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Alright folks, let's just go at it. So starting the respiratory system today, this is the hardest system that I teach. It typically gives students the most difficulty. And so we'll go through it as methodically as we possibly can. This first page over here, you're not going to have to know all these terms, just a few of them euthanasia, hyperventilate, hyperventilate, these are things that I'm going to be talking about more detail at a later time. So there's really no need for me to talk about it much right now. Although I will talk about euthanasia in a little bit, hyperventilate, hyperventilate, you're gonna have to wait for that for other lectures. So we'll move forward. Let's talk about the airways. Now, you guys learned all about the airways and the progression of air through the lungs, all these anatomical structures, we, as physiologists take anatomical structures, and we divide them into what are called zones. And so what I'm going to do is I'm going to draw this except in a different way. And the way I'm going to draw it is a way that you're going to see many times when we talk about the respiratory system. So this tube here is going to represent everything from nerese, all the way down to the terminal bronchial and I'll label it.

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So to draw a big circle. And then I'm just going to draw a dotted line over here.

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If the pen would work, there we go. So all the way from the Nerys down to the terminal, Brock eels and everything in between. We as physiologist lump all that together and call it the conducting zone. I will often just abbreviate it C, z, this is easier to write. Then from the respiratory Brock eels. Again, you should be familiar with all of these terms, all the way down to the alveolar ducts. And I'll just put it all the way down here. We call the respiratory zone. And often I will just call that rz. The reason that I have this as a circle, even though these are tubes, of course, is because within the respiratory zone is located all the alveoli. And so I'll put that over here as well. So contains the alveoli. And we have about a billion alveoli, about 500 million per lung. And so what we're going to first do is we're going to talk about the function of the conducting zone, which is once again all the anatomical structures between the areas down to the Brock eels. And again, we're going to see this picture many many times during these lectures. And so let's first start with functions of the conducting zone controls the volume of area to and out of the respiratory tract. This is going to be mainly the terminal bronchial

that does this. And so what we'll do now is I'm just going to draw a simple tube. And that tube is going to be once again the terminal bronchial and I'll label it. So this is a function of the conducting zone. So this tube right in the middle of the page here, that's the terminal Brock eels, not label it. Those terminal Brock eels have wrapped around them smooth muscle cells just like your blood vessels do. And those smooth muscle cells are going to control the diameter the radius of these airways. And so one of two things can happen. We can dilate. So those smooth muscle cells can relax. And so we have that happening. It's called Bronco dilate. And certainly what that's going to do is it's going to increase airflow. Now, how is that going to happen? There's one way we know because we talked about it and learned it last semester. You get the beta two receptors, if you recall, which are inhibitory memory, it's an even number, it's inhibitory. Then we can go the other direction. And obviously, the opposite is going to happen over here. On this side. We're going to Bronco constrict, so the tubes get smaller. And how does that happen? Well, the smooth muscle contracts. And so of course, we're going to get a decrease in airflow. And we call this Bronco constrict. And how does this happen? It happens and by the way, the beta two receptors aren't the only way that this is going to occur. It happens in other ways as well. The example I'm going to give over here on this side is one of the ways that can happen and the reason I'm going to give this example is because we learned it last semester these would be via the muscarinic receptors if you recall, which are acetylcholine receptors. We learned about this last semester. Now why would the airwaves be done Eating and why would the airways be constricting? I just give a couple of examples so why would we Bronco dilate so for example, during exercise when you exercise you activate your sympathetic nervous system it releases what norepinephrine onto the beta two receptors epinephrine, norepinephrine release from the adrenal gland, they bind to the beta two receptors, we Bronco dilate. Of course, we want to do that when we exercise, we need to get more air into the lungs carries oxygen, we need to get more air out of the lungs, because we get rid of the excess co2 that's produced during metabolism. Now, why would we Bronco constrict? I'm going to put here in quotes, polluted air. And polluted air can mean a number of things. One is just crappy air that we breathe, it's a it's a reflex that occurs that tries to prevent the crappy air from getting into the lungs. A little bit more on that in just a little bit. So once again, this is a function of the conducting zone is controlling air in and out of the lungs by controlling again, airway tone. And so that is here. So what we just drew is, right there. What else does it do? Every single time you take a breath, and that air is going to be at room temperature, I'm sorry, body temperature and 100%. Human. So back when it was what 10 Below,

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we were walking around outside, we are breathing in that air like that that air that got into our airways was warmed to 98.6 degrees. And it happens really, really fast. You can be outside where it is hot as hot can be you go into a hot sauna where the temperature is like 150 160 degrees. When you breathe in that air, when it gets into your airways 98.6 degrees. That was one of the things that people were doing during COVID. They went into these hot saunas thinking that that hot air was going to kill the virus. Complete nonsense. It wasn't working. Because again, the air is brought to our body temperature is very important that it's at our body temperature. And again, it's 100% human. So that's another function of the conducting zone. And how does that happen? It's very well vascularized those airways. So the exchange that happens with those blood vessels is going to keep that air where it's supposed to be 98.6 degrees roughly. What else does the conducting zone do for us? It protects us from the air that we breathe. Now our nose hairs mucus traps very large particles. So as we breathe in air, it gets filtered that way. We Bronco constrict something that I just mentioned when polluted air is breathed. So we're going to go back to the picture that we just drew. Again, this is a protective mechanism. You're breathing in crappy air, the airways constrict to limit the amount of crappy air that you're breathing. If you smoke a cigarette, your Bronco constricted for about 20 minutes. If you're a chain smoker, you're continuously Brockville constricted. I'll go to an extreme

example. You guys ever hear people that die in fires? Typically the fire doesn't kill them, what kills them? smoke inhalation, I'm gonna give you two reasons why one now another one later. It's right here. So the fire that's occurring is creating all this smoke. People are breathing in that smoke and they severely bronchoconstriction they suffocate to death. They cannot get enough air into their air into their lungs. Why? Because this is happening. It's a reflex that occurs as an autonomic reflex. Yes.

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Yep, that's, that's another reflex that occurs. Doesn't have to do with this. It has to do this in what are called irritant receptors that cause that cough reflex to try to desperately get the crap out of their lungs. Yep, the question was, he talked about that later. Next lecture. I do believe we'll talk about that. All right. And I want you to know that, Oh, there's another reason that people die in fires, and it's not the fires carbon monoxide poisoning. So I'll talk about carbon monoxide poisoning and a couple of lectures. Alright, so again, polluted air. polluted air can be extremely polluted like an ice fire can just be something like cigarette smoke or just the air that we breathe, we can be a little bit Bronco, constricted, depending on how dirty the air is. There's another way that we are cleaning the air as we're breathing it in. And that is via the mucociliary escalator I'm going to show you a pretty picture this I used to draw it but they were crappy drawing. So we're just going to look at these two pictures here. The one on the right is a photo micrograph the one on the left is a cartoon picture. We're going to concentrate on the cartoon picture then the same exact thing. So to acclimate you what this is the wall of the the airway right here, so it's like a sagittal section. So we can see what the wall All is made up of. So we have this connective tissue over here. And then we have two sets of cells here we have these cells that have cilia on them. And then we have these cells over here, kind of an orangey, color, tan color, those are called goblet cells. Together, those two cells make up what's called the mucociliary escalator. And so what happens here is this, these two types of cells work together, these goblet cells produce mucus, I think you guys probably learned about goblet cells, right? So they produce mucus. And that's what this is right here. So yeah, that's a little bit darker and easy little things in it. Those are foreign particles that are trapped in those foreign particles, we're in the air that you breathe. Now what kind of foreign particles, pollen, viruses, bacteria, pathogens, just stuff like that, that we don't want in the lungs. So these goblet cells produced the mucus, the mucus traps, whatever it is that we're breathing in, at least most of it. And then what these ciliated cells do, is they cause the cilia to beat in such a way that it moves all that mucus to the back of your throat, and then what you swallowing, you're doing it 24/7, you just don't know it, you are constantly swallowing this mucus, because it's constantly going to the back of your throat. And what that's going to do is it kind of clears the airways. And when it works properly, most of what we are breathing in that contains all these foreign products, go to the back of the throat ends up in our stomach, the essence of our stomach, take care of the rest. And we have some conditions, though, that can affect mucociliary escalator. And so let's talk about cold dry air slows the cilia down doesn't stop them, but slows them down. If you slow the cilia down, it's not going to move the mucus to the back of the throat quite as efficiently. And so what that means is is that the foreign particles, and they could be viruses and bacteria can then increase your incidence of developing a respiratory tract infection. One of the reasons it's not the only reason that people tend to get more respiratory tract infections in the wintertime is because of that is in the winter, or at least up here in the North, cold, dry air. So cold dry air affects the cilia, what else affects the cilia smoking. So smoking not only slows the cilia down, can almost paralyze the cilia to where they don't move very well at all. And once again, if you're a chain smoker, you're going to have a difficult difficult time moving that mucus. If you smoke for a long enough period of time, you won't only just slow the cilia down, you will actually damage the the airways themselves. And then these particular cells right here, the epithelial cells with the cilia are gone forever. They are destroyed. So now I asked you why and by the way, the goblet cells are

completely fine. Smoking does not affect the goblet cells, if anything, smoking increases the production of mucus. So if you don't have the ciliated cells, how are you going to move the mucus to the back of the throat?

13:10

You are not? You do not have the ability to do that. So what happens is that the mucus builds up, builds up, builds up builds up, it activates these irritant receptors and then what do you think you do to get the mucus out of the earlier with you call you hear a smoker's cough. It's very, very wet. They're drowning in their own mucus because they're ciliated cells are gone. And so obviously, they're going to be at a very high risk of developing respiratory tract infections. There's a tremendous amount of inflammation that develops, which then can lead to diseases like emphysema, chronic bronchitis, cancer, all these bad things. Is there anything worse for you than smoking? So anyway, cigarette smoke can not only slow them down, but damage them and damage them permanently. So those were the ciliated cells. Let's talk about the goblet cells. Can we affect the goblet cells we can? Cystic Fibrosis last semester we talked about cystic fibrosis when we talked about the skin remember that? We talked to how you diagnose cystic fibrosis by testing sweat at the level of chloride is five times what it's supposed to be in sweat. Now that person has a cystic fibrosis diagnosis. So cystic fibrosis is a condition where it affects the chloride jam. And that chloride channel is involved in producing the mucus. And that the chloride channel is not working properly. The mucus that is produced is very, very thick. So what happens with cystic fibrosis is we have thickened mucus, we don't have it. We don't have any problems producing the mucus. As you said, the product is very thick, it's very viscous. The ciliated cells are not affected at all. But if we have thick mucus, do you think it's easy to move the mucus? It is not. And so the problem is Cystic Fibrosis one of the process because it also affects the digestive system is that we have trouble moving the mucus to the back of the throat. Typically people who have cystic fibrosis pass away because of respiratory issues, because of this issue right here, and that is thick and mucus. Cystic fibrosis is almost always diagnosed in infancy. And there are two things that are telltale signs of cystic fibrosis number one, failure to thrive, that is the baby doesn't grow because it's affecting the digestive system to lots of respiratory tract infections. So if a pediatrician hears those two things immediately, that pediatrician should think that baby might have cystic fibrosis, they do the test. And they see whether or not now if you have a lot of respiratory tract infections, and you have a baby that's not thriving, it doesn't mean that the baby has cystic fibrosis, but it's certainly one of the differentials that you have to look into. Now, if that baby has that thick mucus and has trouble getting mucus to the back of the throat, there's not a whole lot that can be done to cement it, my thin the mucus a little bit. But one thing that mom and dad are going to have to do frequently during the day is take that baby and do this chest, turn the baby over hit the back, what do you think they're doing? There trying to loosen up the mucus, get there some vests that you can get nowadays that kind of do it, the percussion itself. But we got to get that mucus loosened, why? Because if not, those foreign particles, many of which are pathogens will just sit there and fester. And again, that leads to very complicated things. So no one to mucociliary escalator is no it can affect it. And I think that's it for that. Now what now let's go on to the respiratory zone. So we just spent our time talking about the conductor's conducting zone and in the functions, let's talk about the respiratory zone. And it's one function that we're going to talk about it has other functions, but we're not going to discuss them. This will be the one function that we're going to concentrate on when it comes to the respiratory zone and its gas exchange. So we're going to draw this now. So I'm going to draw that again. So I'm going to draw the conducting zone is going to be part of the the picture itself, but we're going to focus on the respiratory zone. So again, that circle is drawn the way that it is even though again, the bronchial the respiratory brockers In Avila alveolar ducts are tubes. That circle represents all the alveoli. And so I'm also going to put in this picture, a red tube.

17:32

And that red tube is a pulmonary capillary. And you guys know what that is? Least I hope you do. It came from pulmonary artery arterioles, which came from pulmonary arteries, which came from the pulmonary trunk, which came from the right ventricle, right. So why is that in the picture, because we're going to talk about gas exchange. So we're going to have exchange of gases here, the two gases that we're going to exchange our oxygen and carbon dioxide, at least that we're worried about. And this is a way that it happens. Oxygen is going to diffuse from the alveoli into that pulmonary capillary and co2 is going to diffuse from the pulmonary capillary into the alveoli. That's gas exchange. Now, why is that happening? Well, you guys already know because well, you already learned it. That's how we're oxygenating the blood at the lungs, right? Right side of the heart has deoxygenated blood, it goes to the lungs to become oxygenated. I'm showing you how it happens. Now, we're going to go into this in a crap ton of detail in a couple of lectures for right now, this is all we have to know. So gas exchange, why does this occur to oxygenate the blood. And to get rid of excess co2. Obviously, the co2 is leaving the blood, we need to get rid of the excess co2 in the body is going to be part of acid base balance. We'll talk about it later on in the semester. So gas exchange is occurring here. And again, this is the respiratory zone. This is the conducting zone. And so gas exchange occurs here. Because we have alveoli. Without the alveoli, you're not going to get this type of gas exchange and there are no alveoli in the conducting zone. Right. And you guys know that the alveoli don't start until we get to the respiratory bronchioles and you just have a little bit of a smattering here and there. Most of the alveoli are at the very ends of the alveolar duct. So in the conducting zone, there is no gas exchange, at least not this kind of gas exchange. And then I'll just put over here. Why the answer? There are no alveoli present. That's why. So this is the function you have to know of the respiratory zone. gas exchange. It isn't credibly important function of the lungs. So now why? Well speaking of gas exchange in the lack of it, by definition, Deadspace is where no gas exchange is occurring. There's three kinds of Deadspace anatomic physiologic, or I'm sorry, alveolar and physiologic. And so what I'm going to do is to demonstrate this, we're going to put together two or three columns, C, two columns, and three rows. And we're going to talk about anatomic alveolar. And physiologic Deadspace, in a normal individual with healthy lungs, and I'm just going to call it abnormal when it comes to the lungs, and how Deadspace is going to be affected by this. So I will put here anatomic. And then I'll put alveolar here. And then physiologic put down here. So those are be our three rows. Now we're going to have two columns, and the two columns are going to be normal, otherwise healthy lungs. And then over here, I'm just going to put abnormal. And so there are certain conditions that can occur to where it'll affect the dead space. And so we're going to have three rows here. And what we're going to do in each of these is we're going to draw our conducting zone and our respiratory zone. So a little picture here. So conducting zone, respiratory zone, we're just going to keep on doing it over and over again. And by the way, when I draw this, it's representative of both lungs, just so you know. So conducting zone, respiratory zone, two more. And so what I'm going to do here is I'm going to shade in the areas where we have dead space. Now, as I just pointed out the conducting zone, no gas exchange occurs, and the conducting zone is made up of a bunch of anatomical structures and an Aries all the way down to the terminal bronchioles. And so an atomic dead space would be those anatomic structures from the dairies all the way down to the terminal bronchial, so you're conducting zone, no gas exchange. And so that area right there is considered dead space. And because it's anatomical structures, and by the way, it's completely normal.

