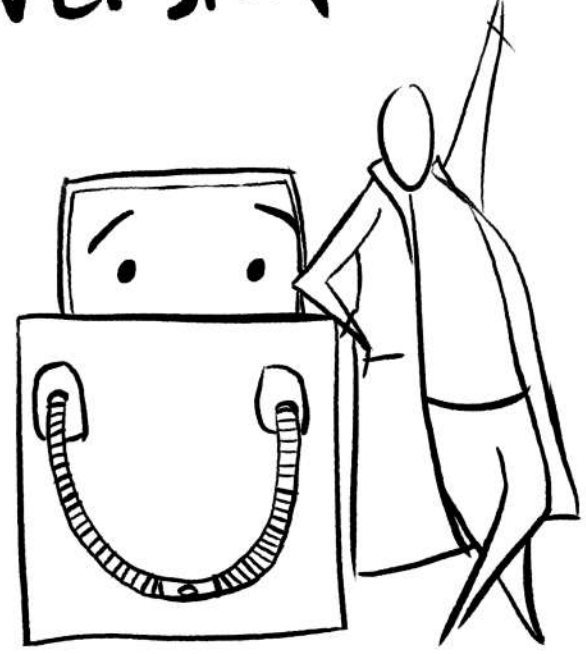
10% Fewer Words

Mechanical Ventilation

For Fun & Benefit

Version 0.8



Khaled Fernainy MD

Mechanical Ventilation

For Fun and Benefit

By Khaled Fernainy, MD

Incomplete - version 0.8

Contents

Dedication

This book is dedicated to the residents and fellows that have struggled in the ICU and to the patients who have struggled with them.

Notice

Legalese I copied from some other book but which seems reasonable

The author of this work has checked with sources believed to be reliable in his effort to provide information that is complete and generally in accord with the standard accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the editors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. For example and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this work is accurate and that changes have not been made in the recommended dose or in the contraindications for administration.

Preface

In 2007 I began a pulmonary and critical care fellowship and I had a secret.

I had just come out of residency and although I had rounded in the intensive care unit and had undergone excellent training I was still uncomfortable with (scared of..) mechanical ventilators.

I knew enough so I could answer simple pimping questions. Hint: The correct answer is always “VQ mismatch”. But I had a pervasive feeling that I had no clear grasp of why fiddling with the different settings helped. I could say “dead space” and “PEEP” but really had no idea how these things were related.

This was no fault of any of the attending or teachers who had done their best to explaining things. It took me my entire fellowship and a few years of being an attending and I slowly became more comfortable with ventilators.

This book is in response to my initial discomfort and I hope it helps residents and fellows get over their ventilatophobia.

Chapter One

Structure and Function

We begin with a section that focuses on structures important to understanding mechanical ventilation. We then review their function and physiology.

In an extremely simplified view, we can think of the respiratory system as being made of just 2 parts. A gas conduction system and a gas exchange system.

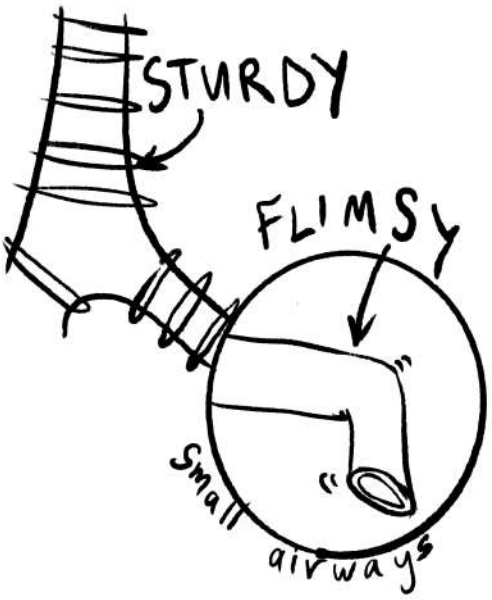
Gas Conduction System

The gas conduction system comprises the hollow airways through which gas moves into and out of the lungs; to and from the gas exchange system. It begins at the vocal cords as the trachea. Like a tree it branches out and divides until it reaches the acini.

The trachea is sturdy, reinforced by cartilage and connective tissues. With each division, however, the airways become narrower, and lose the cartilage and other support elements. After around 16 divisions the airways will have become very numerous, thin, and flimsy.

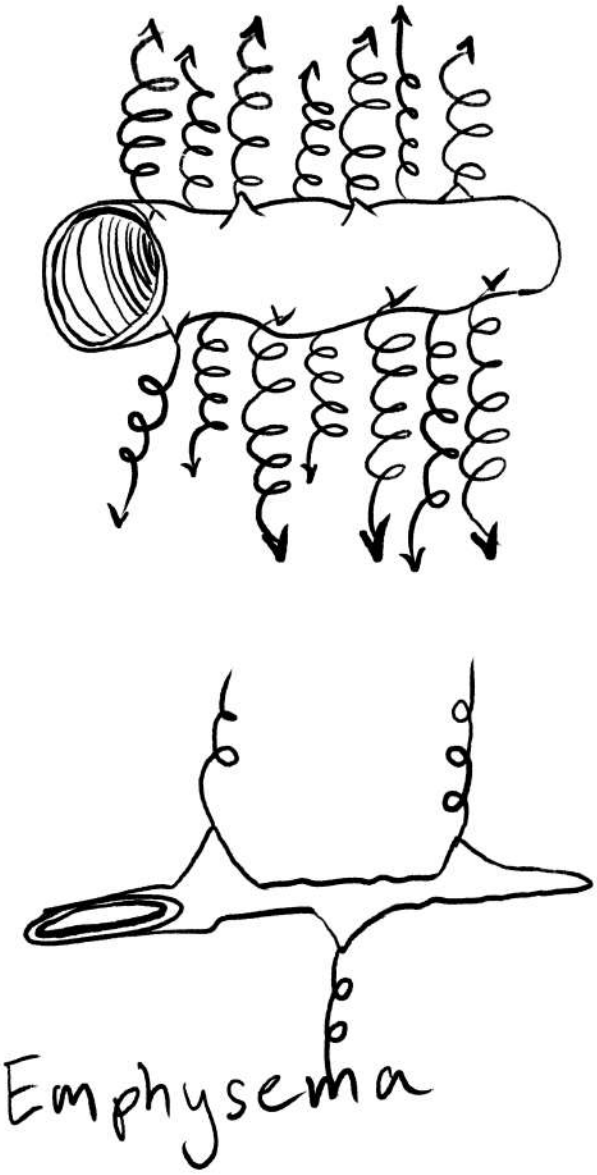
Flimsy

The respiratory system ends with millions of gas exchanging acini. Each acinus has to be reached by the gas conduction system. To fit millions of tiny tubes into the space of a chest it strips these tubes of almost all supportive tissues. This makes them flimsy and prone to collapse. In contrast to the larger airways whose muscular and cartilaginous structure props them open, these smallest airways must be held open by tension from attached surrounding tissues.



The diameter of the small airways depends on how much they're pulled open by the surrounding tissues[1] which depends on how taut the surrounding tissues are.

The tautness of surrounding tissues depends on two things: how expanded the lungs are and the physical properties of these tissues. A full lung will stretch its tissue and pull open the small airways. As the lung shrinks with exhalation the tissues become lax and small airways lose support, shrink and collapse. The tautness also depends on the properties of the surrounding tissues. Loose surrounding tissues (such as in emphysema) will not pull open small airways as effectively as tight tissues.

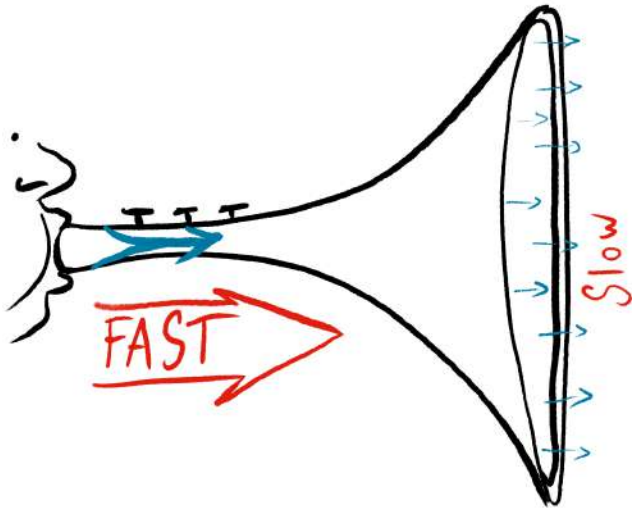


Numerous

As the tracheobronchial tree branches, the number of airways at each level increases. The small airways (…yes that's their name … airways smaller than 2 mm) are extremely numerous.

With the great increase in the number of small airways there is a concurrent increase in their cumulative surface area. The increase in cumulative surface area is like the increase in the diameter of a trumpet as it goes from the mouthpiece to its end.

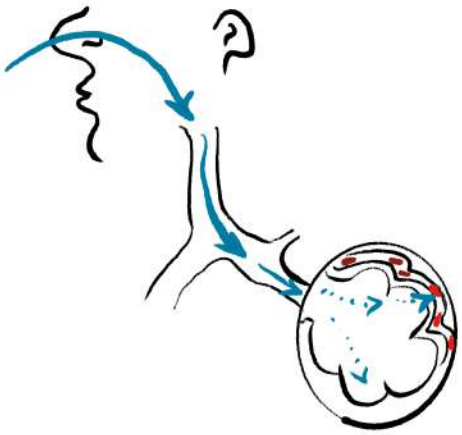
The increased surface area interests us because the air flow through the massive surface area is very slow. The same amount of gas per unit of time traverses a much larger area. It is so slow at the level of the acini that the movement of gas molecules becomes more dependent on diffusion rather than convection. Similarly, the air is fast out of the mouth of a trumpeter into the mouthpiece but slow out the end of the trumpet with its much bigger diameter.



The increased surface area is also important because it explains why these narrow airways do not (normally) pose a great amount of resistance to air flow.

Gas Exchange System

The gas exchange system consists of the acini, alveoli and their surrounding vessels and tissue found at the end of the conduction system.



The lung facilitates gas exchange in an enormous quantity of blood - around 5 Liters per minute and occasionally much more. To allow gas exchange, it brings the blood into proximity with gas.

To closely approximate the large amounts of blood with air, the lung has developed a massive surface area. It packs this massive area into the tight space of the chest by forming the myriad thin bubbles we call alveoli. The alveoli themselves are packed together in clusters termed acini.

Blood percolates around the millions of acini through capillaries which form a similarly massive network.

Gas exchange occurs in the alveoli which are formed of thin, tightly connected epithelial cells, a thin underlying basement membrane, and then the thin endothelial wall. This thin barrier allows blood to get close to gas. The capillaries surrounding the alveoli carry depleted blood from the pulmonary artery and bring it into proximity to air so that gas exchange occurs.

Diffusion drives gas exchange and relies on concentration gradients between blood and the alveolar gas. Oxygen diffuses into the blood from a higher concentration in the alveolar gas. CO2 diffuses out of the blood into the lower concentration of the alveolar gas. It is the maintenance of the gas gradients that allows gas exchange to occur.

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The conduction and gas exchange components work together to perform the functions of the respiratory system. This section discusses the mechanics and physiology of these systems.

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Ventilation

Ventilation replaces exhaust gas with fresh air in the respiratory system. Delivering fresh gases to the gas exchange system allows for the maintenance of concentration gradients and continuing gas exchange. Without a fresh supply of gas to the respiratory system, the concentration difference between the alveoli and the blood would disappear and no further gas exchange would occur.

Mechanics of Breathing

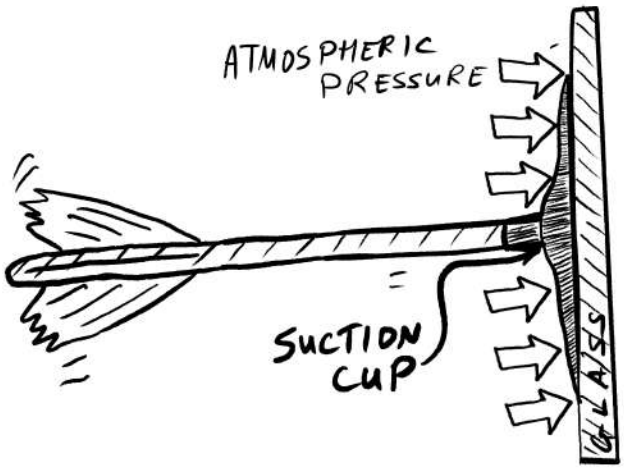
Static Lung Volumes and Pulmonary Mechanics

At rest, the lung and chest wall settle at a particular volume called the functional residual capacity (FRC).

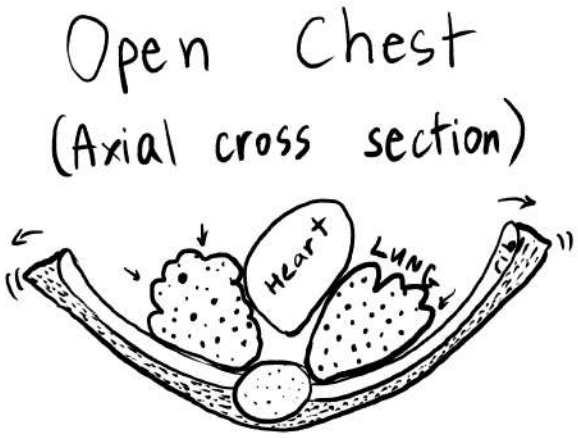
The lung tends to shrink. We can think of it as a rubber balloon that when inflated will always seek to deflate. The lung's tendency to collapse is due to a combination of the surface tension in the alveoli, its micro-anatomy, and elastic components in its connective tissue.

The chest wall tends to expand. We can think of it as a compressed spring pushing to open. Recoil in the chest wall is because of its component ribcage/spine structure and the chest's muscle tone.

The lung and chest wall are tightly connected and expand and deflate together. The connection is due to the vacuum in the chest cavity. The lungs are suctioned up against the chest wall just like a suction cup against a glass window. When the chest wall expands it pulls the lungs open. When the lungs shrink they pull the chest wall.

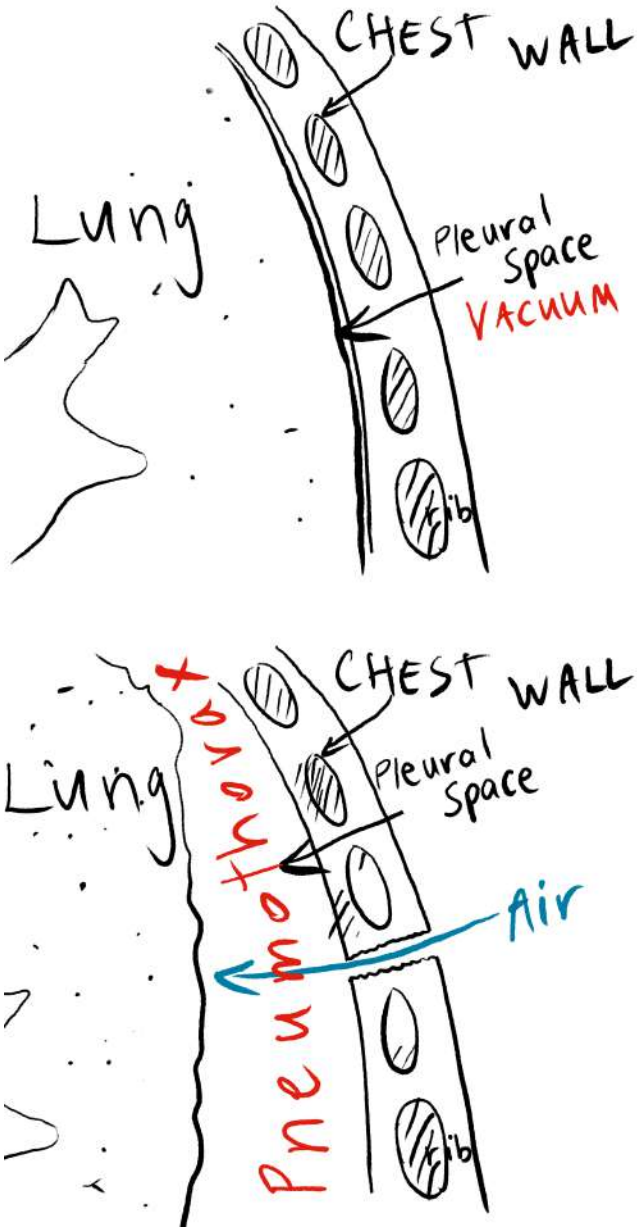


Open a chest by cutting a sternum and the chest wall will spring open as the lungs collapse and shrink. This is because the vacuum, the connection, is lost.



A pneumothorax uncouples the chest wall from the lungs since the vacuum connecting them dissipates. If a hole appears in a person's chest, air will quickly get sucked in as the lung on that side collapses; the chest wall on that side will expand as it recoils

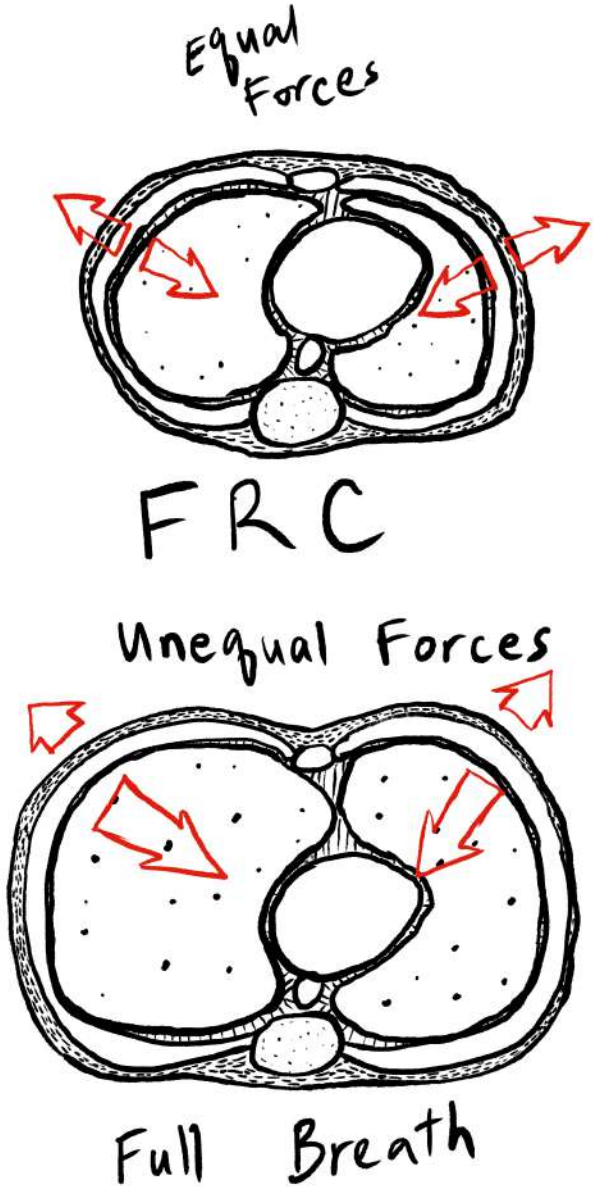
outwards. This leads to the typical unilaterally expanded chest on physical examination and a collapsed lung on x-ray.



Functional Residual Capacity (FRC)

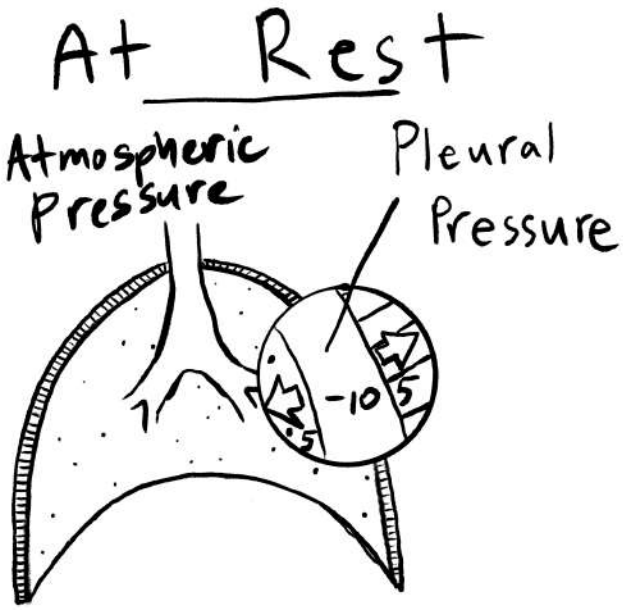
At rest the chest settles at a particular volume. That volume is a balance between the lung's tendency to collapse and the chest wall's tendency to expand and it is called the

functional residual capacity (FRC). It is the volume at which the 2 opposing forces are exactly equal.



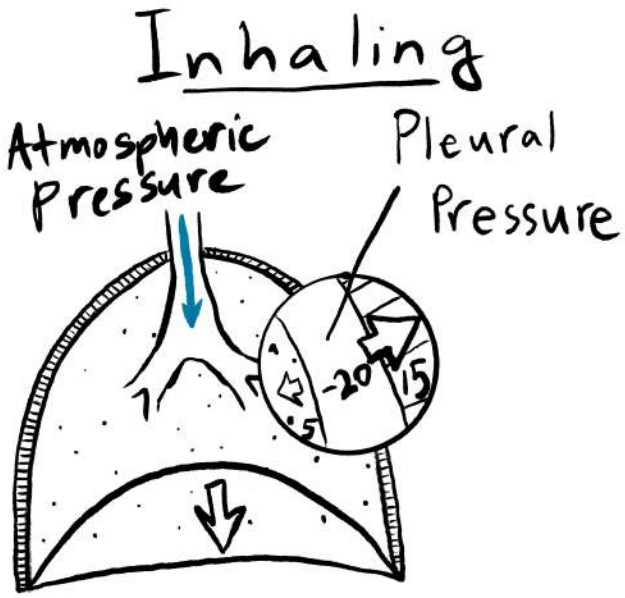
The chest will try to return to FRC when its volume is changed. Similar to stretching a spring, overcoming the equilibrium requires energy. The chest (or spring) stores this potential energy and it provides the force necessary to return the chest to the resting state.

Breathing is the act of moving of gas into and out of the tracheobronchial tree by moving the chest volume away from FRC and then allowing it to go back to FRC.



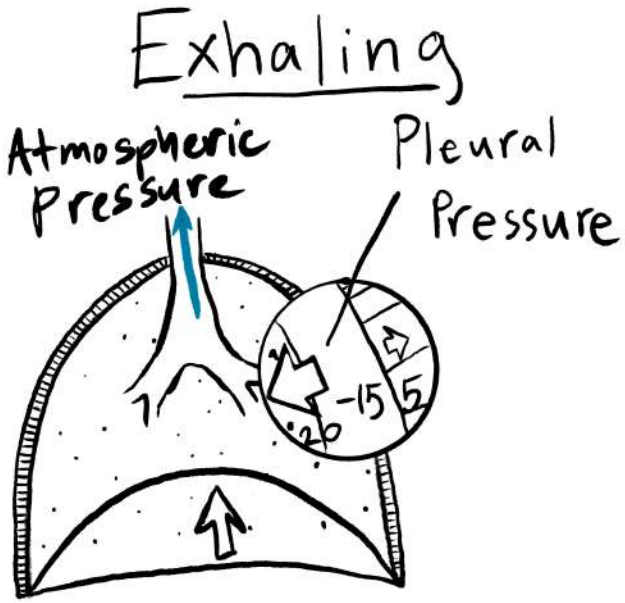
Inhalation

We inhale by generating negative pressure in our chest. We stiffen and expand our chest wall with our chest muscles and concurrently flatten our diaphragm. Air rushes through our tracheobronchial tree to fill the expanded chest. We call this a negative pressure breath because the pressure in the chest, the pleural pressure, at the time of its expansion is less than atmospheric pressure. Air rushes into the chest to equalize the pressures - to return the pressure inside the chest to atmospheric pressure.



Exhalation

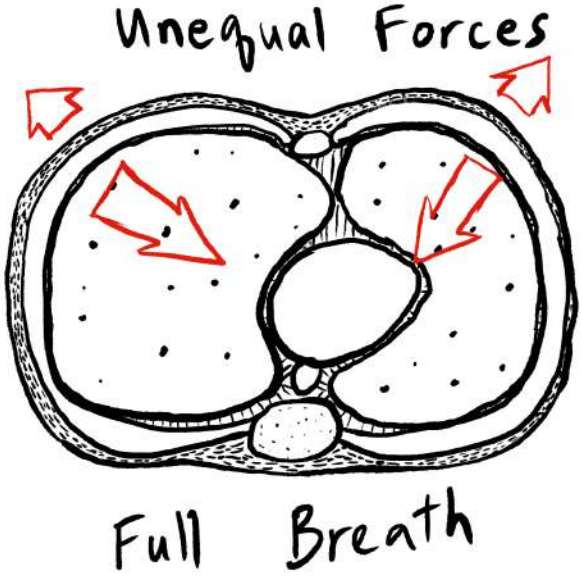
Exhalation is usually a passive process. Once a breath is complete, the diaphragm relaxes and the pull of the shrinking lungs and the pressure from the abdominal cavity push it back up into the chest; the intercostal muscles relax and the chest shrinks. It squeezes out the gas.



There are forces which determine the speed of exhalation. Variations in these forces can prolong exhalation and make mechanical ventilation very difficult. The forces favoring exhalation are the lung's elasticity and the external forces pushing into the chest wall. The forces which oppose exhalation are the resistance to airflow in the airways and the chest wall's outward tension.

Force Favoring Exhalation

Exhalation gets the chest volume back to FRC. The elastic energy stored in the lung when the chest volume is moved away from FRC as well as the pressure built up in the belly behind the diaphragm power passive exhalation.



With a very dyspneic patient there will also be a component of muscular contractions in belly and chest that seek to aid exhalation. We call this active exhalation.

Lung's Elasticity

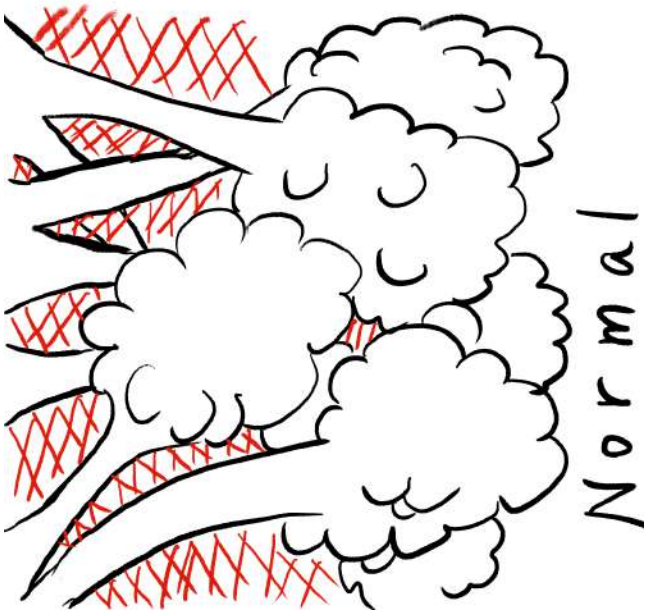
Lung elasticity is due to 2 things - the intrinsic properties of the lung tissues and surface tension in the alveoli.

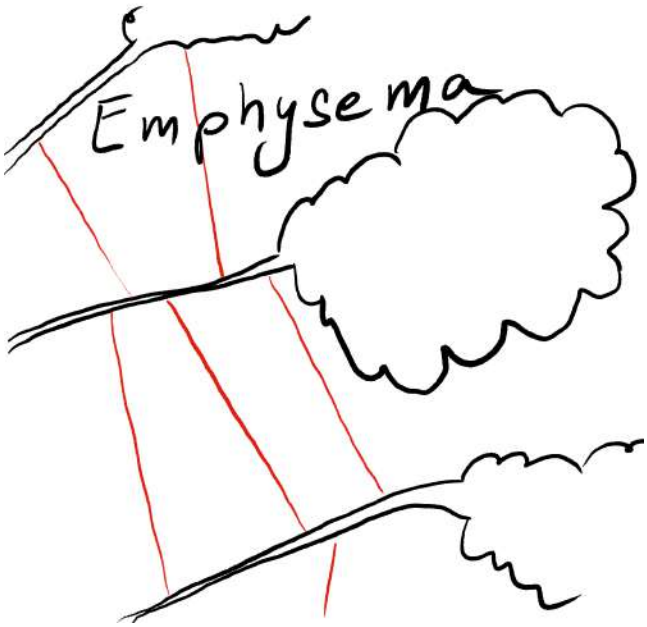
The lung tissue is not just elastic because of the properties of the material it's made from. It is actually mainly elastic because of its microarchitecture. The microarchitecture can be likened to a nylon stocking. A nylon stocking is elastic because of its weave -

where each individual nylon strand is only minimally elastic but combined in the weave become is very elastic.

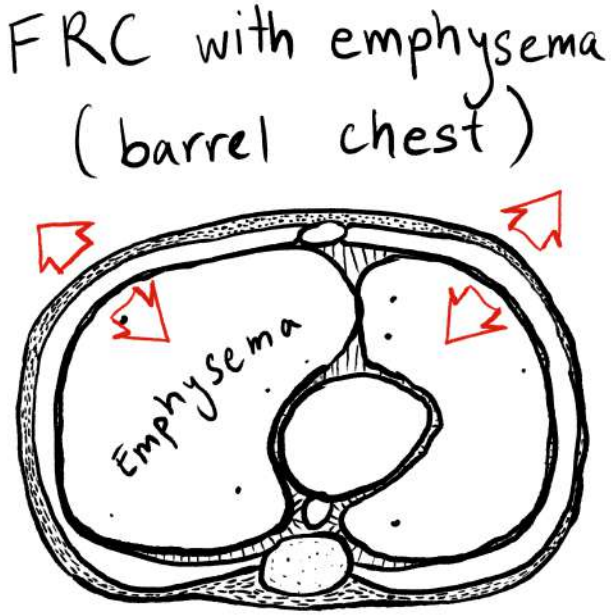
The surface tension is the second very significant component of the lung's elasticity. It comes from the tendency of water bubbles to shrink because the affinity water molecules have for one another. Alveoli are essentially water bubbles and the surface tension of the water is always pulling them to get smaller.

Several things can decrease the elastic recoil of the lung. Emphysema can destroy tissue and change microarchitecture thus decreasing the elasticity of the lungs. This is akin to an old nylon stocking that can become loose due to its weave coming apart and developing large holes.





In patients with advanced emphysema the chest takes on a barrel-like look. This is because of the loss of elastic recoil. The FRC in these patients is at a higher volume than normal because the elastic recoil is weaker and is less able to counteract the chest wall's outward pull. It's only counterbalanced when the chest is more expanded.



Some diseases will increase the elastic recoil. An example is the fibrotic lung of a patient with Idiopathic Pulmonary Fibrosis (IPF) - a disease of progressive scarring in

the lung. Pneumonia and ARDS where the surface tension is much stronger, because of loss of surfactant, also have stiffer lungs.

Extrathoracic Forces: Abdominal Contents and Chest Wall

Other factors can contribute to the force pushing air out of the chest. The diaphragm displaces abdominal contents when the patient takes a breath. These contents weigh on the diaphragm and push it upwards thus contributing to the forces aiding exhalation.

Abdominal pressures can be significant in patients with ascites, abdominal compartment syndrome, or in pregnancy. Non-Abdominal sources of exhalation force can be chest binders, large breasts, tired interns leaning on the patient…

Balancing Forces - the Thoracic Cage

The thoracic cage springs outwards and provides a counterforce to the exhalation. Without this counterforce, an exhalation could completely empty the chest of air. This counterforce strengthens as the chest shrinks until it balances the forces pushing the air out at FRC.

Resistance to Airflow

Resistance in the airways will slow the flow of gas leaving the chest.

Airway Diameter

Airway diameter is the most important factor in determining resistance; resistance increases with the inverse of the fourth power of the radius. Most airflow resistance normally comes from the larger airways (down to division 7). If an endotracheal tube is present then it becomes a point of resistance since it is necessarily smaller than the trachea in which it is lodged. A smaller diameter ETT provides more resistance than a larger diameter ETT.

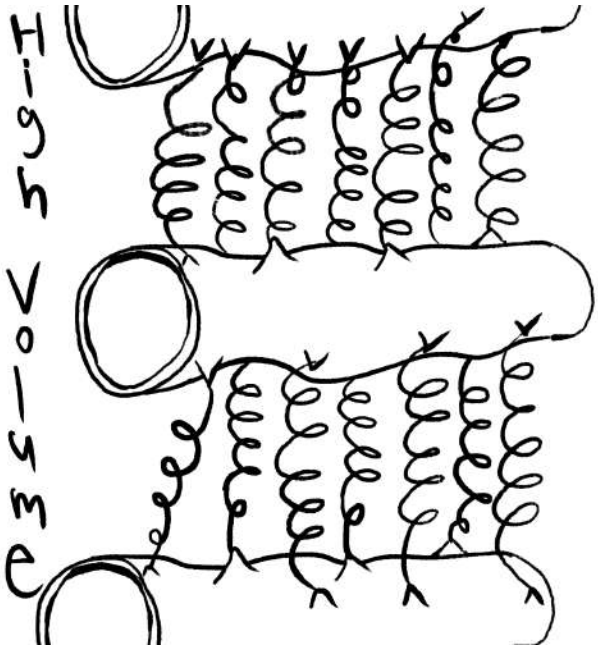
Tracheal strictures, tumors, and foreign bodies can cause narrowing obstructions by posing as physical barriers to flow.

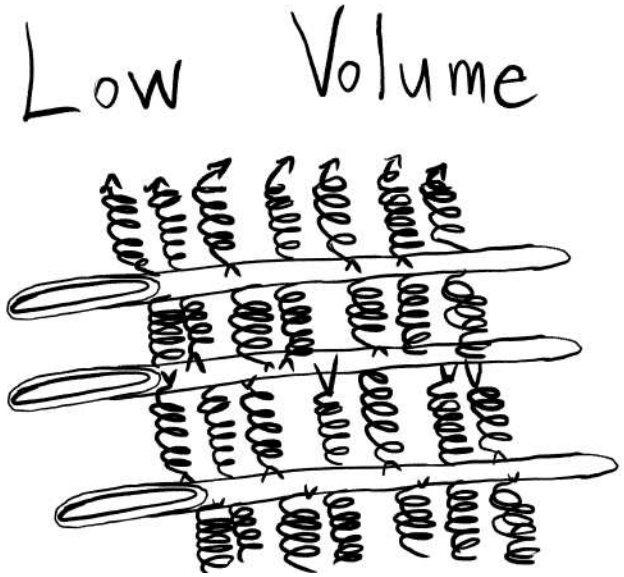
The cumulative cross-sectional surface area of the small airways (around the 16th division) is so large that they normally only contribute a minimal amount to the resistance of air flow. Due their sheer number, however, if the bronchioles are inflamed or narrow they can quickly become a significant source of resistance. This is especially true during exhalation as lung volumes decrease and small airway diameter shrinks with loss of support.

Lung Volume and Phase of Respiration Determines the Resistance to Flow

Diameter of the Small Airways Depends on Lung Volume

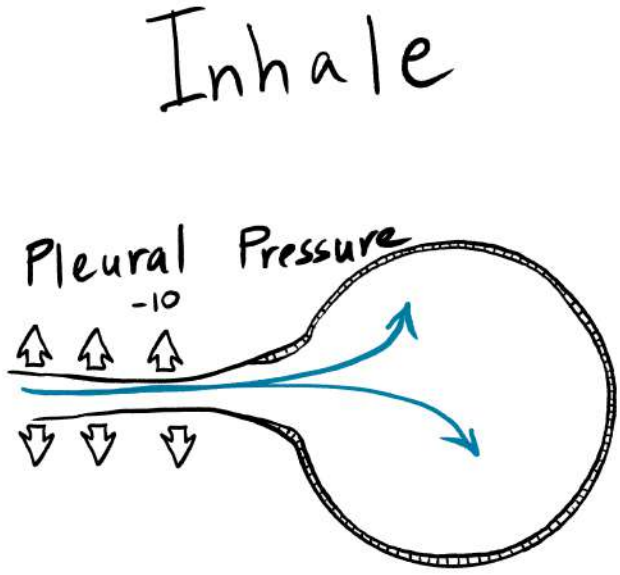
The small airways have no supportive structures. In order to stay open they're pulled open by surrounding tissues. How much they're pulled depends on how taut and stretched out the surrounding tissues are. How stretched out they are depends on the lung's volume - how full it is.

