd like and with the result that **AutoPEEP** starts to develop. At this point the rate dial on our machine is not the primary way we control the overall rate and we need to think about other ways to bring the patient's intrinsic rate down to achieve our goals. Similar to what we said above, strategies here would include further decreasing I-time, addressing comfort, making sure triggers are appropriate, and adding PEEP.

So while we've discussed adjusting RR up to this point as if it were a parameter we have complete control over via the machine interface, recognize that the number of breaths per minute can exceed the value we have set. Patient-triggered breaths are generally of benefit to the patient, but if they threaten overall wellbeing or safety then we ought to take action to get things back on track.[289] Steps to fix this problem include addressing comfort, evaluating triggers, adding PEEP, trailing an increased TV, and paralysis.



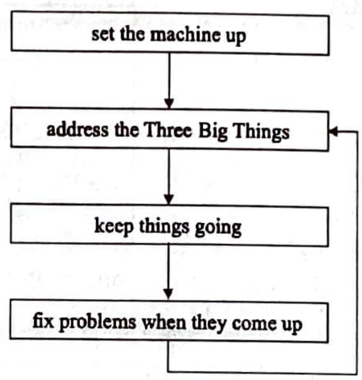
289 This idea was discussed back in the section on **Comfort**

and since this section is a cheat-sheet of sorts that has almost all of the abbreviations, we'll skip the legend here and direct readers back to the rest of the text

A Proposed Protocol/ Flowchart

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

The basic idea of the flow is something like this:



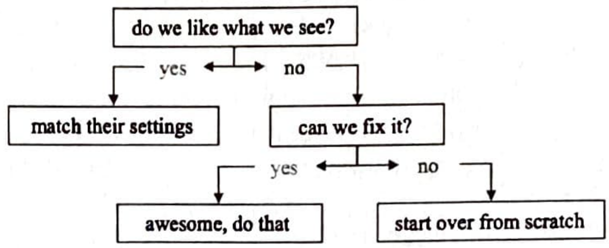
How to do Vent Stuff

I. Set the Machine Up

1. prep
   a. get a report from sending
   b. do some arithmetic: IBW, TV, MV
   c. assess the patient
   d. consider a strategy
   e. check circuit, attach $EtCO_2$ and filter; consider need for HME and/ or suction

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

2. determine settings
   a. patient already on vent (see algorithm, right)
   b. from scratch
      i. turn on machine and leave at default mode and control
      ii. dial in desired TV for 6ml/kg (or PC at 10-15cmH2O)
      iii. adjust RR
         1. adults: to MV goal
         2. kids: using a reference range
      iv. adjust I-time using a reference range
      v. leave all other settings at machine defaults unless one of these considerations applies:



age description	age (years)	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 kids	8 – 9	17 – 25	0.6 – 1.2
preadolescent	10 – 12	14 – 23	0.7 – 1.4
adolescent	12 – 15	12 – 20	0.8 – 1.7
adult	16 and up	12 – 20	0.8 – 1.7

strategy	things to do
obstruction	lengthen I:E (≥1:5) by decreasing RR (and maybe I-time also), then titrate TV (or PC) up to maintain MV as able; consider less PEEP
hypotension	limit PEEP; increase TV and then decrease RR to maintain MV
acidosis	use high end of TV ~~(goal): 8ml/kg IBW~~; increase RR: pre-intubation rate, to get prior/goal $EtCO_2$, or double normal value
lung injury	higher PEEP

3. initiate ventilation (clamp tube if concerned with de-recruitment)