22:26

We call it anatomic dead space. Now, for an average sized healthy adult male. The average dead space and atomic Dead Space is about 150 milliliters. What does that mean? It means that all these

structures together that make up your conducting zone, you could fill it with 150 milliliters of air, that's all it means. And so we have 150 milliliters of dead space. And by the way, the total lung capacity, the amount of air that you can get in an entire lung is a lot a hell of a lot more than that, as we're going to see later on in a lecture. So 150 milliliters now alveolar Deadspace. What does that mean? That means that you would have alveoli that don't do this, they don't get a blood supply. If you don't have a blood supply can have gas exchange, because the exchange is happening between the blood and the alveoli. So if there is a place in the lungs, where we have alveoli, but no blood supply, that's dead space. As far as we're concerned, this is not really the way that it goes. But this is the way that we're going to learn it, we're going to assume that every single one of the billion alveoli in a healthier individual's lungs can go through gas exchange. So the dead space is a big fat zero. What is physiologic dead space is the combination of anatomic and alveolar. So physiologic would be an atomic plus alveolar alveolar, zero. So anatomic and physiologic are going to be the same in a healthy individual because alveolar is nothing. That's good. We don't want extra dead space. Because if we have extra dead space, we're not going to have as much gas exchange, which is what we're going to see in this abnormal individual. And this could be the same individual just at a later point in time in the person's life. And atomic Deadspace we're just going to assume is the same. And by the way, the anatomic Deadspace is going to vary mostly depending on how tall you are. So again, an average size adult male, what about five, eight ish, somewhere in there? If we're talking about Shaquille O'Neal who's seven feet tall, his anatomic Deadspace in 150 milliliters Why is there always a bigger if you're somebody who's five feet tall, small and petite, your airways is smaller, so it wouldn't be 150 Maybe it would be 125. But 150 is the value that I need you to know because that's the variable value that's used in pulmonary physiology. Now An abnormal individual is going to have some alveolar dead space. And so what I'm going to do is I'm just going to shade an area here, how much dead space just depends, I'm going to pick a number just completely out of the air. I don't know 110 milliliters, I made it up. And so what does that mean? It means that this air, this extra air is not going through gas exchange, this extra area is not going through gas exchange. And so now what we're going to have is a larger physiologic dead space. And what the physiologic Deadspace is going to be is simply adding the two together. So now we're at 260. So we have 110 milliliters of extra Deadspace. Now, what is that going to do? What's the consequence of that? Well, let's think about that. So we'll go on to the next page, we'll draw this again. We'll put the blood vessel in the story. And we're gonna say that we don't have any gas exchange over here. And normally what is gas exchange gas exchange is oxygen in, in carbon dioxide out, well, that ain't happening. Because the blood supply is lost for whatever reason, it doesn't matter right now we just don't have a blood supply, we don't have exchange will be here. And so with the decrease Oh, I'm sorry, an increase in Deadspace. Okay, so that is Deadspace. And that would be considered alveolar Deadspace. So with an increase in Deadspace, and I should put physiologic Deadspace, Deadspace DS, what's going to happen is this, when you tell me, if we're not diffusing as much oxygen in the blood, what's going to happen to the level of oxygen in the blood, it's gonna go down, we're gonna have less oxygen in the blood, we're going to have more carbon dioxide in the blood, because I'm not going to get rid of as much. So with an increase in Deadspace, you're going to have a decrease in blood oxygen levels. And an increase in blood co2 levels. And that's bad. We'll talk about why later. Although I think it's probably obviously the oxygen ones obvious, maybe not the co2, one, co2 ones bad too. So this is something that we do not want to have happen.

27:25

So far, so good. So we just drew this. That's what we just drew. Now what I'm not going to talk about this right now, this is in your notes at a later time. The reason I put it here is I just wanted to have a little bit of anatomy over here. And we started talking about the lungs. But this is going to come into play when we talk about how we get air in and out of the lungs, how we actually breathe. So this is

going to be talked about again, in detail. So there's no reason for me to talk about it twice. So on we go. We're going to talk about compliance. Now we already know compliance is right, talked about it with arteries, talked about how compliance, the words that I want you to think of are what's stretchy and stiff. Your lungs have compliance, just like your arteries do your blood vessels do. Your lungs can stretch like a rubber band and do stretch like a rubber band and they snap back like a rubber band recoil like a rubber band. Now the definition of compliance is measure these which structure expands when exposed to pressure. That's the classic definition of compliance. In the end, I just want you to know the word stretchiness. And stiffness, it can be calculated it can be measured. And there's an equation to do this that you don't have to know, which is why there are lines through this equation. I don't need you to know what the normal compliance is of a long either, I don't need you to be able to convert centimeters of water to millimeters of mercury either, which is why that's all crossed off. So don't worry about any of it. So let's talk about compliance. When it comes to the lungs, there are two things two variables that are going to dictate whether or not compliance is normal and we want normal. We don't want it to be too high. We don't want it to be too low. And so I'm going to actually write this down. No, it's in the notes. So normal compliance of the lungs specifically. So number one, healthy elastin fibers, the same elastin fibers in the walls of your blood vessels. We want them to be nice and healthy that way they have just the right enough stretchiness just the right enough stiffness just the right enough recoil when they are stretched very, very important. So healthy elastin fibers. Number two, the presence of surfactant. Now what a surfactant surfactant is It's mainly phospho lipid, but there's some protein in it. So it is a phospho lipid mainly, plus protein, this is something that we produce constantly. So it's produced. I know, you don't know what this is, but I'm gonna throw it down here anyway, by something called type two pneumos sites. Of your alveoli, your alveoli, produce surfactant, your alveoli, the walls of your alveoli are made up of two kinds of cells type one pneumocytes type to numerous sites, that's it. It's just one cell stuck to another stuck to it, your alveoli are one cell layer thick that is it, which is good, because that's going to be optimal for gas exchange, you don't have a thick membrane here, we have epithelial or endothelial cells, and these pneumo sites, and that's it. So we have very, very efficient gas exchange over here, because it's only two cells thick. And the type two pneumo sites, those are the ones that are producing this product, and they produce it constantly. So if right now we stopped producing surfactant, we would run out in about 12 hours, and we would drop dead, because our alveoli would collapse, and we wouldn't be able to get any air into the lungs. And we're actually gonna talk about that. So anyway, let's talk about now. Abnormal, so abnormal compliance. That would be too high or too low. So abnormal Compliance would be a high compliance, we don't want high, we want normal. And so high compliance means lungs are, too. There's that word again, stretchy, and there was a decrease in recoil. They don't snap back as hard when they are stretched. And that's the bigger issue actually. Again, recoil is just snapping back. So that's a high compliance, we already know what low compliance is Dolly, as we talked about it with the arterioles, and the arteries and such, were too stiff now.

32:26

So lungs are too stiff, both are bad. And so what we'll do is we'll first talk about when compliance is too high. We're going to talk about why this would happen. So let's talk about a high compliance. So in increasing compliance, again, that's not normal. Why would this happen? This happens because of one thing, and that is the loss of elastin fibers. So the loss, destruction, they're gone. of elastin fibers. Now, you might be thinking to yourself, Well, Dr. Or if you lose the last fibers round, losing them all, by the way, he lives elastin fibers with alone won't be a stretchy, no, that's not true at all. It'll be more stretchy. And so I'm going to give you an analogy. That's a rubber band right there. That's a thin rubber band. Rubber bands are made up of elastic fibers. This is going to be a thick rubber band. Now, of those two rubber bands, which one do you think is going to be easier to stretch? If I did a little

itty bitty rubber band, I could easily stretch it. If I had a big thick rubber band, I'd have a tough time pulling it. Like if anybody uses bands in the gym. The bands with the greatest tension are the big, thick bands. They're harder to stretch. The little itty bitty skinny ones, the orange ones, little red ones. Those are easy to stretch. Why? Because there's less elastic fibers in it. So we're destroying the elastin fibers, meaning now we have less of them. So now it's just going to be easier to stretch. Why would that happen? I give you emphysema. That's what emphysema is. Emphysema is the destruction of the elastin fibers of the lungs. That's what it is. So for example anthracene see, I'll put it up here. Anybody know the leading cause of emphysema? Smoke smoking. So all that smoke in the lungs causes inflammation you get the release of a bunch of inflammatory mediators, cytokines, all bunch of crap and they just start to eat the elastin fibers. And when you eat those elastin fibers, that's it, you're done. You have emphysema, there's no cure for it, it's just going to get progressively worse. And so, and increase it if you have an increasing appliance and a story, it's emphysema and a story. Now what about we go the other direction, let's keep on talking about the elastic fibers. So now we're going to have a decrease in compliance. Let's put a hard line here. So a decrease in compliance. Now why would this happen, I'm going to give you two examples. One of them is going to have to do with the elastic fibers. So this is going to be damage, not loss, not destruction, loss of destruction means they're gone. Damage means that their damage and scarring of the elastin fibers so now we have scar tissue on top of the elastin fibers, it's going to be very, very difficult for the elastin fibers to stretch and they're not going to. And so now we have a stiff lung. So we already know that. Now why would this happen? disease called interstitial lung disease will cause this to occur. By the way, we're going to be talking about emphysema, interstitial lung disease, and a bunch of others at the end of this chapter. So that's why I'm just briefly mentioning them right now.

36:37

So for example, interstitial lung disease, otherwise known as I L, D, would cause a stiff lung.

36:52

Now we're going to have another example of why we would have a decrease in compliance. So damage and scarring of elastin fibers, and another is the loss of surfactant. Now, when it comes to surfactant, I already told you that it's being produced by the alveoli. And where it ends up is on the inner wall of the alveoli. So let's say that this room was an alveolus. If you just took a paintbrush and just painted surfactant on the wall, that's how it would be. So it's not like it's floating around in the alveolus mainly was in an alveolus is air. So this air, this room is full of air. And on the walls, we're gonna paint surfactant, how did it get there? Well, the cells of the wall produced it and just kind of released it onto the walls. And it's very important that that surfactant is on those walls of the alveoli. If not, what's going to happen is this is that the alveoli will collapse I told you that we stopped making surfactant, we're going to drop dead. I told you that the alveoli are going to be flat as a pancake. I'm going to show you why after we take a break here. So alveoli will collapse that has a special name is called atelectasis. atelectasis is a fancy word for collapse of the alveoli. We'll talk about collapse lungs in the next lecture as well. And so the LV now why the hell would your lungs get stiff if the alveoli are collapsed? I'll tell you after the break. So let's take a little bit of a break. When we come back, we're going to talk about effect.

38:37

Okay, folks, let us keep talking about surfactin here. So the elastan story is done increase compliance,

decrease compliance, a surfactin story, how it's going to decrease compliance is what we're going to discuss now. So what I'm going to do to do that is I'm going to compare and contrast to alveoli, one more surfactant, one without surfactant. So the first one on the left is going to be with surfactant. So this will be normal. And I'm going to show you what surfactant does to keep the alveoli from collapsing. It'll be here it's going to be without surfactant. And by the way, this information, if you're wondering in the notes is this now this equation you're not going to have to know so don't worry about it, but I am going to reference this equation. So we're going to start to talk about collapsing pressure surface tension that right there. And then we're going to parlay into when we have what's called surfactant deficiency disorder where we don't have any surfactant. Nice even put that over here. So surfactant deficiency disorder, we'll talk about it again later. So at the end of the chapter along with a few other disorders, alright, so let's draw it alveolus with surfactant And so we're gonna draw it kind of big. Now, this is also completely normal. I talked about how we paint the walls were surfactant. Well, before we paint the walls was the fact that we're going to paint the walls with fluid. In and alveolus, it is completely normal for there to be fluid lining the walls. Where's that fluid come from? It comes from filtration, we talked about filtration last semester, Chapter Three in the transport, if you recall, it just happens completely normal, most of that fluid is water. So I'm going to put in parentheses there, water. Not all of it. But most of that fluid is water 98% of it give or take as water. Now, we're gonna compare and contrast what about without surfactant we have the same thing going on, we have this fluid. And it's again, mostly water. And it's lining the wall of the alveolus. Now from your chemistry classes, you might remember the term hydrogen bonding. Water molecules love water molecules, especially when there is a liquid air interface. And there is most of that alveolus is air, like this room is mostly air and again, the walls are going to be painted with that fluid. And then on top of that fluid, we're going to paint surfactant that is under normal conditions, we do not want these water molecules to mingle with each other, we want to limit the amount of hydrogen bonding that is occurring between these water molecules. Why? Actually, you know what, let's just go to the abnormal place over here onto the side. So these water molecules are going to be allowed to intermingle with each other if surfactant is not present. And what that's going to do is it's going to create a force called surface tension. So without surfactant, water molecules, and I'm going to use the word mingle, not put it in quotes. So they're there. So they're attached to the wall, they're attached to each other. And they're attracted to each other very, very tightly. And it creates hydrogen bonds. And so what that's going to do is it's going to increase what's called surface tension, this force. And what surface tension wants to do is it wants to collapse the alveoli given the system that we have here. And so what that's going to do is is it increases another force leads to an increase in what's called collapsing pressure. This goes to the places law that we see in the notes that you don't have to know because we're not going to do any type of calculations with this. But if you increase surface tension that's in the numerator, PT is going to go up with that, again, collapsing pressure. Well, we don't want that to happen. Why? Because that collapsing pressure, if it is high, will cause the alveoli to collapse. Again, that's going to occur if we don't have surfactant, because again, the water molecules are allowed to again, mingle with each other. And again, that's atelectasis. And what that's going to do is it's going to cause a stiff

43:36

lung. Now why I'm going to give you another analogy here, anybody ever tried to blow up a balloon? Not with us a tank, just put your mouth on and try to blow, it's always part of blowing up a balloon, right in the freakin beginning, why the balloon is collapsed, it's stiff. Now, there are other reasons that were stiff here. And it has to do with those forces. But we don't need to get into those forces. All we need to do is think about that analogy. A collapsed alveolus is stiff, and it's very difficult to inflate it. Which means it's going to be very difficult to get air into it and you're not going to get air into it.

Okay, not by breathing, you're going to have to have what's called positive pressure ventilation. Most often the people that have surfactant deficiency disorder are premature babies. Why? Because one of the last things that develops during gestation is the respiratory system, including these type two pneumo sites. So those babies don't have surfactant. So they have to be put on a ventilator. And if they're not, they're not going to survive, the babies are going to die. Why? Because it can't get any air in these flat as a freakin pancake alveoli. That's the reason. So the lungs are stiff because the alveoli are collapse. That's the reason. Now we'll be here on this side. We're going to have surfactant. Now this is not exactly The way that it is, but it's going to be a nice visual. So fact is going to be in green. And it just shields the water molecules from each other. That's what it's doing. And so with surfactant water molecules are hidden from each other. Not completely. And so because of this, we're going to have a low surface tension. It's not going to be gone, it'll still be present. It is still a force that wants to collapse the alveoli, but it ain't gonna happen. Because with that low surface tension, we're going to have low collapsing pressure. And with low collapsing pressure, will the alveoli remain inflated?

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Let's not forget what we're talking about here. We're talking about abnormal compliance. That's what we're talking about. And then giving you two instances when we have low compliance, one instance where we have high compliance. Let's not forget, again, why we're talking about this. Don't get caught up in all the weeds here the details, although you do not need to know the details of all these weeds when it comes to the material itself. But don't forget the big picture. Are we good with this? Yes. Oh, and by the way, the green stuff is this fact. Yes. For what you're saying. Thinking that there's a waterproof, it's waterproof in the alveoli. It's just not letting the water molecules do this. Yeah, it's preventing hydrogen, but it doesn't prevent it, it diminishes it greatly. You're not going to get rid of surface tension. If you have an air fluid interface, you're just not going to get rid of it. But it'll be minuscule, Li low. And at the surface tension is low. The collapsing pressure is low, it goes back to the places law. Thank you. Alright. So now what we're gonna change gears, we're gonna talk about blood flow. Not much, because you guys learn this in a lot of detailed lecture Seaver. We're going to talk about Pearl pulmonary circulation that talk about systemic circulation, kind of the one I'm going to spend the most time on and it's going to be about one minute is pulmonary circulation, which you very well know what it is. So let's draw it, just to remind you. So over here, I'm going to have the lungs and I'm going to draw the lungs like I've drawn them up into this point, conducting zone respiratory zone. I'm going to put our blood vessel here the pulmonary capillary. And now I'm also going to put the heart in this picture, the core cyan, this pulmonary circulation. So here's the heart. And of course, that's the right ventricle. And from the right ventricle, we have the white pulmonary trunk. From the pulmonary trunk, we have pulmonary arteries.

48:02

And then we have pulmonary arterioles. See if I can spell that right. So we have our pulmonary arteries here, pulmonary arterioles, which then lead into the pulmonary capillaries. And we'll just label that once again over here, just to remind us that that's what that is.