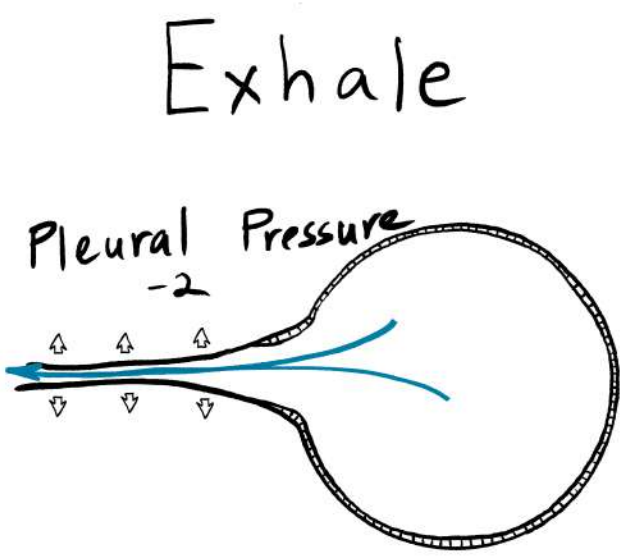




Resistance is Different During Inhalation and Exhalation

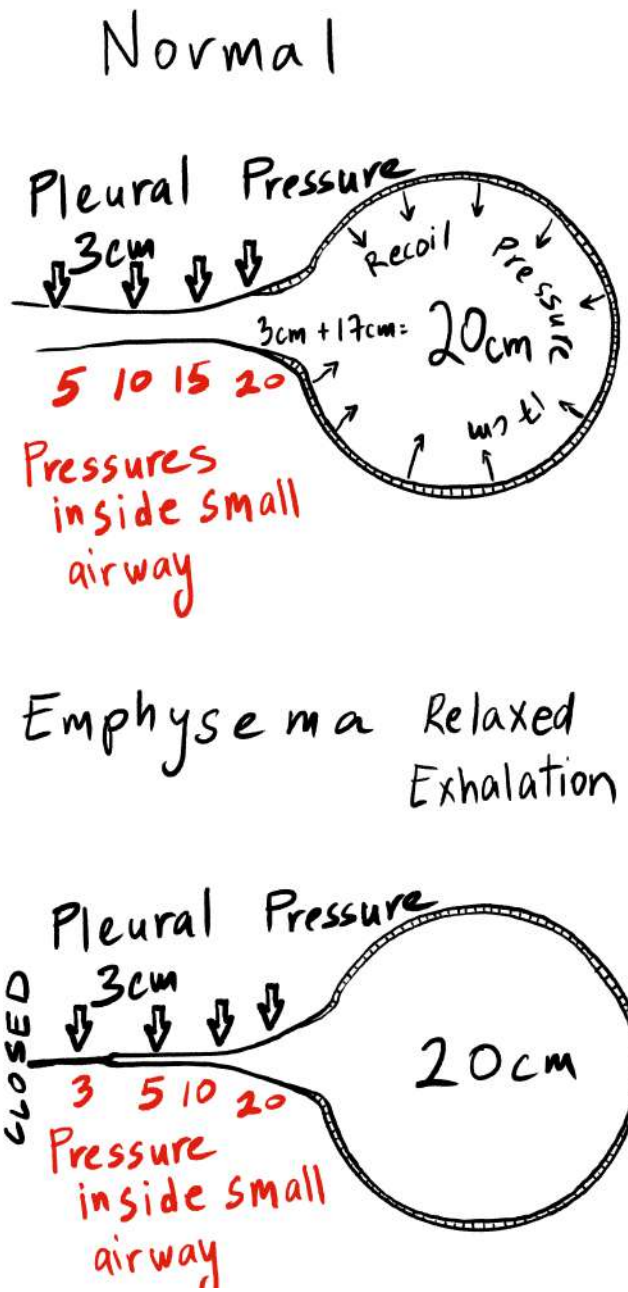
During normal inhalation the pleural pressures are minimal or negative (this is during normal negative pressure breathing). The negative pleural pressures pull open the small airways. During exhalation, however, the pleural pressures become less negative and can be positive in some regions - it pulls the airways open less and may even squeeze them.





The squeezed airways will collapse if the pleural pressures are high enough or if their support is very poor such as in an emphysematous patient.

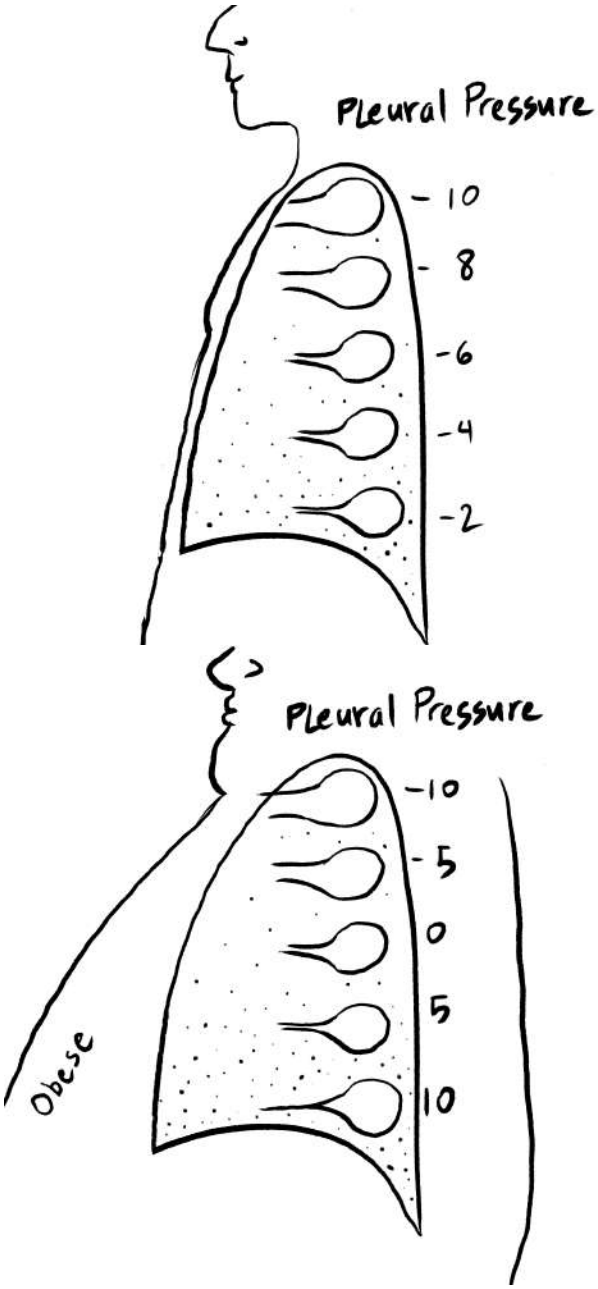
During exhalation the alveolar pressures are higher than atmospheric and the gas leaves the alveoli. Its pressure props open the small airways as it traverses them. As it travels through the small airways the pressure diminishes and at some point can reach the same level as the pleural pressure. This “closing pressure” is determined by how much pleural pressure there is and how narrow the airways are.

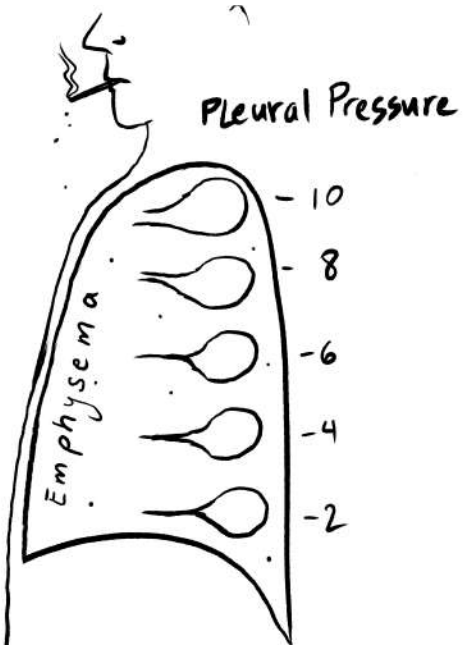


Pleural Pressure is not the Same Everywhere

Pleural pressure in the chest is not the same in all locations. The lung has weight and its shape does not conform exactly with the chest. The weight of the lung makes the pleural pressure at the most dependent portions higher. The lung is slightly larger at the bases relative to the chest wall making the pleural pressures in this region also higher.

This variation in pleural pressures makes it more likely that the small airways in segments of lung in regions with higher pleural pressures will reach the closing pressure. This is particularly true in diseases in which the weight of the chest or belly are high such as obesity or in which the supportive structures are weak such as emphysema.





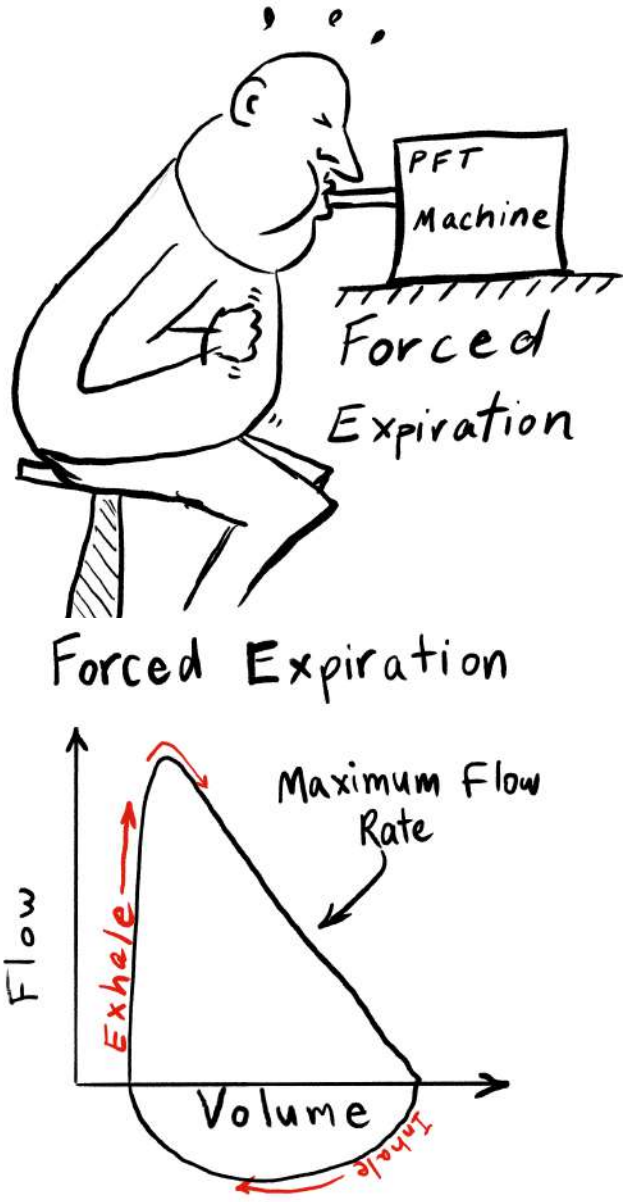
What this also means, when you think about it, is that even though we exhale at a certain rate, we're actually exhaling with myriad small segments each of which has their own expiratory flow. There can be regions that are obstructed and regions that are wide open in the same lung. Keep this in mind when when we talk about VQ mismatch later.

Active Exhalation

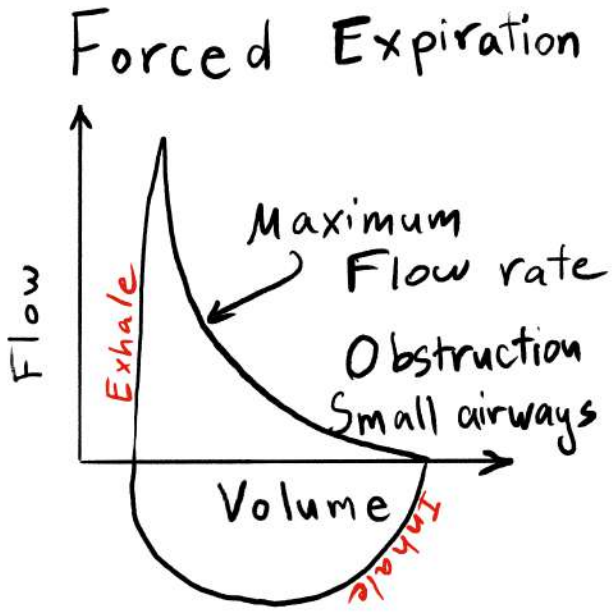
In patients with very elevated respiratory drives, exhalation can become an active process. The air is forced out by tension of the abdominal muscles.

There is a limit to the increase in exhalation speed. This is because the increase in thoracic pressure not only increases the alveolar pressure to push the air out but also squeezes the airways down. At some pressure the squeezing of the small airways, which have no supportive tissue, will obstruct them. The pressure at which this happens is determined by how well these airways are tethered open and limits the speed at which someone can exhale.

A forced expiratory flow test is a common pulmonary function test that can demonstrate the limits in exhalation speed. In this maneuver, a patient blows as hard as possible into a machine which measures flow. The flow and volume exhaled are graphed.



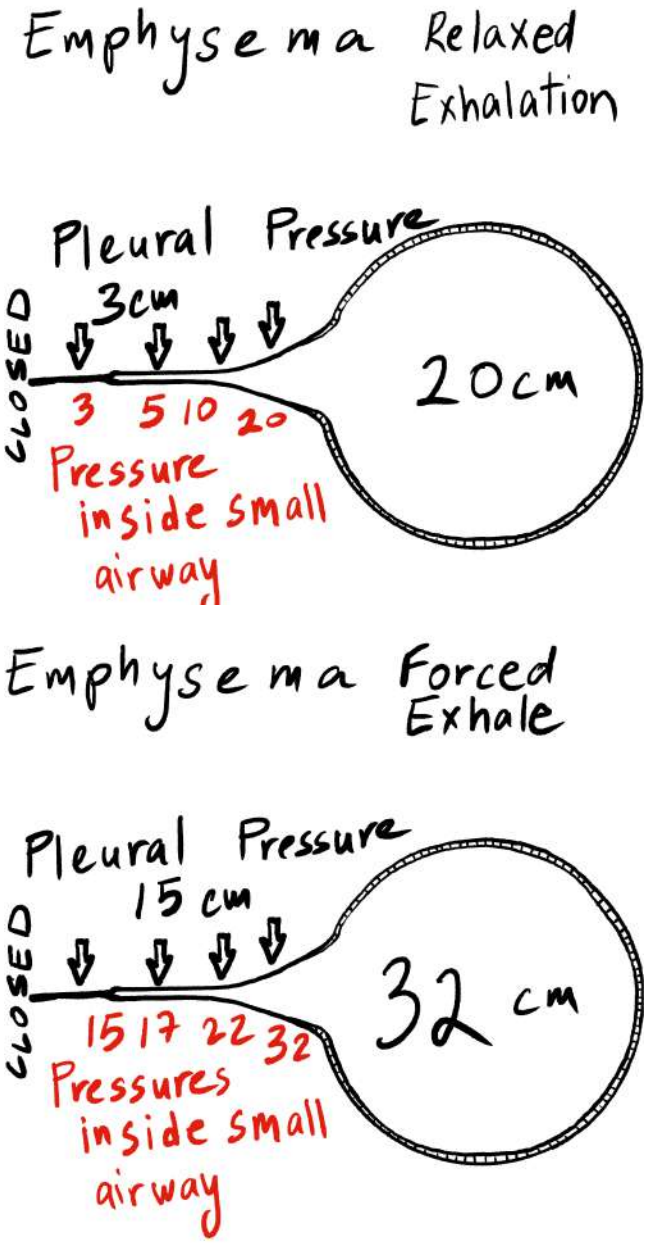
The flow volume loop demonstrates the phenomenon of small airways being squeezed shut with forced exhalations. It shows the maximum flows at various lung volumes.



The second flow volume loop is from a patient with emphysema - it shows that patients with obstructive lung diseases have significant limitations in flows at the various lung volumes.

The limit to exhalation speed is reached very quickly in obstructive diseases, especially emphysema. These patients may be breathing at their exhalation limits even with normal passive exhalations.

Making more of an effort does not increase expiratory flow speeds since the pressure exerted is transmitted to both the alveolar pressure as well as to the pleural pressure. The harder you push to exhale the harder it squeezes shut.



Time to Exhale

The time required to exhale a breath is typically around twice as long as the time to inhale. It depends on a combination of the net force pushing the air out of the chest and the resistance to the flow of the gas.

Diseases which alter the balance of forces in the chest or which cause obstruction will alter the time needed for exhalation.

In diseases which eat away at the lung's elastic recoil such as emphysema not only are the forces pushing gas out of the patient diminished but the resistance to flow is increased. This slows exhalation time greatly.

Several commonly encountered diseases (emphysema, asthma) will cause small airways to become more easily obstructed as the lung volumes shrink and as pleural pressures rise. This significantly prolongs the time required for exhalation.

AutoPEEP

Some of the patients with slow exhalation times can reach a point at which the exhalation time exceeds the time available. These patients are then unable to exhale all that they've inhaled and the chest retains that gas. This is termed dynamic hyperinflation. The retained gas causes an increased pressure within the lung termed autoPEEP.

As the chest expands the lungs and chest wall are placed in a position which favors faster exhalation - the lung is stretched further from FRC and airways are pulled open by the increased volume. A new balance is reached. This new position, however, places the respiratory muscles at a mechanical disadvantage and causes fatigue.

Initially autoPEEP occurs only with faster breathing such as with exertion, but with worsening disease can happen at rest. It can be so significant that it impedes ventilation and causes failure.

Diseases which increase the elasticity of the lungs such as the fibrotic lung diseases will tether the airways open and increase the elastic recoil of the lung. The time for exhalation in these diseases can be shortened.

As we'll see later, this time to exhale and autoPEEP are some of the most important determinants of our limits with mechanical ventilation.

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Gas Exchange

Although our pulmonary system exchanges many gases (as anyone who has eaten garlic can attest) the two gases pertinent to our discussion are oxygen and CO2. In this section we will discuss the exchange of these gases, the similarity and differences.

The exchange of oxygen and CO2 is very similar except for a few important points. To stress these points I've separated them in the explanations below.

Oxygen

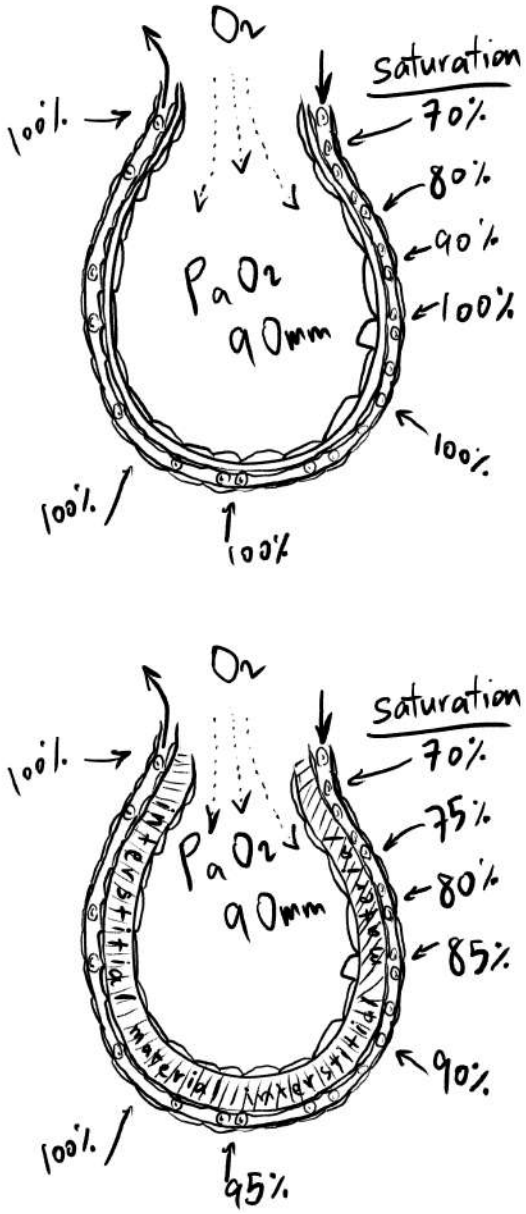
Diffusion

The oxygen molecule takes a particular path to blood. Each breath replaces the used gas in the trachea and large airways. The oxygen moves slowly to the level of the small airways and at the level of the acinus diffusion becomes its primary method of transport. Oxygen diffuses rapidly throughout the acinus, to the alveolar membrane. It diffuses down its concentration gradient, through the endothelium and capillary wall, to attach to the hemoglobin of the red blood cells as they move through the acinar capillary to go to supply the body.

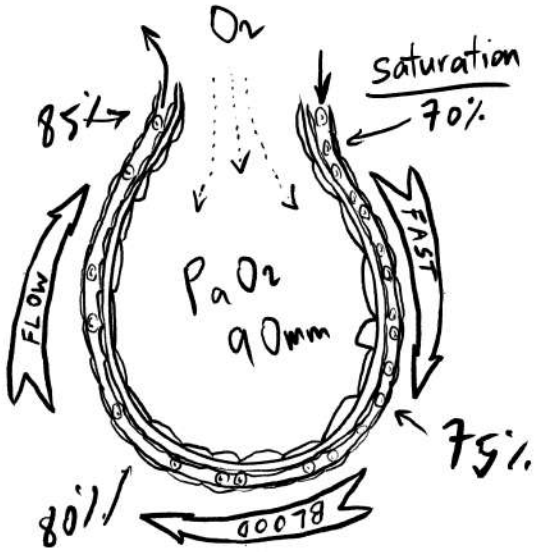
Diffusion, in normal lung, is quick and efficient. A blood cell flowing through the 1 mm long acinar capillary is oxygenated fully (or at least to equilibrium with the acinar gas) in around 0.25 seconds in the first third of its journey (0.75 seconds).

There are factors that can increase how far along the capillary the blood will be before it reaches equilibrium with the alveolar gas. If these factors are severe enough blood may travel the entire length of the capillary and never reach equilibrium. The membrane properties of the acinus and the speed of the red blood cell's movement are the major factors.

In lung diseases that cause the alveolar membrane to thicken the diffusion of oxygen is slowed. Blood takes longer to reach equilibrium in these diseases.



Blood has less time to equilibrate with alveolar gas if it is rushing through the alveolar capillaries at a very high rate - such as during extreme exertion.



The efficiency of the alveolus allows for a great safety margin which makes diffusion an unlikely cause of hypoxemia. With normal diffusion capacity, hypoxemia due to high blood speed is very rare - athletes during extreme exertion. With disorders that cause a diffusion limitation, the contribution of diffusion to hypoxemia increases. It is difficult to tease out how much of that hypoxemia is due to diffusion limitations[2] and how much is due to VQ mismatching (see below).

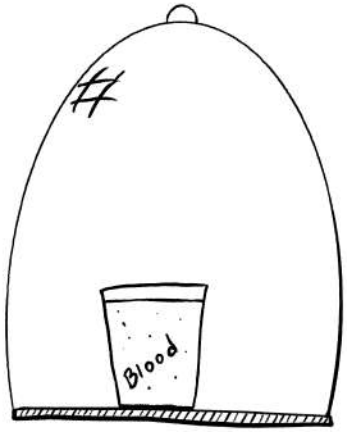
CO2 is not as affected by diffusion limitations because it is 20 times[3] more soluble in water as oxygen. Reaching a diffusion limit is likely to make you hypoxemic before it makes you hypercapnic.

Ventilation Perfusion Matching

The main factor determining the degree to which blood is oxygenated is the amount of oxygen (molecules, pressure of oxygen) in the acinus. Blood perfusing an acinus will reach equilibrium with that acinus's gas. It can only be oxygenated to the partial pressure of oxygen in that acinus. An acinus with a low partial pressure of oxygen will produce blood with a poor saturation.

Blood reaching equilibrium is a sticking point for many folks and so let's do a thought experiment. Take a glass dome (in my head it's a terrarium dome) and fill it with a gas that has oxygen at a partial pressure of 200 mm. The dome is similar to an acinus. Place

a cup of blood into the dome. After a while the blood will reach a saturation of 100%. Take that very same dome and fill it with a gas that has oxygen at a partial pressure of 40 mm. Place a cup of blood into it. After a while that blood will reach a saturation of around 70%. No matter how long the blood sits in the dome the blood remains saturated at 70%. It will not saturate any higher. It is in equilibrium with the gas in the dome.

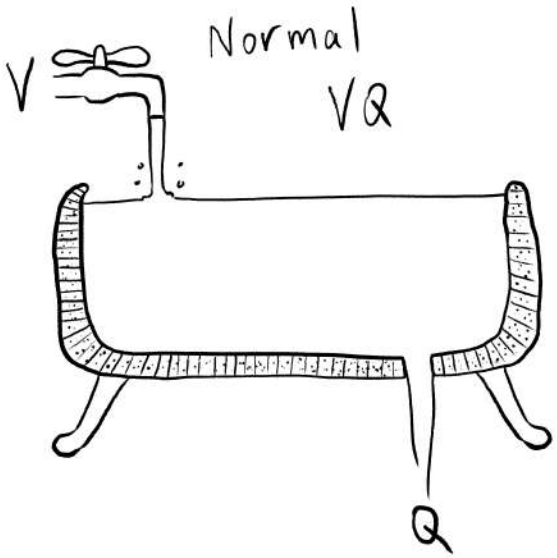


The partial pressure of oxygen in the acinus is determined by the ventilation perfusion ratio.

What is important to understand is that the amount of oxygen in an acinus is in a steady state; oxygen is constantly pouring in and getting sucked out.

Oxygen is sucked out of the acinus by the blood perfusing the capillaries around it. That oxygen is replaced through diffusion from the fresh gas in the bronchiole leading into the acinus. The net amount of oxygen in the acinus is a balance between the amount of oxygen that diffuses in and the amount that is removed. This balance, between these two opposing movements, is termed the ventilation-perfusion (VQ) ratio.

Think of the acinus as a bathtub. Ventilation fills the tub with oxygen. Perfusion drains oxygen from the tub. If there is more drainage than filling, the level of water in the tub will decrease. The VQ ratio is the balance between drainage and filling of the tub. When well balanced, the level in the bathtub is adequate, the VQ ratio is adequate.



Taking this metaphor a bit further, a bathtub can overflow. Too much flow into the tub cannot fill it higher than its rim. An alveolus can only oxygenate blood to a saturation of 100% and no more. Any extra ventilation would be wasted.

An ideal VQ ratio would mean that ventilation and perfusion are matched; a ratio of 1. The VQ match, however, is not perfect and through the normal lung is in the range of about 0.8.

The Alveolar Gas Equation

The alveolar gas equation places the concept of the ventilation-perfusion ratio into an equation:

PAO2 = O2 delivered to the alveolus by ventilation - O2 removed from the alveolus by perfusion

PAO2 = pressure of oxygen in the alveolus O2 delivered = FI(Patm - water vapor pressure) = fraction of oxygen * ambient oxygen pressure O2 removed = estimated by PACO2/R R is the amount of O2 used for the CO2 - typically around 0.8 although varies

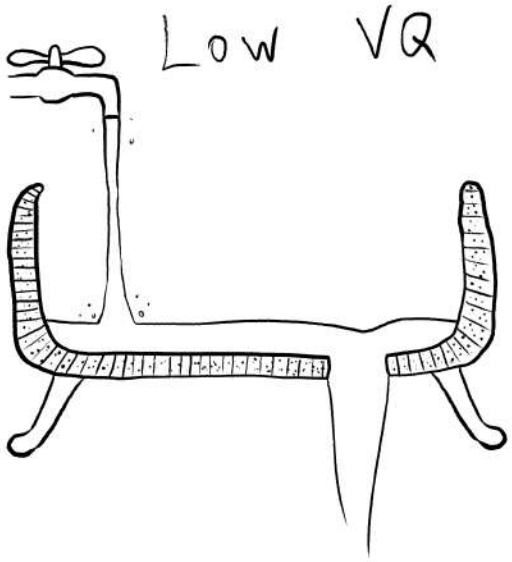
can be rewritten as:

PAO2 = FIO2 (Patm - 47 mmHg) - PaCO2/R

Low VQ Ratio and shunts

Alveoli that have too little ventilation compared to their perfusion will not appropriately oxygenate blood.

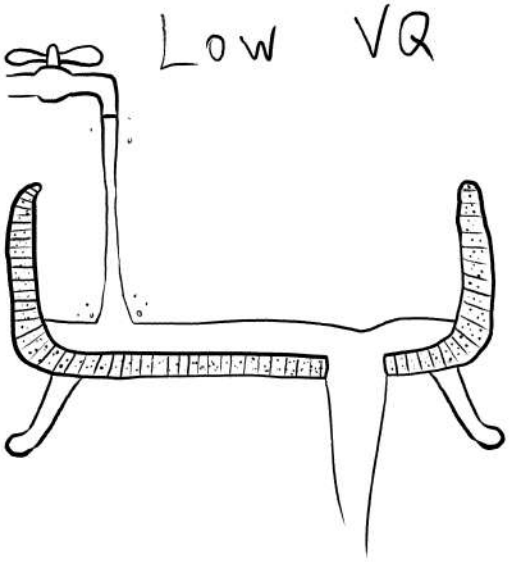
The oxygen pressure in an acinus will drop as ventilation to it decreases relative to perfusion. The oxygen pressure in that acinus will drop as perfusion drains it. The oxygen pressure will approach that of the pulmonary arterial blood as perfusion increases compared to ventilation.

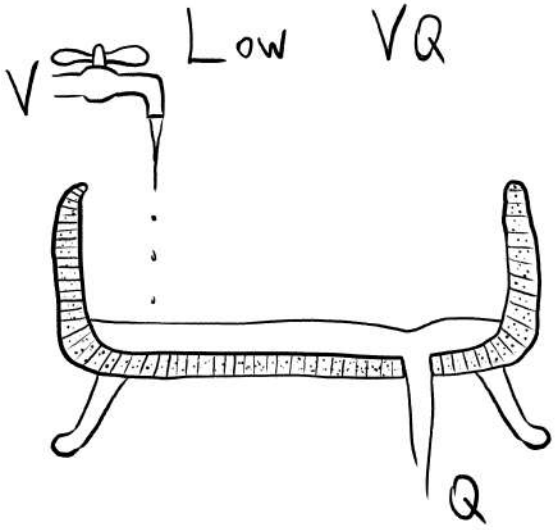


If there was absolutely no ventilation to a segment of lung then no oxygen would enter the associated acini. The oxygen pressure in these non-ventilated acini would equilibrate with and become the same as that in the hypoxemic blood. We call this situation a shunt.

An example of a segment of lung with decreased ventilation is one in which the airflow is impeded such as due to tumor, inflammation, or collapse.

Remember that a segment of lung may be well ventilated but if its perfusion exceeds its ventilation it will still generate poorly oxygenated blood. It is the ratio that is important.

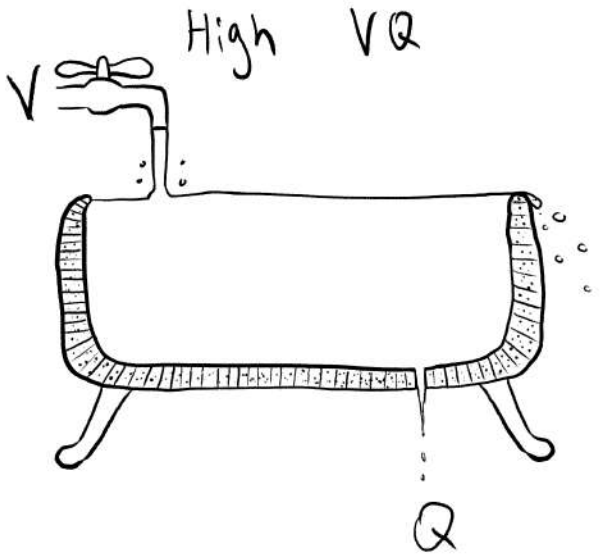




An extreme form of low VQ is a shunt. Shunts can be of two types: Intrapulmonary and Extra-pulmonary. An intrapulmonary shunt is a portion of lung which does not receive any ventilation whatsoever yet is still perfused. An extra-pulmonary shunt is an area outside the lung where blood moves from the venous side of the vasculature to the arterial side without ever running through the lung.

An example of an extra-pulmonary intra-cardiac shunt is a PFO. In a PFO, right heart blood moves into the left side without running through the lung. It does this when right sided pressure rises above left sided pressure such as during certain maneuvers that spike right-sided pressures or in pulmonary hypertension.

A high ventilation perfusion segment is one in which the amount of ventilation is excessive relative to the amount of perfusion. High VQ segments generate blood that is well saturated. There is a limit on how much oxygen can be carried by blood and it depends on Hb. Blood cannot saturate over 100%.



Pulmonary Arterial Blood Oxygenation Matters in Low VQ Segments

To maintain oxygenation, the amount of ventilation must be enough not only for perfusion but also for how hypoxic the blood entering the alveolus is. The more hypoxic the blood, the more oxygen it will pull out of the alveolus, and the lower the PAO2.

The initial saturation of the blood entering the alveolus is the mixed venous saturation of blood and is a reflection of how much oxygen the patient is extracting from blood. An example of a situation with the low mixed venous saturation would be the patient with profound shock, the low saturation reflecting the massive extraction of oxygen in the oxygen starved peripheral tissues.

The low mixed venous oxygen saturation will magnify VQ mismatches. Areas with low V/Q will also see more hypoxemic blood. More drainage if you're still into the bathtub analogy.

High VQ Ratio

Acini that have better ventilation as compared to perfusion cause blood perfusing their capillaries to be very well oxygenated.

The high VQ ratio acini, however, quickly reach a limit in the amount of oxygenation they can achieve. Blood cannot carry any more oxygen after its hemoglobin is saturated. Any excess ventilation above that needed to fully saturate the hemoglobin is wasted effort.

Dead space is an extreme form of high VQ ratio where perfusion is nil and all ventilation is wasted.

As discussed in an earlier section anatomic dead space is due to the conducting airways. Alveolar dead space refers to lung segments with the very elevated VQ ratios.

Different populations

Our lungs are made of thousands of different acini. Each of these is ventilated differently and perfused differently. Each has its own VQ ratio and contributes the blood that it oxygenates to the general pulmonary venous blood pool.

High VQ Ratio Segments CANNOT Compensate for Low VQ Ratio Segments

The final oxygen saturation that the patient experiences is because of all the lung's different segments pouring their blood into vessels that mix into the pool that is the pulmonary venous circulation.

Since the high VQ ratio segments can only oxygenate to the limit of the blood saturation they cannot compensate for low VQ ratio segments. The low VQ ratio segments will, therefore, cause generalized hypoxemia even if there are many high VQ ratio segments.

Dead Space

There are around 3 L of gas in the chest. Most of that gas is in the gas exchange structures - the respiratory airways and acini. The rest is in conducting airways. We move only a small proportion of gas with breathing - around 500cc (the tidal volume).

The gas that we move with breathing comes partially from the respiratory airways and partly from the conducting airways. As you exhale, the first part of the exhaled gas comes from the conducting airways. As the exhalation continues the gas being exhaled comes from the deeper respiratory airways that have taken part in gas exchange.

With each normal breath, the gas that is exhaled mostly comes from the deepest respiratory airways which have participated in gas exchange. Only a small portion is from the larger conducting airways which have not participate in any gas exchange.

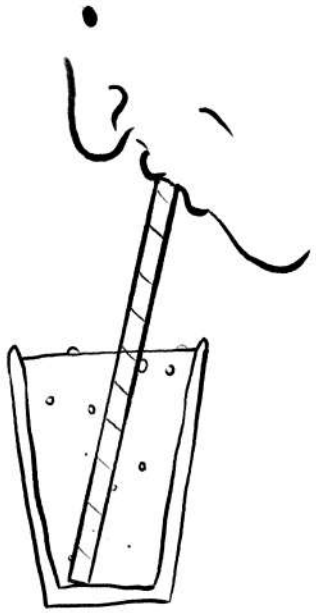
Dead space refers to areas through which gas moves but which do not take part in gas exchange. Some dead space is from the conducting airways which were used to transport the air into and out of the lungs; we refer to this as anatomic dead space. Another type of dead space termed "alveolar dead space" is from gas exchange areas which are not perfused and so are not participating in gas exchange (either due to disease or normal physiology).