II. Address the Three Big Things

parameter	assessment	normal	actions
oxygenation	SpO_2 ~~($\&$ PaO_2)~~	93-99%	*Low:* consider position & suction, increase FiO_2, increase PEEP, increase I-time, consider pathophysiology/ medications *High:* decrease FiO_2 unless contraindicated (i.e. pregnancy, anemia, shock, etc.)
ventilation	$EtCO_2$	35-45mmHg (low end for TBI)	*Any abnormal value:* consider etiology &/ or patient compensation for acid-base imbalance (may be ~~best left alone~~) *Low:* consider perfusion status, decrease RR (monitor MV), then consider decrease in TV *High:* increase TV (~~max 10ml/kg,~~ monitor Pplat), then consider increase in RR; consider permissive hypercapnia
	MV	≈ 100ml/kg/min (2x with acidosis)	*Low:* increase TV and/ or RR; consider permissive hypercapnia *High:* consider patient comfort, decrease TV and/ or RR, consider SIMV
comfort	RASS, ANVPS	at provider discretion	analgesia & sedation, consider settings (MV and I-time), also consider accidental triggering

III. Keep Things Going

1. set (and troubleshoot) all alarms
2. consider pressures (every time vitals get reassessed)

parameter	normal	actions
PIP	<35cmH$_2$O	consider potential causes (**lung** and **airway issues**), check Pplat decrease TV (or PC)
Pplat	<30cmH$_2$O	consider potential causes (**lung issues**) decrease TV (or PC)
AutoPEEP	none	lengthen I:E (lower RR, shorter I-time) consider inadvertent triggering, trial VC if in PC, avoid high PEEP disconnect circuit to allow exhalation
ΔP	<15cmH$_2$O	decrease TV or PC consider more PEEP and permissive hypercapnia consider recruitment maneuvers
P_{aw}	not applicable	monitor for trends and investigate further

3. make adjustments moving forward

strategy	things to do
general stuff	if oxygenation is all good, go down on FiO$_2$ (maybe all the way to 0.40) and reevaluate consider increasing TV to safe Pplat and acceptable ΔP
obstruction	use drugs (in-line neb treatment, consider Ketamine for analgesia/ sedation, etc.) ensure no AutoPEEP develops if hypercapnia develops and/ or no AutoPEEP noted, consider moving towards normal I:E
hypotension	use caution with PEEP to improve oxygenation consider fluid and/ or pressors if perfusion improves, consider working towards normal settings to avoid higher Pplat and ΔP
acidosis	maintain increased MV goal (minimum 200ml/kg/min) also consider Winter's Formula to guide treatment
lung injury	consider titrating TV down to 5ml/kg, then 4ml/kg to maintain ΔP <15cmH$_2$O increase PEEP to maximize oxygenation, consider stepwise approach consider recruitment maneuver if hypoxia persists

IV. Fix Problems When They Come Up

acute deterioration: what to do!?

↓ start with the basics: ABCs
↓ use alarms to guide treatment
↓ consider the Three Big Things
↓ address airway pressures

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

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

Suggestions for Further Study

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

audio/ podcast

EmCrit Dominating the Vent Series
Part 1, Part 2

FlightbridgeED Vent Series
Part 1, Part 2, Part 3

video, vent specific

Strong Medicine Series on Mechanical Ventilation

Hospitalista Series on Mechanical Ventilation

video, physiology

Ninja Nerd Science, section on Respiratory

Khan Academy, section on Advanced Respiratory System Physiology

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text, web-based

Deranged Physiology, section on Respiratory

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

text, free eBook
Principles and Practices of Mechanical Ventilation
by Martin J. Tobin (3rd edition)