48:28

And that's the direction in which blood is going. Why is it going there to pick up oxygen right to get rid of the excess co2. So something that you know very, very well. So we're going to add something to

this story. And that is this. The word perfusion abbreviated with a capital Q with a little.on. top of it. So that dot that you see in your notes above the key was not schmutz. It's supposed to be there. That dot above it is a rate cardiac output. So what is Q? perfusion is q and I'll write it over here and in parentheses, output Q. And so when you hear me use the word perfusion, when you see that q with a.on, top of it, I want you to immediately think cardiac output of the right ventricle. It's taking blood to the lungs for gas exchange. That's what perfusion is to us when it comes to again, pulmonary physiology, respiratory physiology. So I'm gonna say about it for now. More later. There's a lot more later for the material we're talking about today. So springboard to many things to come. Now, what else do we need to know? So what do we just draw us through this? Boom, that's all and then there's a bronchial circulation. You guys know what that is? The lungs are very, very unique, right? They get blood from the right side, the heart The left side of the heart a lot of times, you'll hear people say, well, the right side of the heart takes blood to the lungs, and then the left side, the heart takes the blood to everywhere else in the body, well, the lungs are everywhere else as well. Your lungs have tissue just like any other organ of the body does. And those those tissues need oxygenated blood from the left side of the heart. And that would be the bronchial circulation. And again, I'm not telling you anything new right now. So and of that story done talking about that, because I know you guys know it in a lot of detail. So now what, switch gears completely now, let's talk about what's called spirometry. So what spirometry is a method by which we measure what are called lung volumes in lung capacities. And so what we see in this picture is this little girl, and that little girl has a mouthpiece. And you'll also notice that her nose is plugged in from that mouthpiece is a tube, and then it goes into this computer where it's transducer, and then the computer is going to track the amount of air that that little girl is going to breathe in and out of that tube. And that little girl is going to go through certain types of breathing, that's going to then allow that particular spirometer, which is just nowadays, just a program and a computer. In the old days, it used to be this big SAP, right, that was literally as big as this desk with a bunch of water in it. We've come a long way since those days. And so it's tracking the amount of air going in and out of the this person's airways, breathing in a certain way that allows us to then determine most of these, but not all of these lung volumes and lung capacities. And so you'll see that we have four lung volumes. And we have four lung capacities, and they're bolded, you'll see that we have definitions for each of them. And you'll also see that we have these average values for each of them. You have to know every single word on that page. Now I can tell you this to memorize it. But I'm not a big fan of just memorizing material, you have to memorize it, of course, but we're also going to learn it. So how are we going to learn this, and by the time we're done with this, you're going to know exactly what all this stuff is. And that's gonna make a lot of sense. And you're gonna be able to do it at your desks I want you to because as we go through this little exercise, I'm going to be announcing what I would tell this little girl to do when it comes to breathing into that too. So that we can determine most not all of these volumes and capacities. And by the time it's done, we're going to see a trace that looks something like that except label with everything that we need to know. So let's do that now. So what we're going to do is put together a little x&y axis here, the y axis is going to be volume of air, the x axis is going to be time. And we'll label everything.

52:58

So x axis is time, time in minutes, this is going to be the volume of air. Now, as we draw this trace, we're going to be drawing lines that go up and we're going to be drawing lines that go down. As we draw in the upward direction as we draw in that direction there, that's going to be indicative of inhaling. So breathing in. When the line goes down, that's going to be indicative of exhaling. So the person's exhaling as the trace goes down. So let us begin. So this person has a mouthpiece, we plug their nose and by the way, why are we plugging the nose, the reason that we plug the nose is that we

want to make sure that all of the air through that mouthpiece, none of it is escaping through the nose. That's the reason that that little girl's nose is plugged. And by the way, who's doing this that little girl, I can almost guarantee you and I can guarantee you has has asthma. I doubt she doesn't have that the same at that age product, right? Because at that age, maybe interstitial lung disease maybe, but she probably has asthma who does parametric tests, not healthy people, not that people who have healthy lungs and such people who have diseases like emphysema like as, but like chronic bronchitis like interstitial lung disease. Why? Well, because we want to know how well their lungs are working or not working. We want to see if the treatments are working or if they're not working. So anyway, let us begin. So the first thing that you're always going to want to do is tell them to breathe nice and quietly sitting there. They're going to be sitting probably that's called up Nick breathing. So let's go back to the notes. The very first page, the very first thing in your notes, it's right there you pick breathing, normal, quiet breathing at rest. That's what you're going to ask them to do and you're going to ask them to do that probably for at least a little bit so that they get used breathing in and out of that too, which I'm sure is not that easy to do. So let's see what that would look like. So that person would breathe in, that's an inhale. So it's going to go upwards. Again, normal, quiet breathing, just as everybody's doing right now sitting at your desks. So that person is going to breathe in. And then they're going to breathe out, that has a name is called tidal volume. Tidal volume is the volume of air that you breathe in at rest, it's a tidal volume of air that you breathe out at rest, it's not both of them together, it's each of them. So right now, if you're conscious of your breathing, breathe in, that's the title born, breathe out. That's a tidal volume. So I'm going to label that want to make that look a little prettier. I'm going to label that on both sides. That's tidal volume. That's tidal volume. And for an average size, adult healthy male, it's about 500 millliliters. So you're bringing in about 500 milliliters of air, you're breathing out about 500 milliliters of air. If you're small and petite, it might be only 350. If you're Shaquille O'Neal is seven foot tall, it's probably closer to 700. Give or take. All right. So we've already established number one, we know what tidal volume is now. So one Dawn, seven more to go. Now what you're going to do is, you're going to ask that person to breathe in normally again. So that person will do that. So breathe in, but then you're going to ask that person to stop. You can do it right now, if you want breathing, normally, not stop. That's where the measurement is going to begin. The measurement is going to begin after that normal inhalation, which is why I'm going to draw that line right there, then what you're going to ask the person to do is breathe in as much air as you possibly can. And so, up up, up, up, up, up, up, up, up, up, up, up, up, is going to go until the person has filled their lungs with as much air as they possibly can, they have reached what's called total lung capacity, the amount of air that you can breathe in after you breathe in a normal breath of air, which is what this is. So our measurement is from there to there is called Inspiratory reserve volume, the volume of air that you can breathe in after you breathe in a normal breath of air, it doesn't count tidal volume, which is why I put that line right here. And this person has now reached what's called total lung capacity. They have filled their lungs with as much air as they possibly can. Now, what are you going to ask this person to do? Well exhale, please. So they exhale, down, down, down, down, down, down, down, down,

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we go. Then you're going to ask them, why don't you just take a normal breath in? Why don't you take a normal breath out, and then I want you to stop again. So right now, if you want to exhale, a normal breath of air, that's where the measurement is going to begin, which is why I'm going to put a line right there, then you're going to ask the person blow out as much air as you possibly can. And so now the trace is going to go down, down, down, down, down, down, down, down, down, down. And I'm gonna put a line right there. So that's our reading. That's called expiratory. reserve volume. And by the way, I'm not giving you values yet. So Inspiratory reserve volume, roughly, it's about 3000 milliliters give or take. So about six to seven times as big as the tidal volume Expiratory reserve

volume is about a third of that, roughly about 1000, you can't blow out as much air from your lungs, you have to breathe out a normal breath of air that you can breathe in. After you've inhaled it a normal breath of air, Irv is much bigger than ERP. It's just the way that it is. Now I want you to notice that I did not draw that line all the way to the bottom of the trace. Why? The reason is this. There is a volume of air that is always trapped in your lungs, and there's no way in hell, you're ever going to be able to get rid of it ever, you can blow out as much air as you want from your lungs and I can come up to you and punch you in the stomach as hard as I possibly can. And no more air is coming out of your lungs that has a name. So now we've gotten all four is called residual volume. Now this is what I don't want you to do, do not get residual volume mixed up with dead space. They have absolutely zero to do with each other, nothing, nothing, nothing, nothing nothing. And by the way, residual volume is roughly around 1000 milliliters as well. All of that air pretty much all that residual volume is in your alveoli. It's one of the things that will keep your lb I inflated, your alveoli remain inflated for a number of different reasons. One, the presence of surfactant, we just talked about that. In two, we need some stick and error. And residual volume is going to allow us that's one of the things that residual volume does. Plus, if you hold your breath for a long period of time, you still have air in your lungs. Why are you better, but you need to continue gas exchange. Anyway. You might now be asking yourself, some of you, well, Dr. R, if you can't get rid of that, that air from the lungs, well, then

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oops, what the hell am I doing? I was that little girl to the person who has a mouthpiece going to breathe out that air, how are we measuring it? We're not normal spirometry cannot measure residual volume. Why? Because you can't get that air pass that mouthpiece. Why? Because it's trapped in the lungs. And by the way, your airways will literally collapse and not allow you to

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get rid of that air. They just want. Does that mean we can't measure residual volume, of course we can. Because I already told you what the average value isn't an adult male who's healthy.

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There are three ways to measure residual volume, and we're not going to talk about any of them. It's way beyond the scope of this course you want to go to med school, most of you are probably gonna go to med school PA school, maybe you'll talk about in PA school, you're talking about med school for sure. We're not going to get into it. Alright, just know that you can measure it. It's more complicated than what we're talking about right now. Let the story continue. So now what, ask that person to breathe in, then they're going to breathe out, then you're going to ask him once again, stop. So they breathe out a normal breath of air. This isn't good over here. But instead of blowing out as much air as they possibly can. Now they're going to inhale as much air as it possibly can just as they did over here, but at a different point. So the reading starts, right there after they breathe out normal breath of air. And now in in, in in and they go breathe in, breathe in, breathe in, breathe out until they reach once again, total lung capacity. That's where our reading is going to stop. There. Now let's go to the notes. If you look at the word capacity, capacity is a volume just like these volumes are. So we have four lung volumes than we have for lung capacities, capacities or volumes. So why can't they call them volumes, they call them capacities. Because they're combinations of volumes, they're made up of two or more lung volumes. So every one of these four capacities that you see here, are made up of

at least two of these volumes that you see up here. And so if we look at this spirit metric trace, it's pretty obvious what two volumes This is made up of. And by the way, this is called inspiratory capacity. inspiratory capacity is the amount of air that you can breathe in after you breathe out a normal breath of air, which is different than Inspiratory reserve volume, which is the amount of air that you can breathe in after you breathe in a normal breath of air. So the starting point is different. So let's see what inspiratory capacity as well, we're starting down here. And we're breathing in we get to that point, what's that call? From here to here? What is that? It's tidal volume. And from here to here is what Inspiratory reserve volume. So what is inspiratory capacity inspiratory capacity equals tidal volume plus inspiratory reserve like you can see it two or more volumes make up the capacity, this particular one, two volumes, others are going to be three others are going to be four. We'll see. So now what now you ask this person to breathe out again. And let them take another up big breath. So a tidal volume in a tidal volume out. And now let them go to total lung capacity. So you're just going to ask them doesn't matter where they started, just breathe in as much as you possibly can. That's what you're gonna ask them to do. That's gonna be our starting point. Then what you're going to ask them to do is blow out as much air as they possibly can. So their total lung capacity again, are gonna blow out as much as they possibly can. My going to go all the way to the bottom. Nope, can't get rid of every single volume. The amount of air that you can blow out of the lungs after you have gone to total lung capacity after you have filled your lungs with as much air as you possibly can. is called Vital capacity. visi it's a capacity so it's going to be made up of two or more volumes, in this case three. So let's look at what the three are. This person is blowing up air from up here. So from here, which is what I'm Tracy, we're little arrow is to down here. What's that call from here to here is what Inspiratory reserve volume from here to here is what? Tidal volume from here to here is what? Expiratory reserve volume. So what's vital capacity, vital capacity equals Inspiratory reserve volume plus tidal volume plus Expiratory reserve volume. That's what that equals. What else can we say it is, what's iR V plus vt inspiratory capacity, we just did it over here to the left Irv plus vt. Irv plus vt

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is IC. There you go. I'll throw the residual volume in here again. Alright, we're down to one more, we just covered all we've covered seven of the eight.

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That's that person, breathe in. Breathe out, and then stop again. And that's going to be that. So they're going to stop right there. So you can do it right now exhale, a normal breath of air just stop.

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There's a certain amount of air in your lungs at that time. So from here down to here, it's called functional residual capacity. The amount of air that is left in your lungs after you breathe out a normal breath of air is functional residual capacity. And once again, it is a capacity so it's going to be made up of two or more volumes, what two volumes is made up of. So from here to here is what? The RV right from here to here is from here to here. And from here to here is RV. So what does FRC equal?

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E RV plus RV There you go. I've talked about total lung capacity now three times. What volumes is

ERV plus RV There you go. I've talked about total lung capacity now three times. What volumes is that made up of? All of them?

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We start way up here. From here to here is Irv from here to here is tidal volume from here to here as he RV from here to here is residual volume. What is total lung capacity made up of all of them. Irv plus tidal volume plus ERP plus RV What else could we say it is? Well, what's iR V plus vt? I see. Actually,

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you know what before I do that? What's iR V plus vt plus CR V by capacity? Plus RV. What else can we say it is inspiratory capacity. Plus FRC because FRC is your RV plus RV IC is Irv plus vt.

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Are we good with this? Yes. I'm going to show you another way that this is going to be helpful and actually before I do that, we can't measure residual volume with a normal spirometer we can do it in other ways we can do a normal spirometry. So what that means is this anything that's made up of RV can also not be measured with normal spirometry and we have two capacities right. FRC cannot be measured with normal spirometry. Why because Irv is part of this volume. total lung capacity cannot be measured with a normal spirometer. Why? Because our V is part of its volume. I'm going to repeat that here are the rd cannot be measured. I say normal spirometry I'm going to use a different word. Simple spirometry, because there is a more complex spirometry that can measure

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it and therefore FRC and TLC cannot be measured by simple spirometry because of the mere fact that they're made up of residual volume as well. And by the way, FRC is one of the most important volumes capacities that you can measure in a person when you are assessing lung function.

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So it is routinely measured when people need it to be measured. Now what I'm going to show you another way that will help you realize Eat all of these different things together. And so I'm going to draw a big box here rectangle with four rectangles inside of it. In the rectangle on the far right, we're going to have all four volumes. And I'm going to draw the little boxes or rectangles within the big rectangle here, to scare at least I'm going to give it a shot. So the size of these is going to be indicative of the volume itself. The one in the bottom is residual volume, the one above it is er V, the one above that is VT, and this one's Irv. And if you look in your notes at the average values for these, and I'm just going to use round numbers here. That's about 3000. Actually, it's a little bit more but close enough tidal volume. It's about 500 milliliters, er V again about 1000. A little bit more, but who cares? And we'll go with 1000 there as well. Well, I have three more rectangles, what am I going to put into three other rectangles? I'm going to put a line right here. And then I'm going to put IC here

and FRC here. What is IC equal tidal volume plus Irv tidal volume plus Irv these two rectangles equals that bigger rectangle, er V and RV equal what? E RV and RV equals FRC, these two rectangles equal that rectangle? See how easy that is? Put a line right here. RV? That's vital capacity. What's vital capacity equal? Well, we already know, IR v plus tidal volume plus the RV Irv plus tidal volume plus the RV or IC plus ERD. That's all. What's this one over here. total lung capacity equals everything. This is like put here. So you need to know as I said, what the volumes are, what their definitions are and what the average volumes are. Will really you don't even have to know the inspiratory capacity. You have to memorize these why? Because if you can do second grade math, can't you add these numbers together? Can you add 3500 together and get an IC? Can you do that? I hope you can. Yes. That's one of the ways to use this. It'll help you in to relate everything but it'll also help you with again, the average volumes. Now do you think on the exam, I'm going to put Irv is this VT is these skinny ice you think it's going to be that easy? Of course it's not. So I might do. Let me look here, alright. I can give you VC, I can give you FRC I will give you I see. You tell me what ERP is? Can you do that for me? How would you do that? So let me make sure I did that. Right? Yep, that's right. So let's look at this really quick. So BC. Average value, can you give me one given this table? You know what, I'm not going to do that? Do you think I can change the values on the exam? Of course I'm going to. Alright, these are the average values that you have to know. But of course, I'm going to change the numbers on the exam. I'll talk about somebody who has some pulmonary disease and I'll change the values. So it doesn't necessarily have to be 4500. I don't know I can make this whatever the hell I want. About 6200. I can make FRC which is normally about 2400. Somewhere in there. I don't know, I'm gonna make it 2500. I see. I don't know, I'll make it. Let's go 3200. So how are we going to figure out what ERP is? Well, let's look at this table really quick. If I give you these two, and I give you this, all you need to do is take these two, oops, I didn't say I want to do to get RV not ERP. All you need to do is take these two, right? Add them together, subtract vital capacity. What are you going to be left with? residual volume? Do you see how that how that is? Alright. So how would you do this? Well, you're going to add again, the FRC and the IC. And so what are we going to get? 5700. You subtract it from the 6200 and your RV is

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500. It's low. It's lower than what it's supposed to be so something's not right with this particular patient. So I will do something like that on the exam. All right. Okay, folks, we're done for tonight. I'll see you on Thursday. Good luck to you that are still going to take the exam tonight.