With each breath we ventilate the dead space; this takes energy. The larger the amount of dead space the more gas needs to be moved in order to keep the air in the deeper respiratory airways fresh.

Dead space is an extreme form of high V/Q ratio. The ratio is effectively infinite since there is ventilation but perfusion is nil.

Dead Space and Soft Drinks

Breathing can be likened to sipping a drink through a straw. In order to drink, you must suck at the straw until you evacuate it from air and draw the liquid up into your mouth. The liquid you draw up is analogous to the deep gas from the respiratory airways which has participated in gas exchange. The air you must first suction out of the straw is analogous to the dead space which has not taken part in any gas exchange.



An example of having excessive dead space would be if you were to drink through a longer straw (more dead space). You take longer draws to get the same amount of liquid. This is how an increase in dead space worsens the efficiency of breathing. With a long enough straw you would quickly fatigue just trying to have a drink!

CO_2

Diffusion

CO2 diffuses out of blood into the alveoli using the same path as oxygen (albeit in the opposite direction). CO2, however, is more soluble than oxygen and so encounters less of an obstacle when the alveolar membrane is disrupted making it more resistant to diseases that can affect diffusion.

Ventilation Perfusion Ratio

Removal of CO2 also depends on the VQ ratio just as oxygen exchange does. The concepts are the same except the direction of exchange is reversed.

CO2 is delivered to the alveolus by the blood, diffuses into the acinus, and subsequently to the larger airways where it is moved out.

Ventilation of the pulmonary segment determines how much CO2 it removes. The better ventilated the segment, the less CO2 remains in it and the steeper the diffusion gradient that favors CO2 removal. The delivery of CO2 is determined by the blood flow and the amount of CO2 that is carried in that blood.

Low VQ Ratio

Acini that are poorly ventilated relative to perfusion (low VQ ratio) will have a elevated partial pressures of CO2. The poorer the ventilation in that acinus, the closer the partial pressure of CO2 in that acinus is to the concentration in pulmonary arterial blood (pre lung blood).

Blood perfusing an acinus will reach equilibrium with the gases in that acinus. Blood perfusing a low VQ ratio acinus will lose only enough CO2 to reach equilibrium with the acinar gas. After the CO2 in the blood equals that in the acinar gas then the diffusion gradient is gone and further gas exchange stops. If that acinus is poorly ventilated then blood leaving it will not have lost a significant amount of CO2.

A similar analogy as that used to explain hypoxemia can be used for CO2 exchange. Imagine a bathtub. This time, however, the tap pouring into the tub is delivering CO2 from the pulmonary artery and the drainage is ventilation. We must match the two to maintain the water at an appropriate level.

High VQ Ratio

Alveoli that are well ventilated relative to perfusion will have CO2 levels that are closer to atmospheric.

In contrast to oxygenation a high VQ segment does not easily reach a limit for the CO2 it can remove. The high ventilation will only work to lower the acinar gas CO2 closer to atmospheric and maintain a gradient to drive CO2 out of blood.

High VQ Ratio Segments CAN Compensate for Low VQ Ratio Segments

With CO2 the high VQ ratio segments can compensate for low VQ segments. Low VQ segments may increase the pulmonary venous CO2 level by pouring blood containing high levels of CO2. That high CO2 blood, however, can be cleared efficiently by high VQ ratio segments.

This ability of the high VQ ratio segments to compensate for low VQ ratio segments makes CO2 levels more resilient in the face of ventilation perfusion mismatches.

Control of Breathing

The brainstem controls breathing through integrating multiple signals from various sensory bodies. Blood pH, CO2, O2, noxious, and stretch stimuli from the chest wall and lung are sensed. Inputs from higher centers such as anxiety, pain, and fear also contribute.

The brainstem controls not just the rate of breathing but also the depth and rapidity of inhalation as well as the patient's tolerance to delays and paucity of gas delivery.

Likely due to its function in buffering the body's pH, the control mechanism depends most on CO2 levels, and to a lesser degree O2 and the other signals. The normal patient is not very sensitive to O2 levels as the phenomenon of shallow water drowning illustrates.

To keep the tight range of CO2 levels required to maintain an appropriate pH the breathing mechanism matches the generation of CO2 with ventilation. A rise in CO2

will cause a significant increase in respiratory rate and breath depth. If the patient is awake and unused to the hypercapnia they will feel severe air hunger.

This matching of ventilation to CO2 level to respiratory drive can be overridden in various settings - pain, hormonal influences (cirrhosis, pregnancy), hypoxemia, chest discomfort.

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West, John Burnard, and Professor of Medicine and Physiology School of Medicine John B. West. *Respiratory Physiology: The Essentials*. Lippincott Williams & Wilkins, 2005.

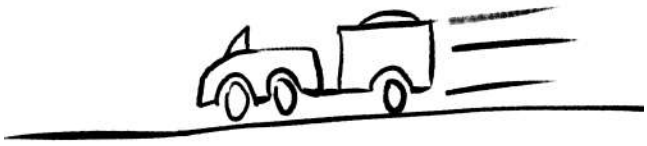
Chapter Two

Failure

Respiratory Failure

Before we discuss respiratory failure, let's agree on what we mean by the word failure. The definition of organ failure is vague. When is an organ in failure? We can define failure of an organ system as its inability to generate the work required in response to a particular demand.

You can liken organ function to a tractor towing a trailer. The tractor pulls a loaded trailer. The tractor is in failure when it is unable to pull the supplied load. It can be relatively healthy but if the load is too great, then it will fail. The tractor can be weak but if its trailer is empty, then it will still work fine.



Similarly, we can define failure in any organ system failure as an inability to generate work commensurate with the current demands. Heart failure is the inability to generate the cardiac output required without preloads that flood the lungs. Renal failure is the inability to clear a required solute or electrolyte load. Respiratory failure is the inability of the respiratory system to sufficiently oxygenate or ventilate.

We classify respiratory failure as hypercapnic failure and/or hypoxemic failure.

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Hypoxemic Failure

Hypoxemic respiratory failure is the respiratory system's inability to bring arterial blood to an adequate saturation of oxygen.

The respiratory system can normally tolerate great increases in demand for oxygen. Cardiac output increases with exertion and can deliver up to 10 times the amount of blood delivered during rest. We maintain arterial oxygen levels within the normal range despite this bigly (too early?) increase in the amount of blood that requires oxygenation.

The body tolerates hypoxemia poorly tolerated and has developed compensatory mechanisms to prevent it. Hypoxemic respiratory failure occurs when these compensatory mechanisms are overwhelmed.

It is most commonly because of ventilation-perfusion mismatches but can sometimes be due to reaching diffusion limitations.

Diffusion Limitations

The alveolus oxygenates blood in the surrounding capillary very efficiently. A blood cell will have travelled less than a third of the way through the alveolar capillary before it is fully oxygenated.

The transit time of blood in the blood vessel surrounding an alveolus is variable and with higher pulmonary blood flow rates this transit time can decrease significantly.

Cardiac output rises with increased demands. The higher cardiac output perfuses the lungs which must then oxygenate that blood. To absorb the new large quantities of blood without posing a significant resistance the lungs use previously closed blood vessels. We term this vascular recruitment.

The increase in blood vessels helps improve the rise in transit time but only up to a certain point. Once we reach that limit any increase in cardiac output causes a decrease in transit time.

Diffusion is rarely a cause of hypoxemia because the cardiovascular system normally reaches its own limit before the transit time ever becomes too short to oxygenate blood.

Some diseases do cause diffusion limitations. Any disease that affects the alveolar membrane or the pulmonary vasculature can make it easier to reach the diffusion limit.

There are many diseases that affect the alveolar membrane. The interstitial lung diseases can cause thickening and scarring of the membrane. Alveolar filling processes can also impair diffusion.

Diseases that eat away at the lung's vascular reserve such as those that cause pulmonary arterial hypertension decrease the lung's ability to recruit blood vessels. This will decrease transit time, particularly in periods of high cardiac output, as blood is pushed through the limited number of blood vessels.

VQ Mismatch and Shunts

Diffusion limitations are only rarely a significant cause of hypoxemia. The primary causes of hypoxemia is VQ mismatch and shunt.

We describe VQ in the section on Function of the respiratory system but basic idea is that the ratio of oxygen influx by diffusion from the larger airways and the oxygen eflux via the perfusing blood determines how well an acinus oxygenates. Shunts are extreme forms of mismatch in which either there is no ventilation to a segment in which perfusion persists, or blood bypasses the lung.

The lung attempts to match perfusion and ventilation by diminishing blood flow to hypoxic segments. This effect is weak and is overcome by high pulmonary artery pressures, by local inflammation that causes vasodilation, and by drugs.

Pulmonary diseases cause VQ mismatches and shunts through a variety of mechanisms. The best way to explain this is to give examples of diseases and describe how their hypoxemia occurs.

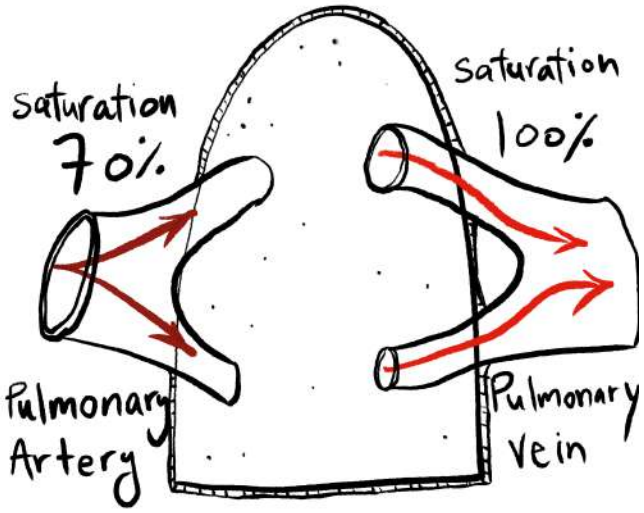
Diverted Blood flow - Pulmonary Embolism

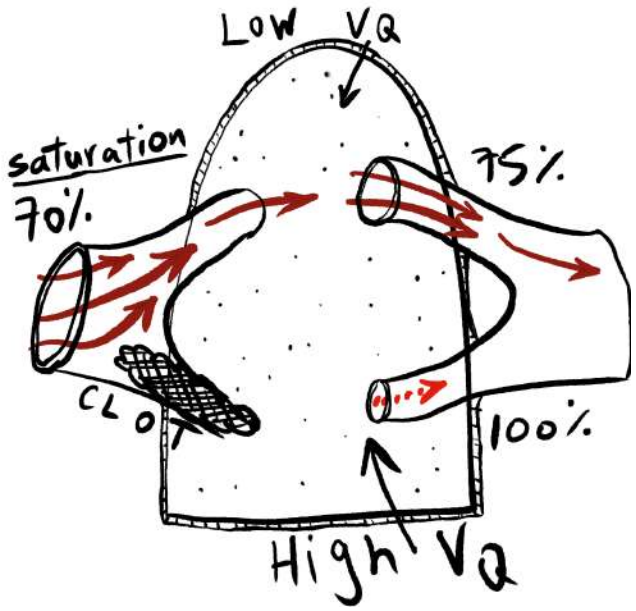
Pulmonary embolism is a favorite way of presenting to an ICU with hypoxemia. A pulmonary embolism is a clot which finds its way into the pulmonary artery and occludes blood flow.

The occluded pulmonary artery had been perfusing multiple segments of lung - segments now without a blood supply. We call segments of lung that are ventilated but no longer perfused and do not take part in gas exchange, "dead space" (more on this later).

Any minimal amount of blood that sneaks past a clot and perfuses this oligemic lung will be fully oxygenated - it will pass through high VQ ratio segments.

Why do patients become hypoxemic with pulmonary emboli? Cardiac output that has been diverted from the occluded segments finds its way to other open pulmonary vessels. Segments fed by those vessels are now hyper-perfused. The VQ ratio drops in the hyper-perfused segments and they pour out hypoxemic blood.



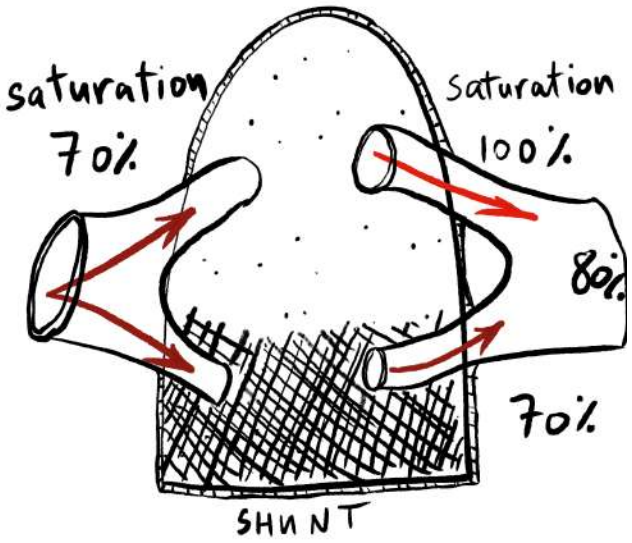


The hypoxemic blood from the low VQ ratio segments gives the patient a low oxygen saturation.

There are other ways in which pulmonary emboli can cause VQ mismatches. Inflammatory substances released by the clotted blood are both vasoactive and cause bronchospasm affecting segmental perfusion and ventilation.

Shunting and VQ Mismatch - Pneumonia and ARDS

In pneumonia and ARDS there is lung injury which causes filling of alveoli. Alveoli filled with debris and pus cannot participate in gas exchange at all; their VQ ratio is zero; they are shunts. Segments of lung surrounding the filled alveoli are poorly ventilated due to inflammation and loss of surfactant.



Blood from shunt segments is hypoxemic and has the same saturation as the pulmonary artery blood.

Systemic hypoxemia is improved when hypoxic vasoconstriction prevents decreases in perfusion of shunts and poorly ventilated segments. The hypoxic vasoconstriction cannot occur in the highly inflammatory milieu of an active pneumonia.

In the widespread damage of ARDS the hypoxic vasoconstriction may be ineffective since there is little healthy lung to shunt blood to.

As we treat pneumonia the inflammation settles down and hypoxic vasoconstriction improves. This is why hypoxia improves before radiographic recovery in pneumonia.

Asthma and COPD

The obstructive lung diseases present with hypercapnic respiratory failure. The patients are also hypoxemic, sometimes profoundly so. Hypoxemia present in obstructive lung diseases is due to VQ mismatches.

In obstructive lung diseases such as asthma and COPD inflammation in the small airways results in worsened expiratory obstruction. This obstruction limits ventilation in the affected segments.

A decrease in perfusion must match the limited ventilation in obstructed segments. If there is no matched decrease in perfusion then the VQ ratio will decrease resulting in hypoxic blood.

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***Summary** * Hypoxemia can be due to diffusion limitations but this is rare. ** Hypoxemia is usually due to shunting and VQ mismatches *** Hypoxic vasoconstriction improves the effects of most diseases but is easy to overwhelm.

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Hypercapnic Respiratory Failure

We know hypercapnic respiratory failure as "ventilatory failure" and can define it as ventilation insufficient to allow the required gas exchange.

We usually see hypercapnic or ventilatory failure as a phenomenon of elevated CO2. Since it affects all gases when ventilation diminishes why do we classify ventilatory failure as a hypercapnic phenomenon? Shouldn't we expect failure of ventilation to produce both an accumulation of CO2 and a decrease in oxygenation? It does.

There are two reasons that hypercapnia is the most prominent side effect of poor ventilation. First is carbon dioxide's resilience to VQ mismatches when general ventilation is adequate. Second is that hypoxemia associated with poor ventilation is easily corrected through a supplementation of oxygen while no such easy therapy is available for hypercapnia.

Ventilatory failure's most prominent clinical effect is an accumulation of CO2 and the only practical remedy to this is to improve ventilation. This makes it understandable that for the most part hypercapnic respiratory failure and ventilatory failure are occasionally used interchangeably.

Hypercapnic respiratory failure can be simplified and explained as insufficient ventilation for the amount of CO2 produced. An imbalance between the amount of CO2 produced and the amount that is removed.

The Amount of CO2 Produced

Metabolism generates CO2. The bloodstream carries that CO2 to the lungs, releases it into the alveolar gas, where it is vented out.

The metabolic rate and diet of the individual patient determines the amount of CO2 generated. A patient can produce enormous amounts of CO2 through vigorous activity or fever. A diet high in carbohydrates generates increased amounts of CO2.

Pathologic conditions such as malignant hyperthermia, hyperpyrexia, agitation from ingestions, or withdrawals cause a massive amount of CO2 generation.

The Amount of CO2 Removed

The amount of CO2 produced is not usually the sole cause of hypercapnia. Increases in CO2 production under expected metabolic loads is usually easily handled. The cause of ventilatory failure is also (or primarily) a limitation in the amount that is removed.

CO2 produced will build up if it is not removed. Its removal depends on its gradient between blood and the acinar gas and so requires adequate ventilation.

Several factors can cause insufficient ventilation. An inadequate drive to breath or a physical impediment in the ability to match ventilatory rate to what is required.

Amount of CO2 Removed with Each Breath - Dead Space Revisited

The gas in the dead space does not take part in gas exchange. A portion of each breath taken, that part that includes the anatomic and alveolar dead space, does not participate in any gas exchange.

Dead space ventilation is wasted ventilation. It does not clear CO2 from the alveolar gas and yet must still be ventilated. More dead space means that whatever remaining

lung is functional must be ventilated more. The higher the patient's dead space, the more ventilation will be required to clear a particular amount of CO2.

If a patient breaths at 5L/min with no dead space that patient would need to breath at 6L/min to compensate for a liter of dead space. This is because whatever dead space ventilation is present does not participate in gas exchange. Only alveolar ventilation, which involves the respiratory airways, participates in gas exchange and removes CO2.

Dead Space Ratio Depends on Breath Depth

Assuming 100cc of anatomic dead space: A shallow breath of only 200cc is 50% dead space. Half the gas in that breath came from conducting airways and only 100cc came from the deeper gases. A deep 500cc breath is only 20% dead space and 400cc is from gas that has participated in exchange.

In the above example we see that 5 L/minute with shallow breaths will clear much less CO2 than 5L/minute with deep breaths. To clear the same amount of CO2 requires much more minute ventilation when the dead space fraction is elevated.

VQ Mismatch in CO2 Removal

VQ mismatches can also contribute to inefficiencies in CO2 removal. Just as a VQ mismatch will cause worsen oxygenation it will cause a worsening in CO2 removal. This is not as important a cause of hypercapnia because of the ability of high VQ ratio segments to compensate for low VQ ratio segments.

Lungs have a mix of high and low VQ segments. A normal averaged total minute ventilation will improve hypercapnia produced by VQ mismatches because some segments - those with high VQ ratios - will clear CO2 well. Hypercapnia occurs when there are no high VQ segments of their number is very low (e.g. low total minute ventilation - drug overdose with a very slow respiratory rate causing all segments to be hypo-ventilated).

Determinants of Total Minute Ventilation

The patient's minute ventilation is linked to the CO2 production through the respiratory control system.

Ventilatory failure occurs when an insufficient minute ventilation is produced either due to a failure of the control system or due to a physical limitation in the ability to generate minute ventilation.

Failure of the Respiratory Control System

Inadequate Respiratory Drive

When there is an inappropriately poor drive to breathe ventilatory failure will occur. The drive to breathe is normally a strong, quick, unrelenting stimulus (as anyone who has tried to hold their breath for an amount of time can attest). With a rise in CO2 your brainstem quickly makes breathing your favorite thing in the world.

There are reasons a patient may not respond to the elevated CO2 levels with an increased drive to breath.

Drugs can blunt the respiratory centers. Among many others, opiates and benzodiazepines are common drugs with this effect.

Catastrophic CNS events can also cause a blunted respiratory drive. A rise in intracranial pressure or a drop in cerebral perfusion can manifest as a loss of respiratory drive.

Alkalosis can diminish respiratory drive. Shallow water drownings occur when a swimmer forces a respiratory alkalosis, then dives and loses consciousness due hypoxemia. The swimmer's respiratory drive was blunted due to the alkalosis and did not kick in before the hypoxia caused them to lose consciousness. Another example is the hyperventilating child that then holds their breath and passes out.

Causes of blunted respiratory drive * Drugs: sedatives, opioids ***Cerebrovascular accidents*** * *CNS tumors* ** Alkalosis

Inability to Generate the Work (Pump Failure)

A mismatch between the amount of work required for ventilation and the amount that can be generated results in ventilatory failure. This is basically a failure of the "gas pump", the set of organs whose role is to move the gases into and out of the lungs.

Work of breathing is what we require to generate pressure gradients that move gas into and out of the chest.

The chest and lungs are normally tuned to minimize the work of breathing. Surfactant reduces surface tension in the alveoli to such a degree that normal lungs are 10 times as compliant as rubber balloons (Respiratory Physiology: The Essentials). The large cumulative cross sectional area of the respiratory bronchioles allows gas flow with minimal resistance. The position of the diaphragm places it at a mechanical advantage.

Several situations can occur that either increase the amount of work that is required or limit the amount of work that can be generated.

Obstructive Lung Diseases

Obstruction is any process that limits air flow. This can be a limitation to inhalation, exhalation, or (if you're really having a bad day) both.

When an obstruction moves to impede only the inflow of air it causes a limitation isolated to inhalation. These obstructions act as valves and are typically extrathoracic in location. An example is a mobile tumor or malacic (soft, unsupported) segment of trachea that gets sucked into the airway only during inhalation.

Some circumstances cause a limitation of flow to both inhalation and exhalation. This is usually due to a very tight obstruction (on the order of millimeters) that varies little with respiratory phase (does not getting pulled or pushed by the air flow). Air moving around such an obstruction encounters significant resistance in whatever direction it is flowing. An example of this is a stenotic scarred trachea, a tumor, or an occluded endotracheal tube.

Limitations isolated to exhalation are the more commonly encountered. Though these can be due to macroscopic causes such as a mobile segment of intrathoracic trachea or tumor, they are more commonly due to small airways disease.

During the usual exhalation phase, when intrathoracic pressures become positive relative to atmosphere, an abnormal, unsupported, mobile segment of intrathoracic trachea gets pushed inwards. This causes the obstruction. When the patient pushes harder to try to expel the air that segment gets pushed further inwards.

Similarly the small airways all act as unsupported trachea during normal circumstances. As discussed previously, the small airways are flimsy and rely on tension from surrounding tissues to hold them open.

With exhalation, as the lungs lose volume, the tethering that keeps the small airways open loses tension. As tension is lost the small airways collapse and are obstructed. In addition to the loss of supportive tension the small airways are also squeezed by pleural pressure ,and during exhalation this pressure rises so exacerbating the obstruction.

The tendency of the small airways to obstruct is worsened by several common diseases. Emphysema destroys the tissue around the airways causing a loss of tethering. Inflammation and mucous in asthma and bronchitis narrow the airway lumen making it more prone to obstruction.

As a limitation to exhalation progresses, the amount of time that is required to deflate that segment increases.

The time required for exhalation can become longer than the time available. The segment then becomes “hyper-inflated” and is unable to exhale fully. As the process continues the segment may become so obstructed that airflow can cease.

Restricted Lungs or Chest Wall

Lungs are remarkably compliant and easy to ventilate. Several processes can make lungs stiff.

In pneumonia and other inflammatory lung processes there is loss of surfactant. This causes an increase in surface tension which works to collapse the alveoli making lungs stiffer and difficult to ventilate.

Scarring lung diseases such as pulmonary fibrosis cause deposition of excess connective tissue in the lung. This fibrous tissue makes the lungs stiffer.

The chest and associated muscles are set up to minimize the amount of work. Rib and vertebral joints allow free and easy movement of the rib cage. The diaphragm is at a level where the muscles are at an optimal mechanical advantage.

Skeletal abnormalities can interfere with the proper function of the chest. The kyphotic chest will not allow appropriate inflation of the chest and places the diaphragm at a mechanical disadvantage. The articulations of the ribs with the spine can become damaged in some disorders decreasing the chest's ability to expand.

Weakness

Breathing requires the work of multiple muscles including the diaphragm and accessory muscles. Diseases that affect these muscles or their nerves will affect the ability to breathe.

An example is the patient with a neuromuscular disease such as Guillain barre. The patient is limited in their ability to maintain ventilation by the progressively denervated muscles.

Cardiovascular Limitations

Inability of the cardiovascular system to deliver the oxygen required by the respiratory machinery will limit ventilation.

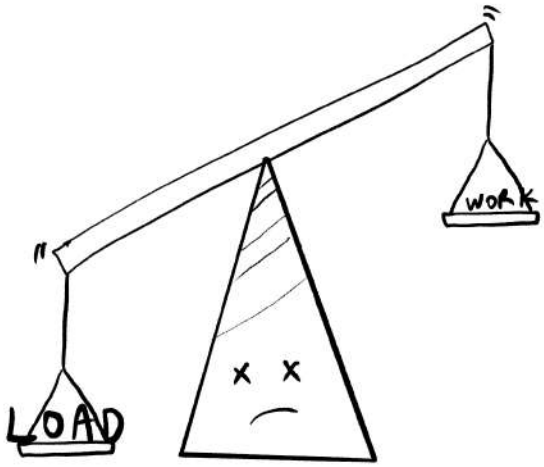
The cardiovascular system is the power source of the respiratory system. Any issue that causes the cardiovascular system to reach a limitation and be unable to deliver the required oxygen will cause ventilatory failure.

Valvulopathy, cardiomyopathy, and coronary insufficiency place limitations on cardiac output. A diseased cardiovascular system which can maintain output during times of

health may reach its limit in times of stress and disease. Sepsis and stress can place demands on the cardiovascular system that it can't meet.

Putting it all together - Balance

Every patient has a limitation to their ability to ventilate. Exceeding this limitation is the definition of ventilatory failure.



A particular patient's limitation may be due to a combination of any of the above mentioned causes. We reach the limitation in a normal patient only when supra-normal amounts of ventilation are required. A diseased patient's limitation is reached more easily through a progressive accumulation of the above factors.

We must keep in mind that any patient can reach their limit and fail. This is obvious in the frail, elderly, and diseased because of the ease with which they reach that limit.

We must also keep in mind that the demands placed on the ventilatory system are variable. The minute ventilation required at rest is low and may be below their maximal capabilities. A fever, however, increases the ventilatory demand and can exceed those capabilities.

A patient with severe kyphosis may be capable of generating a maximum minute ventilation of 5L/minute without issue. As long as this is sufficient for her requirements

then she is fine. If a fever causes the need for more minute ventilation (e.g. 7L/minute) she will fail. If a new impediment to ventilation such as a pneumonia stiffens her lungs lowering her maximum minute ventilation, she will fail.

This concept of particular limitations to minute ventilation is one reason that patient's with severe lung disease can present with respiratory failure when they develop problems that have nothing to do with the lung.

Occasionally a patient with normal lungs and neurologic function can come up against a minute ventilation requirement that overwhelms them. Examples of this are the dysautonomia syndromes such as NMS or malignant hyperthermia where the massive amounts of CO2 production can overwhelm anyone.

Acute and Chronic Ventilatory Failure

Ventilatory failure can be acute or chronic. An insidious onset of any of the above listed limitations to ventilation allows the patient to adapt. Powerful chronic compensatory mechanisms allow tolerance to elevated levels of CO2.

In chronic respiratory failure the blood CO2 level may be high but pH is maintained in the appropriate range through the generation of bicarbonate by the kidneys.

The respiratory system in a patient with chronic respiratory failure is typically functioning at or close to its limit. It takes little to tip it over.

Acute respiratory failure is a failure that has occurred over a time too short for the strong chronic compensatory mechanisms to react. There are myriad causes of acute respiratory failure. A patient in chronic respiratory failure has little reserves and is easily tipped into a concurrent acute failure.

Chapter Three

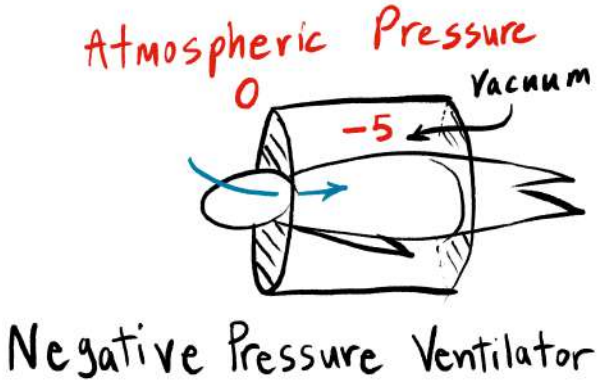
Fundamentals Of Mechanical Ventilation

What is Mechanical Ventilation?

Mechanical ventilation is the use of a machine to administer "mechanical breaths" in order to oxygenate and ventilate.

"Positive pressure" ventilators are the most common types of mechanical ventilators. In a positive pressure ventilator, the mechanical breath pushes gas into the patient. Just as you inflate a balloon, a positive pressure breath is generated by gas being forced into the chest. We call it a positive pressure breath because it is generated by positive pressures (i.e., higher than atmospheric pressure) that push air into the chest.

In a negative pressure ventilator, a patient's thorax is encased in a chamber which is evacuated of air. The vacuum generated in the closed chamber expands the patient's chest and pulls the gas into the lungs. It is negative pressure in that it is a relative vacuum (i.e, a pressure lower than atmospheric pressure) surrounding the chest. Negative pressure ventilators such as the cuirass and iron lung still exist and have a role in mechanical ventilation. My experience with them is minimal and they are not routinely used in the ICU and so I will not discuss them further.



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Parts of a Mechanical Ventilator

A mechanical ventilator, at its simplest, is just an air pump. A simple ventilator can be nothing but a bag that is squeezed to generate gas flow.

Complexity in ventilators are is due to automation, safety mechanisms, gas conditioning, and monitoring devices. This section discusses the basic parts common to most mechanical ventilators.

Most ventilators are made from at least 3 parts:

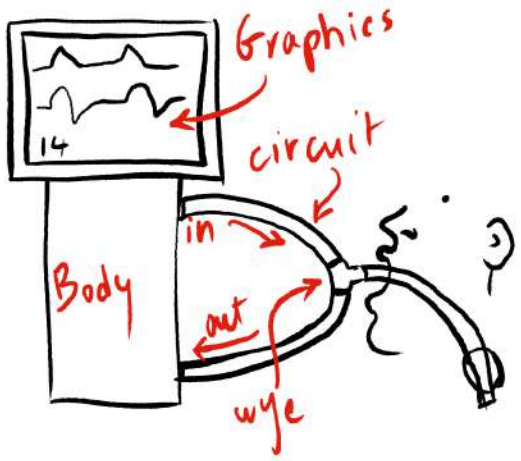
- A screen or user interface - Some method of operating and communicating with the device. In most modern ventilators this is a touch screen although you can still find older ventilators in which the interface is made of knobs and buttons.
- A body containing a blender of gases, perhaps a compressor.
- The ventilator circuit - a set of flexible tubes with associated valves and sensors attached to the endotracheal tube or mask that delivers the gas mixture to the patient.

The most conspicuous part of the ventilator is usually a screen where various graphics are displayed. This functions to display data, alarms, and usually a user interface through which the ventilator can be controlled. Alarms and monitoring numbers are usually displayed on this screen or nearby.

The screen is integrated into or attached to a body. The body houses the components which take in the gases from the wall or atmosphere, filter, mix, and adjust their pressures.

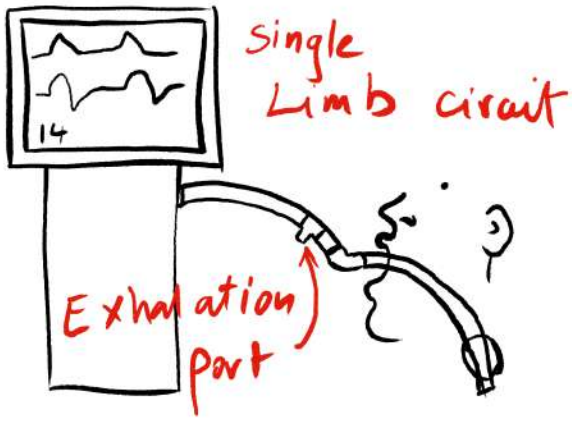
The body has a port for the gases leaving and usually one for those re-entering. An outflow port for gas leaving the ventilator towards the patient and usually an inflow port for gas returning from the patient. If there is no inflow port present the gases are vented somewhere away from the ventilator.

There is flexible tubing running from the ventilator ports. This tubing is termed the ventilator circuit since gas circulates through it. Ventilator circuits can be dual limb or single limb.



The usual ICU ventilator circuit configuration is dual limb and has separate limbs (tubing) for inhalation and exhalation. The tubing attached to the outflow port is called the inspiratory limb because the patient inspires gas through it. The tubing attached to the inflow port is called the expiratory limb and it receives the exhaled gas.

In the single limb configuration the exhaled gas does not return to the ventilator but rather is evacuated from the same limb tubing.



In dual limb ventilators the two limbs are connected with a Y shaped connector (Y junction) to the patient who himself is connected either through an endotracheal tube, tracheostomy tube, or sealed face-mask.

There are various devices which attach to the ventilator circuit in order to condition the gas, filter it, and take measurements. On the inspiratory limb there may be a heated humidifier. Attached to the Y junction there may be a heat moisture exchanger to prevent moisture loss. Nebulizers can be attached to the tubing at various points.

Valves control the flow of gas into and out of the ventilator circuit. The inhalation valve at the origin of the inspiratory limb will deliver gas at varying rates during the different stages of the breath, the expiration valve at the expiratory limb will limit gas outflow to varying degrees during the different stages of the breath.

Pressure and flow sensors measure gas parameters and are present along the ventilator tubing, sometimes within the body of the ventilator.

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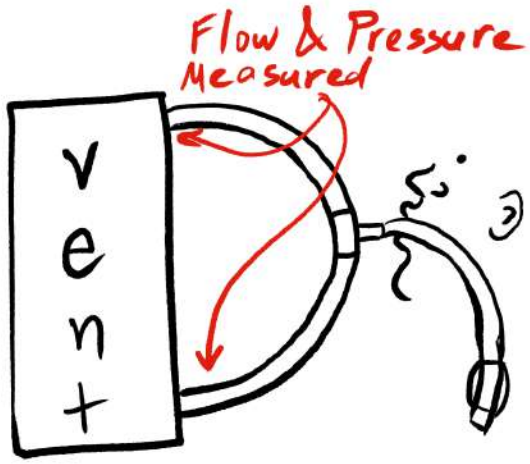
Pressure and Volume

Before we talk about breath characteristics and modes in mechanical ventilation, let's explore the ideas of pressure and volume measurements in the ventilator.

Pressure Measurement

The ventilator is constantly measuring the pressure in the circuit. The pressure measured is used to monitor for safety (a high pressure can indicate an occlusion of the airway) and to adjust ventilator flows for pressure targeted breaths.



The ventilator measures the pressure only in the circuit. There is no direct measure of the patient's intra-thoracic pressure.

The circuit pressure that the ventilator senses while administering breaths is due to factors we can simplify as being 2 separate pressures. A dynamic pressure and a static pressure.

Dynamic Pressure

Imagine that you are blowing through a narrow straw - in my mind I imagine the little white coffee stirrer straws. Blowing through the straw requires great effort and there is a feeling of back pressure as your cheeks puff out and your face gets red. The straw, on its own, doesn't move air. If you stop the blowing, the straw does not blow back at you.

The back pressure felt is caused by the resistance of the straw to the flow of air. If the flow ceases, that back pressure also disappears. If the straw is narrower or longer, the amount of resistance increases. When you try to increase the rate of flow then the back pressure also increases.

Similarly, in the ventilator, there is a pressure that builds up as the ventilator forces gas through the ventilator circuit and ETT. In this case the majority of resistance to inhalational flow is *usually* due to the endotracheal tube or large airways.

We term this dynamic pressure because the pressure experienced requires a flow of gas and ceases when the flow stops.

Static Pressure

There is pressure caused by the elastic recoil of the lungs. The respiratory system once filled will attempt to return to FRC, and in doing so squeeze the inhaled gas thus generating pressure.

Imagine inflating a balloon. Once inflated, the balloon will attempt to push the gas back out. This push-back from the balloon depends on the volume and stiffness of the balloon; it continues even after attempts to inflate the balloon stop.