text, book to buy

Ventilator Management: A Pre-Hospital Perspective by Eric Bauer

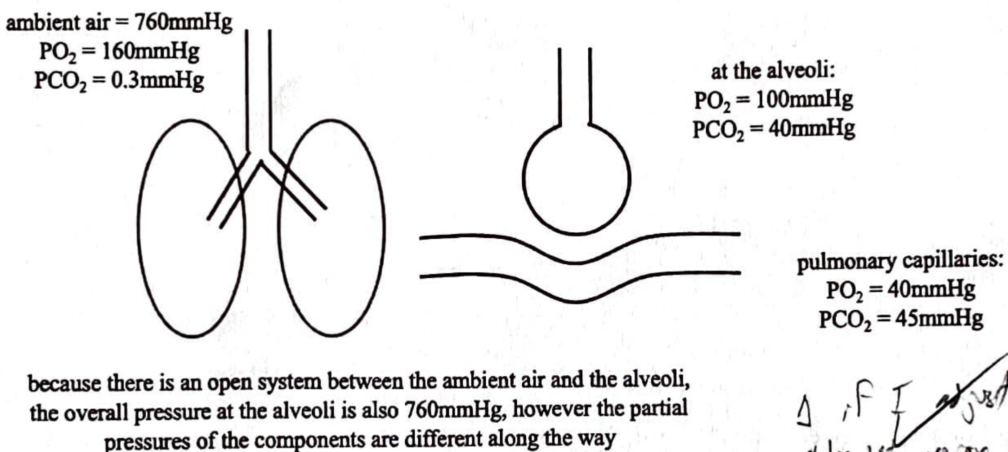
Vent Hero: Advanced Transport Ventilator Management by Charles Swearingen

PCO_2 – partial pressure of carbon dioxide; **PEEP** – positive end-expiratory pressure; **PO_2** – partial pressure of oxygen; **P_{water}** – partial pressure of water vapor at the alveoli and at sea level; **RespQ** – respiratory quotient

Appendix

Alveolar Gas Equation

The alveolar gas equation allows us to calculate the partial pressure of O_2 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:



The equation goes like this:[290]

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

PAO_2 is partial pressure of alveolar O_2
FiO_2 is fraction of inspired oxygen, 0.21 for ambient air
P_{atm} is atmospheric pressure
P_{water} is partial pressure of water vapor at the alveoli, 47mmHg at sea level
$PaCO_2$ 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[291]

given that RespQ = 0.8, we sometimes see the equation simplified as so:

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

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

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

290 Yartsev, 2019 – He's got a good graphic that shows the alveolar gas equation with all parts labeled, maybe makes a bit more sense to the visual learners than how it is represented here
291 Patel & Bhardwaj, 2018 – These guys describe the details behind this respiratory quotient idea; maybe not relevant to our discussion of vent stuff, but good nerdy details for those who want more (another option would be to find an exercise physiology textbook, likely to be some good stuff there)

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cmH_2O – centimeters of water; **FiO_2** – fraction of inspired oxygen; **mmHg** – millimeters of mercury; **O_2** – oxygen; **PAO_2** – partial pressure of alveolar oxygen; **PaO_2** – partial pressure of arterial oxygen; **P_{atm}** – atmospheric pressure

but back to our original equation:

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

other iterations of the alveolar gas equation that we demonstrated in the manual are shown here:[292]

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 (room air)[293]
$$PAO_2 = FiO_2(760\ (+4) - 47) - 50$$
$$PAO_2 \approx 101mmHg$$

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

So we can use the alveolar gas equation to solve algebra problems in an effort to show how things like **FiO_2** and **PEEP** affect PAO_2. And then if we know how much O_2 should be getting to the alveoli and can measure how much O_2 made it into the arteries (PaO_2 from a blood gas), then maybe we can understand something about the efficacy of that exchange. To say it another way, the idea is that we can use values for PAO_2 and PaO_2 to inform us on what is going on with a patient in reference to the movement of O_2 from the input of our vent system into the bloodstream. Values like A-a Gradient and a/A Ratio attempt to do just that. Now there are some limitations to both of these values and their application may be limited in the transport setting, so we won't get into the details here.[294]

292 And this was back in the section on **Oxygenation**

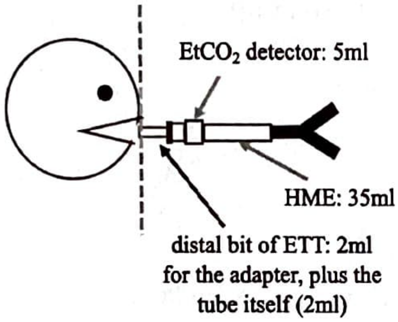
293 Just a friendly reminder that 5cmH_2O is roughly 4mmHg, see chart in **Measuring Pressures**