Resp PM 3-17-22

Thu, 3/31 11:13PM 1:18:19

SUMMARY KEYWORDS

air, pressure, lungs, breathing, oxygen, co2, alveolar, alveoli, respiratory, draw, pulmonary capillary, big, pleural, picture, gradient, barometric pressure, pressure gradients, equal, partial pressure, neurons

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Folks, floss to do tonight, before we move off of spirometry, although we're going to visit it again, we need to talk about one more thing. And that's forced vital capacity. So there's the word vital capacity. Again,

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it's the same exact vital capacity that we see here that we went over. So here's vital capacity right here. And to remind you vital capacity is taking in as big a deeper breath as you can go into total lung capacity, and then just blowing out as much air as you can.

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That volume of air that you're blowing out of the lungs, that's what vital capacity is. Well, there is a special test is called the vital force vital capacity maneuver. So let's go to the notes and look at that. So we have the word forced in front of vital capacity, it's very important that this test be performed forcefully, which means that you take in as much air as you can, and then you blow that air out as fast and as hard as you can for as long as you can. And I'm going to show you the reason why. And if you don't do it that way, the test is worthless. And it doesn't take that long, just seconds. So let us draw a trace.

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So a forest,

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please put SVC test or FVC maneuver, both are correct, I'll just put tests easier to top you'll see right, so we're going to have an extra y axis again.

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Over here is going to be time now I am going to actually put the time scale here and it's going to be in seconds.

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Again, it's only gonna take a few seconds. This is volume, again, volume of air.

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And so what's going to happen is that you'll tell the test subject, you know, maybe take

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a normal breath, breathe in, breathe out, and then go to total lung capacity. So you're going to ask that patient breathe in as much as you possibly can, that's going to be the start of the test, right there. So right there to start the test. So that's going to be time zero, then I'll put some more time points here. That'll be one second, two seconds, three seconds, we'll go out to four seconds, because that's about how long it's gonna take.

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Now they're gonna blow the air out as fast and as hard as they can.

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And so down, it goes down, it goes down, it goes down, it goes down it goes. And then done. Got it down to residual volume.

02:28

This is why it's so important for this test to be performed in a forceful way, because it's very important to know how much air was blown out of the lungs, how much vital capacity was blown out of the lungs after the first second. So what I'll do here is I'll just draw a dotted lines straight up. And I'll mark that. So this volume right here is what we call f e v one the amount blown out in the very first second, you'll notice that it's the majority of it under normal conditions is between 70 and 80%. The vital capacity, and we'll just make it FDC is this whole thing right here.

03:07

And so a calculation is done, a number of calculations are done, but we're just going to talk about one. And that is the ratio of FEV, one to FEC. And I've actually already told you what it's going to be 70 to 80%. So FTB, one,

03:22

over FVC times 100, because as a percentage,

03:29

what's normal is 70 to 80%.

03:33

That's what's considered normal. And what that means is, is that after the first second, you should be able to blow out 70 to 80% of your vital capacity. That's all it's saying. And under normal conditions is what should be done. Now there's another disease where we're going to be talking about him at the end of this chapter where it is normal, but the values are truncated, we're going to see. So not only do you have to look at the percent, but you have to look at the raw numbers as well. Speaking of raw numbers, let's put some raw numbers in here. So an average vital capacity would be somewhere around 5000, give or take.

04:07

So 5000 milliliters of air, that's the FVC.

04:11

And then if that's what that was, for a normal individual, they should be able to blow out about 4000 milliliters of air in that first second. And what's that, say? 80%. So that's just an example of what you might see.

04:26

We're going to come back to this when we get to some diseases. So more on this later.

04:32

Now what now let's move forward.

04:35

And let's talk about pulmonary ventilation.

04:38

Now, what is ventilation ventilation is how much air we move per minute. And so we see here that we have minute ventilation and alveolar ventilation. So to depict both of those, and we see that we have a couple of equations. We're not going to worry about the equations when it comes to calculating anything, but I'm going to incorporate it into discussion here. So ventilation so conducting zone

05:01

respiratory zone.

05:05

And I'll label it conducting zone respiratory zone, we know what both is old are now

05:11

minute ventilation. And we abbreviate that capital V with a little dot above it that does supposed to be there. It's not schmutz. When you put a dot above something, it's a rate, the rate is how much air we're moving per minute,

05:24

subscript II, that's minute ventilation. And so what minute ventilation is, is the volume of air

05:34

into while just put in or out,

05:39

in or out of the conducting zone per minute.

05:47

And so Aryan,

05:50

air out,

05:52

permit, that's all.

05:55

And so we can write this mathematically. And we're just going to assume you click breathing. So we're just sitting here quietly breathing, and we know what your breathing is, because we discussed it in the last lecture. So to calculate it very, very simply

06:08

equals the tidal volume, we know what that is, multiplied by how many times you breathe in a minute, respiratory rate, very simple calculation, although you're not gonna have to do it on the exam.

06:22

That's different than alveolar ventilation alveolar ventilation is going to be the volume of air moving into or out of the respiratory zone. So we're going to define and then we're gonna see the equation and we'll see what the difference is. So alveolar, ventilation capital a

06:42

volume of air in or out

06:47

of the respiratory zone,

06:49

per minute.

06:55

Now, how do we calculate this calculation needs to be different, and this is why. So as this air and we'll just do the air in just

07:04

we'll do one of the two as this air is moving into the conducting zone and through the conducting zone.

07:11

So eventually, that air that is going into the conducting zone is going to be air that moves into the respiratory zone, of course it is. But as this air is moving into the respiratory zone, from the conducting zone,

07:24

we're going to lose dead space. So not all of the air that you breathe in will actually make it into the respiratory zone, a small bit of it is going to be lost to the dead space. Let's remind ourselves with the dead spaces, we did it in the last lecture.

07:42

And we're talking specifically right now, because we're assuming a normal healthy individual that is you're seeing atomic Deadspace. And that's around 150 mils.

07:50

And your tidal volume is what on average about 500. Right. So what the 500, as you breathe into the conducting zone, and only about 350 of it is going to get to the respiratory zone. So in order to calculate alveolar ventilation, we have to subtract out the dead space. So alveolar ventilation, is going to equal tidal volume, minus dead space times respiratory rate. So alveolar ventilation won't be as much as minute ventilation. But that's okay, this is normal. There's nothing wrong with this just the way that it is.

08:25

So please know this. One other thing that you're going to see in your notes, and I'll discuss before we move forward, and that is, it is more efficient to increase your tidal volume as opposed to respiratory rate, what I mean by efficiency is oxygenating the body because that air that we are breathing in, is certainly going to end up in the respiratory zone.

08:47

And that is going to be air available for gas exchange.

08:55

And we know what gas exchange is we're going to talk about it a crap ton of detail today, by the way

09:01

needs to get there, we need to get the oxygen in the alveoli for gas exchange to occur. Plus co2 is obviously

09:08

also part of this story.

09:10

So when I say most efficient,

09:13

just give you a real life example, when people say I want to catch my breath, so you just did something physical, and you're out of breath. If you want to catch your breath quicker, the most efficient way to do it is today is to take big deep breaths, as opposed to smaller breaths. So let's say you take a big deep breath and it's 1000 mils, as opposed to two that are 500 mils. Those two breaths that you take are going to lose 150 mils twice, you're going to lose to the Deadspace more often. Whereas suppose if you take one big deep breath, you're only going to lose to the Deadspace one time.

09:54

That way, you're going to have more air into the respiratory into the respiratory zone for gas exchange. So

10:00

Do you want to catch your breath quicker?

10:03

bigger, deeper breaths, bigger, deeper breaths. I'm not saying to hold your breath after that you're gonna keep on breathing in, but the breath should be bigger. Yes.

10:12

Like when you work out that you have like, your hands above your head is like, what is the reason behind that?

behind that.

10:19

Rather than like crouching down? Oh, just because of the respiratory muscles, it just easier for them to contract. Whereas if you're, you're like this, it's just harder for them to contract. That's all. Okay.

10:30

Are we good on ventilation, we're not done talking about decided, by the way, we're going to talk about it again in the next lecture. But I needed to introduce it to you. So talking about, we've been talking about air moving in another lungs. Now I'm going to show you how it actually happens. I'm going to talk about the breathing cycle. How do we inhale? How do we exhale? Before I do that, there are a number of things we have to talk about first, and here's our good old friend, possibly again.

10:55

passageways law, also is applicable to air moving through tubes, air moving through the airways. Now when we talked about pathways law, in the last chapter, we had a number of variables, we're going to cross one of the variables out, we're not going to talk about viscosity. If we were talking about deep sea diving in different air mixtures, we will talk about that, but we're not going to the only two variables we need to worry about are p one and P two, the gradient and radius. Well, we already did the radius story. We did the radius story in the last lecture. And that was when we were talking about bronchoconstriction and brocco. dilation in this one's pretty easy. Your Bronco constrict less airflow, your Bronco dilate more airflow, no need to talk about it again. We certainly talked about pressure gradients in the last chapter, we're going to talk about it again. And we're going to concentrate on that when it comes to

11:47

this the story of how you how you get air in and out of the lungs. So what I'm going to do is I'm going to

11:54

talk about air moving in and out of the lungs due to pressure gradients. So I'm going to draw three different

12:01

lungs here conducting zone, respiratory zone.

12:06

So I'm gonna do this three times.

12:09

So there's one,

12:12

there's two, and here's three,

12:15

three different scenarios. Now we're gonna have a pressure out here, I'm gonna make this pressure too. So again, let me put up here, airflow.

12:28

And what we need to know of it is going to equal our pressure gradient p one minus P two,

12:36

over resistance, and it's that that we're going to concentrate on. So I'm going to make P two out here.

12:43

And I'm going to make the size of P two exactly the same, because that's just barometric pressure. That's the pressure in the atmosphere.

12:51

P one is going to be the pressure in the alveoli. And the size of the P is going to dictate the size of the pressure. And so what I'm going to try to do here is I'm going to try to draw p one to look exactly like P two, it's the same size, so they're the same. So if p one equals P two, what's p one minus P two,

13:14

it's zero. So how much airflow are we going to have?

13:18

Zero, we're not going to have any. So there's no airflow.

13:24

What I'm going to do over here in the next one is

13:27

going to make p one itty bitty. So P one

13:32

is now less than P two,

13:34

which ways they're going to go into the long down gradient. So that's what we need to have happen if we inhale.

13:43

And then in order to exhale, we need to make p one bigger, that will drive air out of the lungs.

13:51

And that's exactly what's going to happen when P one is greater than P two. So once again, this is inhale.

14:00

And this is exhale.

14:03

So you inhale, you exhale, you inhale, exhale. And actually, as you're breathing, there's going to be two points in time as you're breathing, where air is not going to be flowing in and out of your lungs. So these three scenarios here are happening every time you take a breath. And I'm going to show you how, obviously, what's going to be happening is that we're going to be changing the pressure inside the lungs. And I'm going to show you how

14:27

before I do that, something else we have to talk about, that's just what we drew.

14:33

The ideal gas law, which I'm going to assume most of you are familiar with. So we'll put it up on the screen and we're going to come back to this and what I'm about to put here a number of times when we are talking about the breathing cycle.

14:49

So there is an inverse relationship between pressure and volume.

14:53

So pressure equals nRT

14:57

over V and all we're going to concentrate

15:00

Radon is the V and the P, although I'll mention the numerator a little bit during the breathing cycle, but not much. So what this is showing us is this is that as you get an increase in volume, you're going to get a decrease in pressure. There's an inverse relationship. Again, this is second grade math, this is just a fraction,

15:19

few decreases decrease volume, you're going to increase pressure.

15:22

That's how we're going to change the pressure within the lungs, we are going to change the volume of the alveoli. That's how it's going to happen. And again, I'm going to show you how we'll go back to that a number of times in just a second.

15:35

What else do we need to talk about before I actually teach you how we breathe? We have to talk about these three pressures that are involved in the breathing cycle. There's actually a couple more

about these three pressures that are involved in the breathing cycle. There's actually a couple more but this is good enough for us. So what am I going to do here is conducting zone

15:49

respiratory zone.

15:52

PB is barometric pressure, that's the pressure in the atmosphere. alveolar pressure is the pressure within the alveoli. And then pleural pressure is the pressure on the outside of the alveoli within the pleural space. And you guys know what the pleural space is.

16:10

So now what I'm going to do is I'm going to go over a little bit of anatomy that we had skipped in the last lecture that I told you, we were going to get back to, in this lecture, I'm going to remind you what it is, like find it.

16:26

There it is, we're gonna do this.

16:29

So that's what we're about to talk about. So visceral pleural parietal pleura, pleural space pleural pressure. So I'm going to come together, when it comes to this information that you see up here on the screen, I think it's on the second page of your respiratory notes. So I'm going to draw that.

16:45

So that's the lung

16:47

right there. And as you guys know, there's a visceral pleura that lines along this is a serious membrane.

16:54

I'll draw some ribs here.

16:57

Here's the diaphragm.

16:59

Okay, this is all enclosed. This is not drawn to scale, by the way, not even close.

17:04

Then we have the parietal pleura, I'll label everything pride coupler I'm going to do in blue, so I'll label it.

17:12

So that's the parietal pleura.

17:15

The green one is the visceral pleura, that's the one that's attached to the lungs.

17:22

And then in between those two,

17:24

is a space a very, very small space. But nonetheless, there was a space there and it's not going to be anywhere near as big as the lung. It's like if you put Vaseline on both of your hands, stuck your hands together, that's how much space we're talking about. In Technically, there would be no sphere, there's space in between my hands. If I have Vaseline on both of my hands, then my hands are not touching. So that's how small the space is. It's called a potential space. So again, it's not drawn to scale, but I need to write stuff in here. So this is the pleural space.

17:56

And in that pleural space is a plural pressure

18:00

in the lungs is an alveolar pressure. So there's a relationship between alveolar pressure and pleural

pressure, a very intimate relationship between the two.

18:09

So now what I'm going to do is, I'm going to draw one alveolus, and show you this kind of oriented a little bit differently and talk a little bit about pleural pressure and alveolar pressure.

18:20

So this is one alveolus, just one out of the billion that you have together in both lungs.

18:29

And in that alveolus is alveolar pressure. And outside of that alveolus is pleural pressure. Now, this is what I want you to visualize. I want you to visualize the alveolus as a balloon.

18:42

And I want your hands to be wrapped around that balloon, your hands and the pressure that you're exerting on the outside of that balloon is plural pressure. That's what I want you to visualize.

18:55

Now, under normal conditions, pleural pressure

19:01

is less than alveolar pressure. It has to be

19:06

this is going to help keep

19:09

the alveoli inflated.

19:16

We're going to talk about collapsed lungs after this, by the way, and we'll come back to this point.

19:22

Now, something else that's important to know, to understanding the breathing cycle.

19:28

pleural pressure is going to control the size of that alveolus. Just like your hands could control the size of that balloon. If your hands Squeeze the balloon, if you put more pressure on that balloon, what's gonna happen to the balloon, it's gonna get smaller. If you don't hold on to that balloon is hard, the balloon will get bigger.

19:48

So as you increase pleural pressure,

19:53

you decrease alveolar volume.

19:57

And what happens when volume goes down? 10

20:02

pressure goes up. So as a result of this, we are going to get an increase in alveolar pressure.

20:10

When pleural pressure goes down, the complete opposite is going to occur, we're not going to squeeze the alveoli as hard. So we're going to get an increase in alveolar volume.

20:21

And when volume goes up, pressure goes down.

20:26

So how are we going to change these pressures? I just showed you.

20:32

We're going to change the pressure by controlling the size of the alveoli, which is going to control the pressure within the alveoli. So P one in this picture is alveolar. pressure, P two and is pressure, barometric pressure.

20:46

Pressure on the outside.

20:48

Are we good with this? Yes. Now

20:54

we finally get to go to the breathing cycle. How do you breathe? How do you bring air in and out of the lungs? It's all I don't know what page it's on. But I think it's an entire page.

21:04

And so on that page, you'll see things like expiration during inspiration and of inspiration, and then during expiration. So there are four phases to breathing. We're going to go over each of them. And I'm going to show you how we're guessing the lungs how he gets out of the lungs. I'm going to show you that during two of those phases. Air is not going anywhere. It's not going into the lungs. It's not going out of the lungs. It's all about pressure gradients and creating them. The story starts ended exploration meaning

21:34

you just breathe out in a little breath of air. You are at fr C if you remember what FRC is FRC is the amount of air in your lungs after a normal exploration. So you are at FRC at this time. So what we're going to do is this, we're going to draw four,

21:52

we're going to draw four these. And each of those is going to have pressures and I'm going to raise those and draw a little bit nicer. So the four phases of breathing is right here. That's it's going to be one breath, inhale, exhale.