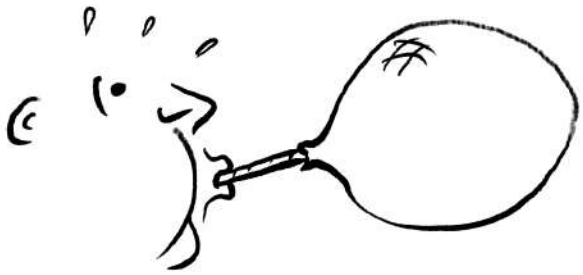
The pressure from the recoil of the lungs and chest will continue even if there is no movement of gas. We term this static pressure.

Total Pressure

At any point in time the total pressure being measured is the sum of the static and dynamic pressures at that point. As a breath is administered to the patient, the two pressures contribute to the total pressure in varying degrees. Initially the dynamic pressure predominates since the lung is still empty but as the lung inflates the static pressure begins to contribute more.

The ventilator/chest combination can be imagined as a balloon attached to a straw. The pressure sensed is what you feel as you blow into the straw to inflate the balloon. When the balloon is still flat all the push-back you feel to flow is all due to the straw. As the balloon inflates, it begins to contribute to that push-back. Eventually once the

balloon is completely full and you stop inflating it, all pressure you sense from the straw is due to the balloon's elastic recoil.



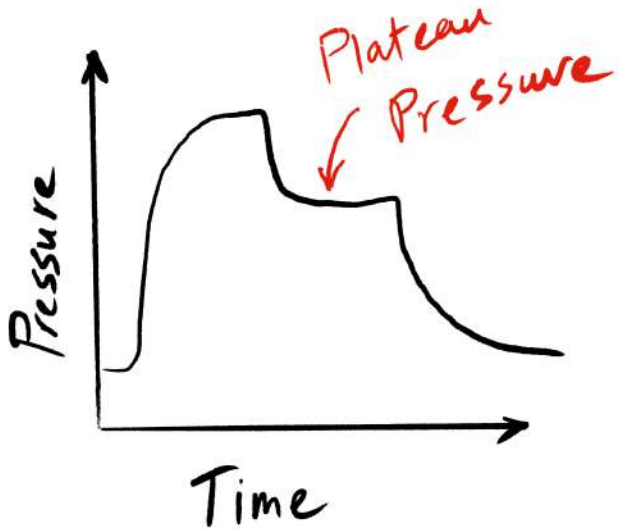
Peak Pressure

Peak pressure is the maximum total pressure measured during the breath and is an important parameter; a rising peak pressure can herald disaster. The ventilator will display the peak pressure measured during the mechanical breath.

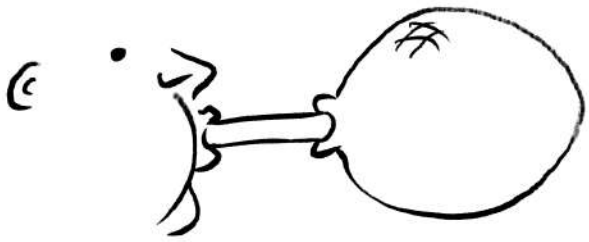
The peak pressure is composed of a sum of the dynamic and static pressures discussed above. Figuring out why a peak pressure is rising is an important skill and requires you to separate out the static and dynamic components.

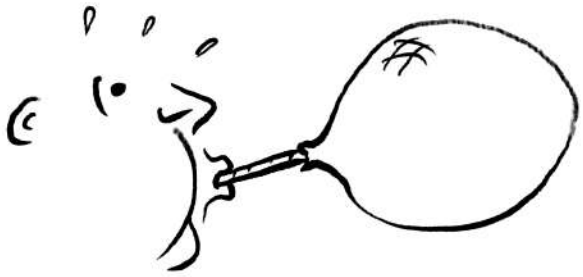
Plateau Pressure

Plateau pressure measurement is a method of trying to tease out the cause of a rising peak pressure. It is the static pressure. It is the pressure measured at the end of a breath when the flow of gas has been turned off. The lungs are inflated with a volume, the breath is paused (typically for half a second) to allow the circuit pressure to equilibrate with the patient's tracheal pressure, and then pressure is measured.



The measurement of a plateau pressure is akin to you holding the straw shut while attached to the inflated balloon and checking to see how much back-pressure is present. A high back-pressure indicates that the balloon is overinflated or stiff. A low pressure indicates that the balloon is fine but that the straw has been posing too much resistance to flow.





In the patient with elevated peak pressures, a high plateau pressure indicates that the lungs/chest wall complex is non-compliant. This may be because of a worsening in the pulmonary process, a pneumothorax, the loss of a lung to mucous plugging…

Elevated peak pressures with a low plateau pressure shows that there is unusual resistance to flow - a kinked tube, mucus in the tube...

Pressure is Measured in the Circuit

The pressure measured by the ventilator is the pressure in the circuit. There is no measure of the pressure inside the patient. This is an important idea.

Imagine a straw attached to a balloon. The pressure measured is in the straw. Now imagine that we inflate the balloon then allow it to deflate slowly. While the balloon is deflating our pressure measurement at the straw is always less than the pressure within the balloon. In fact, our balloon may be horribly pressurized and yet the pressure in the straw may give no indication of this.

Volume

The ventilator measures volume with flow sensors (volume is flow multiplied by time). These can be present at the expiratory port although in some ventilators are present closer to the patient.

Volume is measured as it leaves the ventilator and then again as it returns. Mismatches between volume administered and return can indicate problems.

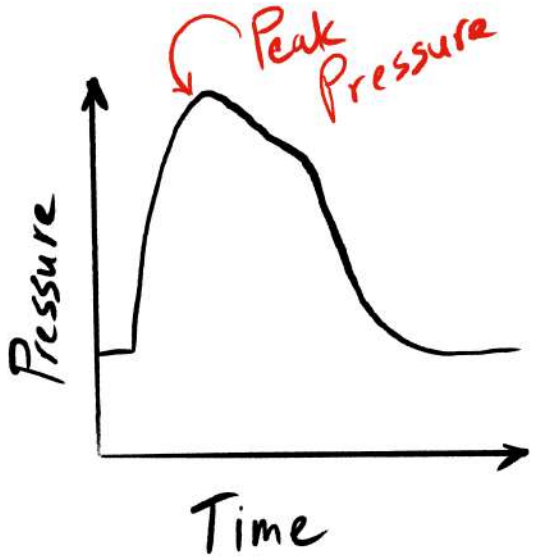
Ventilator Graphics

Most modern ventilators have a screen on which graphics are displayed. The graphics are a good way to quickly evaluate the function of the ventilator and can help troubleshoot and find the cause for alarms.

Several graphics are available.

Pressure Time Curve

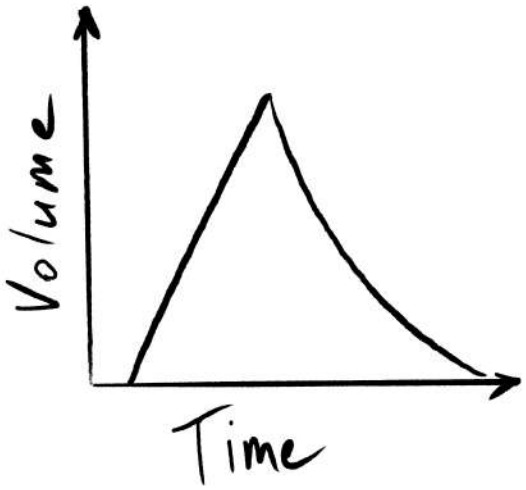
Pressure time graphs give an idea of the pressure over time. A quick look shows what the peak pressure is and when in the breath it is occurs. It can reveal a flow dyssynchrony or elevated work of breathing as a significant drop in pressure before the triggering of the new mechanical breath.

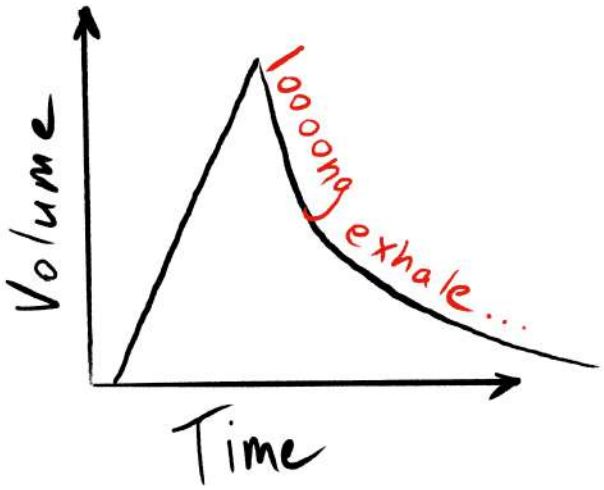


Volume Time

This graph gives the ventilator's measurements for inhaled and exhaled volumes. A quick look at this graph can find a leak, an extraneous infusion of gas, or show volume

variability in support mode. It can identify dyssynchrony such as double breaths. The amount of time it takes to exhale the volume can show the degree of obstruction.

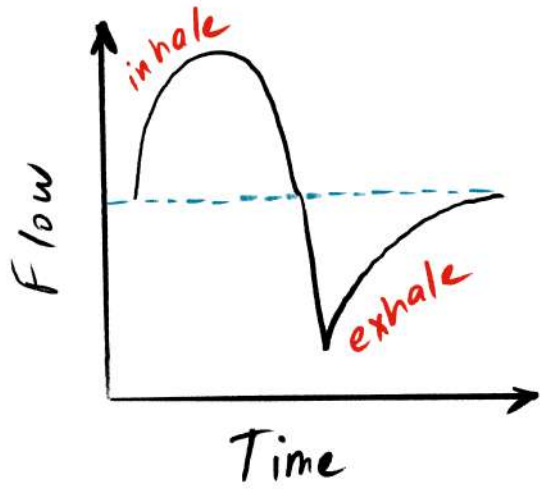




Flow Time

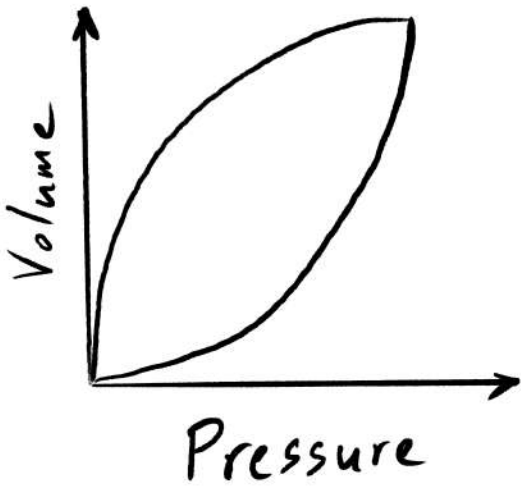
This graph gives the gas flow into and out of the patient. A look at this curve can reveal breath stacking, an extraneous infusion of gas, and in a volume-targeted breath flow dyssynchrony. The speed and pattern of the gas leaving the chest can indicate whether

there is a significant resistance to the flow and, if treated, whether the treatment made a difference.



Volume Pressure

This graph can reveal a lung's poor compliance and whether the PEEP is sufficient to maintain open alveoli. Because it incorporates the dynamic pressure due to the gas flow it does not give the information as accurately as a pressure volume curve obtained by inflating the chest and checking with no flow but it can give an idea of what is going on and raise suspicions if the chest wall or lung are losing compliance.



Flow Volume

This graph shows the flow relative to volume. Because it is not a forced exhalation, it is not the same as the classic pulmonary function test's Forced Flow Volume Loops but still can reveal obstructions with a scooped pattern on exhalation and can show whether this has improved with treatment.

The Mechanical Breath

The mechanical breath is an inflation of the lungs by the ventilator. It's as simple as that. A squeeze of the bellows. Understanding the simple mechanical breath is the first step to understanding mechanical ventilation.

A mechanical breath has two parts: an inhalation and an exhalation. The inhalation is the only part controlled by the mechanical ventilator. The exhalation part, discussed later, is passive and does not rely on the mechanical ventilator.

Because the inhalation is the only part of the mechanical breath that is controlled by the mechanical ventilator, we will use the terms mechanical inhalation and mechanical breath interchangeably.

Inhalation

The 3 Basic Properties of the Mechanical Breath

The mechanical breath has 3 basic properties. We can boil the many of modes that often overwhelm students down to breaths with different mixes of these properties.

The 3 basic properties are the Target, Time, and Trigger.

Property	**Definition**
Target	Parameter the ventilator controls
Time	Duration of inhalation
Trigger	What causes ventilator to initiate a breath.

Target

The target is what the ventilator controls. This is the mechanical breath's set goal; what the breath aims to achieve. It is the parameter controlled by the ventilator.

Two usual targets are volume and pressure. A volume target breath administers a set volume. A pressure target breath aims to generate and maintain a set pressure in the circuit. There can be dual target breaths which we also discuss later.

Administration of the Target

Volume Target

In a volume targeted breath the ventilator delivers a prescribed volume by releasing gas into the circuit through the inhalation valve.

Once the set amount of gas is administered, the patient is allowed to exhale and an exhalation port is opened.

Pressure Target

In contrast to volume target breaths, in a pressure targeted breath the volume is not set. Instead, a driving pressure is set. The machine administers flow into the circuit until the circuit reaches the set pressure. For the duration of the breath, the flow through the inspiratory valve is changed in an attempt to maintain that set pressure.

The pressure in the circuit during a pressure targeted breath is maintained via a feedback loop. If pressure in the circuit drops, flow is fed into the circuit. If pressure rises, flow is slowed down.

For example: If the patient is inhaling, the pressure In the circuit will drop as gas is pulled out of it. In order to maintain circuit pressure, gas is fed into the circuit through the inspiratory valve. If the inhalation of the patient slows then the amount of gas removed from the circuit decreases and the inflow through the inhalation valve also decreases.

Different Target Breaths Guarantee Different Things

In volume targeted breaths, the ventilator will guarantee that the prescribed volume will be pushed into the circuit. That prescribed volume will be given at a set flow rate

and this will generate a variable pressure in the circuit depending on the patient and circuit characteristics.

In pressure targeted breaths, the ventilator will guarantee that the circuit reaches the prescribed pressure. That pressure inflates the lung with variable volumes which depend on patient characteristics.

These differences in which parameter is guaranteed give the different targets particular advantages and disadvantages.

Choosing Targets

Choosing a breath target is largely a cultural/training choice. There have been no proven survival benefits from the use of one target versus the other. There are certain particular advantages and disadvantages to each target.

Volume Target Breath Advantages and Disadvantages

A volume target guarantees that the lungs are inflated with the set volume. This advantage comes with a caveat: the settings do not take into account the pressure changes the administered volume will cause. This can, therefore, result in excessive pressures applied to the lung.

Ventilator synchrony can also be an issue with volume target breaths. During a volume target breath the volume is administered with a set flow rate. If this set flow rate is not matched by the patients inhalation the result is an uncomfortable dys-synchrony with the ventilator.

Pressure Target Breath Advantages and Disadvantages

A pressure targeted breath will maintain the set pressure in the circuit without taking into account the volume being delivered to the lungs. This is an advantage in that it keeps the lungs from experiencing excessive pressure. It is also one significant disadvantage in that lung volumes are not controlled and changes in lung compliance or patient inspiratory efforts can cause them to diminish and result in either insufficient ventilation or excessive and damaging breath volumes.

Dual Target Breaths

Dual target breaths are hybrid breaths that offer advantages of both targets. Typically they are pressure target breaths with some guarantee for volume. The prescriber will set the volume target and the ventilator will adjust the pressure to reach that volume.

The breath's pressure parameters are dynamically altered to meet a set volume target using data from prior breaths. The pressure changes can be between breaths or within the same breath.

Dual Target Breath Advantages and Disadvantages

Because the dual target breath is pressure controlled it carries all the advantages of that target. It has the advantage too of having the pressure change to reach a set volume. What can go wrong, right?

The disadvantage of a dual target breath occurs in patients with air hunger. Initially the patient's effort will cause high volume inhalations. With the high volume inhalation the ventilator will decrease the amount of pressure provided and so decreases its support of the patient. In a patient with air hunger, therefore, the amount of support provided by the ventilator is progressively decreased. This decreased support causes the patient to have to work harder for each breath and negates one important benefit for mechanical ventilation which is to offload a fatiguing ventilatory system. In addition to this, the air hungry patient being given a pressure targeted breath can inhale excessive volumes causing lung damage.

This disadvantage is present in the air hungry patient, generally early in the course of a patient's acute course. In most other situations the dual target breath is a comfortable mode that can be used to improve patient synchrony.

Time

The time property of a mechanical breath is a measure of how long the inhalation lasts. The amount of time before the ventilator stops the inhalation and allows the patient to exhale.

Stopping a mechanical breath is termed cycling. The breath can be cycled by the machine or by the patient.

Machine cycled breaths will cycle based on parameters that are set by the operator. Patient cycled breaths rely on some interaction between the machine and the patient.

Machine Cycled

Volume Cycled

A volume target breath is cycled after the prescribed volume has been administered. The time it takes for a volume to be administered depends on the speed at which the gas in pushed into the lungs, the flow rate of the gas. The same volume can be given very quickly with rapid flow or very slowly with slow flow.

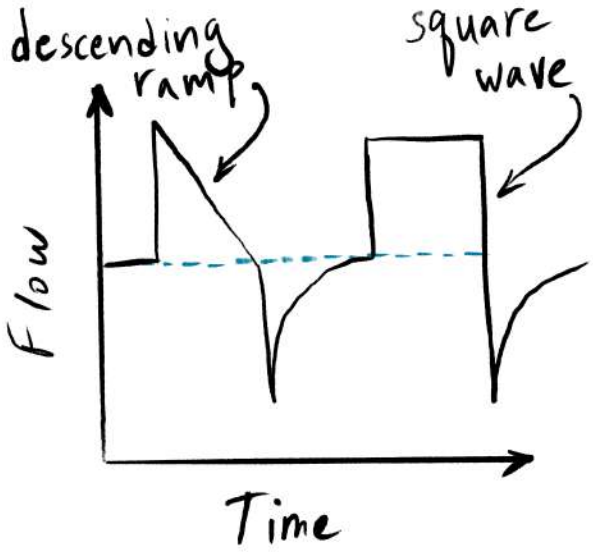
A higher flow rate completes the breath faster but has some drawbacks. It will generate more pressure as it enters the patient. It will meet more resistance at the airway due to the higher speed of the gas. The dynamic pressure rises with higher flow rates - see section on dynamic and static pressure. A decreased flow rate delivers the breath slower but causes less pressure at the airway.

The quicker completion of the breath can also cause issues with synchrony. The patient may want a longer breath. Complete the breath too quickly and the patient will continue breathing past the end of the breath triggering another breath.

The rate of flow can be set to be delivered in a particular pattern. The pattern of change is termed the breath "waveform".

The two most common waveforms are the square wave and descending ramp. In a square wave the flow is given at a constant flow rate until the full volume is delivered. In the descending ramp the flow slows as the breath is administered.

Each waveform has certain benefits. The square wave administers the volume faster. The descending ramp mimics a normal breath where the inspiratory flow slows as the lungs inflate.



Time Cycled

Pressure target breaths can be time cycled; once the set time has elapsed the inhalation ceases. The set time is entered directly (Ti) or in some ventilators is entered as an I:E ratio (with a respiratory rate) to be automatically calculated.

If an I:E ratio is entered the amount of time allowed for the inhalation can be automatically calculated by the ventilator. Assume an I:E ratio of 1:2. If the respiratory rate is 20 breaths/min then 3 seconds are allowed for the total breath time. The I:E ratio of 1:2 then gives us 1 second for inhalation and the remaining 2 seconds for exhalation.

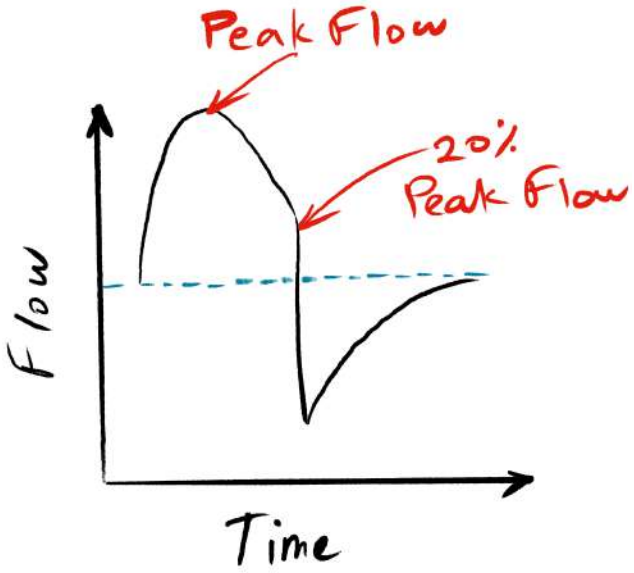
Patient Cycled - Flow Cycled

A patient cycled breath is ended by the patient. Patient cycled breaths are usually "flow cycled". Since a flow cycled breath relies on changes in flow it is set as a property of pressure targeted breaths.

In a flow cycled breath the pressure is usually applied to the ventilator circuit during the patient's inhalation effort. In order to support the inhalation effort the mechanical breath needs to be administered only when the patient is inhaling and needs to stop once the inhalation is over.

How does the ventilator know when the patient wants to stop inhaling? As the patient inhales, inflow increases up to a peak (the peak inspiratory flow), then declines as the chest fills and the breath approaches completion. Once the decline in flow reaches 20% of peak inspiratory flow the breath cycles and the support pressure ceases.

Flow cycled breaths allow for variability in breath time. A patient is allowed to take longer or shorter breaths. This is both comfortable for the patient but also, as we'll see in the section on ventilator dys-synchrony, gives information regarding fatigue.



Trigger

Trigger is what causes the ventilator to start a breath.

Triggers can be patient triggers or machine triggers. Patient triggers are caused by some interaction between the patient and the ventilator while machine triggers are set by the operator.

Common trigger types are:

- Time trigger
- Flow trigger

- Pressure trigger

Machine Trigger Time

The simplest type of trigger is a time trigger or machine trigger; a timer that is set to go off every few seconds. This trigger is used to set the minimum required rate. It is especially needed if the patient is expected to be very sedated or paralyzed. A backup rate with a time trigger is usually set in the case of a patient who may develop an unreliable respiratory drive such as someone who is is severe shock.

Other types of triggers allow for some interaction between the patient the ventilator.

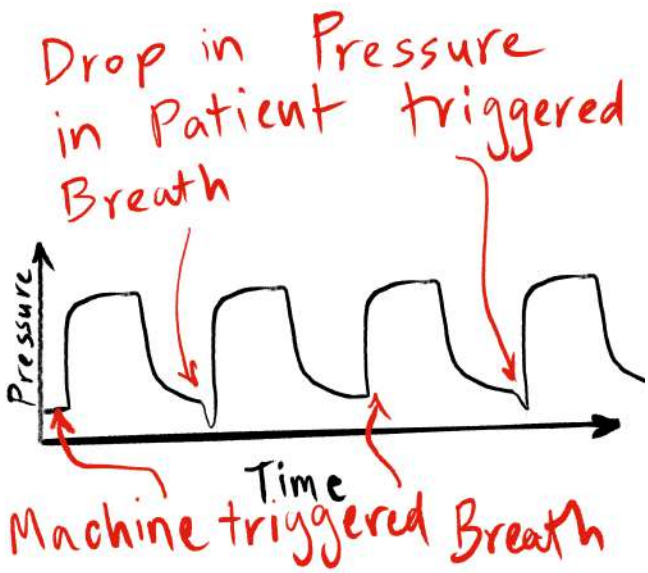
Patient Triggers

Flow and Pressure Triggers

The most commonly used patient triggers are the flow and pressure triggers.

A flow trigger is set off by the patient inhaling gas from the ventilator circuit. The ventilator senses a loss of flow and starts a breath.

A pressure trigger is set off when the ventilator senses that the pressure in the circuit drops.



The sensitivity of the ventilator to these triggers can be set.

There are minor differences between these two triggers. The main differences were present years ago when pressure triggers took longer to activate and so caused patient discomfort and worsened air hunger. Current ventilators respond very quickly to even pressure triggers and the differences are minor as long as the thresholds are set thoughtfully.

NAVI

Less often used, in this method of trigger a special lead inserted as a gastric tube senses diaphragmatic electrical activity to initiate breaths. It sounds interesting but I have little experience with this and so will not discuss further.

Breath Types

The described properties of breath types can be put together in different combinations. Particular combinations of the above characteristics can be grouped in to breath types.

Mandatory Breaths and Spontaneous Breaths

We classify mechanical breaths as either mandatory or spontaneous depending on how much control the ventilator or patient has. This classification is important as we discuss modes.

A spontaneous breath is one in which the patient has control of both the trigger and of the time of the breath; It is a breath that is both patient triggered and patient cycled (usually flow cycled).

A mandatory breath has at least one property controlled by the ventilator. It is machine triggered and/or machine cycled. A breath that is time cycled but patient triggered is still a mandatory breath. A breath that is patient cycled but machine triggered would

also be classified a mandatory breath. The machine controls some aspect of this breath.

The terms assist and control are common in ventilator parlance and refer to types of mandatory breaths. An assist breath is simply a mandatory breath that is initiated by the patient. A control breath is initiated by the ventilator.

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Modes

A mode is the set of rules that the ventilator uses to determine when and what type of breath to give the patient.

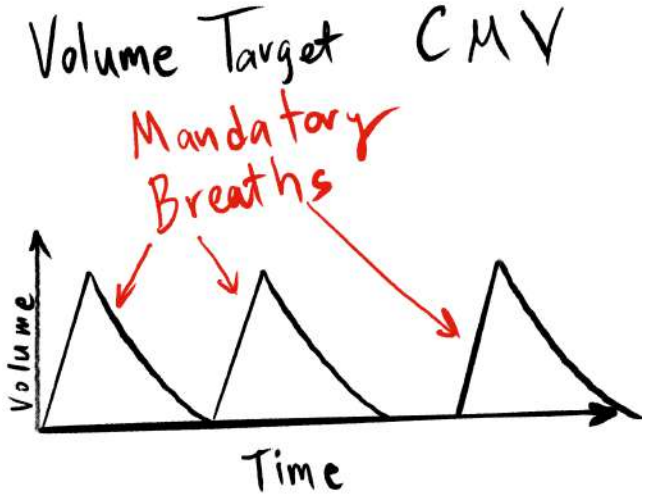
The different modes are simply just mixes and matches of breaths with different characteristics and type.

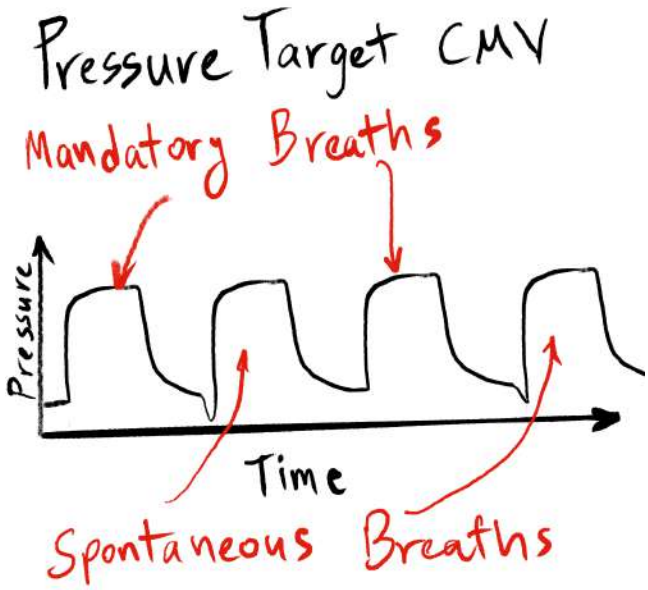
Continuous Mandatory Ventilation (CMV)

This mode consists only of mandatory breaths. A time trigger is set but a patient trigger is also present so that the patient can trigger supplemental breaths. The breaths may be any target (pressure, volume, dual) but they are always machine cycled. The patient can trigger breaths but the breaths given are always mandatory breaths and never spontaneous.

This mode is also known as Assist-Control Mode since it gives both types of mandatory breaths - assist breaths and control breaths. This is in contrast to an archaic mode that is no longer present in modern ventilators in which only control breaths could be given.

When the CMV mode is used with volume targeted breaths you will hear it called “Volume Control” mode. When used with pressure targeted breath you may hear it called “Pressure Control” mode. These are all the same CMV mode. (Pulmonologists like to make things complicated - just ask one to talk about interstitial lung diseases and prepare for a nap.)





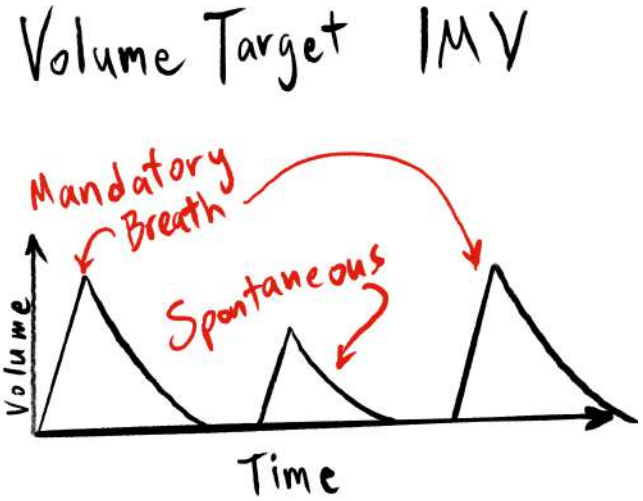
Intermittent Mandatory Ventilation (IMV)

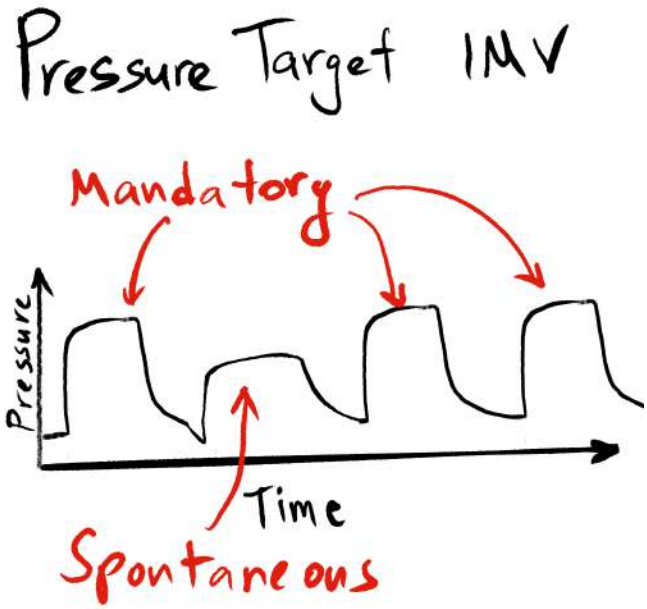
This mode is similar to CMV mode in that both machine and patient triggered breaths are administered. The difference is that the patient triggered breaths are not mandatory as in CMV mode. They are either unsupported or spontaneous breaths (patient triggered, patient cycled).

Sometimes this mode is referred to as SIMV (synchronized IMV) since the two breath types are synchronized so as not to overlap. If it is almost time for a mandatory breath, then even if the patient triggers a breath, he will be given a mandatory breath.

IMV or SIMV was used for weaning in the past. The machine set rate would gradually be decreased until the patient was only receiving spontaneous breaths. This proved to prolong the process of removing the patient from the ventilator and so is now discouraged in ICUs. In long term care facilities some slower weaning protocols still utilize this mode.

SIMV is also used in particular settings such as neurologic ICUs were strange breathing patterns are relatively common and patients can vacillate between rapid deep breaths and apnea at erratic. This mode can help guarantee some minute ventilation without the excessive dyssynchrony caused by the periods of rapid deep breathing.

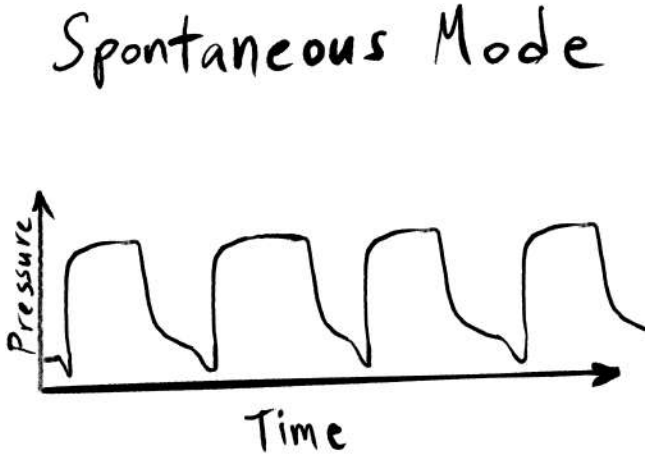




Continuous Spontaneous Ventilation (CSV)

This mode consists of only spontaneous breaths. There are never mandatory breaths given. When the breaths are pressure target then it is referred to as pressure support mode (PSV) and is a popular wean mode.

This mode allows the patient to set their own rate and their own minute ventilation. It is very comfortable for the patient when the patient can reliably breath and it is safe to use.



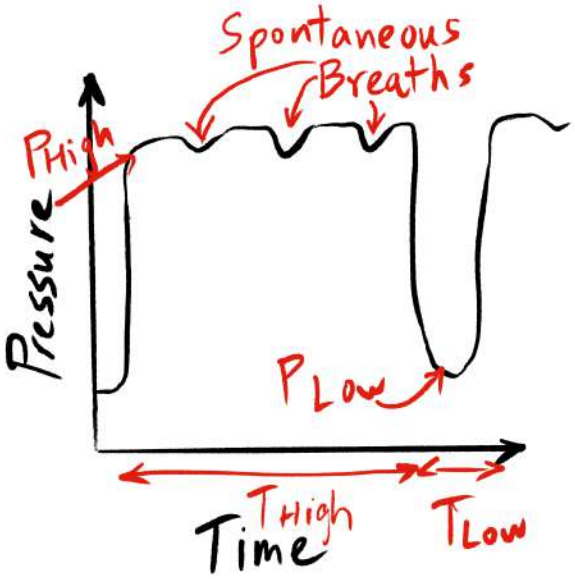
Newer Modes and Methods

There is are many modes of mechanical ventilation that are not used on the usual patient with many certainly on their way. With a good understanding of the fundamentals of mechanical ventilation these modes can be understood without much difficulty.

APRV Airway Pressure Release Ventilation

This mode is a continuous spontaneous ventilatory mode - CSV with a bit of a twist. The PEEP alternates between a high level with brief periods of low PEEP.

Airway Pressure Release Ventilation (APRV) is continuous spontaneous ventilation with a high PEEP and a low PEEP set. The high PEEP allows for alveolar recruitment (see chapter on hypoxia) and the low PEEP allows for ventilation. During any of these periods the patient is allowed to breath spontaneously which is supposed to improve patient comfort.



Oxygenation Parameters

The oxygenation parameters describe the settings that are manipulated in order to change the concentration of oxygen in the ventilator's gas mix or to increase the PEEP. These will be elaborated further in the section on initial settings.

The fraction of inspired oxygen is set by choosing the percentage of the gas mix that will be oxygen. It can usually vary from 30% to 100%.

The PEEP is the pressure maintained in the ventilator circuit between breaths.

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Exhalation

The mechanical ventilators commonly used in the ICU do not augment exhalation. Exhalation is up to the patient and exhalation is important to understand. Please review the section about exhalation in "Function of the Respiratory System".

Physical limits can be reached during mechanical ventilation and are usually related to the exhalation phase. In some common situations this limitation poses the biggest risks to the patient.

AutoPEEP

The main cause of ventilatory limitations is auto-PEEP. This happens when the time given for exhalation is insufficient and a patient receives a breath before the previous breath has been fully exhaled.

More and more gas accumulates with each breath in the patient with autoPEEP. Each breath adds to the lung volume over FRC. This higher volume increases the expiratory forces and stretch open airways until eventually the exhalation time decreases to match the time available.

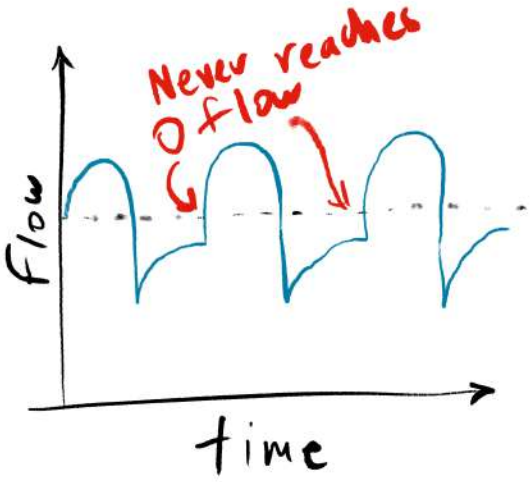
Any patient can develop autoPEEP if mechanical breaths are given fast enough. All mechanically ventilated patients with COPD experience some degree of autoPEEP.[4] In patients with airflow obstruction autoPEEP occurs quickly and at relatively slow respiratory rates.