294 Strong, 2014; Yartsev, 2019 – The first is a video that explains A-a gradient and reviews a number of the concepts we discussed previously; the second is an articles that discusses these types of measurements and identifies issues with their application to clinical practice

%TaDP – percentage of time at decreased preload; **ΔP** – driving pressure; **ARDS** – acute respiratory distress syndrome; **CO_2** – carbon dioxide; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **$FECO_2$** – fraction of exhaled carbon dioxide; **HME** – heat & moisture exchanger; **IBW** – ideal body weight; **kg** – kilogram; **L** – liter; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume

Mechanical Dead Space

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



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

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

Let's say we want to figure out to what effect 50ml of added **Dead Space** impacts **Ventilation** in our patients. Now this gets a little weird and the math takes a few leaps of faith along the way, but let's follow along and then compare what we come up with to data after the fact. Also, note that we are going to introduce a few new ideas here and that we will get more into those in the very next section:[295]

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

now we already said a few things about this:
alveolar TV = TV – anatomic dead space
and this dead space is approximately 1/3 of TV
so alveolar TV = 260ml
VA = RR x alveolar TV
in this case VA = 4420ml/min

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

[295] And to review these ideas: **Ideal Body Weight**, **Tidal Volume**, **Respiratory Rate**, and **Minute Volume**

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OK – alright; **P_{atm}** – atmospheric pressure; **PC** – pressure control; **$PECO_2$** – mean partial pressure of exhaled carbon dioxide; **pH** – power of hydrogen; **Pplat** – plateau pressure; **P_{water}** – partial pressure of water vapor at the alveoli and at sea level; **RR** – respiratory rate; **TV** – tidal volume; **VA** – alveolar minute volume; **VC** – volume control; **V_D** – dead space; **VTe** – exhaled tidal volume

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

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

Now there are two versions of this formula that use $PACO_2$ and $PaCO_2$ rather than $EtCO_2$, but it has been proposed that this representation might be of value in calculating dead space in practice.[296] So simply for the sake of this example, we will go with that. Now that $PECO_2$ value is something we haven't discussed yet – it is the mean partial pressure of CO_2 during exhalation. A normal value is around 30mmHg and it could also be calculated based on the idea that a normal fraction of expired CO_2 ($FeCO_2$) is about 4%:[297]

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

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

only anatomic dead space:

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

with mechanical dead space added in:

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

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

%TaDP – percentage of time at decreased preload; **ΔP** – driving pressure; **ARDS** – acute respiratory distress syndrome; **CO_2** – carbon dioxide; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **$FECO_2$** – fraction of exhaled carbon dioxide; **HME** – heat & moisture exchanger; **IBW** – ideal body weight; **kg** – kilogram; **L** – liter; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume

Now a difference in $EtCO_2$ of 10mmHg doesn't necessarily mean that a corresponding quantity of CO_2 remains in the blood and impacts the body. The purpose of this exercise was simply to show that the potential exists for a buildup of CO_2 in the alveoli. In the transport setting where $EtCO_2$ monitoring is routinely used to assess ventilation, we would simply increase MV to bring that second value into a normal range. But let's suspend that idea for just a moment longer and consider what impact this might have if we failed to do that. Researchers looked at this very problem and determined that removing 115ml of dead space from a circuit resulted in a decrease in $PaCO_2$ of 11mmHg and an increase of pH from 7.30 to 7.38.[298] Furthermore, they were able to do that with less MV. Now this was in patients with ARDS in which one of our concerns is the amount of air needed to maintain ventilation and consequences of that air on the patient's pulmonary system, but the findings are pretty significant.