22:08

So let's do it.

22:12

So the first one is end of expiration, that's where the story is going to start.

22:18

Now our assumption is that we are at sea level. And it's you pick breathing,

22:25

if it was a different elevation, if we were exercising, things would be different when it comes to the numbers. So our assumption is again, we're in Dayton, Ohio, and we're sitting in a classroom listening to Dr. R teach about breathing.

22:41

Now we have a barometric pressure.

22:45

We're gonna the units that are used are centimeter of water, as opposed to millimeter of mercury as it is with blood pressure. For example, why is that? Because the people that put this together a long time ago, use centimeter water, it's just a it's just a measure of pressure. It's like how tall are you? six feet tall or 72 inches tall, you're the same height, just the units are different. That's all centimeter water is a different unit, the millimeter of mercury. So at sea level, barometric pressure is roughly it's not exactly this, but it's a nice round number. So that's the one I'm going to use. It's about 1000 centimeter of water, which would equate to about 760 millimeters of mercury, give or take, I mean, technically 740 millimeters of mercury, but it doesn't matter.

23:29

Now, this is the end of exploration.

23:32

And what that means is, is that you've just breathed out breath of air. When you do that you can do it at your desk right now is air moving in and out of your lungs.

23:44

No, you breathe out a breath of air air has stopped moving. If that's the case, tell me what alveolar pressure is Tell me.

23:56

Tell me what alveolar pressure is.

23:59

It's got to be 1000. There's no gradient air is not moving in and in and out of the lungs. P one has to equal P two barometric pressure has to equal alveolar pressure it has to.

24:13

So at the end of expiration, alveolar pressure is also 1000. It has to be because there's no airflow. Right now if I told you to hold your breath is any air going in and out of your lungs?

24:27

No, you created an environment where you caused the pressure in the alveoli to be exactly the same pressure as it is in the atmosphere. Now you didn't do it consciously. You just didn't contract your respiratory muscles.

24:40

No pressure gradient, no airflow. Now, I also stated that under normal conditions, pleural pressure has to be less than alveolar pressure. And it is well how much less roughly about five centimeter water. So plural pressure is equal to 995

25:00

Give or take has to be less though.

25:04

Now

25:06

we're going to inhale.

25:09

So we're gonna draw another one of those.

25:12

So here we are.

25:16

Conducting zone respiratory zone, barometric pressure is not going to change relative to the lungs. So we're just going to keep it at 1000.

25:24

Now,

25:25

there are a number of things that we need to do now stepwise to show you how we get air into the lungs, how are we going to get alveolar pressure to go down, I'm going to show you.

25:36

So what we're going to do is two columns here, the column on the left is going to be during inspiration that's taking air into the lungs.

25:46

And the one on the right is going to be during exploration exhaling.

25:53

So what needs to be done to get air into the lungs what needs to be done to get alveolar pressure to go down? I'm going to show you first thing that has to be done contraction

26:04

of respiratory muscles

26:08

in a short net because I'm running out of room respiratory muscles contract.

26:15

Now what respiratory muscles Well, we're talking about youth breathing here, quiet breathing, and the only two muscles that are involved their diaphragm, external intercostals you guys know the diaphragms dome shape, right? And when it contracts, what does it do?

26:29

It flaps and yesterday intercostals contract, what happens pulls on the ribcage, so we're going to make the thoracic cavity bigger. So when these respiratory muscles contract, you get an increase in thoracic

26:44

cavity volume.

26:47

Now let's go back to a picture I just drew a little bit ago.

26:51

Something else I didn't mention, I'm gonna mention it now.

26:54

Your lungs are always slightly stretched. It's not like a rubber band. If you just dangle a rubber band, that's not how your lungs are.

27:03

So take a rubber band and slightly stretch the rubber band. Even if you blow all the air out of your lungs that you can, it's still going to be slightly stretched, at least a better be

27:11

does that rubber band feels like it wants to snap back like it was to recoil. Of course it does, your lungs are doing the same thing. Your lungs are recoiling, because they're always slightly stretched. So

they're creating a force that goes in that direction.

27:25

Your thoracic cage by the way, is doing the complete opposite it wants to expand, it's just not allowed to. And so what happens as a result of that, and that's the reason that per pleural pressure is a little bit lower than alveolar pressure is because it makes this space a little bit bigger than it normally would be volume up. Pressure down it creates a vacuum in this space. That's why this portal pressure is lower. So I throw it out there for you guys. Now, if we are expanding the thoracic cage, we're going to cause more stretch to occur. It's going to make this space even slightly, slightly bigger.

28:05

So we increase thoracic cavity volume we are going to increase pleural space volume.

28:15

And what's going to happen the pleural pressure, volume up

28:19

pressure down. So that's going to cause your pleural pressure to go down. Well, what happens when pleural pressure goes down? Well, we already know

28:27

it makes your alveolar volume go up, I discussed it already. It's controlling the volume of the alveoli. So this is going to cause an increase in alveolar. Volume, you're not going to squeeze the alveoli as tightly just like your hands, you're just going to kind of let go of the balloon a little bit and let it expand.

28:48

And so of course, then what's going to happen the alveolar pressure

28:52

is going to go down. PV equals nRT. Do we now have a pressure gradient? Yes or no?

29:00

Yeah, we do. Now, how low did it get how loaded total pressure get plateau pressure started at 995.

Total pressure is going to go towards 992 not going to change that much.

29:17

What's the pressure in the alveoli going to be

29:20

alveolar pressure

29:22

is going to go towards 999. It changes one centimeter of water, which is peanuts. But it's still enough to get air into the lungs. That's how small the resistance is within the airways under normal conditions, of course, do we have a gradient? We've already established we do. And as a result of that

29:44

we inhale

29:47

down our gradient so this is during

29:51

inspiration.

29:53

So what do I need to put in here?

29:56

I need to put in hail here. Look at that.

29:59

I bet you

30:00

Almost all of you in this room thought that when you do this,

30:05

you breathe air into your lungs and you expand your lungs, I bet you almost everybody in here thought that it's the complete opposite. The lungs get bigger first, and then the air comes in. That's how it works. It seems like this is blowing up your lungs, like you do a balloon. But it's not all of this stuff has to happen first, before the air gets into the lungs, because we need to create a pressure gradient. It's all about pressure gradients.

30:35

So now what we're going to breathe air into the lungs. So eventually, we're going to be at the end of inspiration you're going to stop breathing in.

30:45

And so let's draw this now.

30:49

So this is the end

30:52

of inspiration, barometric pressure is still 1000.

30:58

Now at the end of inspiration, is air going in and out of the lungs, yes or no?

31:03

No. So tell me what alveolar pressure is, please.

31:06

It's 1000.

31:10

Well, wait a second, Dr. R.

31:14

alveolar, volume went up,

31:17

pressure went down. And certainly, this is going to be as big as the alveoli you're going to get, which means alveolar pressure should be as low as it's going to get. So how in the hell did alveolar pressure go up from 999 to 1000? If the alveoli are now as big as they're going to get,

31:37

because the numerator got bigger, okay, we have two variables here. Well, I mean, three in total volume controls pressure, but so does NRT Avogadro's number a bunch of other stuff, we're not going to get into it. As air is going into the lungs were adding molecules in the lungs, it's filling in that extra space. It's just basic physics stuff.

31:58

The reason that we go from 999 to 1000, is because the numerator gets bigger because we're bringing air into the lungs until we've reached a new equilibrium, that's all.

32:08

So it is going to get to 1000. And when it does, that's when inspiration stops. Now what's plural pressure, we actually reached 992, that arrow is indicating that we're going towards it, we finally reached it.

32:23

So now no more breathing in, because there's no gradient.

32:28

Now we want to exhale.

32:31

Oh, and by the way, I'll never buy never like nevermind. Alright, so let's do this. Now, last one.

32:38

So this is going to be during

32:42

expiration, barometric pressure does not change, it's going to be 1000. Now we have to go through a little flowchart again.

32:50

So now during expiration, what's going to happen the complete opposite of what we saw with during inspiration, which makes it nice. Now the respiratory muscles are going to relax.

33:02

And as a result of that

33:08

thoracic cavity volume is going to go back to its original size.

33:12

So this is the passive process. By the way.

33:15

Inspiration is an active process, we're actively contracting the muscles, this is going to be a passive process, we're just relaxing the muscles, everything just falls into place. So decrease in thoracic cavity volume.

33:30

Everything's the opposite, just change the arrows. Now we're going to get a decrease in pleural space volume.

33:36

And eventually, what's going to happen is that we're going to create an environment where

33:41

we have a pressure gradient once again, but this time, it's going to be in the opposite direction. The alveoli are going to get squeezed.

33:50

So the pressure is going to go up. And then we're going to exhale. So again, the last thing that happens is the movement of air but in the opposite direction.

34:00

Now,

34:02

how high does pleural pressure get? And how high does alveolar pressure get? Well, let's look.

34:08

pleural pressure started at 992. And it's going to go back towards 995.

34:15

alveolar, pressure started at 1000.

34:18

You know, all that's gonna do is go towards 1001. These pressure changes are itty bitty, and we're still moving 500 milliliters of air right? Isn't that a normal tidal volume and an average size adult healthy male? Yes. So we have a pressure gradient.

34:36

And we go,

34:38

we exhale

34:40

until we've removed enough air to reach a new equilibrium.

34:45

And it starts all over again. And we do this anytime anywhere from 12 to 18 times a minute, that's normal respiratory rate. Okay, at rest that is.

34:56

So that picture right there and all these words here and these two things

35:00

Charge is all the words that you see. I don't know what page it is. But it's all these words right here. And again, in order to truly understand that we need to know passageways law, we need to know gradients, we need to know, ideal gas law. But fortunately, they're all very, very simple things.

35:17

No calculations with the ideal gas law, no calculations with possibly law.

35:22

Just know it qualitatively to understand how you inhale and exhale, air, how you ventilate, very, very important.

35:31

Now what

35:33

let's talk about when things are abnormal, let's talk about collapsed lungs pneumothorax. What causes a pneumothorax is excess air in the pleural space, that's what causes the pneumothorax. And so what we're going to do is that we're going to draw this picture right here, but we're going to do

something to the picture to cause the pneumothorax. So this is going to be a simple pneumothorax. And then we're going to talk about tension pneumothorax.

36:05

So here's our lung.

36:07

Within the lung alveolar pressure, of course, we have our visceral membrane here,

36:14

ribs diaphragm, parietal pleura, all the stuff that we had before.

36:21

And we have our pleural pressure right here, inside of the portal space. So how does a simple pneumothorax occur, going to have some type of trauma to be any number of things, I can stab you in the chest, shoot you in the chest, bust, your ribs, something's going to happen, you can have these blisters on your lungs, by the way, with certain lung diseases that just pop. And all of a sudden, we start to introduce air into that space that doesn't belong in that space. But that air is going to be allowed to equilibrate through that traumatic area. But it's still going to be air in the lungs. So we're going to get an increase in air in this space, they don't belong there. And if we get an increase in air, we're going to get an increase in pleural pressure.

37:13

So the pressure around the alveoli is going to go up to the point. And this will be a simple pneumothorax where pleural pressure equals alveolar pressure, boom, those alveoli are gonna be flat as freakin pancake. Now, you might think to yourself, Well, why dodger? If the pressure on the outside is equal to the pressure on the inside? Why would the alveoli collapse? Because there's another force that wants to collapse your alveoli and tell me what it is.

37:41

Thanks, surfactant.

37:45

So in the last lecture, we talked about the importance of surfactant, I think I just started surface tension. Yep, the surface tension goes away completely.

37:55

Surfactant simply lowers surface tension and surface tension as a force that wants to collapse your alveoli, well, Imperial pressure is lower than alveolar. Pressure, that's enough to overcome the surface tension.

38:08

But if all of a sudden those two pressures are even, while surface tension is going to when the plural pressure and the alveolar pressure cancel each other out, and now you have this force, that's gonna smash your alveoli like pancake. Surface Tension does not go away, it's just lowered vice effect. That's all. Which is why when pleural pressure and alveolar pressure equal each other, you collapse your lungs.

38:34

Okay?

38:36

Now, oh, and by the way, that's gonna suck. Because how you going to ventilate the lung now. Now, another thing too, is that you might not collapse your entire lung, you might just maybe 10% of the lung gets collapsed. And certainly, the lungs are completely separate from each other. So one lung could be completely collapse and the other one's completely fine. It's still going to suck. So you're not going to be able to ventilate as well, certainly. And certainly, this is not going to be a pleasant situation, but it's probably not life threatening, or at least not yet. The tension pneumothorax However, life threatening

39:09

if it's severe that is, so this is attention NUMA.

39:15

Oops,

39:17

oh, yeah, I spelled it right. Damn it.

39:20

So we're going to draw the same picture, except the trauma is going to be different.

39:27

So long,

39:30

visceral pleura, and all the other structures that we're going to draw.

39:35

For idle flora. Now we're going to have trauma again.

39:39

But this time, the trauma is going to result

39:43

in a different kind of injury to where the air can get into the pleural space, but it can't get out. With the simple minimal the air was allowed to equilibrate, we suck out extra air in this space, but the air was allowed to equilibrate here, it will act kind of like a valve

39:59

like valves on the

40:00

allow blood flow to flow in one direction. This is this is an injury to where air can get in, but it can't get out.

40:08

And so what we'll do is we'll just have a couple of areas here. And we're just going to have arrows coming in.

40:13

So air can only get in, it'll just keep coming and coming and coming and coming in. And how much

So air can only get in, it'll just keep coming and coming and coming and coming in. And how much depends on how severe the injury is. As a result of this,

40:24

our flow pressure is going to go way up. So no longer is it equal to alveolar pressure, it's greater than pleural pressure, or I'm sorry, alveolar pressure. So pleural pressure is now greater than alveolar pressure.

40:37

Now, if it's severe, and pleural pressure has gone up greatly.

40:43

It's life threatening within minutes. And it has to do with the cardiovascular system and things that we learned in the last chapter, the circulation chapter, so we're going to go over that now. So severe

40:54

tension pneumo.

40:58

I'm gonna show you a picture of it. It's an actual

41:03

x ray.

41:05

So we have in this picture is a severe tension pneumo over here on the right in the right lung. Okay, this is an actual x ray, you see how dark that is compared to what it is kind of opaque over here, it's really dark over here, that darkness is actually air. And that's not the way it's supposed to look. That is a huge tension pneumothorax right now, it's a crap ton of air where it's not supposed to be.

41:27

And what will happen is, if it's severe enough, it's gonna push the heart, where's the heart supposed to be?

41:33

Like the middle, you're sticking chest? Can you see the hardness picture? It's over here on the left side of the chest, why? It's being pushed by all that pressure, it's being squeezed by all that pressure. So as a result of this severe tension pneumo

41:48

we're gonna squeeze the heart.

41:51

How well do you think the heart is going to be for squeezing it?

41:56

Probably not very well. So as a result of this, we get a huge decrease in heart contractility.

42:05

And when you have a huge decrease in heart contractility, what's gonna happen to stroke volume, tell me,

42:10

it's gonna go way down.

42:13

And when stroke volume goes down, what happens to cardiac output?

42:17

It goes way down. Now, is there a way that the body can respond to this? What do you what do you think's gonna happen to heart rate, it's gonna freakin skyrocket.

42:26

So at this point, you're going to get an increase in heart rate. And if you are monitoring this patient, their heart rate would be freakin tachycardic as hell, but it will only be able to do so much. And so instead of four arrows down, I don't know, I'll go two arrows down. What happens when your cardiac

output goes down? What happens the blood pressure,

42:44

ma P equals cardiac output times TPR ma P equals cardiac output? Did I tell you that this equation is important to tell what happens, blood pressure goes down?

42:56

What happens when your blood pressure goes down? Blood pressure gradients get low, maybe you reach critical closing pressure,

43:03

decrease in blood flow,

43:05

sag good.

43:09

organ failure.

43:13

Death.

43:15

Attention pneumo is dangerous, not because of ventilation issues. But because of cardiovascular issues.

43:23

And a decrease in blood pressure. How do you think you can treat this

43:29

get the air the hell out of that space, you're gonna stick a big, you're gonna stab him with the big ass needle. And you will literally hear the air leaving the needle and then you will see your patient's blood

needle. And you will literally hear the air leaving the needle and then you will see your patient's blood pressure come up, and you will see the heart rate come down, you just save your life. Now, then you got to fix the problem, you know, you just bailed the water out of the boat. You got to get to patch things up. So that stops happening. Here's another point before I'm gonna let you go for a break.