While a little autoPEEP can be tolerated, the amount of pressure that can build up in the chest can be significant. The pressure can build up enough to cause discomfort, rupture lung, and even cause obstructive shock and death.

AutoPEEP can easily kill patients if it isn't considered, particularly when ventilating patients with airflow limitations.

Detecting and Measuring AutoPEEP on the Ventilator

AutoPEEP's serious consequences make it important to detect. There are several ways of detecting autoPEEP. Simply looking and listening to the patient: If you hear air exiting the patient at the point when a new breath starts then you've detected autoPEEP. The ventilator graphics can give you a clue. If the flow graph has not returned to the baseline, this may indicate autoPEEP.



The End-Expiratory Pause

At the end of the time allowed for exhalation (just before a new breath is given) the pressure inside an autoPEEPing patient's chest remains higher than circuit pressure. This residual pressure left inside the patient's chest is the intrinsic PEEP and can be measured. Under normal circumstances without autoPEEP the pressure inside the patient's chest would be the same as the circuit pressure and no flow would occur.

The ventilator's pressure sensor measures the pressure in the ventilator circuit. We cannot detect autoPEEP by looking at it since it is not inside the patient where the intrinsic PEEP builds up.

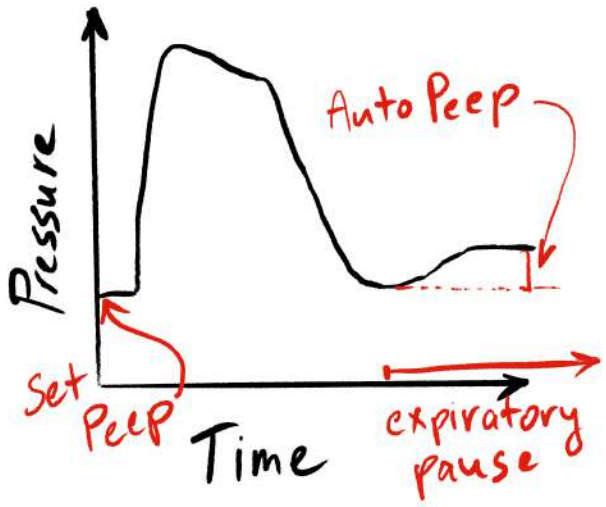
The level at which the autoPEEP occurs is beyond the reach of any pressure sensor practically in use. It occurs beyond the level of expiratory resistance which is usually the smaller airways. How do we measure pressure in an inaccessible space?

Imagine a balloon deflating through a straw. How would we measure the pressure inside the balloon without placing a sensor inside the balloon? If flow is allowed to continue through the straw, any pressure measurements would always be somewhere between atmospheric pressure and the balloon pressure. The one way to get an accurate balloon pressure would be to pause the flow, allow the balloon to pressurize the straw, and measure the straw pressure.

In order to detect the pressure inside the patient the flow will need to be stopped and the pressure across the tubing will need to equalize.

The principle used to measure end-expiratory flow is important. It is used to measure the pressure inside any inaccessible structure. It will be revisited when we talk about plateau pressure. It will be seen when you discuss Swan Ganz catheters. It would be a good idea to take a few minutes to try and understand it.

The procedure to measure the pressure inside the patient at the end of expiration (the intrinsic PEEP) is called an end-expiratory pause. The flow out of the circuit is halted at the time when a new breath is about to begin. The pressure is allowed to reach equilibrium and is then measured. That measured pressure is the intrinsic PEEP or autoPEEP.



Managing AutoPEEP

If the patient is in shock due to autoPEEP then disconnect the ventilator and allow the patient to decompress. This will improve the patients hemodynamics but only buys a short amount of time since when you re-connect the patient to the ventilator the issue will recur. A more durable solution is needed.

The key to managing autoPEEP is remembering that the phenomenon is due to a mismatch between the time available for exhalation and the time required for

exhalation. Management will involve either increasing the time available for exhalation or decreasing the time required to exhale.

Increase time available for exhalation - Change flow rates

Increasing inspiratory flow rates will shorten the inhalation time - either increasing the maximum inspiratory flow, changing to a more rapid waveform such as a square wave, or in case of a pressure target breath simply changing the Ti.

The amount of time saved by this method is usually small. Patient intrinsic breath rate usually increases as flow rate increases[5]. Furthermore, the increase in flow rate causes a rise in the inspiratory pressures - due to the rise in dynamic pressure. An increase to a high flow rate of 100 from the default of 60/70 only gives a fraction of a second more even in cases were the patient is paralyzed and cannot increase the respiratory rate. This method of correcting autoPEEP can be tried but is generally not very effective.

Decrease respiratory rate

Decreasing the respiratory rate is another means of allowing more exhalation time. This is easier said than done.

Patients who are breath stacking are generally agitated and uncomfortable and have a high respiratory drive. Sedation/analgesia will help in this situation. Paralytics can be used acutely once the patient is very sedated if control of the rate cannot be achieved and the patient is in extremis. Paralytics should only be used as a last line maneuver and very sparingly until the process which is causing the autoPEEP can be reversed. Never use paralytics without excellent sedation. Decrease time needed for exhalation Decrease the tidal volume administered Decreasing the tidal volume is one way of doing this. If the volume administered is smaller, the time to exhale that gas will be shorter.

Decrease Tidal Volume

A smaller tidal volume means that there is less gas to exhale. This has to be balanced with the required minute ventilation for acid-base status.

Decrease Airway Resistance

If the resistance to airflow is at the level of the larger airways placing a larger endotracheal tube or suctioning secretions may be a solution. If the resistance is at the level of the bronchioles, as it usually is with COPD and asthma, then reversing any underlying bronchospasm is a solution. This can be achieved with bronchodilators and steroids.

Ventilator Settings for Exhalation

Exhalation, in many ways, poses more of a problem in mechanical ventilation than inhalation. We have little control of a patient's exhalation and so ventilator parameters need to be set carefully. These characteristics can change and so ventilator parameters need to be fluid and reactive.

Ventilator settings such as rate need to take into account the expiratory time. The available expiratory time is easy to calculate. For example, a breath rate of 10/min will allow for 6 seconds of total breath time. If 2 seconds are taken to administer the breath then 4 seconds are available for exhalation.

Exhalation may not require all the time available. If it requires more time then breath stacking will occur. Mild breath stacking is usually tolerated and acts simply as extra PEEP. Significant breath stacking, however, as discussed above can be very detrimental.

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Ideally the mechanical ventilator would give a patient a breath whenever requested and that breath would be as deep and as long as the patient wants. In order to achieve this state of patient ventilator "synchrony" the mechanical ventilator must initiate a breath immediately upon patient request, the breath must flow at a rate acceptable to he patient, and must cease when the patient stops the inhalation.

Dyssynchrony occurs when the above ideal situation does not take place and the patient either cannot initiate a mechanical breath or gets a breath that is too slow, too short, or too long.

Dyssynchrony is harmful. It can increase mortality.[6] It causes patient agitation and discomfort thereby significantly increasing sedation needs. It generates undue stresses on the lungs when the ventilator is triggered against an exhalation or when a double breath is initiated. It leads to prolonged mechanical ventilation and is a miserable state.

Fortunately, understanding ventilator dyssynchrony can help in its management.

The mismatch between the patient's intrinsic respiratory drive and the ventilator settings causes dyssnychrony. This is occasionally unavoidable in our attempts to protect the lung from excessive volumes or pressure. In many situations adjusting our ventilator settings can better match the patient and improve the situation.

Forms of Dyssnychrony

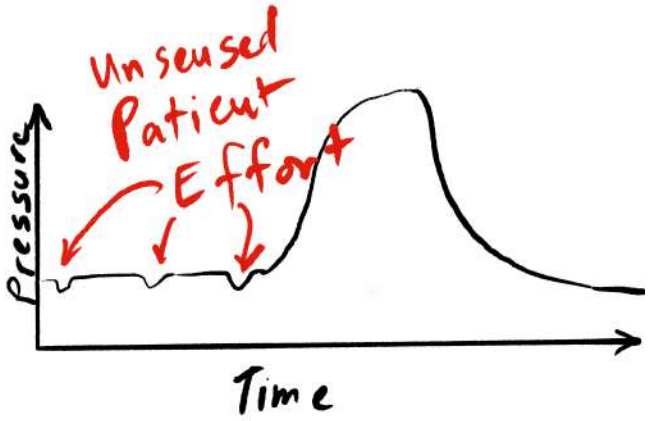
There are several types of ventilator dyssynchrony: failure to trigger, flow dyssynchrony, time dyssynchrony, and autotrigger.

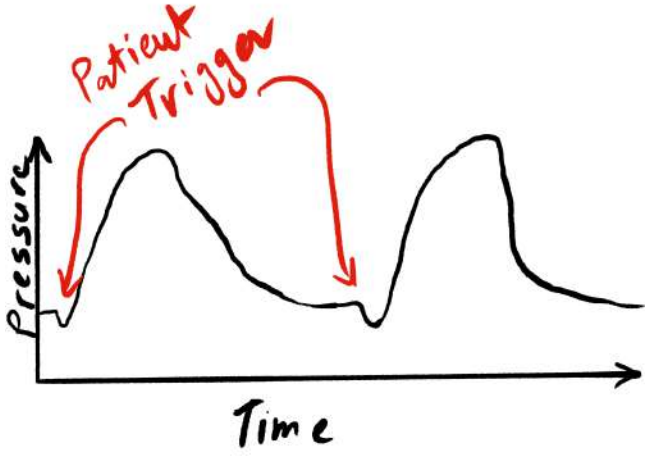
Dyssynchrony	**Type of mismatch**
Failure to Trigger	Patient unable to trigger ventilator
Flow dyssynchrony	Unable to get enough flow.
Time dyssynchrony	Breath too short or too long.
AutoTrigger	Ventilator administers unwanted breaths.

Failure to Trigger

Failure to trigger is a common form of ventilator dyssynchrony. It occurs when the patient is unable to trigger or needs to exert excessive efforts to trigger the mechanical ventilator. This is a very uncomfortable state.

Most failure to trigger is not due to neuromuscular weakness but rather to autoPEEP. The development of an intrinsic PEEP will generate an expiratory flow that the patient needs to overcome in order to be able to generate the negative flows that trigger the ventilator. The hyper inflated chest is also at a mechanical disadvantage. The worse the autoPEEP and the more the hyperinflation, the more difficult it is to trigger the ventilator.





The management of failure to trigger due to autoPEEP is the same as that for autoPEEP. It mainly focuses on increasing the time available for exhalation. This entails treating any obstructive process that is slowing exhalation, decreasing the tidal volume or respiratory rate, and possibly increasing the flow rate if the patient is getting breath types that allow this.

Increasing the set PEEP can help improve the ventilator's ability to sense the patient's respiratory efforts. An increase in set PEEP will decrease the amount of pressure that the patient is required to overcome since the difference between the extrinsic and intrinsic PEEP shrinks. An increase in extrinsic PEEP does not affect intrinsic PEEP much as long as it remains substantially less. If the PEEP is increased too much then it will worsen autoPEEP as it poses an additional pressure inhibiting gas outflow.

Any increase in PEEP has to be done carefully and slowly with continuous observation of the patient's response. In general the set PEEP can be around 3/4 of the estimated intrinsic PEEP.

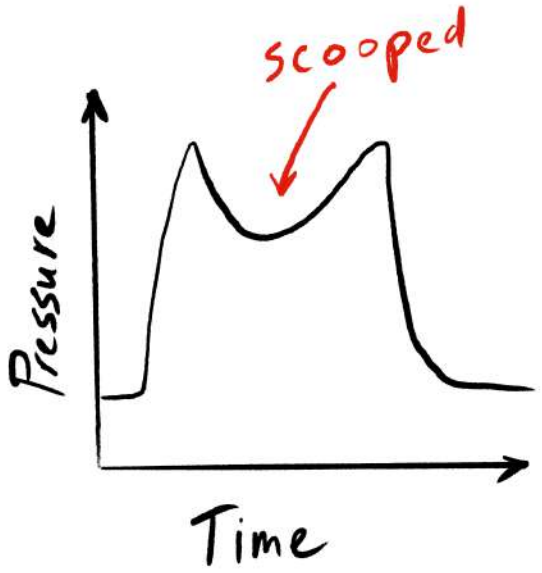
Flow Dyssynchrony

Flow dyssnychrony occurs when the ventilator's flow rate does not match the patient's flow requirement. It is present only in flow limited breath types (ie. volume targeted breaths) which limit flow rates to what is dictated by the setting and flow pattern.

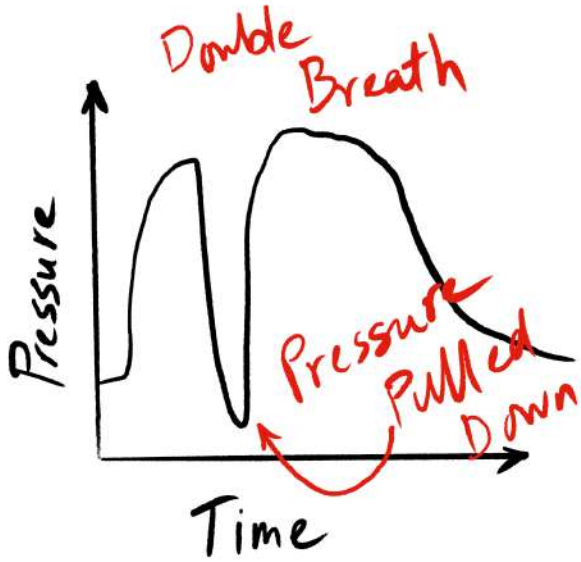
Flow dyssynchrony, when severe, causes double triggering in which the ventilator administers a second breath without any period of exhalation. This exposes the lungs to double the prescribed tidal volume. Even when a double breath is not administered the effort at ventilation that the patient makes pulls at lung tissue. The lung tissue that gets pulled by the diaphragm is disproportionally the basilar tissue which locally over-distends[7] and due to its atelectasis and disease does not transmit its stress to other portions of lung. The negative pressures generated in the lung with vigorous inhalations also cause high transmural pressures on the vasculature which can leak and can cause pulmonary edema.[8]

Patients with elevated respiratory drives are most likely to become flow dyssynchronous. Pain, agitation, acidemia, and anemia all contribute to a high respiratory drive and can make a patient more likely to become flow dyssynchronous.

In flow dyssynchrony the patient pulls gas out of the circuit faster than the ventilator is supplying it. This results in a drop in circuit pressure. If this drop in pressure and flow leaving from the circuit is severe enough it can trigger another breath. Flow dyssynchrony is uncomfortable and causes patient agitation and worsens work of breathing as the patient pulls flow from a limiting source (think of how uncomfortable it is to breath through a stuffy nose).



The remedy to flow dyssynchrony needs to come from both the patient and from the adjustment of the ventilator.



On the patient side the cause of the high respiratory drive should be managed. The process generating the acidemia should be treated. If the patient is in pain or anxious a dose of either an anxiolytic or analgesic should be administered. Fever, anemia, and any other possible pathologies should be addressed.

There are several changes that can be made on the ventilator side of the equation.

The flow rate can be increased if the breath type allows this. This will result in a faster flow rate but at the expense of duration of breath. This can become counterproductive if the ventilator then cycles before the patient is done inhaling giving a double breath. The flow pattern can be altered to see if there is one that better matches the patient.

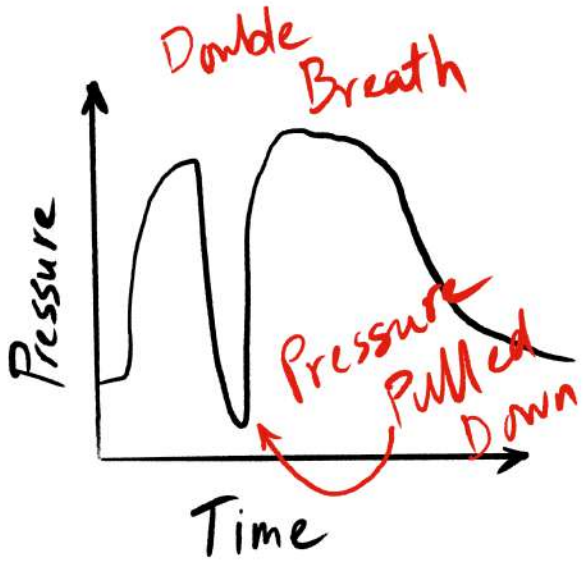
Another remedy to flow dyssynchrony is to switch to a mode that doesn't limit flow rates such as a pressure controlled breath. This, however, can have a significant downside if the patient begins to take excessively large breaths which risk traumatizing lung. It might be appealing to change to a dual target breath type but in this breath type the ventilator will treat an air hungry patient with progressively decreasing support pressures to maintain the low tidal volume and so increase work of breathing.

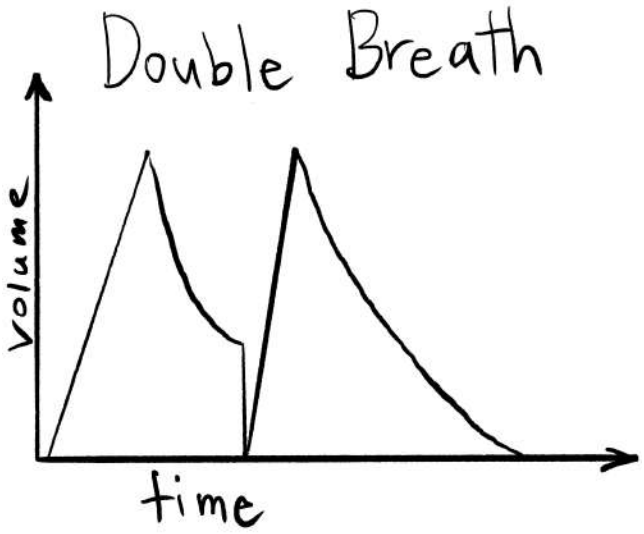
In cases of significant flow dyssynchrony and double breaths a more liberal set volume may be acceptable - at least until the process causing the air hunger is controlled.

Time Dyssynchrony

Time dyssynchrony is due to a mechanical breath time that is longer or shorter than what the patient's intrinsic drive requests. If the mechanical breath time is too short then the patient will continue inhalation even after the mechanical breath is complete. This can trigger another breath before exhalation has occurred causing a "double breath" or "double trigger". If the breath is too long then the patient may begin exhalation while the machine breath is still ongoing. This causes high airway pressures.

Ventilator graphics allow us to easily detect time dyssynchrony. If the mechanical breath time is set too short the flow time curve's flow tracing may not reverse or only reverse partially when the inhalation is complete. The pressure may drop below baseline in the pressure time curve. A double breath will occasionally be seen. If a breath time is set too long the pressure at the end of the breath will spike as the patient begins to resist further inflation.





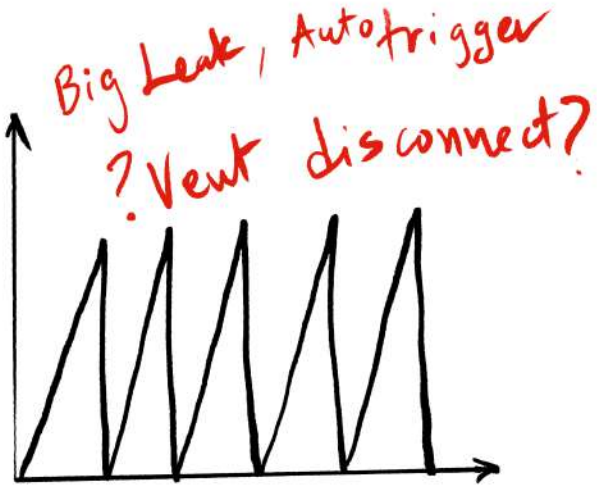
The remedy to time dyssynchrony is to try to match the breath duration with what the patient is requesting. In a volume targeted breath this entails adjusting the flow rates or manipulating the flow pattern. In a pressure targeted control mode this would entail adjusting the Ti. The easiest way to synchronize a patient who is very time dyssynchronous may be to switch to a flow cycled mode such as spontaneous pressure support mode. In the spontaneous pressure support mode the patient sets the breath duration.

Air hunger is one reason for a patient to request long inhalation times. Air hunger will cause patients to request long deep breaths. Any reversible pathologies leading to air hunger such as acidosis, fever, and pain should be addressed.

Autotrigger

Autotrigger occurs when the mechanical senses the need for a breath without the patient requesting a breath. The trigger mechanism in the modern ventilator is very sensitive. In order to keep the patient comfortable and minimize work of breathing minute alterations in the flow or pressure in a circuit are meant to quickly trigger a breath. This makes the ventilator sensitive to other causes events that can alter circuit flows. This can be due to non-breath effects such as hiccups, tremors or even from the vigorous pounding of a heart. It can occur due to leakage from the ventilator circuit in

which there is a loss of pressure or flow from the circuit.

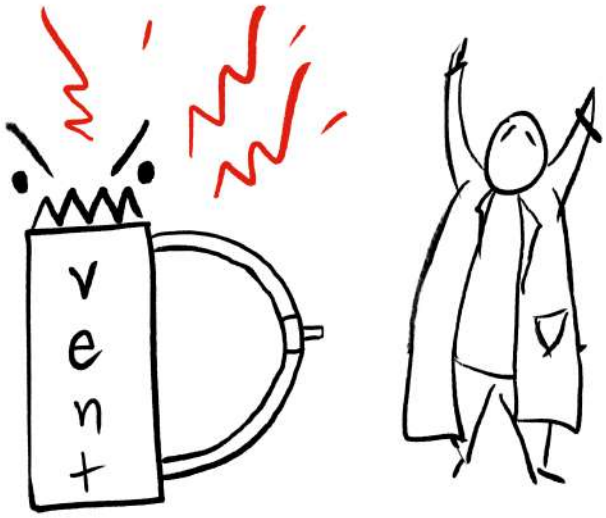


Autotrigger can result in hyperventilation and respiratory alkalosis. Respiratory alkalosis can cause a variety of problems including spasms, seizures, and arrhythmias.

Autotrigger can be detected when the patient is being ventilated above a set rate without any visible respiratory effort. A respiratory alkalosis while on mechanical ventilation should always prompt an evaluation for respiratory alkalosis.

Alarms and Troubleshooting

Ventilator alarms are a common cause of anxiety for both family members as well as those caring for the patients. This section will review some common alarms and how to troubleshoot them.



If the patient is deteriorating or you cannot easily remedy the cause of the alarm then the best thing to do is to disconnect the patient from the ventilator and bag.

- Peak pressure or High Inspiratory Pressure
- Leak alarm
- Low pressure
- Patient disconnect
- Low tidal volume
- Low minute ventilation
- High minute ventilation

- Apnea
- High exhaled tidal volume
- Low exhaled tidal volume

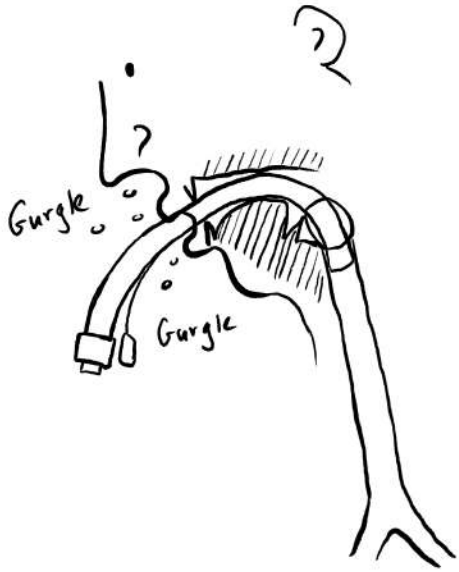
Peak Pressure or High Inspiratory Pressure

The ventilator is sensing high pressures in the circuit. Anything that pushes gas into the circuit or impedes the outflow of gas can trigger this alarm. It is commonly caused by the patient coughing into the circuit. It can also (and more importantly) be caused by a rise in either the dynamic or static pressure measured in the circuit. These causes need to be investigated and remedied.

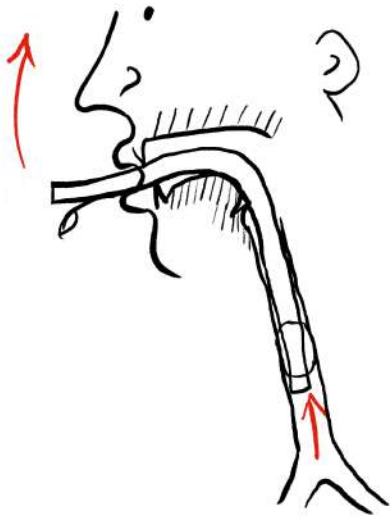
Assess for obvious kinks in the tubing or for the patient biting the tubing. Attempt to pass a suction catheter into the trachea and suction briefly to assess for obstruction and suction any significant phlegm. Listen to lungs to ensure that there is symmetric air entry. Order a chest x-ray or ask for the bedside US to check to assess for pneumothoraces. A pneumothorax in a mechanically ventilated patient can become a tension pneumothorax and may need to be treated emergently. A plateau pressure can be checked to tease out whether the high pressure is due to high resistance to flow, such as kinked obstructed tubing, or due to loss of compliance in the chest.

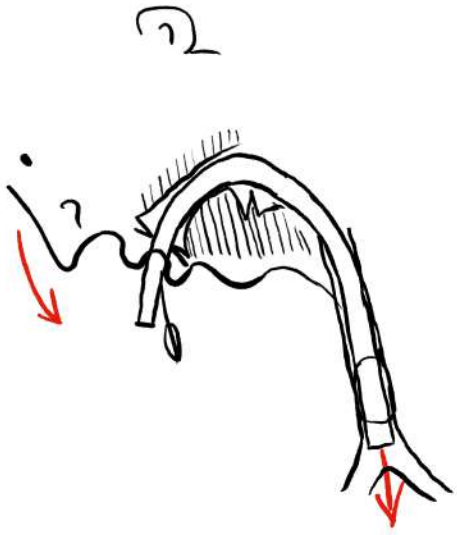
Leak Alarm

The ventilator is sensing a loss of gas from the circuit. The ventilator measures flow into the circuit and back out. If there is a mismatch then flow has been lost somewhere along the circuit. This is usually due to a leak or a disconnect. Check the circuit for leaks. These can be heard or felt. If there is bubbling or gurgling from the patients mouth be sure that the endotracheal tube has not migrated out above the vocal cords or is malfunctioning with a torn cuff.



If the endotracheal tube has dislodged then it is an emergency and the patient has an unstable airway. This can happen if the endotracheal tube has migrated out of the the trachea by being accidentally pulled or due to changes in patient position. The ETT does move with patient head position - looking upwards pulls the ETT out, looking down pushes it in. (The ETT follows the nose). Look at x-rays done to ensure the ETT is 2-4 above carina, at or below the level of the mid clavicles. If the ETT has been dislodged then call for help since the patient may many need to be re-intubated.





A leak from the ventilator circuit can be seen on the graphics. The ventilator will measure the amount of gas administered into the circuit and the amount of gas coming back. If the amount of gas being pushed into the circuit is less than what comes back this can be visible on a volume-time curve as a step in the exhalation portion. Frequently the leak will cause auto triggering as in the cartoon below.

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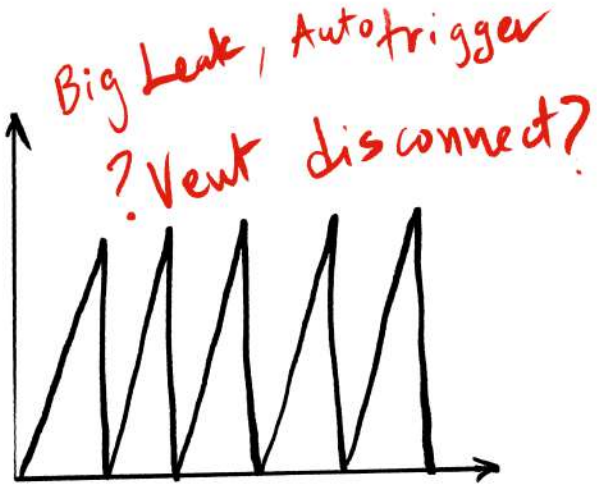
Low Pressure Alarm

This is a more extreme form of the leak alarm with the ventilator sensing a low pressure in the circuit. It generally stems from a larger leak but can occur if the patient's inhalations are powerful enough to drop the circuit pressures during the exhalation phase. The same troubleshooting as for the leak alarm should take place.

Patient Disconnect Alarm

An extreme form of the low pressure alarm. The ventilator has sensed a significant decrease in resistance to flow and an extreme persistent drop in circuit pressure. Generally indicates that the patient has been disconnected from the ventilator or has become extubated.



Low Tidal Volume Alarm, Low Minute Ventilation Alarm

The ventilator senses the flow at the exhalation port and measures the returned volume. If the returned volume is below a particular value then these alarms are triggered. The low tidal volume alarm will sound when particular breaths return too

little volume. The low minute ventilation alarm will sound when the aggregate returned volume estimated over a minute is below the set alarm value.

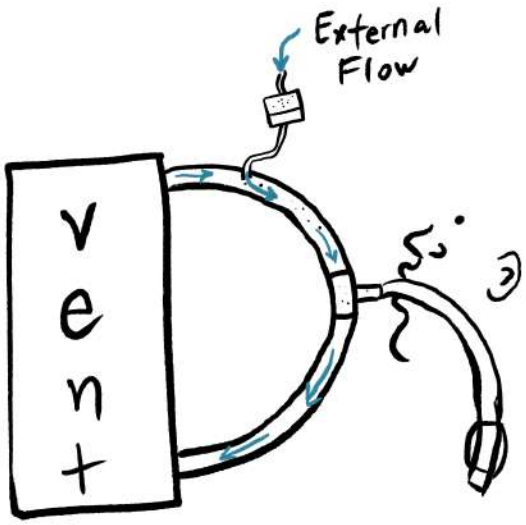
The reason for low tidal volume and low minute ventilation alarms needs to be investigated. If the patient is being ventilated with a pressure targeted breath then that patient may be receiving too little support. If on a wean trial this may indicate patient fatigue. If on a mode that is using volume targeted breaths then this is even more ominous and may indicate that the ventilator is cutting off breaths due to a concomitant high pressure alarm.

High Minute Ventilation Alarm

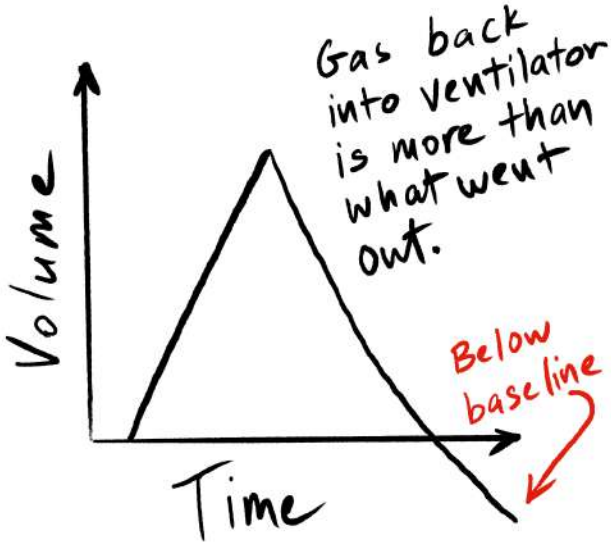
The ventilator is sensing that the minute ventilation measured is above the set alarm value. This may be due to the patient becoming tachypneic due to pain, anxiety, or a developing metabolic acidosis. Another possible cause is auto-cycling of the ventilator and this phenomenon is discussed in the section on ventilator dyssynchrony.

High Exhaled Tidal Volume Alarm

The ventilator senses a large volume at the exhalation port. This may be due to a patient exhaling a large amount of volume due to a prior stacked breath but may also be due to gas flow being bled into the ventilator circuit. Check for flow into the circuit from an extraneous source such as a forgotten nebulizer.



The high exhaled tidal volume can be seen on a volume-time graph with the return gas being more than the administered flow.



Apnea Alarm

Sounds when the patient does not trigger a breath after a particular set amount of time. Generally sounded when the patient is on a spontaneous mode such as during a wean trial.

The reason for the apnea needs to be elucidated. There are several reasons for apnea including over-ventilation while on the previously chosen mode, over-sedation, and catastrophic neurologic event. These things need to be investigated and, if possible, treated.

Chapter Four

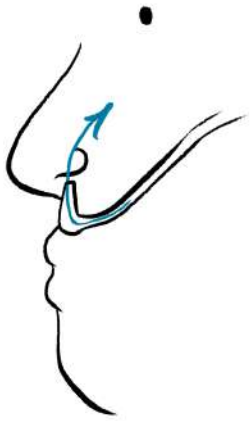
Fixing Failure

Correction of Hypoxemia

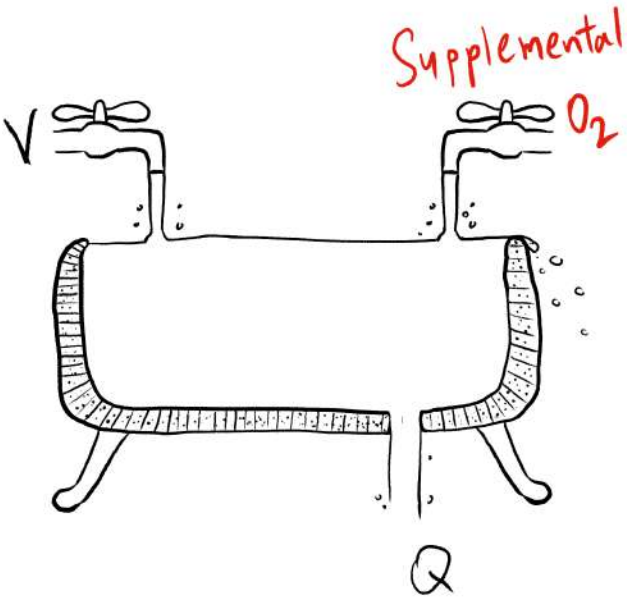
Hypoxemia is very poorly tolerated and is an emergency. Fortunately, hypoxemia is usually easy to correct and several options are available. In this section we'll go over a few methods of supplementing oxygen before we discuss correction with mechanical ventilation.

Passive Oxygen Supplementation

Adding oxygen to inspired air will improve the oxygen pressure in even poorly ventilated alveoli thereby correcting VQ mismatch.



Supplemental oxygen will also improve oxygenation issues in alveoli with diffusion problems as the higher oxygen level in the alveolar gas generates a steeper diffusion gradient.



Shunt hypoxemia will not improve with supplemental oxygen. Segments of lung that have shunt physiology are not ventilated at all. Blood perfusing shunt segments will not interact with the supplemental oxygen.

Oxygen can be delivered through several "non-invasive" devices.[9]

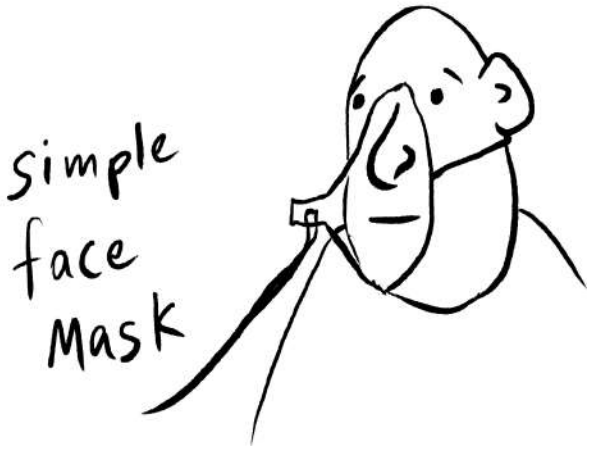
Limitations in oxygen delivery through these devices comes because room air is entrained while the patient breathes and this dilutes the oxygen mixture.

The amount of air entrained will depend on the flow rate of the patients inhalation as well as the respiratory rate. This will vary throughout the day with activity, fever, eating… This change in the amount of entrained room air can significantly change the amount of oxygen delivered.

Nasal cannulae are the most common and least obtrusive. A tube with prongs to fit into the patient's nose. Flow from the wall is generally limited to 5 L/min for regular cannulae to 10L/minute with the high flow cannulae. The concentration of oxygen delivered to the patient is variable and depends on the amount of ambient air entrained during the high flow phase of inspiration.