Back to our discussion and application to the transport setting: we said just a moment ago that we could potentially avoid this increased CO_2 retention by monitoring $EtCO_2$ and increasing MV to accommodate, but the truth is that doing so isn't always a benign thing. Going up on TV or pressure control (when in PC) will increase pressure (**Pplat** and **ΔP**), while going up on RR has the potential to cause discomfort and increase that %TaDP concept.[299] If we can promote CO_2 removal while simultaneously avoiding all of those things, this seems like a pretty good reason to be conscious of adding unnecessary things into the vent circuit whenever possible.

One last thing about all of this with regards to pediatrics and VC ventilation. We mentioned way back when that it's OK if our calculated MV is larger than our goal MV because of some complications posed by dead space.[300] We want to revisit that to show why that is and how we can mitigate it all. The example was a 4-year-old kid of 18kg:

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

$$MV\ goal = 100\text{ml/kg IBW/min}$$
$$MV\ goal = 1800\text{ml/min}$$
$$MV\ goal = 1.8\text{L/min}$$

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

298 Hinkson & friends, 2006 – Small sample size, but significant findings that support the idea of limiting mechanical dead space
299 Also refer back to **Comfort** and **Hypotension**
300 In the section **A General Vent Strategy**

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OK – alright; P_{atm} – atmospheric pressure; **PC** – pressure control; **PECO₂** – mean partial pressure of exhaled carbon dioxide; **pH** – power of hydrogen; **Pplat** – plateau pressure; P_{water} – partial pressure of water vapor at the alveoli and at sea level; **RR** – respiratory rate; **TV** – tidal volume; **VA** – alveolar minute volume; **VC** – volume control; V_D – dead space; **VTe** – exhaled tidal volume

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

we'll go with a TV of 6ml/kg (108ml) and a RR of 24
MV calculated = 2592ml/min
VA = RR x (TV – dead space)

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

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

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

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

Just to be clear, this isn't completely the case. As TV decreases we likely get less anatomic dead space as airway structures don't flex and expand as they normally would with the delivery of normal-sized breaths. We mentioned before that we assume dead space is constant when going up on TV, but there is some variation here and it is most notable the extreme end of low.[301] Knowing to what degree this type of thing happens isn't generally possible in transport, but the takeaway point still stands – be sure to consider these things when ventilating in VC with small volumes.

301 Yartsev, 2019 – And yet again, this resources offers even more insight on this idea

%TaDP – percentage of time at decreased preload; **ΔP** – driving pressure; **ARDS** – acute respiratory distress syndrome; **CO_2** – carbon dioxide; **$EtCO_2$** – end-tidal carbon dioxide; **ETT** – endotracheal tube; **$FECO_2$** – fraction of exhaled carbon dioxide; **HME** – heat & moisture exchanger; **IBW** – ideal body weight; **kg** – kilogram; **L** – liter; **min** – minute; **ml** – milliliter; **mmHg** – millimeters of mercury; **MV** – minute volume

One last thing to consider is the idea that if we are using uncuffed ETTs with our kids, some TV may get lost as air moves back past the tube to the oropharynx.[302] So the moral of the story here is that we should either ventilate these patients in PC (to bypass this vent circuit stretch dead space concept) or start at a higher end of normal TV and be ready to quickly go up on MV as soon as initiating ventilation in VC (based on VTe and $EtCO_2$). As we said before, there is no right or wrong to this, so long as we know the consequences and correct actions associated with whatever choice we make.