43:55

You are not going to take somebody to X ray if they have a tension pneumothorax if you do, you should be sued for lots of money and fire.

44:02

Because this is an emergency situation that can be dead within minutes. So how the hell did this X ray come about this person more than likely had a simple pneumothorax who then was taken to X ray to see why the simple pneumothorax happened and during X ray, they developed attention NUMA Alright, so again, you're not going to take somebody to X ray with attention NUMA with a simple demo. That's okay, tension pneumo. You're fine. Alright, let's take a break. When we come back, we'll continue.

44:28

Let us continue. So what are we going to do next?

44:32

So we just talked about breathing. And the story started

44:38

right here at the muscles well what's controlling the muscles,

44:42

control the muscles you control breathing. So let's talk about that now.

44:47

So regulation of breathing, so what's going to happen so that the nervous system is going to do and there's a special area in your in your brainstem?

44:57

It's called the respiratory center. And there are a number of

45:00

things that are influencing that respiratory center, which is going to influence the way that you breathe. And so I'm just going to put a little Trier a rectangle here, this is going to represent that area in your brainstem that's responsible for regulating your breathing.

45:16

And there is a ventral part of this area and there is a dorsal part of this area, so just a bunch of neurons. And so I'm just going to put a little imaginary line here, this will be the ventral part.

45:28

And this will be the dorsal part. So Dr. G is dorsal respiratory group. VRG is a ventral respiratory group. And together those neurons and there's a bunch of them

45:42

make up the respiratory Senate.

45:47

Now within the VRG is a special small kernel of neurons, not a lot like 1000. There's a funny side, by the way, this is bilateral, so on both the left and in the right side of the of the brainstem, there is a very, very special part of the VRG is called the pre besting or complex, those two dots, by the way, belong on top of the oh, I want you to think of the pre Bezier complex as the SA node. The Priebus here complex is pacing your breathing. You guys think about breathing. Soon, you probably don't. You might know. But you don't think when inhale, now, I'm going to exhale, I'm going to inhale, I'm going to exhale, you don't do that. Why? Because these neurons are taking care of it for you. These pre Besig neurons are firing action potentials, which will eventually cause

46:38

these muscles to contract, and then off to the races we go. And then the muscles are going to relax. And it just happens all over again, just like the SA node generates action potentials at a certain frequency. So do these actions are so to these particular neurons over here, but not as frequently as the SA node does, because we breathe about 12 to 18 times per minute or take 1218 breaths permitted you nicly. Now just like the cardiac conduction system that's influenced by a number of things. Well, these neurons are influenced by a number of things. And so what are those things? Well, one of the things that influences these neurons is your cerebrum conscious thought. So that's the cerebrum, I'm just going to have a synapse there.

47:23

So what's that mean? It means right now you can breathe however the hell you want.

47:28

If I asked you to hyperventilate, right now you can hyperventilate, if I asked you Take three breaths, and the next five seconds, and then hold your breath for four seconds, and then take 10 breaths for the next 20 seconds, you could do that. Because you have thoughts. Those thoughts are action potentials, right? We learned that last semester, those action potentials will cause the release of some neurotransmitter here just the right way. And you're going to depolarize hyperpolarize. These neurons when you hold your breath, you've hyperpolarize these neurons.

47:55

So you can control the respiratory center, literally just by your thoughts. That's all. How else is the respiratory center controlled through emotion.

48:05

And that would be the hypothalamus.

48:10

And so this is through emotion.

48:13

Over here, the cerebrum, your thoughts? Again, we know action potentials are thoughts. So we're going to synapse with these neurons. And so you get excited about something, you might start to hyperventilate a little bit.

48:27

When you're stressed.

48:30

You actually decrease your ventilation, you inhibit the respiratory centers, when you have a high level of stress, you might even notice that you're really really stress. Think about your breathing for a second, you're probably holding your breath at that very point in time. You slow down your breathing

when you're stressed. So that's emotions. What else controls these neurons, which will again control the breathing cycle because they're eventually going to control these muscles over here.

48:53

Chemo receptors.

48:56

We learned about chemo receptors briefly last semester when we talked about the autonomic nervous system. These are autonomic receptors, chemo receptors, their job is to detect some chemical

49:07

oxygen and carbon dioxide chemicals. And so these chemo receptors

49:14

detect

49:16

the level

49:17

of co2

49:20

and oxygen. In the arterial blood, I'll just say the blood

49:26

and we have what are called Central chemo receptors.

49:31

And peripheral chemo receptors. The central chemo receptors are in the brain and they're actually intermingled within this area of the brainstem as well. And then the peripheral chemo receptors are in

intermingled within this area of the brainstem as well. And then the peripheral chemo receptors are in your carotid bodies in the carotid arch. But it's it's a

49:48

it's not too terribly important to know. But this is very, very important for you to know and it's in bold in your notes. So I'm going to show it to you.

49:56

By just a couple things we don't have to know by the way, I'll point that out just a second.

50:01

co2 is the main stimulus to breathing well, what does that mean?

50:05

So again, at detecting the level of co2 and oxygen in the blood

50:11

the main drive to breathe, to ventilate

50:18

is the level

50:21

of co2

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such that

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with an increase in co2,

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you stimulate breathing with a decrease in co2, you inhibit breathing.

50:38

co2 is the main stimulus to breathing under normal conditions. Not really, really, really, really low levels of oxygen, that'll start to take over the breathing. But that's not common.

50:50

I want you to know this. Central chemo receptors are the neurons that I spent a whole bunch of my time on. When I was the scientists I used to, I used to study stuff in the brain. And that's one of the neurons, one of the main neurons, I tried to figure out how to how they work.

51:04

hated it.

51:06

Which is why I'm here in front of you guys today. So again, we're controlling the respiratory center. And then there's only one more thing in the notes that we're going to worry about. Those are irritant receptors.

51:17

irritant receptors are within the airways themselves, and they detect irritants, like smoke, just whatever something that makes you cough. It's a reflex, it's an autonomic reflex. Oh, and by the way, I'm going to put positive and negative signs here indicating that these things can inhibit

51:36

and stimulate the respiratory centers, irritate receptors or Mondego, it's going to be that so what they're going to do is cause coughing.

51:46

That'll be our angle, it's not the only thing, but that's going to be our angle. So reflex to get crap out of the airways that don't belong in the airways.

51:55

There are a number of things that we aren't going to talk about one of which are two of which I should say pneumo tactic and F new stick centers, don't worry about it, I'll put this on pilot, don't worry.

52:06

And then the appropriate receptor is not going to worry about those either, just the ones that I have in the picture.

52:13

Okay, now what changing gears once again,

52:16

we're going to talk about gas exchange finally, in a lot of detail. And we know a gas exchange is the exchange of oxygen and co2. We saw it in the respiratory zone, but we're also going to see it at the cells. Before we do that, though, we have to talk about partial pressure.

52:34

What is partial pressure pressure is either by the exerted by gas or the mixture of gases. What does that mean? Let's take the atmosphere, for example, which is what I have as an example here in the notes. So the atmosphere has a certain pressure already already talked about how it's 1000 centimeters of water at sea level. For now we're going to go back to millimeters of mercury. At sea level, it's about 760 millimeters of mercury. That's the pressure that we are under right now in this room in Dayton, Ohio.

53:04

That total pressure is due to two things, the gases in the air and gravity.

53:12

What are the two main gases in our atmosphere?

53:15

Oxygen, Nitrogen. How much oxygen about 21% Give or take? How much nitrogen 79% Give or take. And there's a bunch of trace gases like radon and carbon dioxide and carbon monoxide, blah, blah, blah, blah, blah. But they're just Trace trace trace trace amounts to main gases, oxygen,

53:36

and nitrogen. So 9% 21%. So each of those gases in the air is exerting a partial pressure, it's part of the total. That's all it means. Guys, there's nothing more magical about it. So if the total pressure in the air 760, the partial pressure of oxygen and the partial pressure of nitrogen have to equal 760.

54:01

How do you calculate it? It's very simple partial pressure is calculated as

54:07

the total pressure which in this case is 760. times the percent of the gas, which in the oxygens case is 21%. Nitrogen case is 79%. So what's the partial pressure of oxygen in Dayton, Ohio? 760 millimeters of mercury times point to one equals 160. Nitrogen 760 times point? 796 100 600 plus 160 is 760. That's all that is all it is. All right.

54:39

You're gonna have to do those calculations on the exam.

54:42

Easy money, second grade math. It's multiplication, that's all. Now, can we change partial pressure? Of course we can. There's two variables. What are the two variables? The two variables are the total pressure and the percent of gas

55:00

Make sure gases, the percent of the gas,

55:04

we can change. So the total pressure is atmospheric pressure. Well, how the hell do we change atmospheric pressure, there's four ways to do it, we only have to know one of them is across three of them all. And so let's talk about that one, we'll draw a picture here. So right here is going to represent sea level. This is good old day in Ohio.

55:27

And that sea level, we have a total pressure, a barometric pressure of about 760 millimeters of mercury.

55:36

And we have 21% Oxygen. And so we know that our co2 in good old Dayton, Ohio, is 160 millimeters of mercury, we just calculated it two seconds ago. Right?

55:50

Now,

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if we get on a plane,

55:55

and let's take Denver again, and we go to Denver, Colorado,

56:00

so Denver, SEO, which is about a mile above sea level, so roughly about a mile above sea level

56:09

5280 feet,

56:12

the partial pressure of oxygen in Denver is not 160. Why not? Because oxygen concentration whether I'm sorry, percentage went down. As long as you are within Earth's atmosphere, we could be on the top of Mount Everest right now, which is about 30,000 feet,

56:29

oxygen will still be 21%. It doesn't matter where you are. As long as you are within Earth's atmosphere. Oxygen is 21%. Nitrogen is 79%. So why is it there as much oxygen in Denver? Because the total pressure isn't 760. The barometric pressure in Denver is instead

56:48

about 630. Why? There's less gravity. As you go up in elevation, gravitational force goes down. So it's

639 760. So what's the co2

57:05

in Denver?

57:08

About 130 millimeters of mercury.

57:12

People in Denver don't have as much oxygen as we do. What do they do to acclimate Tell me one thing that we learned in the blood chapter,

57:21

they increase red blood cell concentration to make up for the hypoxic conditions. So one of the ways that we can change the p O to or any partial pressure is just go up in elevation. And the higher you go, the lower that PB gets.

57:37

The other ones we're not going to talk about don't have time, although it's all kinds of cool to talk about it, it really is.

57:44

deep sea diving is all kinds of cool to talk about hyperbaric and hyperbaric chambers, it's all kinds of cool to talk about, we just don't have time to talk about it. Plus, it's beyond the scope of the course. Alright, I taught it in med school, and it was all kinds of fun.

57:58

The other variable is the percent of oxygen or the gas that we have. Now, you doctor argue to said that the oxygen is always only always 21%. It is. But you can give a patient supplemental oxygen. So we're going to talk about that now. So we can change again, partial pressures by changing the total pressure, but we can also change partial pressure by changing

58:22

the percent of the gas or supplemental oxygen.

58:28

Now,

58:30

actually, before I do that,

58:32

I'm going to put what happened there.

58:38

Good. So I'm going to put

58:42

room air.

58:45

So if a patient is breathing room air, that patient has available to them 21% Oxygen, of course, but that might not be enough.

58:57

And so the level of oxygen in that person's body might be too low. Why? Well, because they have some pulmonary condition, maybe cardiovascular condition, something's wrong toward them, that oxygen at the body very well. And so in that case, you give them what's called supplemental oxygen.

59:13

So what percent are you going to get the patient it depends on the patient. But it can be whatever the hell you want it to be. You can make supplemental oxygen 30%

59:22

Or you can make it 40%. Or you can make it 50%. Or you can make it 100%. And what's the partial pressure going to be and we're going to assume that we're in Dayton, Ohio.

pressure going to be and we're going to assume that we're in Dayton, Ohio.

59:34

So you just simply do the calculation, P O two equals 760 times 0.3

59:44

or 0.4, and so forth.

59:48

I can do one of these in my head. I'm good at math, but I'm not fantastic at math when it comes to this on the fly. But we'll do a couple of these so 50% What would that be?

59:59

After three or seven?

1:00:00

6380. So our feel to would be 380 is way bigger than 160.

1:00:05

The po two could be 760.

1:00:11

That's a heck of a lot of oxygen. But some patients need that. Although you don't want to give the patient 100% oxygen for very long, maybe a couple of days, because if you do, they'll develop a condition called art's really, really high levels of oxygen caused the production of a free radical called superoxide, which is incredibly damaging everybody in here, I'm going to assume has healthy lungs, including myself, if we were put on 100% oxygen for more than two days, we would probably develop arts and be in danger of dying.

1:00:39

So why the hell would you give somebody 100% oxygen because you don't have a freakin choice, because if you don't, they're gonna die. Like people on COVID that had COVID. I guarantee you a crap ton of them were on 100% Oxygen before they died, they were just desperately trying to keep them alive.

1:00:53

A big point here is is that you want to give your patient as lower level of oxygen as possible. Well, how much do you know how to get started 30. If your oxygen levels don't go up titrated up to 40. They're actually levels don't go up high enough titrated up to 50 small increments until you get their oxygen levels at least matter or bearable for the patient's cell.

1:01:15

So

1:01:17

know this, this is another way to change partial pressure. And it's done in clinical situations. It's done when people walk around in Meijer. You ever see people with the little tank on their wheelchair, any little cannula knows that supplemental oxygen? Why are they doing it? They probably have COPD, or some cardiovascular disease, whether or not I should add the body as well. So they need that oxygen or things aren't going to work out too well for them. All right. Okay, so that's partial pressure.

1:01:46

We're not going to worry about water vapor pressure is another level of complexity that is not needed for this course. So we're not going to worry about

1:01:55

what we do worry about is all these partial pressures that you see here up on the screen.

1:02:00

So what we have here is partial pressure in the air, partial pressure and alveoli for both oxygen and co2. Well, where are they headed co2 come from doc or Dr. Arc, we didn't talk about it yet. Do we produce co2 in our body.

1:02:13

during aerobic cellular respiration, there's a whole bunch of co2 in our body. And it's a partial pressure for it that is normal. So there are partial pressures of co2 in the alveoli in the arterial blood, the venous blood, and it's just a space in ourselves, and you got to know every single one of those values. But you're not just going to memorize them, you're going to learn them.

1:02:33

Because we're going to go over that.

1:02:36

And we're going to go over that. And when we go over that, and that every single one of these field values will make complete sense. So let's do that. And so we're going to do gas exchange. Now, oxygen from the alveoli, enter the blood co2 from the blood into the alveoli. And we're going to do gas exchange at the cells oxygen into the cells co2 Out of the cells and into the blood

1:02:59

that's happening at the same time, oxygen and co2 out. But we're going to separate them, I can't put both of those things on one figure, it will be the biggest hottest mess you've ever seen. When it comes to a picture. I'm separating them, but do understand that the two pictures that we're going to draw are happening at the same time. All right, the first story is going to be the oxygen story. So let's draw it, it's gonna be a big picture, by the way, two big pictures before we leave.

1:03:24

So in order to talk about oxygen, we have to start at the lungs because that's where the oxygen is coming from.

1:03:29

It's coming from the lungs.

1:03:34

And we're gonna put some familiar picture figures here there's our pulmonary capillary, actually, I'm going to be all kinds of fancy, half of its going to be blue and half of its going to be red, the blue part is going to be deoxygenated the red part is going to be oxygenated.

1:03:48

So let's pulmonary capillary.

1:03:52

I'll label everything.

1:03:54

Down here we're gonna put our cell

1:03:58

doesn't matter what cell pick yourself.

1:04:01

We're also going to have a capillary down here, there's going to be a systemic capillary.

1:04:11

And then we have to have the heart in this picture.

1:04:15

That's all one. So let's put our heart right here.

1:04:19

I'll label everything. So this is a pulmonary capillary.

1:04:26

This is a systemic capillary.

1:04:31

And I also have to put here arterial end of that pulmonary capillary venous end

1:04:40

of that pulmonary capillary over here.

1:04:44

It's going to be arterial end

1:04:49

of this pulmonary or I'm sorry, systemic capillary.

1:04:52

And this is the venous and well why just switch it Dr. R because blood vessels are running Every Which Way But Loose

1:05:00

Inside the body, they go left to right up and down diagonal doesn't matter. The reason I do it this way is because it makes the picture prettier.

1:05:06

Now let's make sure that we understand what arterial end and venous end mean, I think you do, but just in case you don't.

1:05:12

Okay, this is your circulatory system and more or less how it's put together.

1:05:20

That's an artery.

1:05:22

This is an arterial.

1:05:25

This is a capillary.