A benefit to nasal cannulae is that the gas can be humidified. In fact if a patient is on nasal cannula at higher than 1-2 L/minute then that oxygen should be humidified for comfort. One of the more frustrating aspects of medicine is trying to convince a patient (typically an elderly man) to put on their nasal cannula once they have decided they don't need it and that it is irritating their nose. (…But you're turning blue Mr. Smith…)

Face masks are a more obtrusive method of delivering oxygen. The benefits of these devices is that they entrain less air if the patient's mouth is opened. This improves oxygenation and decreases the frequency of the ever annoying and unhelpful "breath through your nose!" emitted by concerned staff and family members.



The oxygen concentration in a face mask can be controlled using a venturi device. This device functions using the Bernoulli principle - flowing gas exerts less pressure than still gas. The flowing oxygen from the wall suctions air from the room and mixes with it. The

amount of air suctioned depends on the flow rate of oxygen and the size of the opening in the device. Mixtures can be set from 30% all the way to 90%. The amount the patient actually receives still dependent on the patient's flow requirement as room air dilutes whatever oxygen mix the mask delivers.

With very high oxygen requirements the need to deliver more oxygen mandates that a decrease in the amount of entrained air. There are several methods to do this non-invasively. The reservoir devices and the very high flow devices.

Reservoir devices include a bag or container that will administer a volume of gas upon initial inhalation. The rapid inspiration will pull gas out of this bag instead of pulling room air and helps decrease the amount of entrained room air that is inhaled.

There are reservoir devices in the form of nasal cannula - Oxymizer is one company making these and has ones with the reservoir immediately below the prongs or hanging as a pendant. The face mask reservoir device is typically called a non-rebreather mask. The reservoir is typically attached to the mask. One big limitation of these devices is that the reservoir precludes humidification of the gas since the humidity rains out into the reservoir container. This makes these devices more likely to dry out secretions and be uncomfortable. The non-rebreather is especially drying and should only be used for brief periods of time - such as during transport.

The high flow systems are another method of dealing with the entrainment of outside air. The high flow systems can deliver flows up to 40-50L/minute and come close to exceeding the flow requirement of the patient. The high flow delivery devices can be through a mask or nasal cannula and is humidified.

Passive supplemental oxygen can only go so far to oxygenate a patient and in some patients further support is needed.

Mechanical Ventilation

Mechanical ventilation is the inflation of the lungs with a gas pressure. This is termed "positive pressure ventilation" in that breaths are formed with the application of gas pressure. A more detailed explanation of mechanical ventilation will take place in the section on mechanical ventilation.

Mechanical ventilation offers several mechanisms which help improve a patient's saturation.

Mechanisms through which mechanical ventilation improves oxygenation

- Delivery of oxygen
- Recruitment of lung tissue
- Decreased oxygen utilization
- Improve Hypoventilation

Improved Delivery of Oxygen

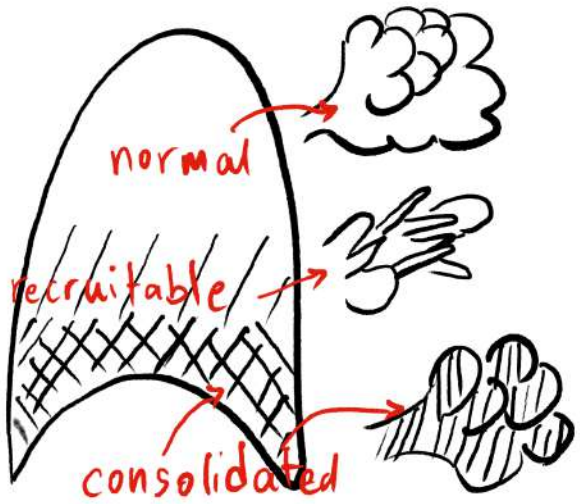
There can be no entrainment of ambient air with mechanical ventilation since any interface between the ventilator and patient is sealed. The patient is guaranteed to receive the prescribed concentration of oxygen.

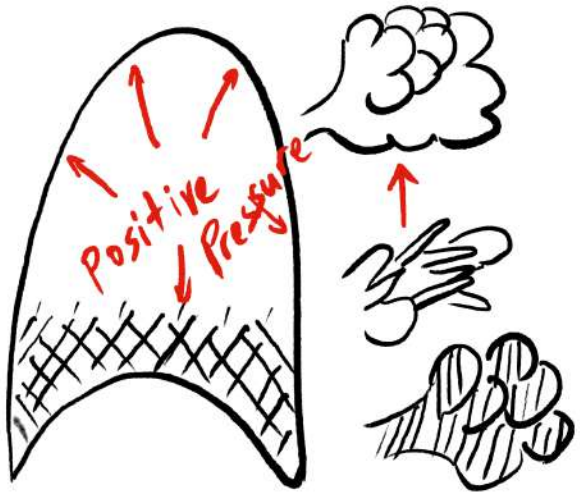
Recruitment

Diseased lungs will have segments which are not full of pus and debris but are collapsed (atelectasis). This collapse can occur due to loss of tethering of small airways in low lung volumes and under external pressures (belly, chest wall), due to loss of surfactant, or due to a high oxygen concentration in the inspired gas (resorption atelectasis).

The collapsed segments act as shunts and do not participate in gas exchange. Shunt worsens the patients oxygenation.

Recruitment is the inflation of the collapsed segments. The mechanical ventilator recruits collapsed segments through the application of airway pressure which stents them open. Once recruited these segments can participate in gas exchange and no longer function as shunts.





We call the pressure that pops open an atelectatic lung segment its "opening pressure". Once segments are popped open we must keep them open.

PEEP

Positive End Expiratory Pressure is airway pressure maintained between breaths. This functions to prop open alveoli between breaths and can even. The PEEP is chosen to maintain lung segments open between breaths, and prevent the segments from cyclically opening and shutting which can cause atelectotrauma.

Increasing PEEP, however, will prop open and recruit alveoli up to a point. Too much PEEP will only function to apply pressure to the lung and tend to over distend lung. See the section on Initiation and Adjustment of the mechanical ventilator for PEEP titration recommendations.

Diminished Oxygen Utilization

Oxygen utilization is another factor that can affect a patient's oxygen saturation. High oxygen utilization due to activity and metabolism decreases a patient's mixed venous oxygen saturation. This drop in mixed venous oxygen saturation will magnify the effect of shunts and low VQ areas as pulmonary artery blood starts of with a lower saturation.

The respiratory system consumes large amounts of energy and oxygen. This is especially true when the lungs are diseased, stiff, and hard to ventilate.

A mechanical ventilator functioning in appropriately allows the unloading of respiratory muscles and decreases the patient's oxygen needs.

An agitated patient in distress is also using a lot of oxygen. Mechanical ventilation through an endotracheal tube allows for the safe deep sedation and even paralysis of patients. This further decreases oxygen usage.

Correction of Hypoventilation

As a patient fatigues their ability to ventilate diminishes. Insufficient ventilation will aggravate hypoxemia. Mechanical ventilation can guarantee ventilation despite the patient's fatigue and takes effort and ability out of the equation with the only limitation to ventilation being the mechanical properties of the lung. This ability to hyperventilate thus improves oxygenation.

More Oxygen More Better?

Oxygen is a caustic gas that is highly reactive and places exposed tissues to oxidative stress. The lung can exhibit pathology and is injured at high oxygen doses.[10] It is very well absorbed into blood and can cause "absorption atelectasis" when it is pulled out of alveoli too rapidly and not enough nitrogen is present to keep them propped open.

In non-invasive oxygen supplementation, a more conservative approach to oxygenation - keeping the saturation between 94-98% vs higher improved patient mortality[11]. This conservative approach has yet to show any benefiting in mechanically ventilated patients[12].

Keep the dose of oxygen as low as possible while keeping the oxygen saturation around 94-98% even in mechanically ventilated patients.

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Correction of Hypercapnia

Hypercapnic respiratory failure is due a mismatch between the patient's minute ventilation and the minute ventilation they require to clear CO2.

Unlike oxygen which can be supplemented in order to improve its diffusion gradient, CO2 is already a trace gas in the ambient air. There is nothing that we can supplement air with that would help improve CO2 removal.

Other than treating reversible causes of poor ventilation and hoping that the patient recovers, there is little that can be done to directly improve CO2 removal except mechanical ventilation.

Mechanical Ventilation

The mechanical ventilator is an artificial support for the patient's "gas pump" described in the section on ventilatory failure. With this in mind, mechanical ventilation offers several means which can improve a patient's CO2 level.

Guaranteed Minute Ventilation

In patient with a poor respiratory drive the mechanical ventilator can guarantees some set amount of ventilation.

Increased Ability to Generate Work of Breathing

The mechanical ventilator can support a patient unable to do the required work of breathing. This is its principal role because it augments and can even replace the patient's intrinsic ventilatory mechanisms. Mechanical ventilation can supply a minute ventilation limited only speed at which air can be exhaled and inhaled into the lungs.

Recruitment of Lung Tissue

The recruitment of lung tissue with positive pressure with will improve the removal of CO2. The previously collapsed or minimally ventilated (low VQ) lung units become better ventilated thereby clearing CO2. Minimize CO2 production

Decrease CO2 Production

The mechanical ventilator can allow for safe sedation and even paralysis of a patient. This decreases metabolic demands and decreases CO2 production.

The administration of drugs that sedate or calm is dangerous and only done with extreme caution in a patient with hypercapnic respiratory failure that is not on mechanical ventilation .

Summary

** There is no supplement we can add to air that will increase CO2 removal. ** Enhancing CO2 removal requires mechanical ventilation. ** Mechanical ventilation aids CO2 removal through several mechanisms

Chapter Five

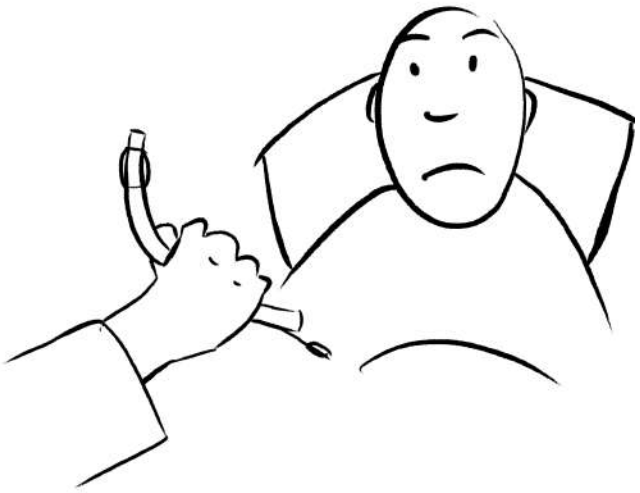
Initiating and Setting Mechanical Ventilation

When to Consider Mechanical Ventilation

There are several reasons to start mechanical ventilation. A common reason is respiratory failure. Another is airway protection during surgical procedures or when mental status is poor.

Not all patients with respiratory failure or poor mental status will require mechanical ventilation.

The decision to initiate mechanical ventilation is a clinical one. It integrates information regarding a patient's vital signs, lab work, disease, expected time to respond to therapy, and clinical trends.



We must put thought into the decision to initiate mechanical ventilation since the procedure is not without potential harm. The initiation of mechanical ventilation can

cause hemodynamic shifts precipitating a decompensation. If an endotracheal tube is required, the intubation procedure places the patient in potential harm. Mechanical ventilation raises risks of pneumonias and other complications associated with the bedbound state.

Another danger is waiting too long before initiating mechanical ventilation. Progressive deterioration should always prompt very serious consideration for mechanical ventilation. Waiting while a patient worsens is a recipe for disaster. Once a patient is in profound failure, the intubation becomes much more difficult, non-invasive ventilation is much less likely to help, room for error becomes slim, and the patient is more prone to a post intubation crash.

Mental Status - Airway Protection

Airway Protection

Up to 20% of ICU patients are intubated because of airway protection.[13]

A patient not capable of protecting his airway is at high risk of deterioration due to apnea, hypoventilation, or gross aspiration. One simpler way of deciding on a patient's ability to protect the airway is the GCS. A GCS score of < 8 ("GCS of 8, intubate"). This number comes from trauma literature that indicates that these patients are at higher risk for deterioration with apneas/aspiration/ and hypoventilation.[14]

Airway protection is a vague term. Although a patient whose GCS is 8 is usually considered unresponsive enough to intubate this number needs to be taken in the context of the patient. A patient who is vomiting or has secretions may need to be intubated despite a mental status that is not strictly GCS 8. Similarly, a patient who is hypoxic and septic with a diminishing mental status may also need to be intubated before that mental status is 8.

The gag reflex is not reliable as an indication of the ability to protect the airway. A better approach is to assess the response to voice commands which will give information about the level of consciousness and ability to handle secretions. If a patient is pooling secretions, gurgling, snoring, or requiring a maneuver to maintain

the airway (jaw thrust) they will need intubation for airway protection unless the condition is quickly reversible (e.g. opioid overdose).

Respiratory Failure - Hypoxemia

A patient with progressive hypoxemia with increasing doses of oxygen required should be intubated before the hypoxemia is at a point where it requires an high levels of supplementation.

This is especially important if we have placed the patient on non-invasive mechanical ventilation for hypoxemia. If the oxygen requirement is rising, then waiting in such a patient places them at risk for complications at the time of intubation which requires that they be taken off non-invasive ventilation and made apneic. An already severely hypoxemic patient who is requiring large doses of oxygen and positive pressure will desaturate quickly after induction and if there is any difficulty in placing the endotracheal tube a severe hypoxemia can result.

Highest Risk Patients

Although in extremis any patient can suddenly arrest, some patients are particularly prone to this - either from sudden catastrophic respiratory muscle failure or due to a propensity for silent failure as CO2 builds up.

Signs of Respiratory Fatigue

Rapid and shallow breathing

Erratic breathing

Complaints of dyspnea

Diaphoresis

Agitation or (more ominous) somnolence

Hypertension

Tachycardia or development of arrhythmias

Paradoxical breathing and use of accessory muscles

Shock

Patients in shock are specially prone to sudden arrests because their respiratory muscles are becoming starved of oxygen. In a patient with progressive shock, for any reason (sepsis, myocardial infarction), we should consider intubation earlier.

High Work of Breathing

Another group of patients that are high risk of sudden failure are those in whom the work of breathing is particularly high - asthmatics in status are an example of this. These patients may be young and can continue generating the work of breathing only to suddenly deteriorate and arrest.

We should closely observe any asthmatic in status and a normalizing CO2 or signs of fatigue should prompt an intubation. The asthmatic patient is also at high risk of complications from intubation.[15] The decision to intubate or defer is particularly difficult. Neuromuscular disease

Neuromuscular disease

Patients with neuromuscular diseases with progressive ventilatory failure are also a population that warrants close observation. Respiratory arrest in these patients can come on suddenly after a period of silent failure. This is especially true when the patient is being watched overnight and is expected to be sleeping.

Patients with neuromuscular diseases can develop a combination of factors that occur simultaneously.[16] A difficulty in clearing secretions, inspiratory muscle weakness, atelectasis, These factors may occur slowly and in variable amounts in different patients. Remember that in these patients the subjective signs of respiratory distress may not be obvious.

We can make monitoring for acute respiratory failure in neuromuscular disease more objective by checking respiratory parameters.[17] The Forced Vital Capacity (FVC), Negative Inspiratory Force (NIF), and Maximal Expiratory Pressure (MEP) are usually followed. In Guillain Barre syndrome (GBS) the "20-30-40" rule says that a patient with an FVC below 20, a NIF below -30, or a MEP below -40 should be considered for intubation.[18] We should also consider a patient unable to count to 20 with a single breath for intubation.

Other signs of failure include complaints of dyspnea, tachypnea, paradoxical breathing, using accessory muscles, speaking in shortened sentences. If a patient is progressing rapidly they should be intubated.

Hypoxemia and hypercapnia are late findings and you should not wait for these. An emergency intubation is much worse than a controlled intubation.

Some neuromuscular diseases, such as Myasthenia gravis, are relatively quickly reversed with therapy and a trial of non-invasive ventilation is reasonable. Some, such as GBS, can progress quickly and take time to recover. Patients with Guillain Barre syndrome should be intubated early.

** Signs and symptoms of failure in neuromuscular disease table

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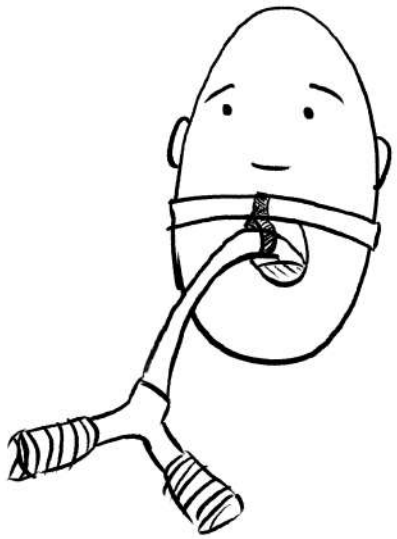
Invasive and Non-invasive Mechanical Ventilation

The mechanical breath is delivered to the patient through an interface that is either invasive or non-invasive. Invasive indicates that the interface between the patient and the ventilator is either an endotracheal tube or a tracheostomy tube that bypasses the upper airway. Non-invasive (NIV) means that the interface is a well sealed face mask.

While the 2 methods of delivering mechanical breaths confer the benefits of mechanical ventilation there are advantages and disadvantages unique to each.

Invasive Mechanical Ventilation

The endotracheal tube and tracheostomy offer several advantages. With an endotracheal tube the delivery of the gas is no longer reliant on the patient maintaining an open airway. The upper airway is bypassed by the endotracheal tube which seals the lower airway with its cuff. This allows deep sedation without the worry of the patient's airway occluding. This also allows the delivery of whatever gas mix is prescribed without the worry that the patient will entrain room air and dilute the mixture. The pressure prescribed will be guaranteed to be delivered to the deep airways without worry that it is actually transmitted to to the face or cheeks instead. The patient is protected from massive aspiration events due to the inflated cuff. The patient's lower airways can be suctioned through the endotracheal tube so patients with ineffective cough can undergo pulmonary toileting.



The disadvantage of the invasive interface is that the upper airway and its protective reflexes are bypassed. This seems to increase the risk of developing pneumonias. There is also the risk of placing the endotracheal tube - the intubation - which comes with its own set of risks and possible difficulties.

Non-invasive Mechanical Ventilation

The mask through which the patient is mechanically ventilated has the advantage of not bypassing the normal protective mechanisms of the upper airway. This allows the

patient to continue to swallow and cough and prevent small aspirations. Because the mask is less uncomfortable than an endotracheal tube there is typically no need to sedate the patient and so complications of sedation are avoided.

The lack of a secured sealed lower airway means that it is up to the patient to maintain the airway's patency. A patient with an unstable mental status or someone who requires heavier sedation will not be able to undergo non-invasive ventilation safely. Due to the lack of a seal protecting the lower airways the non-invasive interface does not protect the patient from aspirations - particularly of massive aspiration such as from vomiting.

There is another disadvantage to non-invasive ventilation. The mask, when in use, prevents access to the patients mouth and presses onto the soft tissues of the face and nose. If the respiratory failure is expected to require a prolonged time to recover then use of NIV is relatively contraindicated. The reason for respiratory failure should be one which resolves relatively quickly.

The best studied uses of non-invasive ventilation are in COPD exacerbations, pulmonary edema, and immunocompromised pneumonia where it has been shown to reduce the risks of intubation, hospital stay, and mortality.

Other studied uses include attempts at preventing intubation of high risk post operative patients such as those with advanced COPD. It can be used in an attempt to allow the weaning of the ventilator in a COPD patient.

The use of NIV in the setting of other acute hypoxemic failures is a little trickier . The major risk is that endotracheal intubation will be delayed and that this will harm the patient. The risk of NIV failure in severe pneumonia is high and there hasn't been a proven benefit except in patients with underlying COPD. It is OK to trial a short period of NIV with very close observation in patients with no comorbidities (shock, unable to protect airway…) with an understanding that the patient will be observed very closely in an ICU and that an evaluation for improvement be done in 1-2 hours after institution with a move to intubation if there is a lack of improvement.

Some patients are more likely than others to improve with non-invasive ventilation. Particularly those with initially mild failure. Advanced failure with high minute ventilations are likely to fail. The HACOR[19] score (heart rate, pH, P/F, RR, and GCS) can integrate several of these signals and predict failure. A high HACOR score makes it more likely that the patient will fail a trial of non-invasive ventilation.[20]

After non-invasive ventilation is initiated the patient should be observed closely. Signs of improvement are a steadying of the respiratory rate, the patient's subjective feeling of improved dyspnea, improved saturations/blood gases. A patient with trends to worsening should be intubated rather than being watched as they progress to overt failure. Intubating a patient overtly failing non-invasive ventilation is dangerous.

The use of non-invasive ventilation should, therefore, be done judiciously in an alert patient without any of the contraindications listed below:

Contraindications to Non-invasive Ventilation

Hemodynamically unstable

Unable to protect airway (GCS < 8)?

Excessive secretions

Uncooperative, agitated

Recent surgery on UGI or airway (ask surgeon)

Can't fit the mask

Respiratory arrest

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The setting up of mechanical ventilation requires some thought. The patient's internal settings which are functioning to maintain homeostasis are supplemented or overridden by the mechanical ventilator settings. A mismatch between what is required and what is provided may result in patient injury or death. The practice of placing a patient on "default" settings without thought should be avoided.

The initial ventilator settings first depend on what type of mechanical ventilation has been started. Invasive vs. Non-invasive. The initial settings in both cases seek to administer adequate ventilation and oxygenation to the patient.

Invasive Mechanical Ventilation Settings

The first step in initiating mechanical ventilation is choosing the correct mode of ventilation.

Choosing the Initial Mode

In the ICU, the main reasons for invasive mechanical ventilation are shock, acute respiratory failure, and airway protection. Also, a patient just intubated will, for some time be under the influence of paralytics and sedative agents weakening their respiratory efforts.

These situations imply that upon initiation of mechanical ventilation the patient is unable to reliably ventilate on their own and requires some guaranteed minute ventilation.

The modes that guarantee minute ventilation are the modes that utilize mandatory breaths: CMV and IMV. The mode generally used in the ICU is the CMV mode.

Choosing the Target

Breath target selection is largely a cultural choice as there has been no proven benefit of one mode over another. An institution gets used to one particular mode; staff become better trained to deal with a ventilator using that mode. There are some situations which we will discuss which contraindicate a particular target.

We adjust settings to normalize blood gases. This relies on achieving appropriate ventilation and oxygenation.

Adjusting Settings to Ensure Adequate Ventilation

An adult patient should be ventilated with at least 4-6 Liters/minute. A patient, once on mechanical ventilation, will generally trigger breaths and adjust minute ventilation to an appropriate level.

Relying on a patient's ability to self-adjust the minute ventilation, however, is not always reliable. Immediately post intubation, a patient may be paralyzed or still under the effect of strong sedative agents. A patient may be suffering from a catastrophic intracranial event which makes their ability to adjust their minute ventilation impossible. The initial settings should take into account that a particular patient may initially not respond to hypercapnia appropriately and ensure that an adequate minute ventilation is still supplied.

The peri-intubation period is especially dangerous if the patient requires a compensatory respiratory response for an acute acidosis. Intubation with its sedatives and paralytics will cause bradypneic period which will cause a precipitous fall in pH and a possible arrest. Pre-intubation Kussmaul breathing should be matched with a setting giving a similar high minute ventilation.

Similarly, A chronically hypercapnic COPD patient should not have the hypercapnia corrected too rapidly. Chronically hypercapnic patients have developed a compensatory renal alkalosis. The setting of an elevated minute ventilation would rapidly raise pH to dangerous levels. Contrary to popular belief, a high pH is quite poorly tolerated.

Setting an adequate initial minute ventilation will require ensuring an adequate tidal volume and an adequate breath frequency.

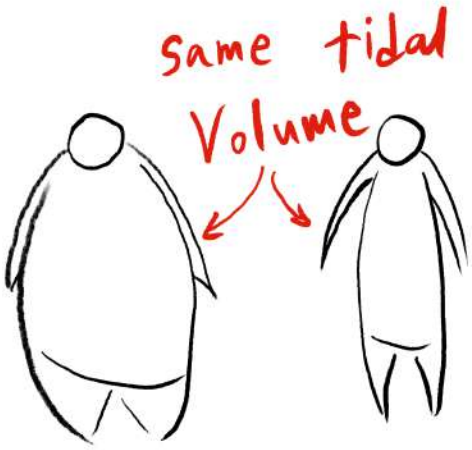
Setting an adequate tidal volume will depend on which breath target we select.

Setting an Appropriate Tidal Volume in the Volume Target Breath

The initial tidal volume setting takes into account the need to ventilate the patient and the need to protect the patient from high volumes and pressures which are associated with poor outcomes.

Any patient, especially one with diseased lungs, should have a tidal volume set in accordance with the recommendations for low tidal volume ventilation. The tidal volume used is one that is based on the patient's ideal body weight and targeting around 6cc/kg.

The ideal body weight can be found using the ARDSNet table. It's important to remember that it's the ideal body weight that determines the tidal volume target. Two patients with the same height and gender will have the same target tidal volume.



While the recommended tidal volume for ARDS is around 6cc/kg of IBW. The initial settings for a patient may not be that low. We should approach the ARDSNet volumes in a controlled fashion after the patient is stable on settings adequate to ventilate.

Typical initial settings are with a tidal volume of around 8cc/kg and a targeted minute ventilation of around 7-8L/minute. Over the next few hours (less than four) reductions are made to tidal volume as detailed below.

Setting the Pressure Target Breath to Deliver an Appropriate Volume

Driving Pressure

The appropriate driving pressure in a pressure target breath allows for adequate ventilation but limits the amount of pressure the lung is exposed to.

The driving pressure is usually set at 20cm of water and adjusted to get to a tidal volume of around 6cc/kg ideal body weight.

The pressure the lungs are exposed to is the sum of the driving pressure and PEEP. This should be limited to around 30 cm of water.

Inspiratory Time and I:E Ratio

The inspiratory time or the I:E ratio will need to be set for a pressure control breath. The inspiratory time determines the amount of time the driving pressure will be applied to the ventilator circuit. The I:E ratio is a ratio of inspiratory time (I) to expiratory time (E) and when coupled with a respiratory rate essentially assigns a time for inspiration.

Determining an appropriate inspiratory time will need to take into account the respiratory rate since we must allow for an adequate amount of time to exhale. A patient breathing at 15 breaths per minute has 4 seconds to inhale and then exhale. If the patient can breath in at around 60L/minute (about average) then the breath will take around 1 second to inhale. This leaves 3 seconds to exhale which is an inspiratory to expiratory ratio of 1:3.

A good I:E ratio will be around 1:2 which mimics normal respiration. Obstructive diseases may require longer E times and hypoxia may necessitate longer I times.

Setting the Respiratory Frequency

After the tidal volume range is determined the frequency of breaths is determined. The frequency is typically set in order to target a minute ventilation around 5-7L/minute. For patients with obstructive lung diseases or a propensity to higher exhalation times see section on COPD and Obstructive lung diseases.

The patient can trigger the ventilator and set their own rate. Unless there is a particular reason to keep the patient extremely sedated or paralyzed then they should be allowed to set their own rate.

If a mechanically ventilated patient is not over breathing the ventilator then a reason should be found. It can be that the patient is paralyzed, sedated, or is being over ventilated.

Oxygenation

Oxygen Fraction

The settings focussed on oxygenation are the fraction of inspired oxygen (Fi) and the PEEP.

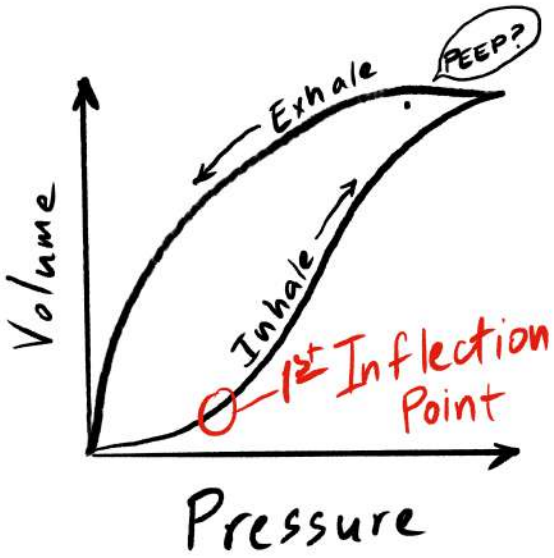
Upon initiation of mechanical ventilation in the crashing patient the Fi is usually set at 100%. We titrate this value down until the patient is on the lowest Fi that maintains oxygen saturations above 90%. This titration should occur as soon as possible.

High fractions of oxygen are quite irritating (especially to the pulmonologist). Oxygen is a very caustic gas and an excessive concentration can result in tissue injury and microatelectasis. There are indications that hyperoxia in particular scenarios is detrimental - e.g. the post arrest patient.

To PEEP or not to PEEP

It's a matter of debate how much PEEP to use. An initial PEEP of 5 cm is acceptable and if the patient has no significant oxygenation requirements is usually sufficient. When we have a hypoxemic patient, we put more thought into choosing the "correct" PEEP.

PEEP is used to open atelectatic alveoli. Alveoli that repeatedly opened and closed are damaged - atelectotrauma. The ideal PEEP level would be the least amount that opens atelectatic alveoli and keeps them open for the duration of the breath. The ideal PEEP would be one above this "opening pressure" or lower inflection point of the pressure/volume curve. Ideally, making this curve, however, requires you to inflate lung and measure pressure with no flow at several points to remove the element of dynamic pressure.



At the bedside this is not a practical maneuver. Finding the correct PEEP, therefore, requires a bedside titration. This can be done in several ways.

The PEEP is slowly increased in step with FIO2 to maintain oxygenation while keeping plateau pressure acceptable. There are titration graphs which can be used e.g. the ARDSNet PEEP titration graph.

In general PEEP is increased stepwise with the FIO2. This can be done using the ARDSNet PEEP/FI chart.

Another method, once popular, to set PEEP is to perform recruitment maneuvers and monitoring the lung compliance. PEEP can be set to maintain recruitment. This method, however, has been shown to increase mortality and isn't recommended.[21]

A higher PEEP will improve oxygenation but there is no survival advantage to the patient.[22] The higher PEEP also comes with the disadvantage of worsening hemodynamics and lowering cardiac output particularly in those that are volume depleted or have right heart failure.

There is some benefit to a minimal amount of PEEP, the typical is around 5 cm. This has been shown to decrease, of all things, VAPs and minimize the number of times a patient becomes hypoxemic.

Recruitment Maneuvers

Recruitment maneuvers are the temporary increase in the mean airway pressure in order to open atelectatic segments of lung. We maintain the recruitment if successful by raising the PEEP.

For the recruitment maneuver we temporarily place the patient on higher pressure ventilator settings or we Ambu bagged with a PEEP valve to raise mean airway pressure.

If the mechanical ventilator is used to recruit then the pressure and mode used are typically part of an institutional protocol. One example is a BiLevel mode where the high PEEP is set at around 40 cm and the low PEEP set at whatever the patient's original settings are. The maneuver is maintained for up to 20 seconds and is terminated immediately if the patient does not tolerate it by either developing hypotension, hypoxemia, or arrhythmias.

Once the maneuver is performed, we observe the patient looking for an improvement in oxygenation and lung compliance. If we see an improvement, we can titrate the PEEP to maintain this new higher SPO2 and lung compliance. If the procedure fails to improve the oxygenation and compliance we can repeat it at higher pressures until it is successful or the patient proves to be non-recruitable.

De-recruitment occurs when the patient loses alveolar recruitment. Previously inflated alveoli that were open due to the elevated airway pressures collapse and no longer participate in ventilation. This can occur when PEEP is lost such as when the patient is disconnected from the ventilator or suctioned.

Another method is to attach the patient to an Ambu bag and bag at higher inflation pressures. This method is not recommended since the patient is likely to de-recruit as soon as the Ambu bag is disconnected to place the patient back on the ventilator.

Recruitment maneuvers can cause hypotension, pneumothoraces, barotrauma, and are not proven to improve a patient's outcome. They are not recommended for routine[23] use.

Adjusting Mechanical Ventilation After the Initial Settings

Mechanical ventilation is not a static treatment. The patient's condition will change. Lung stiffness will change. Their minute ventilation and oxygenation requirements will change. Adjustments will need to be made after the initiation of mechanical ventilation and, on occasion, quite frequently afterwards.

Initial ventilation requirements are always, to some degree, guesses. Around 15-30 minutes after initiation of mechanical ventilation (and with any changes) a blood gas should be obtained.

Ventilation will need to be adjusted to get the pH close to a normal range and in order to ensure that the patient's tidal volumes meet low tidal volume criteria if that is possible.

Adjusting to get pH to Normal

In general, increasing minute ventilation will decrease blood CO2 levels and, thereby, increase pH.

The adjustments in minute ventilation that happen in attempts do not always have predictable results.

The minute ventilation can be calculated by the product of the rate and the tidal volume. The change in settings required for a particular change in CO2 is not readily calculated because of changes in dead space that occur with adjustments in tidal volume and respiratory rate.

Even though two settings generate the same minute ventilation, they may clear CO2 differently. This is because of the relative amount of dead space being ventilated. A smaller breath will have a higher amount of dead space relative to its volume - it will have a higher dead space ratio.

Imagine you are receiving 25 mL breaths. These breaths move gas in and out of the large airways but do not ventilate the deeper respiratory airways and so will not clear

any CO2. Whatever the respiratory rate, these breaths will not clear CO2. The deeper the breaths the more CO2 is removed thus making each breath more efficient.

A smaller tidal volume may also change the amount of recruited lung. With the smaller breath less lung is opened up for ventilation and so ventilation/perfusion matching can be impaired.

A change in respiratory rate may also, paradoxically, change CO2 clearance if it gives obstructed lungs, which take longer to exhale, less time to empty. The rapid respiratory rate may then cause a decrease in minute ventilation and worsen issues.

Adjusting Tidal Volume to Reach ARDSNET Recommended Volumes

An important aspect of mechanical ventilation is to attempt to get the patient to the lowest tolerated tidal volume - 7cc/kg then 6cc/kg.

During titration of volume downwards we should check blood gases to make sure that the pH remains acceptable. If the plateau pressure remains above 30 cm, then further attempts at decreasing the tidal volume can be made to as low as 4cc/kg.

In order to reach the lower tidal volumes a patient may develop a respiratory acidosis. This acidosis is generally well tolerated in most patients when it is mild (pH > 7.2) and so this level of acidemia is allowed. This is termed “permissive hypercapnia”. The actual CO2 level is less important in this case. A more severe acidemia may be buffered in order to allow the low tidal volume ventilation.

An important thing to know about permissive hypercapnia is when it is NOT permitted. There are consequences to the elevated CO2 and low pH. Hypercapnia is vasodilatory to brain vessels and is contraindicated in conditions associated with high intracranial pressures. Hypercapnia can decrease myocardial contractility and increase sympathetic activity and so if a patient is hemodynamically unstable or having difficult to control arrhythmias it is contraindicated. It is contraindicated in pregnant patients as the fetus relies on the low CO2 level in the mother’s blood to help clear his own. It is contraindicated in patients with severe pulmonary hypertension due pulmonary vasoconstrictive effects.

Lower tidal volumes are uncomfortable for the patient. Most patients will want deeper breaths. Low tidal volume will, therefore, cause ventilator dyssynchrony. More on this in the chapter on dyssynchrony.

Another consequence of lower tidal volumes is a worsening of pulmonary mechanics and oxygenation. This is to be expected since the lower tidal volumes are more prone to atelectasis. Remember, in the longer term the protective strategy pays off.

Limitations in Mechanical Ventilation

There are limitations on the minute ventilation that a mechanical ventilator can provide. These limitations are imposed by the patient's expiratory phase. This topic is discussed at length on the chapter on exhalation.

Adjusting to Reach the Appropriate Plateau Pressure

Along with the tidal volume, the plateau pressure should be monitored and kept under 30cm.

In volume targeted breath modes this will require the intermittent checking of the plateau pressure. If the peak pressure is below 30cm and the patient is not air hungry and pulling large volumes then it can be assumed that the plateau pressure is below 30cm.