[302] Chambers, 2017 – For more information on that, take a look at this paper

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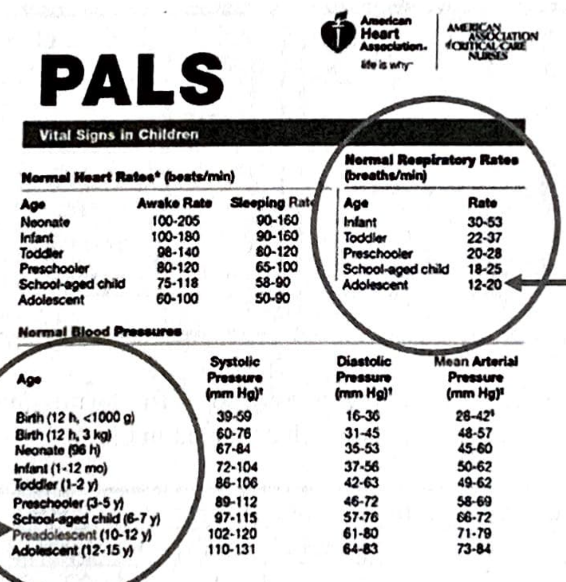
I:E – inspiratory to expiratory; **I-time** – inspiratory time; **PALS** – Pediatric Advanced Life Support; **RR** – respiratory rate; s – second

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
   b. Normal RR range for an adult is 12-20 (cited in many, many sources)
   c. A normal I:E at rest/spontaneous respiration is 1:2, but we often work with a ratio of 1:3 for vented patients[303]

2. Fill the gaps in the PALS Normal Respiratory Rates data set:
   a. What gaps?[304]


PALS

American Heart Association. life is why

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

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

no data for preadolescents

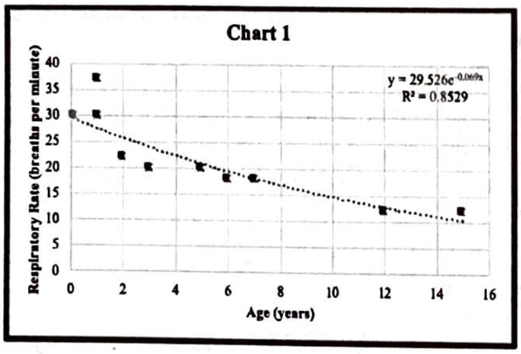
no info for the 8-9 year range

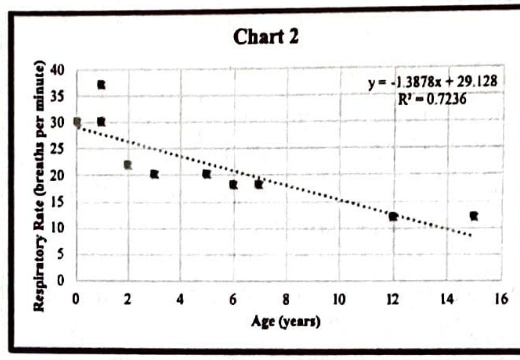
[303] And this may be by convention of leaving I-time set at a given value, not necessarily because that's the thing we ought to be doing; but regardless, we'll get a range of possible values using both 1:2 and 1:3

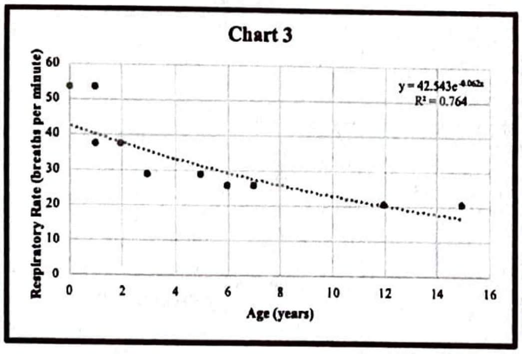
[304] American Heart Association, 2016 (image) – And we said already (section on **Respiratory Rate**) that we chose to use these values not because they are intended for use with vent management, but because they represent normal values by age and are from a reference that most of us are familiar with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