1:05:28

That means that's a venule. And that's a vein, right?

1:05:33

1:05:33

That's the arterial end of the capillary. That's the venous end

1:05:40

of the capillary. That's all it means. You guys already knew this. But I just wanted to remind you, all right, let the story begin. Our assumption is, we're in Dayton, Ohio, sea level. So we have a P O two

1:05:54

of 160.

1:05:56

And we're going to inhale that, we know how that happens.

1:06:00

So we're going to bring that oxygen or I'm sorry, that that air into the lungs, it's oxygenated.

1:06:06

And that air is going to make its way into the respiratory zone into the alveoli. So we're going to have alveolar

1:06:14

oxygen levels, but it's gonna go down to 100.

1:06:20

Well, Doctor, what the hell happened? How did it go from 160?

1:06:24

To 100? There's one main reason and I'll show you what that isn't one second. Now, we're going to have blood at the arterial end of the pulmonary capillary. Is that blood oxygenated to deoxygenated Tell me please?

1:06:39

It's deoxygenated, right? It's venous blood. It's venous blood, VP vo two is 40. Why is it 40? Why is the partial pressure 40? I'll tell you later, later, as in about three minutes. All right. blood is flowing in this direction. By the way, do we have pressure gradients? Is 100 different than 40? Tell me which way oxygen is going to go. Of course, it's going to go into the blood.

1:07:05

So oxygen is going to diffuse into the blood. That's why po two is low in the alveoli because the blood is taking so much oxygen. That's why it's low.

1:07:16

We're adding oxygen to the blood we are oxygenating the blood at the lungs. This is how it happens. By the time we get to the venous end of this pulmonary capillary. partial pressure of oxygen is going to equal the partial pressure of the alveoli. Under normal conditions, that's going to be the case on the exam. I can change P and by the way, big A is alveoli, you're going to see little a as arterial, if I made this 116 This is 116. If I make this 103, that's 103 Whatever alveolar vo two is is what the venous Enfield two is going to be under normal conditions because it calibrates please know that?

1:08:00

Where does this blood go? Of course, it goes to the left atrium, correct via what the pulmonary vein

1:08:08

no label that. So to the left atrium, it goes via the pulmonary vein.

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pulmonary vein has oxygenated blood

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and then to the left ventricle and then to the

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aorta, of course. And so there's the order.

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Now we have arterial blood, by the way

Now we have arterial blood, by the way.

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arterial blood is not arterial blood until it hits the great artery, the aorta, not the pulmonary artery, the aorta. And so now it's not just P O two, notice I didn't put anything over here to designate anything. I didn't put a big a nipple a little b and put anything there. Because you really don't call this blood anything other than oxygenated blood. But now

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I'm going to put a little a there because that's for arterial, it's not for aorta is for arterial. Oh 290.

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Like what? How did we lose 10 millimeter mercury from the time we get to the pulmonary capillary to the time we get to the aorta? It's called the anatomic shut. I'll show you where is your notes. It's right here, you're going to notice that it's all crossed out. It's a level of complexity. We don't have to know it's completely normal. Everybody has an anatomic shut. It will lower your oxygen levels. It's completely fine. We're living just fine. But we're not going to worry about it. Just know that it happens. That's all.

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So that's the aorta. arteries. Arterioles,

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arterial end of a pulmonary or I'm sorry, systemic capillary, where we still have arterial blood. So P little a oh two is still 90. Well, how are we going to get that oxygen into that cell? Well, the P O

1:10:00

In a cell, it's quite low. It's about 20.

1:10:05

Why is it so low because that cell is using oxygen to make ATP. That's why she's in a whole bunch of oxygen. Well, we have a fluid out here, the interstitial fluid and the co2 in the interstitial fluid is 40. Do we have gradients? We sure do. Oxygen is going to diffuse from the blood into the interstitial fluid and from the interstitial fluid into the cell. Beautiful. Now the cell can live.

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Blood flows in this direction,

1:10:30

from an arterial end to a venous, and by the time we get to the venous side, we now have venous blood. So now it's P vo two and the PBO two is exactly the same as interstitial fluid, P O two. And I will change that on the exam, they're going to match each other under normal conditions. So if p O two and interstitial fluid is 43, P vo two is going to be 43. Whatever PDO. Two is, is what interstitial fluid po two is because it is allowed to equilibrate. It's just simple diffusion.

1:11:03

Where does this blood go? So obviously gonna go back to the heart venules.

1:11:10

veins, vena cava, right atrium, right ventricle, pulmonary trunk, pulmonary arteries, pulmonary arterioles.

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Arterial and have a pulmonary capillary and it starts all over again.

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So this is oxygen diffusion gradients. And now you understand why it is that the P O TOS are what they are in this picture. We have P big ao to P layo. To interstitial fluid ao to or I'm sorry, oh, to sell o to look at this, we just covered half of them.

1:11:47

We covered half of them. Now let's do the co2 story.

1:11:51

The co2 story is easier. And why is the co2 story easier because we don't have to worry about the anatomic shock. Why? Because we just don't, it just doesn't come into play. If you want to know why I can talk to you about it in my office, but it's not going to be on the exam.

1:12:05

1:12:05

Now, the co2 story starts at the cells.

1:12:11

Because that's where co2 comes from, we don't breathe co2 in the air, we just we breathe very, very Trace trace trace trace amounts of co2.

1:12:20

Unless you put your mouth around the tailpipe, you're not breathing very much co2. So we're going to start at the cell.

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With this story,

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we're going to put the same anatomical structures in this story by the way, that's a cell pick yourself, the PCO two and a cell and you just have to take my word for it. It's about 50, give or take.

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And so we have to put our systemic capillary in this particular picture as well. So this is going to be the end that's oxygenated.

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This will be the end that's deoxygenated nothing different. Anatomically, here, we need to put the heart in the picture. I'll label that systemic capillary.

1:13:05

I'll also put

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arterial end this as I did before,

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this is the venous end.

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The hearts got to be in this story as well. So let's put our little heart here.

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And we have to put our lung

1:13:30

and we're deoxygenated on this side.

1:13:43

Okay,

1:13:45

pulmonary capillary

1:13:49

arterial, or I'm sorry, venous.

1:13:55

And arteriole, and on the other side. All right. So let the story begin. Now you have to take my word for this, but you're going to see why.

1:14:04

P little 802. That is the amount of co2 the partial pressure of co2 in arterial blood is 40. You're going to see why.

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That P or the P co2 in the air initial fluid.

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Oops, is 45.

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Do we have gradients yes or no? Yeah. co2 is going to diffuse out of the cell into the interstitial fluid. And co2 is then going to diffuse

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into the blood. So we're going to add co2 in the blood again, gas exchange, right? co2 into the blood at the cell.

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oxygen into the cell, right. There's a gas exchange right there, oxygen into the cell, co2 out of the cell.

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Blood flows in this direction. What do you think P V co2 is going to be

1:15:01

What do you think?

1:15:03

45 what it is in the interstitial fluid just like oxygen equilibrates With interstitial fluid at the cell. So to co2.

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co2 levels in your venous blood are higher. Why? Because the cell added a little bit of co2 to it at the cell. co2 comes from aerobic cellular respiration. Where's this blood gonna go? Well, it's going to go back to the heart.

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So venules veins, being Akiva right atrium, right ventricle, pulmonary trunk, pulmonary arteries, arterioles, arterial end of a pulmonary capillary, we still have venous blood, of course we do.

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What's P big a co2 equal to you just have to take my word for it. It's 40.

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We're still 45 Over here. Do we have a gradient?

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Of course we do. co2 is gonna diffuse into the capillary.

1:16:00

There's gas exchange co2 in the alveoli.

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oxygen from the alveoli into the blood. That's gas exchange. Again, I just didn't put the pictures together. Could you imagine this picture in this picture combined in one picture? It would look ugly. I tried. It does look ugly. Blood flows in this direction. Why don't you tell me what PCO two is it? The venison. Tell me what it is.

1:16:25

It's 40. It's what it is in the alveoli? It's allowed to equilibrate. Now we're going to breathe that co2 off. We call that expired co2, e p e co2 actually equals 30. So how do we go from 40 to 30?

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We lost it to the to the sum, it has to do with the dead space and then air coming in mixing with air going out. It's not that important, at least not for this class. It's not. I do want you to know that expired co2 is 30. So if you measured the expired co2, somebody at rest, it would be about 30.

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This blood

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flow in every vein,

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left atrium, I'll label that pulmonary vein.

1:17:17

left ventricle, aorta.

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As I said before, we don't have to worry about the anatomic shot when it comes to co2. So the co2 level isn't going to change. It's a simpler story. So P little a co2, although I already told you what it is, is 40. I told you what it is way over here when we started our story. So why is this 40 Because it's 40 in the alveoli, which means it's going to be for you to be instead of a pulmonary capillary. That's just what it is.

1:17:48

aorta,

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arteries, arterioles, arterial end before of a systemic capillary. And it just keeps on going round and round and round.

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So now we understand why it is that all these co2 values that I have listed here are what they are every single one of them except this over here. We're not going to worry about partial pressures in the conducting zone. We're just not worried about it. All right. Okay, guys.

1:18:14

If you go out tonight, be safe.

Resp PM 3-22-22

Thu, 3/31 11:13PM 1:14:17

SUMMARY KEYWORDS

hemoglobin, oxygen, arterial blood, lung, co2, venous blood, carbon monoxide, ventilation, perfusion, total lung capacity, fev, air, dissolved, normal, blood, cells, called, diseases, means, lecture

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All right, folks, let's get started. So before we go on to new material, just as a reminder, in the last lecture, at the end of it, we were talking about these diffusion gradients to both oxygen, carbon dioxide, the bulk of which we discussed how both of those gases, oxygen and carbon dioxide are travelling through the circulatory system. What we're going to do now is discuss how those two gases are transported within the blood. And so let's do that. So gas transport oxygen, and that's what we're going to start with is transported in two different ways. So when oxygen makes its way into the blood, and that starts over here at the lungs, of course. So as this oxygen makes its way into the blood, some of it is going to get dissolved. Not a lot, because only one and a half percent of the oxygen that's carried in the blood is carried in its dissolved form. And so now what we want to do is and we're going to concentrate on dissolve right now we're going to be talking about hemoglobin, that's going to be actually in more detail, we're going to talk about something called Henry's law, solubility coefficient of oxygen and such. So there's not a whole lot of oxygen dissolved in the blood, why it just doesn't dissolve very well. That's the reason it's solubility coefficient is very, very low, you don't have to know that number, which is why it's crossed out. So again, all those things that you don't have to know for the exam, I actually have already put it on pilot. So on pilot or other things in PowerPoint form your slides, and I have stuff crossed out, it's all done for you already. Oxygen just does not dissolve very well in water, but it does. And and this is an important point, dissolved oxygen is responsible for its partial pressures and body fluids. What does that mean? Well, if you go back to this picture, these co2 values that we see throughout, it's because oxygen is dissolved, that's what's exerting the partial pressure is dissolved oxygen, if oxygen did not dissolve in any of these fluids, po two would be a big fat zero. So that is a very important point. Now, something else that I want to make clear here, there is a direct relationship between dissolved gas and the partial pressure of the gas such that so if you have an increase in the partial pressure of notice, do it this way. Let me first just put Henry's law here, but we're not going to do the whole equation. Let me go back to that slide really quick. This equation right here that we see the concentration of dissolved gas equals the partial pressure of the gas tank solid, because we're not we're not going to do calculations. But we want to know, and we want to be aware of that there is a direct correlation between the amount of dissolved gas and the partial pressure of the gas. So I'm just going to put that little teeny part of the equation here. So the concentration of dissolved gas, whatever dissolved gas, that is oxygen, carbon dioxide did matter is proportional to the partial pressure of that dissolved gas such that if we have an increase in dissolved gas, see there's two s's in there, dissolved gas, then we have a higher partial pressure of that gas, it makes it go up. Another way of saying that is if we have a higher partial pressure of a gas, that means there is more dissolved gas. And then we'll be thorough. And we'll say if there is a decrease in dissolved gas, then you're going to have a lower partial pressure of that particular gas. And then that's the same thing as saying if we have an increase in the partial pressure

of a gas that means more of it is dissolved. Very important correlation that you need to understand. Now what else do we need to know here? For now, that's about it. Now let's go on to hemoglobin. This is where most of your oxygen is going to be as it's traveling throughout circulatory system. Hemoglobin is what's carrying most of the auction how much but 90 and a half percent. It's a little bit higher than that a little bit lower than that when we compare arterial blood with venous blood, but we're really not going to get into those nuances. We will co2 Because the differences are vast, but with hemoglobin, but 98 and a half percent whether it's venous blood or arterial blood, now let's draw hemoglobin. And when I draw hemoglobin, as I've done in the past, I was draw a box with four boxes inside of that box. So there's hemoglobin we have our four little sub units, this is adult hemoglobin.

05:04

And what we can do too is we can put our little teams on here if we want. And on each of those teams is where we're going to bind oxygen. So we have four binding sites for oxygen on a hemoglobin molecule. And when we do, we say that the hemoglobin molecule is fully saturated. So you can have no oxygen 123 or four oxygens when you have four is considered fully saturated. Now we can measure that. And we measure that as SEO to so SEO to saturation of oxygen is a measurement.

05:51

Or I should say is a measure of the percent the percent of fully saturated hemoglobin molecules, so of hemoglobin that are fully saturated, that is look like this. And so sa oh two is measured as a percentage. Now we can measure it clinically in arterial blood, we do that with what's called a pulse ox. So a pulse ox pulse oximeter, I'll just write out the full word. Most often, it's just called a pulse ox, the pulse ox measures sh O two, and it measures your pulse, it measures your heart rate, it's that little thing that you have on the end of your patients finger you can actually go by when am I right now for like 40 bucks, do it yourself at home as often as you'd like. So a pulse oximeter measures that co2 in strictly arterial blood, not venous blood. Now, the physics behind that how it does just the arterial blood, I don't know how that happens. I don't know if it's because of how arterial blood it pulses. Since I have no idea it doesn't matter. But in arterial blood normal values. 95 to 100% is considered normal. What does that mean? In your arterial blood 95 to 100% of your hemoglobin molecules look like that. So nearly 100% of your hemoglobin molecules are fully saturated good. Because where's that blood going? To your cells to give the cells oxygen, most of the oxygen that your cells are going to receive come from hemoglobin, we can sustain the tissues of the body with dissolved oxygen, we cannot. It's not it's not nearly enough, you can sit on your couch and do absolutely nothing and lay as still as you possibly can. If we didn't have hemoglobin, we'd be dead in like a minute or two. We need the hemoglobin to carry the oxygen to ourselves because dissolved oxygen simply is not enough, which is why we have evolved this beautiful protein to get ourselves the oxygen that our cells desperately need to make ATP. Now there is obviously a relationship between oxygen and hemoglobin. Hemoglobin is bound or binds, I should say oxygen. So now we're going to talk about that hemoglobin oxygen binding curve. So I'm going to draw this curve very, very quickly. And I'm going to talk about left and right shifts and what they mean. So let's draw this, we need to understand what it means before we get into the specifics about how we can change the relationship between oxygen and hemoglobin. So we have an X and a Y axis. Our Y axis is Sai oh two, we know what that is now measured as a percent is going to be as high as we can go. I'll put here 75%. You'll see why in a little bit. And those are going to actually be the only two valleys I guess I can put 0% there on the x axis is going to be P O two that is dissolved oxygen. And so 1020 3040 is going to be one magic number that I have there 5060 7080 90 will be another. So those are our P O two values.

Now how do we generate this curve, the sigmoidal curve, this S shaped curve, what we do is and this has been done experimentally is you change the pH so you have an environment where there's hemoglobin, you change the co2 and then you measure what the SA oh two is. So at a PO to a 40. We're about 75% saturated. What does that mean that appeal to a 40 75% of your hemoglobin molecules look like this. It doesn't mean that we have three or four of these sites bound by oxygen. That's not what it means. It doesn't mean that hemoglobin molecule 75% saturated, it means that 75% of hemoglobin molecules are fully saturated again, that's what I say oh two is be sure that you understand that. So at appeal to a 40 75% of your hemoglobin molecules look like this.