In pressure targeted breaths the plateau pressure is simply the set PEEP + the driving pressure. Keep it below 30cm. Lower if the tidal volumes creep high.

Setting Spontaneous Mode

In some situations such as weaning trials or in awake patients who are comfortable you may choose to put the ventilator on spontaneous mode.

The benefits of this mode are that the patient gets breaths that are as long and as deep as they like.

In spontaneous mode (also called pressure support ventilation PSV) a pressure support pressure is set on top of a PEEP. For wean trials the pressure support is generally chosen to be about 5-8cm. A typical setting would be for a PSV trial of 5/5. For other patients it can be whatever pressure the patient finds comfortable - within reason. Remember that the plateau pressure (in this mode the pressure support + PEEP level) should be kept as low as possible and preferably below 30cm.

As discussed in the section on modes this mode is flow cycled and

Non-invasive Mechanical Ventilation Settings

Non-invasive mechanical ventilation settings are very similar to those for invasive mechanical ventilation.

When a non-invasive method of mechanical ventilation is chosen the choice of mode is simplified. The choice is for either CPAP mode or BiLevel ventilation. BiLevel is essentially the same as spontaneous mode in the invasive ventilator. CPAP is simply a mode that maintains a PEEP in the ventilator circuit.

The choice between CPAP and BiLevel (BiPAP) depends on patient comfort, which non-invasive ventilator is available, and whether we want to try to guarantee a minute ventilation.

CPAP can be used for patients with acute congestive heart failure. The positive pressure will recruit lung, increase FRC, and decrease work of breathing as we diurese and control blood pressures. The patient will breath against a set pressure continuously - called the EPAP (expiratory positive airway pressure) - which is just another name for PEEP. In CPAP some patients will complain of feeling suffocated due to a feeling of constant impediment to exhalation.

BiPAP can be used for patients uncomfortable with CPAP or for those in which a backup rate is felt to be important. The BiPAP mode is the same as the spontaneous mode in invasive mechanical ventilation. With each patient inhalation the ventilator will increase the circuit pressure then decrease it again once the breath is over.

The setting for bipap, however, is a bit different. In invasive mode we set a pressure support on top of the PEEP. e.g. we set a pressure support of 5cm on a peep of 5cm. The total circuit pressure during an inhalation will be 5+5 = 10cm. In non-invasive mode we set an IPAP separate from the EPAP. Ex: we set an IPAP of 10cm and an EPAP of 5cm to get the same pressure settings as a PSV 5/5. To set a non-invasive ventilator to CPAP we would set the IPAP and EPAP to the same value.

In general, we set the BiPAP to 10/5 and observe the patient. We target a tidal volume of around 5-7mL/kg PBW. IPAP can be increased in slow increments to get to the target tidal volume. The non-invasive nature of the mask lend itself to leaks and the tidal volume measurements are not always reliable. The consistent need to increase the IPAP to get to target tidal volumes may indicate that the patient is failing BiPAP and that invasive ventilation may be needed.

For oxygenation in non-invasive ventilation EPAP can be adjusted upwards in increments. The adjustment should be made slowly as patients will not tolerate large changes to the settings without a period of time to adjust.

Monitoring Ventilation

A capnograph can show a change in ventilation with adjustments as CO2 in exhaled gas changes with better or worse ventilation. With hypoventilation the exhaled gas would contain higher and higher concentrations of CO2. With hyperventilation the exhaled CO2 would decrease.

The only reliable method of ensuring that adjustments are having the desired result, however, is to rely on blood gas measurements. Blood gases, therefore, should be checked intermittently on the mechanically ventilated patient and with adjustments in the ventilator settings.

A blood gas gives information on CO2 and pH. We adjust the mechanical ventilator to keep pH within an acceptable range. Adjustments to a mechanical ventilator are accompanied by a blood gas around 30 minutes later to allow for CO2 levels to equilibrate.

Adjusting and Monitoring Oxygenation

Oxygenation is followed in real time with the ubiquitous pulse oximeter and the dose of oxygen is adjusted to keep the patient's oxygen saturation above 92%. The pulse oximeter can occasionally be wrong and we should obtain at least one blood gas to make sure it's being honest.

Some situations in which the pulse oximeter are wrong include - severe tricuspid regurgitation with the pulse oximeter on the earlobe, interfering nail polish, carboxyhemoglobinemia/methemoglobinemia.

A discussion of correcting oxygenation is in the section on "Fixing Failure>>Correcting hypoxemia".

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The heart and lungs are intimately connected. Positive pressure ventilation emphasizes and alters this connection. This is particularly true during the immediate post intubation period.

In order to understand the hemodynamic consequences of mechanical ventilation we'll review heart lung interactions.

Heart Lung Interactions[24]

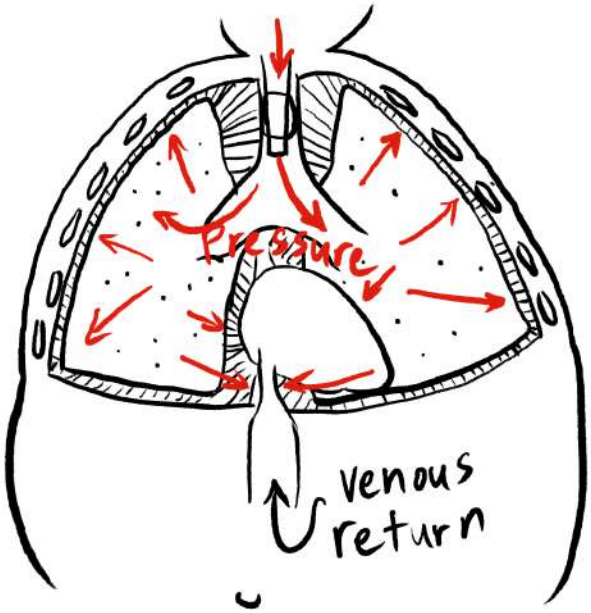
The Respiratory Pump and Right Heart Preload

Normal breathing generates a negative pressure in the chest. This negative pressure pulls air into the lungs. It also pulls the blood from outside the chest. Blood gets sucked into the chest through the large veins that sit outside the chest which are under atmospheric pressure.

This "respiratory pump" serves to augment right heart preload by filling the right heart.

The initiation of positive pressure ventilation reversed the respiratory pump. Instead of pulling blood into the chest, the positive pressure that occurs with each breath impedes venous return. This impediment to venous return is counteracted somewhat

by the increase in abdominal pressure that occurs with each breath - the increased pressure squeezes intraabdominal capacitance veins and augments the venous return.



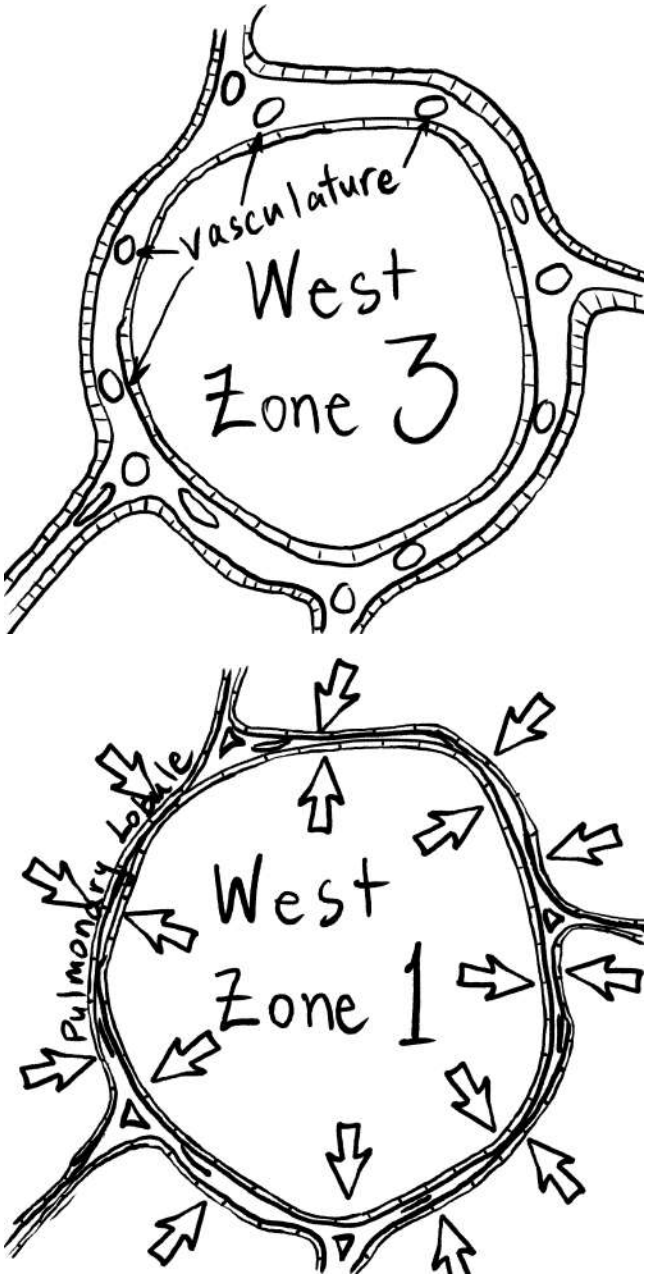
In patients who are hypovolemic, the drop in venous return can be catastrophic and can result in a severe drop in cardiac output and blood pressure.

Pulmonary Pressures and Right Heart Afterload

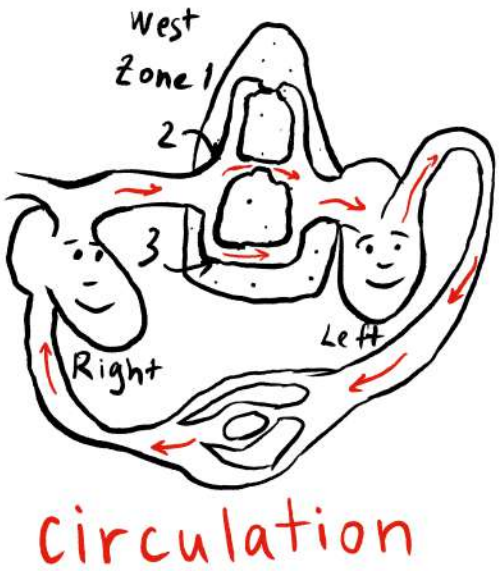
The pulmonary circulation is contained entirely within the chest and so changes in pleural pressures don't directly alter it. The changes in lung volumes, however, do.

The pulmonary vascular resistance changes depending on lung volume. It is highest at the extremes.

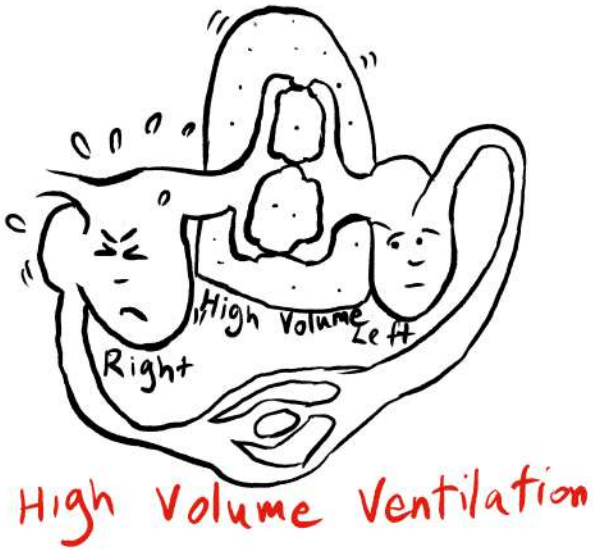
Because the alveolar pulmonary circulation is a low pressure system the weight of blood makes a difference in pressure. Normally, the blood flow into the alveoli is divided into 3 zones (West's lung zones).



In zone 1 the alveolar pressures are higher than vascular pressures and the vasculature collapses and resistance is very high. This zone is usually at the most elevated part of the lung. In zone 2, lower down, the alveolar pressures are high enough to collapse alveolar vasculature incompletely and resistance is high. In zone 3 the vasculature is open and resistance is low.



With mechanical ventilation, if volumes are high, more of the lung's segments are converted from zone 3 to zones 2 and 1. The pulmonary vascular resistance rises.



With low lung volumes the patient develops atelectasis. Extra-pulmonary vasculature collapses into the atelectatic segments and resistance, again, rises.

The ideal lung volumes with respect to pulmonary pressures seem to be near FRC.

Right heart syndrome, cor pulmonale

The right heart has little ability to tolerate increases in afterload and responds by dilating.

The left and right ventricles are interdependent. They reside inside the pericardial sac and share a septum. Changes in volume of one ventricle take the space from the other ventricle.

As the right ventricle dilates it encroaches on the left ventricle. This encroachment decreases the diastolic size of the left ventricle and can impede left ventricular cardiac output thus dropping blood pressure.

The right ventricle's perfusion relies on the gradient between its pressure and the arterial pressure. As it dilates and pulmonary pressures further rise due to left heart failure/hypoxemia/acidosis, the systemic pressures drop. This combination of dropping systemic pressures and rise in pulmonary pressures cause further ischemia and a progressive cycle of deterioration starts.

Left Heart Afterload

The left heart is within the chest but pumps into vasculature outside the chest. Its function is, therefore, affected by the chest's pressure changes.

In normal negative pressure breathing the heart is pulled on by the negative pressures in the chest. These pressures impede its output to the extrathoracic vasculature. This impediment is not an issue and easily compensated for.

In situations of extreme intrathoracic pressure swings, such as very stiff lungs in ARDS or with an obstructed upper airway (such as with stridor) the pressure swings can be severe enough to cause a drop in cardiac output and left heart failure.

In contrast to the right heart, the switch to positive pressure ventilation improves the situation for the left heart. The positive pressures in the chest now reduce afterload by squeezing the left heart and augmenting output. This can be of tremendous help to a failing left heart.

Post Intubation Hypotension

The initiation of mechanical ventilation is often accompanied by a period of hemodynamic instability. This isn't surprising seeing as how closely the heart and lungs interact.

The hemodynamic instability post intubation can be due to several factors.

- Changes in adrenergic tone and response to induction/sedation drugs.
- Changes in preload.
- Changes in afterload.
- Changes in oxygenation/ventilation parameters around time of intubation.

Changes in Adrenergic Tone and Drug Effects

The induction agents that allow a comfortable and safe intubation can be hemodynamically active.

The sedation received by the patient removes the intrinsic adrenergic drive that may have been keeping blood pressure elevated and so result in a drop in blood pressure. The patients most likely to experience this drop in blood pressure are those whose adrenergic tone is elevated prior to the intubation.

The sedatives and induction agents used can also be directly vasoactive in that they may be negatively inotropic or cause some degree of vasoplegia. The choice of drugs used to intubate patients is beyond the scope of our discussion but some drugs are more likely to cause hemodynamic instability than others.

Changes in preload

As discussed above the initiation of positive pressure ventilation will impede right heart preload. This impediment can be severe in the hypovolemic patients.

Changes in Afterload

This is usually an issue in the patients suffering from pre-existing right heart failure. The initiation of positive pressure ventilation presents a sudden rise in pulmonary vascular pressure and a struggling right heart may drop its cardiac output.

Changes in Oxygenation and Ventilation Around Time of Intubation

With the administration of induction medications and paralytic agents the patient becomes apneic and loses FRC. This results in the rapid development of hypoxemia and hypercapnia if the intubation takes any degree of time. Patients with low FRCs such as pregnant or morbidly obese patients are at particular risk for peri-intubation hypoxemia. This hypoxemia can result in hemodynamic compromise.

Remedies to the Post Initiation Hemodynamic Issues

It is important to anticipate and prepare for the hemodynamic compromise that occurs after initiation of mechanical ventilation. Never leave the patient's side immediately after you inititate positive pressure ventilation - wait until you are sure they are stable. Fluid boluses should be readily available and in the case of the patient who is suspected to be hypovolemic (lack of PO intake, diuresed, or having diarrhea) a bolus of fluids should be started as soon as a decision to intubate is made. Pressor agents should be available to be started - if the patient is already somewhat hypotensive these agents should be primed and either started at a low rate or ready to go. Push dose pressors if they are available in your ICU can help too.

Chapter Six

Complications of Mechanical Ventilation

Mechanical ventilation is not benign. This section details complications of mechanical ventilation which will delay or even completely abort a patient's recovery.

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The mechanical breaths damage lung tissue. Every mechanical breath administered stretches tears at the lung. As soon as we start a patient on mechanical ventilation we begin accumulating this damage. We expect that the patient's pathology will recover faster than the damage from mechanical ventilation accumulates. If this is not the case then we end up with a patient who cannot be liberated from mechanical ventilation.

One way mechanical ventilation damages lung is through the pressures and stretch applied to the delicate tissues. This trauma causes alveolar damage, leakage, and leads to progressive failure which, after a time, can look indistinguishable from ARDS.

Volutrauma

Volutrauma is the over-distention of lung through the application of excessive volumes. This is particularly a problem in processes, such as ARDS, in which heterogenous patches of lung tissue become so consolidated that they are no longer ventilated. The balance of the volume of an administered breath over-inflates whatever ventilated "baby" lung remains.

The remedy to volutrauma is the judicious use of tidal volumes. The ARDSnet volumes have been proven to lower mortality[25] and should be followed closely. This is particularly true in patients with ARDS but should be considered in anyone on a mechanical ventilator.

Barotrauma

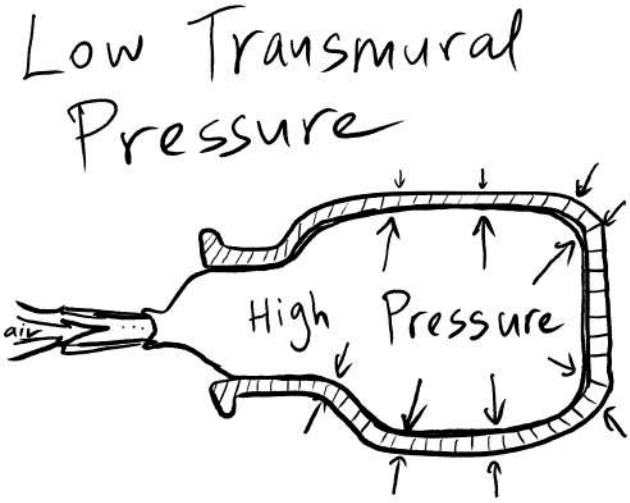
Barotrauma is the application of excessive pressures upon lung tissues.

The damaging pressure that is applied is the transmural pressure. This pressure is the difference between the pressure inside the lung and that outside the lung. A low transmural pressure is not damaging irrespective of what the pressure inside the lung is. A high transmural pressure is damaging, also irrespective of what the inside pressure is.

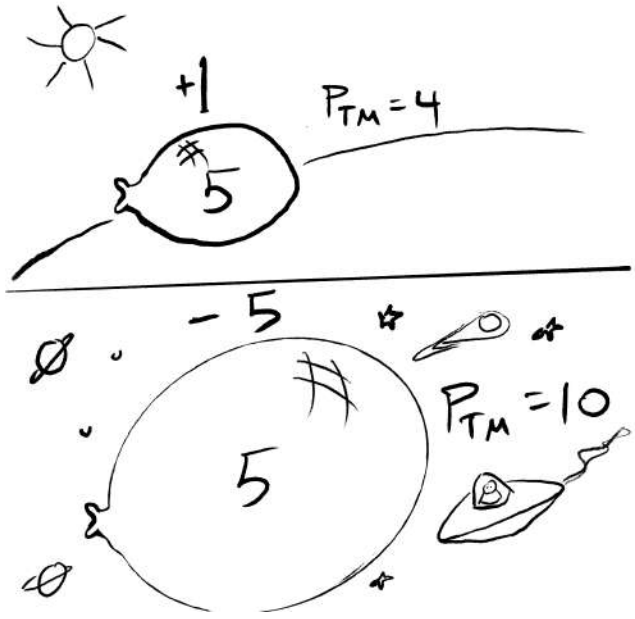
An example of high intrapulmonary pressure with low transmural pressures is that of the extremely morbidly obese patient. The intrapulmonary pressure may be high to allow for ventilation. That pressure is counteracted by the large weight of the belly and chest. The lung in that case is not damaged.

An example of low intrapulmonary pressures with elevated transmural pressures is of the air hungry patient generating large thoracic pressure swings to pull air into the chest. The intrapulmonary pressures are very low. The very negative pleural pressures, however, cause great trans pulmonary pressure gradients that damage lung tissue.

One way to think about how transmural pressure is important is to think of a rubber balloon. If you place the balloon inside a glass bottle and inflate this balloon at some point the balloon will have taken up the entire inside of the glass bottle. The pressure you exert on the balloon at that point can be raised immensely and the balloon will not rupture. You can keep inflating and inflating and raising the pressure up until the glass bottle itself explodes. Until that point the balloon will have experience minimal transmural pressures. The pressure exerted on the inside of the balloon will have been countered by an equal pressure exerted on the outside by the walls of the bottle.



At the same time a balloon filled with a minimal amount of gas but taken into the vacuum of space will explode. The transmural pressure there will be very large due to the extreme negative outside pressure.



Excessive transmural pressures can cause rupture of lung tissue manifesting as pneumothoraces, as pneumomediastinum, or subcutaneous emphysema. The pressures can induce inflammation and as with volutrauma a progressive failure of the lung.

Plateau pressures, used a surrogate for intrapulmonary pressure, kept below 30cm and attempts at keeping driving pressures below 15cm limit barotrauma. These measures are not perfect because as explained above they don't take into account what the trans pulmonary pressures are.

Atelectotrauma

The cyclic collapse and re-inflation of alveoli can lead to tissue damage. This is termed atelectotrauma.

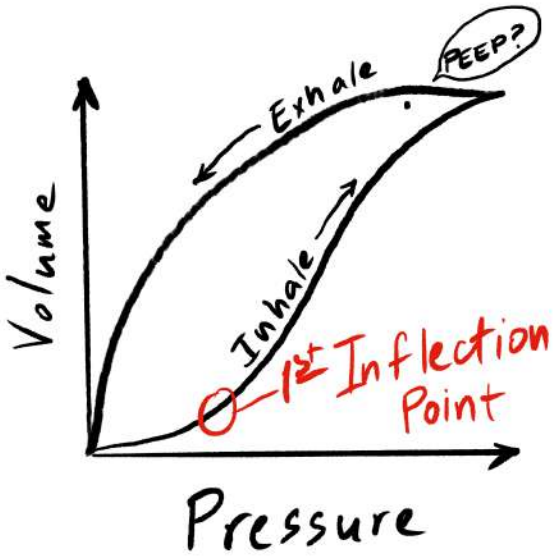
In order to minimize atelectotrauma we either keep the alveoli closed or open but try to prevent the cyclic opening and closing.

Low tidal volumes and plateau pressures minimize the chance of opening collapsed lung segments. PEEP can be applied to keep alveoli open even between breaths.

The ideal method of finding the ideal PEEP is unclear. One method is to use the Pressure-Volume curve. The lower inflection point is the point at which alveoli are fully recruited. PEEP levels chosen above this point decrease atelectotrauma.

❖

The lower inflection point is the point at which the lung compliance improves as alveoli convert from collapsed to open. Below the inflection point alveoli are collapsed and the pressure builds up until they pop open at which point pressure inflates them.



VAP

The usual scenario is of your patient steadily making progress. Then, one day, you notice that the oxygen requirement has increased. Or that there is a bump in the white count that had been dropping steadily. You take another look at the x-ray…

Mechanical ventilation can be complicated by infections. When delivered through invasive airways (tracheostomy, ETT) it bypasses the respiratory system's usual protective mechanisms. Patients have trouble clearing secretions due to an inability to generate adequate cough and due to the required sedation.

A ventilator associated pneumonia (VAP) is a pneumonia acquired after 48 hours of intubation. VAP is a serious consequence of mechanical ventilation and is associated with significant morbidity and mortality - up to 10% depending on the type of patient. It has been associated with longer hospital stays and increased use of antimicrobials.

The incidence of VAP increases with duration of mechanical ventilation. It is highest initially and the rate tapers off from around 3% per day initially to around 1% per day after day 10.[26]

VAP is suspected when the patient develops a worsening clinical picture, worsening or new imaging or exam findings, worsening ventilator settings, secretions, or evidence of infection (white count, fever…)

Diagnosis and Treatment of VAP[27]

After VAP is suspected, samples from the lower respiratory tract (an endotracheal aspirate) and blood cultures should be obtained to guide therapy. The patient's prior exposures and underlying diseases can given an idea as to their risks or resistant organisms. Empiric therapy with antimicrobials chosen to treat the most common institutional organisms should be started using the hospital antibiogram.

Prevention of VAP

There are several strategies that decrease the risk of developing VAP.[28] Use on non-invasive ventilation in appropriate patient populations, elevating the head of the bed, subglottic drainage of ETTs, the daily evaluation for readiness to extubate, oral care with or without chlorhexidine.

Strategies to prevent VAP

Use of noninvasive ventilation in appropriate patients.

Daily sedation vacations and assessment for extubation.

Early Mobility

Subglottic drainage of ETT

Mouthcare (with chlorhexidine in cardiothoracic ICUs)

Change ventilator circuit only if visibly soiled

Elevate head of bed to 30-45 degrees

Airway Injury and Complications of the Endotracheal Tube

The complications of mechanical ventilation are not just from the mechanical breaths. There are complications that are the result of the necessary devices needed to deliver the mechanical breaths.

The endotracheal tube, its placement, and the devices that secure it can also cause damage to the patient.

The placement of the endotracheal tube (intubation) can be a dangerous procedure. Its complications are beyond current scope. Briefly, they include damage to the teeth, mouth, and upper airways. This is particularly true when the procedure is done emergently or hurriedly (as in an ICU patient with little reserve).

The endotracheal tube is secured to the patient using either tape or, more appropriately, a tube holder. These securing devices exert pressure on the patient's skin and trap moisture. The underlying skin can deteriorate and develop pressure ulcers.

Prevention of damage from the endotracheal tube holder requires vigilance and routine examination and shifting of pressure points. The area beneath pressure points is frequently examined, dried, and relieved.

The endotracheal tube, itself, can cause damage. It lays on structures in the oropharynx and can cause pressure ulcers on the lips and mouth structures. The endotracheal tube passes through the delicate vocal cords and laryngeal mechanism and can cause pressure ulcers or dislocate the joints. The cuff, which seals the airway to prevent leaks and gross aspiration, exerts pressure on the delicate trachea. The pressure exerted diminishes the blood flow and in some cases (hypotension or high cuff pressures) can cause ischemia. The ischemic trachea reacts with the formation of stenoses or malacic (soft) segments. The cuff can exert pressure on the recurrent laryngeal nerve and cause unilateral or even bilateral vocal cord paralysis.

Prevention of complications from the endotracheal tube involves several steps.

Initially choosing an endotracheal tube that is sized appropriately is the first step. A smaller endotracheal tube complicates ventilation but a too large tube exerts pressure on the glottic structures. A general rule is to use a 7-5 tube for an average size female

and an 8-0 tube for a man. These size tubes will allow ventilation and bronchoscopy when needed. A larger endotracheal tube is rarely needed.

Using the minimal volume and pressure in the tracheal cuff helps minimize tracheal damage. There are several ways of doing this. A cuff manometer is usually used to check the cuff pressure and keep it in a safe range. The "minimal occluding volume" involves filling the cuff until air leakage is obliterated then withdrawal of gas while making sure that no leak recurs. The "minimum leak" technique of filling the cuff is another method used in which the cuff is inflated until a leak is obliterated, then deflated to allow a minimal amount of air to leak around the cuff. The worst method of determining the amount of air to put in a cuff is to palpate the pilot balloon for "softness".

Removing the endotracheal tube as soon as feasible is an important part of minimizing complications of this device. Many of the complications associated with the endotracheal tube become more common as the endotracheal tube remains in place.

Weakness and Deconditioning

Within as little as 4 days a mechanically ventilated patient can develop weakness.[29] This weakness can complicate the ICU stay, make for a prolonged recovery, and is associated with an increase in one year mortality.

The weakness can be the result of development of critical illness myopathy, due to drugs, due to deconditioning from being bedbound, or most likely due to a combination of all these things. The workup is beyond the current scope.

Mobilization in adults mechanically ventilated for > 24 hours is now recommended Early physical therapy and mobilization even during mechanical ventilation has been shown to decrease time on mechanical ventilation.[30]

Oversedation and Delirium

Invasive mechanical ventilation is very noxious. It requires sedation to prevent the patient from experiencing discomfort and attempting to self extubate.

The sedatives given, combined with the patient's illness, make the patient prone to delirium. The sedatives, if given in excessive doses, will also prolong the period of time the patient remains mechanically ventilated.

Protocols should be in place to allow for minimizing sedation and to allow awakening in patients who will tolerate this.

Gastric ulcers and bleeding

Patients under stress of critical illness and mechanical ventilation are at risk for gastric ulcers since the gastric mucosa loses its protective mucous layer.

In those mechanically ventilated for more than 48 hours it is recommended that we start stress ulcer prophylaxis with an H2 blocker or PPI. PPIs are more effective but seem to be associated with a somewhat higher risk for C. Difficile infection.[31]

Ileus and Constipation

Sedation with narcotics, immobility, and poor perfusion makes mechanically ventilated patients prone to constipation and ileus. A bowel regimen should be instituted early on and the patient monitored for bowel function. This is particularly true for those patients needing paralytics and deep sedation.

Keep in mind that these patients are at risk for intra-abdominal diseases too and that the physical exam becomes limited due to sedation and especially limited when paralytics are used.

DVTs and Pulmonary Emboli

The mechanically ventilated patients are sedated and immobilized and so despite DVT prophylaxis DVTs occur in up to a quarter[32] of the patients particularly those with malignancy or central lines.

Keep this in mind when looking for the cause of a failure to wean, unexplained fevers, or edema. Have a low threshold to look for DVTs.

Be Aware

Many of the complications listed above can be avoided, or at least improved upon if you are aware of them and institutional protocols address them.

Chapter Seven

Discontinuing Mechanical Ventilation

In the ICU, one of the chief roles of the medical team is the discontinuation of mechanical ventilation.

The consideration of when to discontinue mechanical ventilation begins as soon as we initiate it. The longer a patient remains mechanically ventilated, the more likely they are of developing a complication.

There are many complications associated with mechanical ventilation. Ventilator associated pneumonia rates are around 1% per day[33] in the intubated patient and carry a very high mortality of around 20-50%. Mechanical complications pose another threat - accidental dislodgment, damage to the delicate laryngeal and tracheal structure. Sedation and the bed-bound state is another source of problems. Dyssynchrony with the ventilator is associated with oversedation, lung, and respiratory muscle injury.

ICU ventilator management is aimed at discontinuing mechanical ventilation as soon as possible. This requires frequent assessment for readiness to discontinue mechanical ventilation.

Premature extubation is associated with significant consequences.[34] Re-intubation risks loss of the airway and a period of time during which the patient is at risk for Hypoxemia and hemodynamic instability. Extubation failure is a significant independent risk factor for death, development of nosocomial pneumonia, prolonged ICU stay, and an increase in the average hospital bill by nearly 35000$.[35]

When is it Time?

It is important, given the above risks, to know when a patient is ready to have mechanical ventilation discontinued. We should assess patients for readiness to extubate once they meet several criteria.

- The underlying cause of respiratory failure must be reversed.
- The patient must have adequate oxygenation - typically an Fi of not more than 0.4 on not more than PEEP of 5cm.
- The patient must be hemodynamically stable.
- The patient must be capable of initiating inspiratory effort and protecting the airway.

Assessing Readiness to Extubate

Clinical judgement by itself is a poor predictor of a patient's readiness for extubation. We must combine it with clinical parameters. The best information is extracted during spontaneous breathing trials with minimal ventilatory support.

SBT

The role of the SBT is to see if the patient can ventilate and oxygenating themselves in a sustained manner and without the aid of the mechanical ventilator. To see the fitness of the patient, support must be withdrawn and the patient must perform the work of breathing.

Several SBT techniques are available. The most common technique is the pressure support trial. In a pressure support trial, we place the patient on spontaneous mode with a support pressure of 5-8cm. In this mode, as discussed previously, the patient is supported in such a way that we minimize the resistance of the endotracheal tube. The spontaneous mode allows us to see the patient's respiratory rate as well as the depth of breaths. Another technique is the T-piece trial in which we detach the patient from the ventilator and allow them to breathe through the circuit with no support.

The different techniques of SBT have certain advantages and disadvantages. The pressure support SBT trial is recommended as it has been shown to decrease the duration of mechanical ventilation. Since the patient is attached to the ventilator alarms and a backup rate will save the patient in case of a catastrophic failure. The T-piece trial is used occasionally, particularly is the setting were we are suspicious that the minimal PEEP used is helping to prevent respiratory failure that would manifest once we extubate the patient.

Failed Breathing Trial

A failed SBT manifests as evidence of fatigue in the patient. Generally, it is the development of rapid shallow breathing. The tidal volumes decrease and the respiratory rate increases. Rapid shallow breathing can be quantitated to allow a more objective assessment by staff. The rapid shallow breathing index (respiratory rate divided by tidal volume in liters) is used in many ICUs as a measure of how well a patient is doing on an SBT. The lower the number, the better. An RSBI of more than 105[36] is generally considered failure.

The RSBI is good at predicting failure of extubation but is less adept at predicting success (an RSBI of <105 had a negative predictive value of 95%). In the original study the RSBI was calculated in patients placed on T-piece in which there was not ventilatory support. In its current form there is support from the ventilator in the form of pressure support. This decreases the RSBI and can mask failing patients.

In some patient populations, the RSBI is even less useful. In COPD patients early in the wean while they're still autoPEEPing, the RSBI may be low because of failure to trigger. In neurosurgical patients, in whom the intubation was for airway protection, RSBI did not correlate with successful extubation.[37]

Other signs of failed SBT include: the development of significant hypertension >180mm or hypotension less than 90mm, tachycardia, anxiety, diaphoresis.[38]

It can occasionally be difficult to tell whether a patient is failing because of their anxiety at having a large plastic tube in their mouth or due to an inability to breath. Examination of the patient (widespread crackles or wheezing generally indicate a

failure), a mild sedative to allow a longer period of time to query, or, occasionally (especially in the young anxiety prone - typically overdose - patients) an extubation with close observation.

A failed spontaneous breathing trial is a valuable source of information. It may indicate insufficient recovery from the primary process but can give clues to new emerging pathologies such as volume overload, VAP, oversedation, ischemia… Never let a failed SBT go to waste.

A failed SBT is typically not repeated more than once every 24 hours. Unless the cause was something imminently reversible whatever caused the failure is unlikely to correct rapidly. A failed SBT causes some degree of muscle fatigue that takes some time for recovery. Institute any changes that are necessary and repeat in 24 hours. Between SBTs full support has been shown to aid weaning.

Passed SBT

If a patient is able to tolerate a spontaneous breathing trial without signs of fatigue or the development of derangements in oxygenation, then they are likely to tolerate the work of breathing they require. Consideration for extubation should take place.

There are other causes of extubation failure other than ability to perform work of breathing. Once an SBT has been passed we should assess the patient for these other factors that can cause failure - airway protection and patency.

Airway Protection

Airway protection consists of two parts. Alertness and Ability to clear secretions.

Alertness essentially means their ability to protect their airway from emesis, from foreign bodies and saliva being aspirated. This is typically easy to assess simply by talking to the patient and observing their reaction. An alert patient will protect their airway.

While a patient may be alert, they may not have the strength or ability to clear secretions - a weak cough on suctioning or a severe dysphagia that limits the ability to expectorate secretions once they are brought up to the glottis are signs that the

patient may not clear their lungs. This is relative. A patient with a very poor cough but without secretions may do well, as might a patient with a strong cough but many secretions.

The decision to extubate a patient who is not alert is nuanced. Over 80% of those with a GCS < 8 do well with extubation.[39] The decision needs to take into account multiple factors.

Airway Patency

Airway patency is another consideration. If the patient was intubated traumatically or has been intubated for some time they are at risk for extubation failure because of airway patency. One test for airway patency is the "leak test" in which we deflate the endotracheal balloon and measure the amount of gas leakage around it. A leakage of more than 110cc indicates some airway patency. A failure of air leakage around the endotracheal tube when the balloon is deflated shows that there may be a risk of loss of airway patency with extubation. This is not always reliable but is recommended[40] for those at risk for airway edema (traumatic intubation, prolonged intubation).