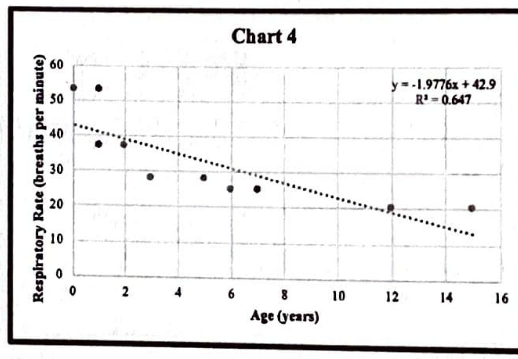
I:E – inspiratory to expiratory; **I-time** – inspiratory time; **PALS** – Pediatric Advanced Life Support; **RR** – respiratory rate; s – second

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









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

age description	age (years)	RR
infant	.083 (1 month) – 1	30 – 53
toddler	1 – 2	22 – 37
preschooler	3 – 5	20 – 28
school-aged child	6 – 7	18 – 25
big kids	8 – 9	17 – 25[305]
preadolescent	10 – 12	14 – 23
adolescent	12 – 15	12 – 20
adult	16 and up	12 – 20

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

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I:E – inspiratory to expiratory; **I-time** – inspiratory time; **PALS** – Pediatric Advanced Life Support; **RR** – respiratory rate; s – second

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 (years)	RR	I-time (s)
infant	.083 (1 month) – 1	30 – 53	~~0.3 – 0.6~~ 0.7
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 kids	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

2x ✓ the math

low end RR w/ 1:2 (÷3) high end w/ 1:3 (÷4)

infant	.67	.28
toddler	.91	.4
preschool	.91	.54
SAC	1.11	.6
big	1.13	.6
pre a	1.43	.65
ad	1.67	.8

PEEP – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **PPV** – positive pressure ventilation; **RR** – respiratory rate; s – second; T_{total} – amount of time per breath; **TV** – tidal volume; **VA** – alveolar minute volume

Hypotension Strategy Math

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

But before we get there, one other thing to mention. PEEP is also a contributing factor to hypotension in the susceptible patient, so we want to keep that to a minimum. While it may seem like a good idea to drop PEEP to zero in the hypotensive patient (especially in light of the P_{aw} calculations we'll show in just a moment), recognize that **Oxygenation** is also super important and PEEP is one of our tools to maintain that. Other specific benefits of PEEP that'd we'd like to maintain in these patients include ease of triggering spontaneous breaths and alveolar recruitment. Last thing: the PPV/ PEEP → decreased preload → decreased CO sequence of events can be mitigated by fluid resuscitation.[306]

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

306 We mentioned this very same idea in **How is Positive Pressure Different?**

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%TaDP – percentage of time at decreased preload; **CO** – cardiac output; **I-time** – inspiratory time; **kg** – kilogram; **ml** – milliliter; **min** – minute; **MV** – minute volume; **OK** – alright; P_{aw} – mean airway pressure

Now for the math, starting with how the lower RR, higher TV strategy decreases dead space. Let's assume another 65kg patient and see how it looks. We've shown the calculations here working from the assumption that anatomic dead space doesn't change with TV, but recognize that this idea is based on a number of factors and may not be the case for all situations:[307]

general strategy (6ml/kg TV)			
TV 390ml		**MV = 6630ml/min**	**MV is basically the same**
- anatomic dead space 130ml	**x 17/min**	**(dead space = 2210ml/min)**	
alveolar TV 260ml		**VA = 4420ml/min**	**dead space is more in the general strategy**
hypotension strategy (10ml/kg TV)			
TV 650ml		**MV = 6500ml/min**	**VA is greater in the hypotension strategy**
- anatomic dead space 130ml	**x 10/min**	**(dead space = 1300ml/min)**	
alveolar TV 520ml		**VA = 5200ml/min**	

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

The next concept to discuss is P_{aw}. The airways and lungs live inside the thoracic cavity, so if we put pressure into the respiratory system then we see changes to pressure in the thoracic cavity. The idea is that P_{aw} directly correlates with a concept called intrathoracic pressure and intrathoracic pressure, in turn, is the thing that causes all those hemodynamic changes associated with PPV.[308] Now it gets exponentially more complex than that, as pressure at specific components within that thoracic cavity, all of which are tied to hemodynamic function, vary significantly (in terms of influence on function, not necessarily quantitatively), but the simple interpretation of the idea is that pressure we put in via the vent can disrupt hemodynamic function and result in less CO.[309] So theoretically, if we limit P_{aw} we can minimize these potential negative consequences.

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

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

[307] Yartsev, 2019 – We mentioned this in **Ventilation** and referenced this same article then; also refer back to **A General Vent Strategy** if need be

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

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

PEEP – positive end-expiratory pressure; **PIP** – peak inspiratory pressure; **PPV** – positive pressure ventilation; **RR** – respiratory rate; s – second; T_{total} – amount of time per breath; **TV** – tidal volume; **VA** – alveolar minute volume

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

see all page!

general strategy

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

hypotensive strategy

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

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

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

general strategy

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

hypotensive strategy

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

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

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%TaDP – percentage of time at decreased preload; CO – cardiac output; I-time – inspiratory time; kg – kilogram; ml – milliliter; min – minute; MV – minute volume; OK – alright; P_{aw} – mean airway pressure

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

We didn't consider a Δ in RR w/ general → ↓BP "

So I did w/ a RR of 17 vs. 10, r… to also include 13

general 13–17
↓BP 10

General (1) @ 13, (2) @ 17; ↓BP (3) @ 10

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

(1) 1.0 s / (60 ÷ 13)
4.62
0.22 × 0.5 = 0.11

(3) 1.0 (60 ÷ 10) ✓
6
.17 × 0.5 0.085

(2) 1.0 / (60 ÷ 17)
3.53 ✓
0.28 × 0.5 = 0.14

A Personal Reflection

When I started putting this all together I thought I knew a fair amount about vents. At least I thought I knew enough to effectively manage patients in transport and that my comprehension of it all was adequate to simplify it for others. Turns out I still had (and have!) a long way to go. In spite of this realization, the process of putting in all down in words and images has helped me learn way more than I thought I would've needed to. And I think this final product will satisfactorily help others achieve a better understanding of vents with the ultimate outcome being improved care for the patients we move around.

Another thing that came up in this process was the awareness of how choice of language can contribute to a project like this. One could say that I have a baseline aversion to formality and convention – my preferred venue for this chat about vents would've been a backyard patio with beer in hand. My initial drafts reflected this a bit more at the potential cost of alienating readers. I've tried to find a balance, so we'll see how that turns out. And mad props to both Ben and Bruce for being frank with me about that.

Carrying on with that idea, Ben made the point that my readers are likely professionals in a niche setting and, because of that, it may help to reference certain concepts that all of ought to keep hidden away in the back of our collective brain. For example, I was reluctant to include references to both gas laws and the oxyhemoglobin dissociation curve, as I didn't want to fall into the trap of putting out content specific for test takers or to be seen as taking away from really good material that's already out there. But the point was not lost on me that there is, in fact, a middle ground, so I've tried to accommodate those ideas.

Last thing I've come to realize is that organizing thoughts coherently is quite a chore. I wanted a sequential progression of concepts from start to finish, but also a format that allows for quick referencing and jumping between sections. Thanks to Dan for pointing out that something as simple as a legend at the top of each page can be a game-changer with this process. And then within that overall framework there were countless explanations that got erased and then rewritten multiple times. Same goes for those graphics I used to try and replace words. Bruce – I appreciate your feedback on that front and am sure that things will be clearer for folks because of it.

The goal from the beginning was that this is to be an ongoing project. I'm sure there are errors and misunderstandings hiding in plain sight, but that's all part of the learning process. If you come across something that needs attention or even if you just want to get involved in the next version, don't hesitate to reach out. There's no reason that this sort of thing should be a one-man project. So let me know what you think, feel free to touch base any time, and check out the website for more.

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