If we suspect upper airway edema then a trial of steroids may help.[41] If the patient is ready for extubation and there is concern about loss of the airway then we can try to extubate with an endotracheal tube exchanger in place. A tube exchanger is a hollow tube that we keep in the airway to guide the replacement of an endotracheal tube. This allows a higher risk extubation as we can re-intubate the patient over the tube exchanger and, if that fails, we can oxygenate until we obtain a surgical airway.

Once they meet all criteria, the patient passes an SBT, is protecting the airway, and has a patent airway: then extubation can take place. We remove the endotracheal tube; we give the patient some supplemental oxygen source - typically humidified and we monitor closely for stridor.

Remember that we must adjust medication doses for the now extubated patient. Remove any high dose of PRN sedatives (an experienced nurse will not administer these but…).

Extubation Failure

A successful wean trial does not guarantee a successful extubation. We should carefully watch patients post extubation. We should defer procedures off the unit until the patient has declared themselves stable post extubation. Deconditioned muscles, poor nutrition, airway edema from the laryngeal manipulations, poor secretion clearance, mental status due to persistent drug levels can all lead to extubation failure. Laying a patient flat for "bathing", a small amount of emesis, an extra dose of narcotic all can decompensate a newly extubated patient.

Non-invasive ventilation can rescue a patient who is teetering after extubation. It must not be used when the patient is already in failure but rather when the risk is deemed high such as a patient with COPD or risk for pulmonary edema who is just extubated. Once a patient is in respiratory failure non-invasive ventilation will only delay a necessary procedure. This delay makes that necessary procedure riskier.

Prolonged Mechanical Ventilation

For some patients, liberation from the mechanical ventilator is difficult and protracted. Around 1-5% will become ventilator dependent.[42]

Prolonged mechanical ventilation results from the patient's primary disease process as well as the accumulation of complications from being mechanically ventilated. Weakness and deconditioning, VAPs, and delirium can all add to the difficulties the patient has in being removed from mechanical ventilation. As mechanical ventilation continues, the complications continue to accumulate making this a tough situation to correct.

Prolonged mechanical ventilation is variably defined as needing more than 21 consecutive days of mechanical ventilation (over 6hr/day) to needing more than 7 days of weaning after initial SBT.[43]

In a patient who is recurrently failing ventilator weans, we perform an aggressive search for the complications of mechanical ventilation. Look carefully for alternative diagnoses

that may not have been considered - look for occult pulmonary emboli, an undiagnosed neuromuscular disease, an untreated infection.

If the patient looks like they will need a very prolonged time on the ventilator, we arrange for a tracheostomy and perhaps a percutaneous feeding tube.

Tracheostomy

A tracheostomy offers the patient a more comfortable interface with the ventilator thus decreasing sedation requirements. It decreases the amount of dead space in the circuit and makes it easy to suction the patient.

Placement of a tracheostomy is a surgical procedure with all the complications associated with making a hole in someone's neck. Bleeding, infection, and damage to delicate tracheal structures can all happen.

The decision of when to place a tracheostomy has been controversial for some time. If placed too early patients may undergo a procedure that is unnecessary, if too late then complications from the endotracheal tube accumulate.

In some patient populations the decision to place early is clear - those with devastating neurologic damage or with neuromuscular disease such as Guillain-Barre syndrome in which the recovery will be very slow. In others we wait around 10 days and consider the procedure if the patient does not look like they will come off the ventilator.

Around the 7 day mark, a discussion with decision makers should happen about the tracheostomy. This is another opportunity to discuss goals of care. A prolonged period of time for recovery and the high burden of suffering as the lifestyle changes that entails may be something the patient would never have wanted.

Alternative Weaning Strategies

Different weaning strategies for those who are not coming off the ventilator include progressively decreasing pressure support or progressively longer periods of time on T-piece or tracheostomy.

Chapter Eight

Ventilation of Particular Scenarios

There are scenarios that emphasize particular aspects of mechanical ventilation. These are a few of these scenarios with a few tips.

ARDS

ARDS is a syndrome manifesting as an acute (within a week) lung injury, pulmonary infiltrates, poor oxygenation, and that is not pulmonary edema.

Many disorders that injure lung cause ARDS. ARDS can result from a primary pulmonary insult such as a pneumonia or inhalational injury. It can be due to a systemic inflammatory response that damages lung as can occur in pancreatitis or severe generalized inflammation.

ARDS results from diffuse alveolar damage which causes areas of consolidation and intrapulmonary shunting.

We divide ARDS into severity categories based on the ratio of PaO2 to FiO2. P/F ratio.

Severity	**P/F ratio**
Mild	<300
Moderate	<200
Severe	<100

There is no particular treatment for ARDS. We treat the underlying cause. The patient, however, has to survive long enough for the underlying cause to resolve and the lung to repair itself.

To do that, we need to be able to oxygenate the patient. Unfortunately, ARDS is a disease that can cause difficulty in oxygenation and ventilation due to its severity.

Mechanical ventilation of ARDS can, itself, cause damage, and most times there is a race between the damage we cause with mechanical ventilation and the speed at which the ARDS resolves (see Complications). Ventilation with low tidal volumes and limits on plateau pressure is the best way we have to control the damage we cause the lungs. Strict adherence to ARDSNet tidal volumes is very important.

In ARDS there are significant portions of lung that are consolidated. This consolidated lung tissue does not ventilate and so the tidal volumes given by the mechanical ventilator all go to whatever lung is not consolidated. This "baby lung" is very prone to injury from high tidal volumes and so low volume ventilation is particularly important in ARDS.

The management of severe ARDS beyond low tidal volume ventilation involves the management of refractory hypoxemia and the right heart.

Refractory Hypoxemia

If we cannot improve a patient's oxygenation through simple application of PEEP (<10cm) and FIO2 (<60%) we consider them as having refractory hypoxemia.

We use our understanding of VQ mismatch to change the patient physiology to overcome the hypoxemia. Several, but definitely not all, of these maneuvers also result in improved patient outcomes.

A more comprehensive review of ventilation modes is in a separate section. This section will just cover basic concepts used in common rescue therapies in refractory hypoxemia.

Increase Mean Airway Pressure

Increasing mean airway pressure will help oxygenation by recruiting and maintaining lung segments.

PEEP and Recruitment

An increase in PEEP will improve mean airway pressure, prop open airways, and recruit lung. This will usually improve oxygenation. Up to a certain point.

There are situations where PEEP can become detrimental. PEEP can squeeze shut pulmonary vasculature and decrease cardiac output. This can cause hemodynamic instability - particularly in patients who are volume depleted or have RV dysfunction. The lung with vasculature squeezed shut also contributes to dead space since it is no longer perfused and so decreases CO2 clearance.[44]

In some situations high PEEP can even cause worsening oxygenation by shunting blood away from ventilated lung and into consolidated (and thus protected from PEEP) lung.

PEEP should be raised carefully, in increments, ideally with the physician standing at the bedside monitoring. If capnometry is available it can help. If we are able to recruit more lung there will be a period of increased CO2 exhalation. A drop in exhaled CO2 happens if instead of recruiting lung we generate dead space or if cardiac output drops.

Recruitment maneuvers, as discussed, will help speed up an oxygenation improvement because of lung recruitment. Recruitment works with PEEP to aid oxygenation and on its own provides only temporary improvements. They are associated with hemodynamic instability and are no longer recommended for routine use.

Increasing I Time

Increasing inspiratory time can augment mean airway pressure and improve alveolar recruitment. In extreme cases we can even make the inspiratory time longer than the expiratory time - we term this inverse ratio ventilation (vs normal ratio ventilation with i longer than e). A discussion of the ventilation modes is in a separate section.

Minimize Oxygen Utilization

Agitated or dyssynchronous patients use a lot of oxygen. Febrile patients use a lot of oxygen. Steps to minimize oxygen usage help in the patient with refractory hypoxemia.

Sedation is the first step in decreasing the excessive oxygen utilization from agitation and dyssynchrony. We should use combinations of analgesics and anxiolytics until the patient is comatose. If there is still significant dyssynchrony, then we can start paralytics.

Treat fevers and shivering with antipyretics. Cold compresses can help bring down fever.

Paralytics

Paralytics are drugs that will relax skeletal muscles. When given during mechanical ventilation they allow for much improved ventilator synchrony and minimize use of oxygen by skeletal muscles.

Paralytics used early for 48 hours have been shown in one smaller study to improve survival without increasing muscle weakness.[45] The reason for this is unclear, but one hypothesis is that the decrease in transpulmonary pressures with the silencing of the patient's respiratory efforts plays a role. This is not a consistent finding, and other studies[46] showed no benefit. If a patient has underlying reasons not to use neuromuscular blockers, we should withhold them.

Longer use of paralytics is associated with muscle weakness and have significant downsides.

Only use paralytics once generous doses of sedation have made the patient unconscious.

Prone Positioning

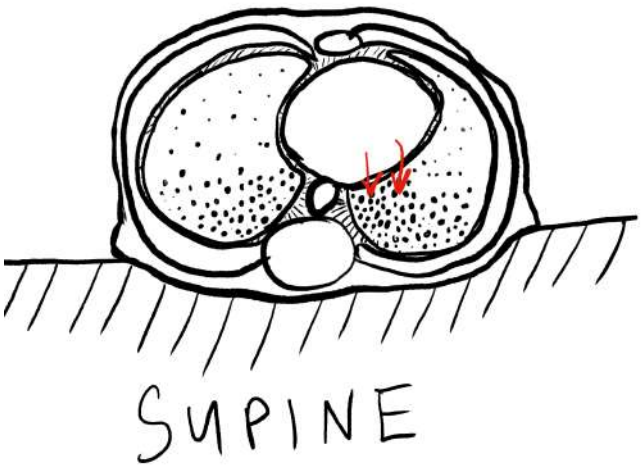
Prone positioning of the patient has the effect of improving oxygenation in a significant portion of patients with severe ARDS. If used early in severe ARDS prone positioning can improve survival.[47]

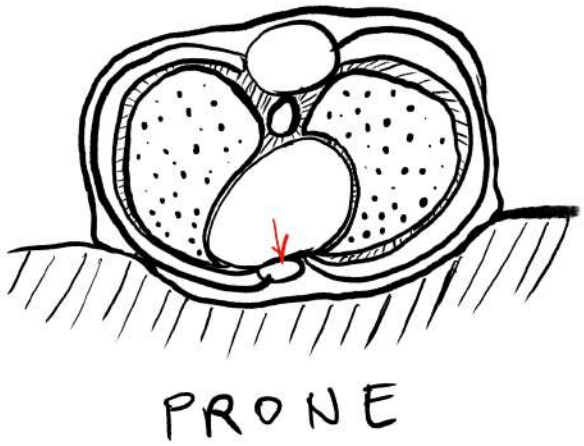
Reasons for Benefit[48]

During normal breathing, the flattening of the diaphragm ventilates the dependent bases of the lungs. This dependent region is very well perfused and so the extra ventilation helps maintain ventilation to perfusion ratios. In contrast, during positive pressure ventilation in the supine position, the ventral chest wall is more mobile, ventilates more, and so leaves the dependent caudal lung poorly ventilated. Prone positioning eases this somewhat, making the ventral chest less mobile and allowing better ventilation of the caudal region.

The lung tissue sags. In the supine position, the weight of the lung puts more tissue in the dependent caudal region. This density of tissue with its dense vasculature sits in poorly ventilated regions. To aggravate the situation the weight of the heart also sits on the left lower lobe also further compressing it.

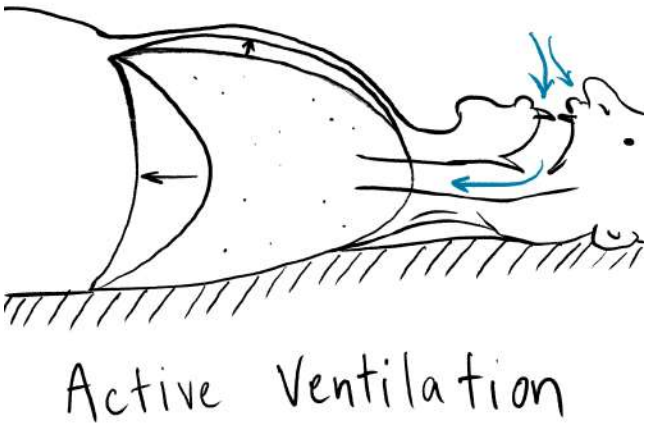
In the prone position, the distribution of both ventilation and tissue is more even. The caudal region continues to receive more blood (explained as due to regional differences in vascular diameter and NO production) but is better ventilated.

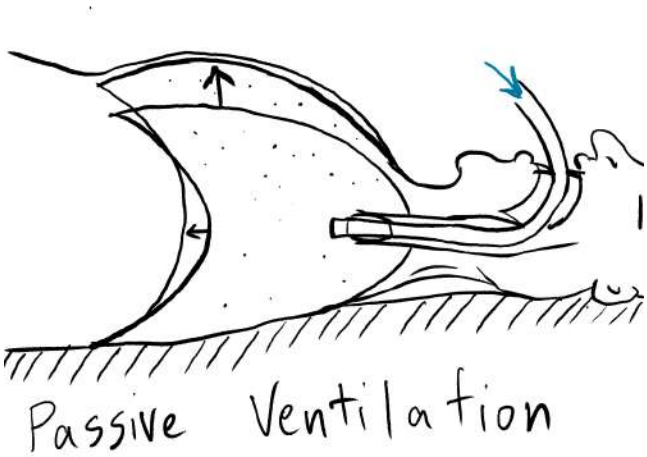




The improved ventilation/perfusion seems to explain why some patients, particularly early in the ARDS process, respond to prone positioning with an improvement in oxygenation.

There are other benefits of prone positioning which include better mucous drainage and a relief of abdominal weight off of the caudal diaphragm.





Inhalational Vasodilators

Inhalational vasodilators can increase flow to well-ventilated areas of the lung. This improves blood flow in these areas and steals flow from areas which are less ventilated. Improving blood flow improves VQ matching and helps correct hypoxemia.

Inhalational vasodilators can improve oxygenation in particular patients. They have never been proven to improve survival and are expensive. They are useful to buy time, however, while the patient improves or we make arrangements for alternate therapies such as ECMO.

The available agents are inhaled Epoprostenol or inhaled nitric oxide and use depends on institution. If using nitric oxide then methemoglobin levels need to be monitored.

HFOV

One option to raise mean airway pressure is high frequency oscillator ventilation. This is a means of ventilation using extremely small breaths (smaller than dead space) administered at a very high frequency.

The purported benefit is a high mean airway pressure with good recruitment and little atelectotrauma as the tidal volumes vary little. The modality improved oxygenation in some patients.

The major drawback is that the high mean airway pressures and high frequencies can cause autoPEEP, barotrauma. The high pressures can also impede right heart function and even generate dead space.

No mortality benefit in adults and in one study increased mortality, this mode of ventilation is no longer recommended.[49]

ECMO

Extracorporeal membrane oxygenation is an option in the refractory hypoxemia patient with the prospect of relatively rapid improvement.

In ECMO, we place large catheters into the patient. We remove blood from the patient, pass it through an oxygenator (a set of thin tubes through which blood comes into proximity with oxygen) and then return it.

ECMO is expensive, labor intensive, and can come with multiple complications. The placement of the required catheters can cause mechanical complications such as tears and bleeding. The catheters can get infected. The circuit used to move blood can clot

or disconnect. The patients are placed on anticoagulants to prevent clotting of the circuit and this can cause bleeding.

The complications accumulate over time and so once we initiate ECMO the patient has a finite amount of time to recover and get off before some complication occurs. For this reason the patient should have a relatively quickly reversible cause of the respiratory failure such as pneumonia or ARDS from a reversible cause.

The only exception to requiring a reversible cause is in patients listed and awaiting a lung transplant. In those cases, a bridge to transplant with a brief period on ECMO is acceptable.

To help decide on whether a patient would benefit from ECMO we can estimate their survival using the RESP Score[50] at www.respscore.com

Patients must also be able to tolerate anticoagulation and have vasculature amenable to cannulation.

Although ECMO itself hasn't been proven to improve mortality in ARDS transfer to an ECMO center did improve mortality of patients with severe ARDS[51] although the study has been criticized for the higher use of lung protective strategies in those transferred.

Extra-pulmonary Shunts

In a patient with refractory hypoxemia, and without significant parenchymal disease to explain it, or in whom there is a paradoxical worsening of oxygenation with PEEP you must think about the possibility of an extra-pulmonary shunt.

These shunts can remain silent until the right-sided pressures rise sufficiently to push them open and allow for admixture of right-sided blood with left-sided blood.

There are many factors associated with mechanical ventilation and lung disease that can raise right-sided pressures. The intubation and gas pressure applied to the lung collapses pulmonary vasculature and raises right-sided pressures. Hypoxemia, hypercapnia, and lung disease itself can cause right-sided pressures to rise.

We can diagnose extra-pulmonary shunts, such as a PFO, with a bubble study. A small amount of agitated saline is injected while imaging the heart. The agitated saline forms bubbles visible on echo. If these bubble cross from the venous to the arterial circulation there is a shunt. The timing of the crossing (early vs late) can help identify whether the shunt is intrapulmonary or extrapulmonary). Intra-cardiac shunts show left sided bubbles usually within 3 heart beats.

In the case of an extra-pulmonary shunt medical management is limited to decreasing right sided pressures: minimizing oxygen utilization, optimizing hemodynamics by decreasing the right sided pressures through diuresis, limiting PEEP, and correcting reversible factors such as acidosis.

The Right Heart in ARDS and Cor Pulmonale

The heart lung interactions discussed previously are especially relevant in ARDS. In severe ARDS the right heart is stressed and its failure leads to hemodynamic instability and organ failure. The development of right heart failure (cor pulmonale) is an ominous sign associated with worsened mortality.[52]

Factors that lead to failure of the right heart in ARDS[53] are:

- Worse oxygenation
- Hypercapnia
- High ventilator pressures
- Pneumonia as a cause of the ARDS

Hypoxemia can pulmonary vasculature to constrict. The lung's vasculature is unique in that it vasoconstricts in hypoxemic regions. This functions to redirect blood flow away from these regions and so improve ventilation to perfusion matching. In ARDS hypoxemia can cause the vasculature to constrict and increase pulmonary pressures and impede right heart function.

Acidosis can cause pulmonary vascular vasoconstriction too and this can be a problem with ARDS in which there is significant dead space and a respiratory acidosis develops.

In ARDS, lung volumes can be an issue. The smaller amount of ventilated lung means that tidal volumes given by the ventilator are all concentrated in a smaller area. This can function to squeeze shut the vasculature in that area and so impede the flow from the right heart.

Decreased lung volumes help with pulmonary vascular resistance. Lung compliance is associated with the amount of consolidated lung and how much lung is being ventilated.

With competing parameters (plateau pressure, tidal volume, PEEP) one parameter seems to link the others and correlates with mortality - the driving pressure. The driving pressure is the difference between the plateau pressure and the PEEP. A driving pressure kept below 18mm was associated with improved survival.[54]

The right heart needs to be managed in ARDS. A strategy[55] of low tidal volumes, driving pressures below 18cm, maintaining PCO2 less than 50mm, and use of prone positioning has been proposed in patients with severe ARDS to protect the RV.

Fluids and ARDS

We usually treat the hypotension that can occur from the right heart strain and the patient's primary disease with fluid boluses. Fluids in this situation will place a load on the right heart and may worsen things. A right ventricle in distress bloats and enlarges.

The left and right ventricle are contained within the pericardium and share the septum. This causes the two ventricles to be interdependent. The enlargement of the right ventricle due to failure and fluid overload will squeeze the left ventricle eventually cause it to fail.

Even low levels of vascular impediment and shock will cause the release of stress hormones that favor fluid retention. A patient in ARDS will retain fluids and over time become volume overloaded. Anyone on mechanical ventilation and under stress will develop volume overload given enough time.

Patients with ARDS who are treated with a conservative fluid management strategy have improved survival.[56] If the ARDS patient is not hypotensive, then we should give diuretics to keep fluid balance negative to even to avoid volume overload and right heart strain.

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Severe Obstructive Lung Disease and COPD

In patients with severe asthma exacerbations or COPD, mechanical ventilation runs into particular difficulties. The principal problem is the limitation set by the high expiratory time. These patients require a lengthy period of exhalation time to allow the lung to deflate through the obstructed small airways.

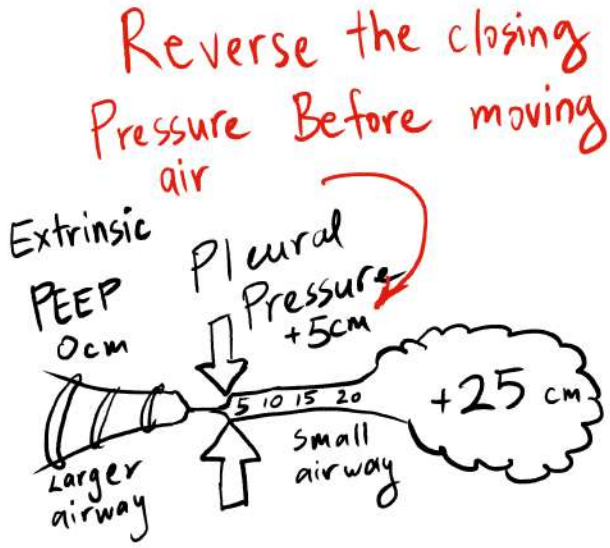
In many patients with obstructive lung diseases, some degree of autoPEEP is unavoidable. The effect of the autoPEEP is a raised mean airway pressure with all its consequences.

Raising extrinsic PEEP can help decrease work of breathing and correct dyssynchrony

Patients with significant outflow obstruction can have very high work of breathing causing fatigue and discomfort. The autoPEEP that most of these patients experience can even cause failure to trigger.

For a patient to trigger the ventilator they must decrease the pressure in the circuit below ambient pressure. The ambient pressure in the circuit is the set PEEP. The pressure inside the patient's chest, however, is the autoPEEP value. The patient will need to overcome the autoPEEP and then after that continue and overcome the set PEEP to trigger the ventilator. This is a significant amount of extra work.

Increasing the PEEP level brings the set PEEP closer to the autoPEEP making it easier to trigger the ventilator and thus decreasing work of breathing.



The explanation as to why any PEEP does not impede exhalation in obstructed patients is that the set PEEP remains behind closed airways. The small airways that drain obstructed segments are collapsed at regions were the internal pressures are overcome by external pleural pressures. PEEP behind this obstruction will not worsen the air trapping. We explain this with a metaphor - a waterfall.[57] We can think of the large airway pressure as the lower level, the alveolar pressure as the upper level, and the distance the water falls as the difference between PEEP and autoPEEP. Whatever the height of the lower level, as long as it is below the upper level, water flow is unaffected.

This is unreliable because we don't know what the opening pressure is, and how many segments of lung are obstructed. We should do it with caution. An increase in PEEP, in the wrong patient, can worsen autoPEEP by impeding exhalation.

We increasing PEEP in an obstructed patient very carefully. We do it slowly, standing next to the ventilator. At each increment we monitor tidal volumes and measure plateau pressure. If the plateau pressure (in a volume-targeted breath) is rising (or the delivered tidal volume in a pressure-targeted breath decreases) then the patient is experiencing an impairment of exhalation and we should abort the procedure. If the plateau pressure does not rise then we can continue slowly until we find a good extrinsic PEEP. That is usually around 80% of intrinsic PEEP.

Increasing PEEP may even improve exhalation in patients with flow obstruction. The lungs segments are heterogeneous. There are areas with higher pleural pressures because of the weight of the lung. This generates segments of lung that are more prone to autoPEEP due to the being exposed to higher closing pressures. The application of PEEP can help open these segments thus help even the distribution of inflow gases.

Increasing PEEP in a patient without flow obstruction, however, does not improve autoPEEP and will increase hyperinflation and barotrauma.

Elevated Dead Space

In obstructive lung diseases there is significant ventilation/perfusion mismatching because of uneven distribution of ventilation in the diseased lungs. Segments of lung with obstructed small airways are ventilated without the gas reaching the acini. Resulting high VQ segments waste ventilation - increase dead space ventilation.

The increase in dead space means that even supra-normal minute ventilation may not be enough to maintain normal CO2 levels. In some disorders such as cystic fibrosis the amount of dead space from the ventilation/perfusion mismatches and obliterated small airways can be quite significant[58].

Fortunately, we can tolerate elevated CO2 levels and allow for "permissive hypercapnia". Permissive hypercapnia entails allowing minute ventilation that does not correct the hypercapnia but suffices to prevent hemodynamic compromise.

Permissive hypercapnia allows for a pH down to 7.2 as long as there are no significant adverse events and no contraindications.

In patients with severe obstructive lung disease that has resulted in renal compensation, we should adjust the patient's ventilator settings to the pH and not the CO2. If we mechanically ventilate a patient with chronic compensated hypercapnia and normalize his CO2, after some time then his renal compensation will resolve. This patient will subsequently have trouble once extubated. He will now need to maintain the normal CO2 level and may not be able to do this. Allowing the patient to keep his usual elevated CO2 will help him keep the renal compensation.

As discussed in the section on Setting the Mechanical Ventilator there are contraindications to hypercapnia.

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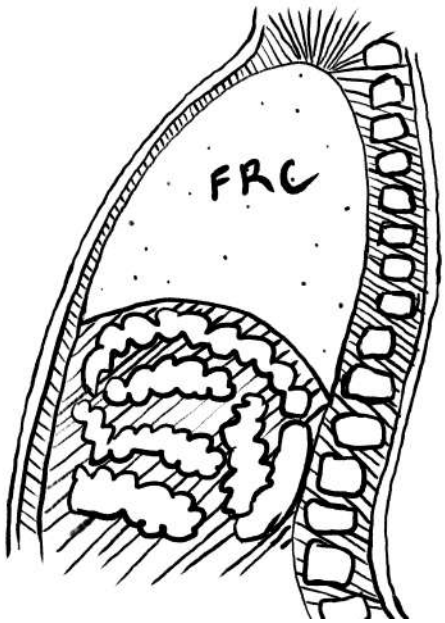
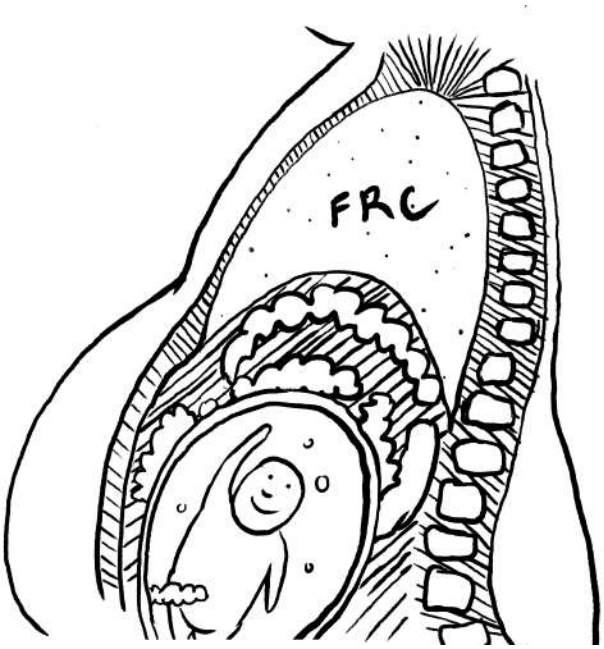
Pregnancy

Pregnancy is associated with several physiologic changes in the respiratory system. There is a decrease in FRC due to the gravid uterus, a decrease in chest compliance, and an increased utilization of oxygen because of the parasitic fetus. These changes make the pregnant patient less tolerant to disturbances that stress the cardiopulmonary system and place a usually young and resilient population at risk for requiring mechanical ventilation.

Normal pregnant physiology causes an increase in tidal volume and a resultant mild respiratory alkalosis. Normal pCO2 is 27-34 due to the stimulatory effect of progesterone. The decreased FRC due to the gravid uterus as well as the higher oxygen utilization from the fetus cause a tendency to rapid desaturation on induction.





Endotracheally intubating the pregnant patient is also more risky - the pregnant patient is always considered a difficult airway with a full stomach. The rapidity with which the patient desaturates as well as mucosal edema and capillary engorgement make the airway difficult. The engorged mucosa also requires a smaller endotracheal tube size.

In pregnancy there is a loss in the lower esophageal tone, an increase in intra-abdominal pressures, and a delay in gastric emptying. These changes place the patient at increased risk for aspiration. If we choose to use non-invasive ventilation the patient needs to be alert, have brisk protective airway reflexes, and stable hemodynamics. Non-invasive ventilation in these patients requires especially close monitoring because of the higher risk of aspiration and should only be used for rapidly reversible causes of respiratory failure.

For pregnant patients on mechanical ventilation the goals are to maintain a PaO2 of around 70mm,[59] an oxygen saturation of around 95% as opposed to the usual recommendation of > 92%.

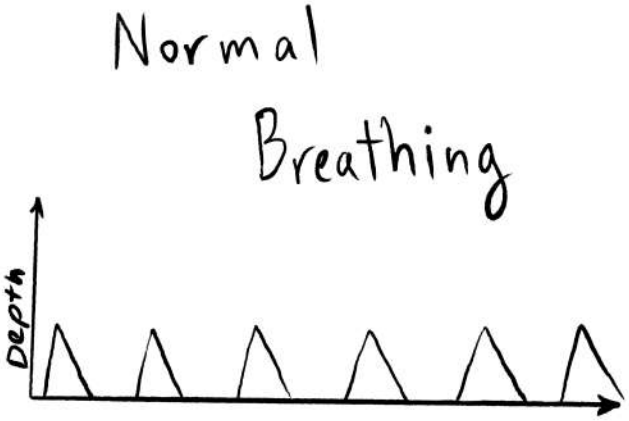
The fetal CO2 removal requires that there be a CO2 gradient of around 10mm. Permissive hypercapnia can result in fetal acidosis and has not been studied in pregnancy. Respiratory alkalosis above 7.48[60] can cause uterine artery constriction and also should also be avoided.

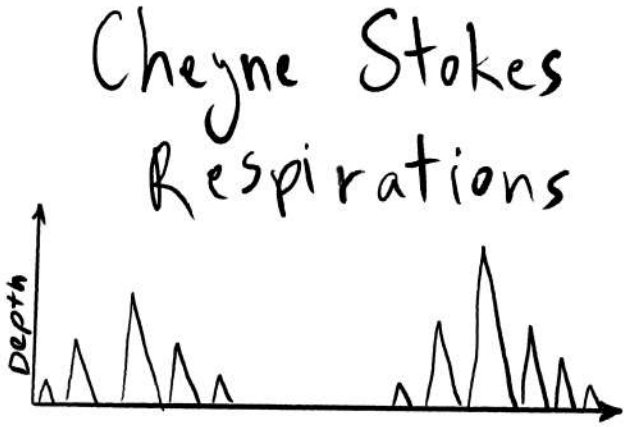
There is a loss of chest wall compliance and a rise in the diaphragm with pregnancy. Although this decreases transmural pressures and likely protects the lungs from overshooting the ARDSNet target of 30cm of plateau pressure (See section on barotrauma) some recommend use of the ARDSNet Pplat targets anyways to ensure safety and to reach normal CO2s by increased respiratory rates.[61]

Abnormal Breathing Patterns

Cheyne Stokes Breathing

Cheyne Stokes breathing is characterized by cycles of increasing breath depth and frequency followed by gradual decrease until there is a period of apnea; a crescendo-decrescendo pattern.





If is important to recognize this abnormal pattern because the ventilator will alarm during the various cycles with different alarms and without recognizing this abnormal breathing pattern the result is usually an increase in sedation and escalating complaints from staff that the patient is dyssynchronous.

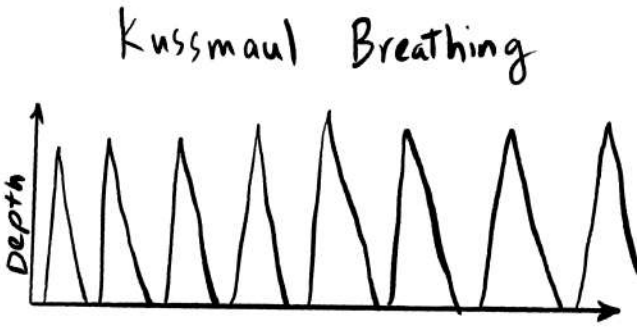
The ventilator will cycle between the deep frequent breaths with resultant flow dyssnychrony and double triggered breaths to the apnea period with complaints of patient apnea.

The mode chosen should not be a dual target mode since the varying respiratory efforts will result in varying levels of support. A mode that guarantees some minute ventilation (a assist/control mode). Using a pressure targeted breath type would alleviate the flow dyssynchrony associated with the crescendo phase.

During wean trials the ventilator will trigger the apnea alarm during the apnea phases and this alarm can be extended somewhat to allow the wean trial to continue while the patient is being monitored.

Kussmaul Breathing

Kussmaul breathing is the deep fast breathing associated with a severely air hungry state. It is seen in situations of significant metabolic acidosis such as in those patients with DKA.



The problem with Kussmaul breathing is the associated ventilator dyssynchrony. The associated extremely deep breaths are necessary to maintain an adequate pH and prevent hemodynamic compromise. Forcing the patient to a more normal breathing pattern will have the effect of causing a severe metabolic acidosis and its subsequent complications.

The remedy to Kussmaul breathing is to correct the underlying cause of the metabolic acidosis. A temporizing method of generating synchrony with the ventilator is a low pressure support mode which will allow for the deep breaths without generating the double triggering.

Patients on ECMO

ECMO is a modality that is becoming more common in patients with refractory cardiogenic shock or hypoxemia. In this modality, an external oxygenator replaces lung function. One of the great benefits of this is that we can avoid injuring the lung with aggressive ventilator settings.

Once the patient is on ECMO, we should down rapidly wean ventilator settings to the minimum support that allow maintenance of oxygenation and ventilation. In a patient with sufficient blood flow through the ECMO circuit, we can completely replace lung function. We should then place this patient on a low pressure support mode with a PEEP of 5-10cm, just enough to keep the lungs inflated. If the patient is awake and can protect the airway, then we should consider extubating the patient.

Pneumothoraces and Bronchopleural fistulas

In a patient with a pneumothorax with a persistent leak or a bronchopleural fistula, there is a continuous leakage of air through a puncture in the lung into the pleural space.

Mechanical ventilation can worsen this situation by generating large pressures that prevent the puncture from closing and healing. There is no specific ventilation mode or method that is recommended.

The ventilator settings in the case of a bronchopleural fistula should avoid elevated pressures and volumes. We should use a minimal amount of PEEP. We prefer a low volume-targeted breath in cases of sizable leaks. This is to prevent the continued inflation of the lung that will happen with pressure-targeted breaths as pressure is lost through the puncture.

Patients with significant leaks are also at risk for auto-cycling as the loss of volume from the leak triggers the ventilator. This can cause respiratory alkalosis and all its implications. If the leak is sufficient to cause this then the ventilator settings need to be adjusted.

Right Heart Failure and Pulmonary Hypertension

In patients with pre-existing pulmonary hypertension and right heart failure intubation and mechanical ventilation are very high risk procedures.

The stresses on the right heart discussed in the sections on heart-lung interactions and ARDS apply here too. In this scenario the patient's have pre-existing pulmonary hypertension and right heart failure.

The initiation of mechanical ventilation in these patients risks precipitating an acute decompensation. The rise in pulmonary vascular resistance can devastate an already struggling right heart.

We should anticipate the hemodynamic compromise. Vasopressors should be readily available.

Morbidly Obese

The morbidly obese patient shares some of the characteristics of the pregnant patient. The FRC is reduced due to the heavy belly and chest. Oxygen consumption is increased due to the increased work of breathing.

The accumulated comorbidities and age in this population make it more likely for the lung parenchyma to be lax. In addition to that the pleural pressures will be higher due to the weight of the chest and organs. The closing pressure for lung segments may be reached quickly, particularly in the supine patient. This makes these patients more prone to autoPEEP with increased respiratory rates.

Ventilator setting is similar to the non-obese patients. The ARDSNet trial excluded patients who weighed more than 1kg/cm but an analysis of the trials found similar outcomes in a 200 obese patient subgroup.[62] We should calculate tidal volumes using the ideal body weight which may be substantially less than the actual body weight in these patients.

We may need higher PEEP and plateau pressures due to the heavy belly and chest. The transpulmonary pressures may be acceptable because of the restricted chest but high PEEP has not been studied in this population.

We have used prone positioning in morbid obesity and is effective.[63] There may, however, be more technical difficulties with ECMO because of the body habits.

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