10:18

And then what we also do is that these different points, and so I'm just going to put some so on appeal to have 10, we have a saturation of about that, that appeal to of 25, of saturation of about their PR to 30. It's about there at 90, it's nearly 100% saturated. And so we have all these different points and all these different po tubes, and then you fit the line. And the line looks like that it's a six, not a straight line. It's a sigmoidal curve. That's telling us that as co2 goes up, that's a crappy looking line there. That's better. As P O two goes up, saturation goes up, that should make sense. If P O two goes up, are we getting more and more dissolved oxygen? Yes or no? Of course we are, we just established it. If the partial pressure of a gas goes up, that means there's more dissolved gas. If there's more dissolved gas in the blood, there's more oxygen for hemoglobin to grant. And if we have more oxygen for hemoglobin to grab as our co2 levels go up, well, then we're going to be more and more saturated. Should make perfect sense. And so what this template what this is telling us is this is that P O two affects

11:38

sh O two. This is an important point but sh O two does not affect P oh two. P two is affected by other things like ventilation that we're going to be talking about again in the lecture today.

12:01

Now what about these left and right shifts? What do they mean? Well, what I'm going to do right now is I'm going to take a little line, dotted line from a p O to a 40 minute draw straight up, then I'm going to draw straight over and show that we have a 75% saturation. Now why did I pick a peel to a 42 to highlight and why am I picking a peel to have 90? Anybody want to take a guess? Feel to a 40 is the co2 in the venous blood PO 290 is about co2 and arterial blood. So What's that telling us? You know, arterial blood, we are nearly saturated? Do we already know that? Yeah, I just told you that co2 and arterial blood is between 95 and 100%. Under normal conditions, I've already told you that. I'm just showing it to you on this curve right now. In venous blood, we're still 75% saturated, by the way, 75% saturation would be awful. If we were an arterial blood, when you get peel to SEO tubes of less than 90 in your arterial blood, your patients in trouble. Something has gone wrong. Okay. And that's actually stated in the notes as well, I think, yeah, pulsa 90% is considered very abnormal, something is wrong. Now let's move on. Let's talk about a right and a left ship, what is a right and a left shift, meaning I'm going to define it for you. But then I'm going to show you in this graph, how we get that term, this is a right shift, I'm going to draw a right shift in green, the curve is shifted to the right, a left shift is going to be shifted to the left there, it got shifted to the left. And so a right shift, the curve is shifted in this direction. So for the same po two values, we're going to get different saturation value.

So let's just do it for appeal to a 40. So if we draw our line up from 40, and then draw it over to our Sal to line that's much less than 75%. Whereas if you do the same thing for a less ship, well, clearly, we are much more saturated. And so once again, that's going to be a left shift. Now why is that? This is the reason if you here, right shift what that and not just for this relationship, but any relationship that Sigmoidal when it comes to the oxygen hemoglobin are not the only two molecules that have relationship with each other. When you hear the term right shift, what it means is in this particular case, because we're dealing with hemoglobin and oxygen, what it means is is that hemoglobin is not binding. Oxygen is tightly. No, of course it's not look at the curve. It's not holding on to it as tightly which is why there aren't as many hemoglobin molecules that are fully saturated. Left Shift is the complete opposite. A left shift tells you that hemoglobin is binding oxygen more tightly. So that's what those two terms mean. And those are terms that I will use those terms that you'll hear a lot. And it just comes from this curve. Shifting right, and shifting left, right shifts and left shifts of the relationship between hemoglobin and oxygen are 100%. Normal, I need you to understand that as well. I don't want you to think that these shifts are abnormal. So what's going to be happening here is, is that we have a change in affinity. Hemoglobin just doesn't bind oxygen is tightly. Why? Because it just has a lower affinity for it now. Whereas here, hemoglobin is going to have a higher affinity for oxygen. And why is that because hemoglobin is changing its shape a little bit. And there are things that cause hemoglobin to change. It's confirmation. Confirmation is a fancy word for shape when it comes to a protein. We learned that last semester, I think it was the second lecture of last semester. So let's talk about those things that cause a right shift and those things that cause a left shift, we're going to draw one picture, I'll show you the words that we're going to draw, we're going to draw these words right here. From here, all the all that that's what we're about to draw, I'm going to show you how co2, pH and temperature affect the affinity of hemoglobin, for oxygen, I'm going to show you how they cause left and right shifts whether they go up or whether they go down. I'm going to start by drawing a picture that we should be familiar with, because we drew it in the last lecture, the very end of the last lecture, we're going to add the lungs up here. So there's our respiratory zone and our conducting zone, here's our pulmonary capillary. We don't have to put the heart in this picture, but we are going to put a cell in this picture. So this is a cell doesn't matter what cell

16:51

and he was going to be a systemic capillary right there. All right, now let's a story begin. So this cell is going to be going through these metabolic processes. So we have a higher metabolism will be here at the at the tissue. So we have higher metabolic rates at at the tissue. At the lungs, the metabolic rates are going to be a little bit lower at the tissue, they're going to be a little bit higher. Well, during these metabolic processes, these cells are going to in part be making lots of ATP they have to for these metabolic processes, and in so doing aerobic Li, they are going to be producing some products, such as co2 anaerobically, they will be producing some acids, like pyruvate, and lactate. During these processes, they will also produce heat, the cells need to make cell a little bit bigger, so I can put the word heat here. So these are products that metabolism, now the cell has gone to all the co2 and all the acids and all that heat cell is going to die. cells don't want to die. So what they're going to do is that they're going to transport these products into the blood. And so into the blood, these products are going to go. And so as a result of that at the tissue, we're going to have higher co2 levels, we're going to have more acid, which means we're going to have a lower pH. And we're going to have more heat and increase in temperature. These are three variables that can affect the relationship between hemoglobin and oxygen. And it's so happens that higher co2 levels lower pH, by the way, co2 is also an acid, lower pH and higher temperatures each of those individually caused right shifts, all three of them together are going to cause a bigger right shift. So they're going to cause a right shift. Let me write it the same color as I had on the other page. I'll write it in green. So you tell me do we want to right shift at the cells? Yes or no? Let's remind ourselves what a right shift is. Hemoglobin is not going

to bind oxygen as tightly do we want to right shift to the cells? Of course we do. Whereas oxygen has to go. It has to go into the cell. Do you want hemoglobin to bind tightly? Of course you don't. We want hemoglobin to give it to ourselves. And that's exactly what's going to happen more aptly, because we have a right shift at the cell. So again, right shift and let's just completely normal. And so these products over here are changing the shape of hemoglobin in such a way that hemoglobin just simply does not bind oxygen is tightly It is a beautiful thing. The complete opposite is going to happen at the lungs. And this is the lungs over here of course conducting zone respiratory zone. We're going to have a lower co2 level what are the main reasons is our didn't last lecture. The main reason is co2 is diffusing into the alveoli that's going to take off that's going to take some co2 from the blood, we're going to have a lower metabolic rate at the lungs as well. So we're not going to have as many of these metabolic process or these products, so we're going to have a little less acid over here, temperature is going to be a little bit lower, so our pH is going to be a bit higher, these same variables are going to cause a left shift, because lower co2, higher pH, lower temperature just so happens to cause a left shift. And I'll write that in red. Do we want to left shift at the lungs? Of course we do. As oxygen is diffusing into the blood at the lungs, as we learned in the last lecture. And oxygen needs to be grabbed by hemoglobin and is going to be grabbed tighter by hemoglobin because of the left shift that's occurring. So these are completely normal things. And these shifts aren't all or none. We can have a bigger left ship, a small enough ship, the bigger right ship, the smaller right ship, for example, with the ratio, let's say you were exercising, do you think you'd have a higher metabolic rate? And let's say your skeletal muscle? Do you think you'd make more co2? More acid? More heat? Yeah. Do you think you'd have a bigger right shift? Yes. Do you think that would be a good thing? Of course it would, because you need even more oxygen to go to the cell. So you certainly don't want hemoglobin to bind oxygen anywhere near as tightly as it did before that. So just let go of it more. Right shift left shift completely normal.

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Now, there's a couple of other variables that I haven't noticed only one of which we're going to discuss. Again, this is already on pilot, it's crossed off. It's posted, all is good. You don't have to feverishly cross this stuff off. If you want to go ahead. We're not going to talk about BPG but we are going to talk about carbon monoxide. So I'm going to show you how carbon monoxide poisoning kills. So let's do this now. Now we know that we have a normal amount of carbon monoxide in our blood right? We learned that in the blood chapter catabolism of heme, we produce carbon monoxide and a certain amount of carbon monoxide is found to hemoglobin at all times. We call that hemoglobin what carboxyhemoglobin remind you that so we're going to draw another hemoglobin molecule here. And that hemoglobin molecule initially is going to have oxygens bound to it. And so let's put our oxygen molecules bound to the hemoglobin. Alright, that's hemoglobin. Now this particular person, unfortunately, is going to be exposed to high levels of carbon monoxide in the atmosphere, maybe they're in a house fire. Whatever it space heater that happens to be propane starts to screw up and it starts to produce a whole bunch of carbon monoxide. They're breathing it in. And they don't know if by the way carbon monoxide, odorless, colorless gas, you have no idea there could be carbon monoxide in this room right now we'd have absolutely no idea until everybody started getting nauseated and headaches and start to pass up. Unfortunately, hemoglobin loves carbon monoxide a hell of a lot more than it does oxygen. So I've had a choice between the two, it's going to grab carbon monoxide, the affinity for carbon monoxide is about 200 fold greater for Khara as compared to oxygen that's in your notes 200 to 250. See what the value is think it's 250 that I used in the notes I did 250 times greater affinity for carbon monoxide. That's unfortunate. The reason is this, don't have to have a ton of carbon monoxide to start knocking these oxygen molecules off. We could knock one of them off two of them all three of them all four of them off. For this picture, I'm going to knock two of them off, just because I want to know I'm going to knock these two off. So they're going to go bye

bye. They're no longer there. Instead, what's there? Now all right, a couple of lines, right? I'll draw a couple of lines indicating that the bond is a little bit tighter. Now we have carbon monoxide in those sites. Now you might look at that and say, well, that's clearly a right shift. It's not that looks too ugly for me. Let me just, I'm just gonna cross out the oxygens. And I'll do it in blue. So you're gone. Now we have carbon monoxide there. Instead, it's found a little bit tighter. And again, you might think to yourself and see that well, doctor, that's clearly a right shift. It's not. When we talk about a right and a left shift. When we talk about the affinity of hemoglobin for oxygen, we have to talk about it. If there's an actual relationship between there's no relationship between oxygen and hemoglobin on these sites in oxygen is gone. The only thing that we can talk about when it comes to a left and a right shift is where oxygen is actually bound to hemoglobin. And that would be these sites over here. So what's going to happen to the oxygen that is bound to hemoglobin on these sites where oxygen is actually present. That's going to happen. Hemoglobin is going to bind the oxygen even tighter. So when carbon monoxide starts to knock off oxygen molecules, the oxygen molecules that are present are going to be bound, tighter. So we're going to get a left shift, when we draw that and read a left shift at the sites where oxygen is present, I'll give you an analogy. Let's say you're held holding two bags of candy. And some jerk took the bag from your left hand, and you don't want to lose the bag from your right hand, what are you gonna do? You're gonna hold it like this? Well, you're gonna grab your candy really, really tightly. So the jerk doesn't take your other bag of candy. Hemoglobin just got his candy taken away, and it doesn't want to lose the candy that it has. So it grabs it tighter. Now we have two huge problems. What are our huge problems here? So two big problems.

26:01

Number one, do you think our SEO tool is going to go down? Of course it is as co2 is what the percent of hemoglobin molecules that look like this? Well, that hemoglobin molecule. Sure. So don't look like that anymore. It's no longer fully saturated. So number one, we have a big huge decrease in co2. Now how big it depends on how much carbon monoxide was just breathe in. Number two, what's our other big problem? Hemoglobin is carrying this to the cells carrying the oxygen to the cells, but it's grabbing the oxygen very, very, really, it's grabbing it tightly isn't gonna let go. No. Hemoglobin does not let go of oxygen, it's not going to get delivered to the cells. And so both of these problems together mean a big huge decrease in oxygen to the cells. That's what kills you with carbon monoxide if you breathe enough of it in. Now, we know that carbon monoxide that is bounded to or I'm sorry hemoglobin that is bound to carbon monoxide is called carboxy. Hemoglobin we know that. So hemoglobin or carbon monoxide bound to hemoglobin co bound to hemoglobin is carboxy. Hemoglobin we learned that in the blood chapter. And we know that under normal conditions, but 1% Right. But 1% of your hemoglobin that's not sa oh two that's just telling us about 1% of the hemoglobin molecules in your blood have carbon monoxide bound to it anyone time. That's what that says. Smokers it's what between five and 10% give or take, which makes things pretty bad, right? Smokers, their hemoglobin doesn't deliver their oxygen to the tissues as well as somebody who's normal. You know, I'll say the word normal. I think you're abnormal if you smoke. No offense, anybody smokes. But that's not going to cause symptoms that carbon monoxide poisoning does with carbon monoxide poisoning. If you start to get up to about CO poisoning

28:35

you get to about 20 ish percent you're going to start to get a headache you're going to start to feel nauseated why it has to do with not getting enough oxygen to the cells in your brain for example, you get about 50% coma death so about half your molecule hemoglobin molecules are have Hema carbon

monoxide bound to them. Not good at all. Yes. Beautiful I was given the next question. You tell me how he treated was that? Bingo supplemental oxygen. Number one, get them the hell away from wherever the highlight carbon dioxide levels are, but yet supplemental oxygen. So what is the treatment for this supplemental oxygen? How much do you think when we talked about supplemental oxygen, we talked about 30% 40% 50% Anything you don't give your patient? You better give 100% You give them everything you have. I told you that really high levels of oxygen can cause damage over time. We're not worried about that at this point. It's going to take two three days cause kind of damage that I was talking about that develops into a condition called Arts hours a day that's not going to happen. If you have a hyperbaric chamber, that would be ideal hyperbaric chamber, pumped up to about two or three atmospheres plus 100%. Oxygen now, it'd be beautiful. But not all hospitals have this, unfortunately, although it's becoming a bit more mainstream. supplemental oxygen is really what I want you to know, we didn't talk about hyperbaric chambers. But that would be ideal if you could do that. And so when you give them the hot and supplemental oxygen, all that oxygen will start to knock off some of the carbon monoxide molecules and you'll have more dissolved oxygen. Okay, you're going to have about eight times as much, well, seven times as much dissolved oxygen, which will certainly help sustain the tissues of the body much more so than what little you have when it's one and a half percent. dissolved, there'll be a bit more than that, with 100% Oxygen. Are we good with this? Left Shift? Not right shift. So that is, again, our oxygen story. Now, what are we get to talk about? Well, before I move forward, this chart, I think it's in your notes, if it's not as on file is just showing what we just showed, what causes right left shift. Some more things that you get cross out, don't have to do it. Now if you don't want to because it's already on pilot, couple of new terms Oh to capacity and oh to content we already know no to saturation is. Now let's talk about these other two terms. Now the words capacity and content means the exact same thing and physiology as they do in everyday life. Capacity is the most of something that content is actually how much of that something is actually present. So in this room, for example, I don't know maybe the capacity in this room is 90. That would be the capacity of this room, 90 students, there's 90 chairs, I have no idea if it's 90, but let's just go with let's go with 100. That's a nice even number. We don't have 100 students in this room, we might have 40 students in this room, that would be the content. So oh to capacity is the most amount of oxygen that can be carried in your blood flow to content is actually the amount of oxygen that is carried in your blood. And it's going to be different between arterial blood and venous blood when it comes to content, not capacity. The capacity is going to be based on and this is our this is going to be our angle. What carries oxygen the most, which is hemoglobin, right? Hemoglobin Clarys, nearly 99% of the oxygen in your blood about 98.5%. So the capacity is going to be based on how many hemoglobin molecules you have. And how many sites can bind oxygen. That's going to dictate what the capacity is. Okay. Now, where's the hemoglobin? Send the blood, but specifically, where is it in the blood? It's in your red blood cells? Correct. So your red blood cell concentration is going to dictate hemoglobin concentration, which is going to dictate fotu capacity. Do you buy that? I hope so because that's all fats? Does your red blood cell concentration change between your venous blood and your arterial blood? No, if you took venous blood and you did a measure him adequate, you took arterial blood and you measured homematic rate, it would be exactly the same. red blood cell concentration is the same in your arterial blood as it is in your venous blood. So what does that mean? It means that oh to capacity is exactly the same in your arterial blood and your venous blood, do you buy that?

33:39

Yes, hopefully, because that's the case. Content, however, is going to be different. So to demonstrate this, or to give you a visual of this, what I'll do here is is I'll do o to capacity here. So we'll just put together a little chart. And we'll put o to content here. And then we will put arterial blood, venous blood. Now the numbers that I'm going to put here in this chart, are actually calculated values based

on all this stuff that you see here. That is crossed out. And you see that number 20. There, it's an actual calculated value, I did it. But we're not going to get into that kind of detail here. We're not going to worry about it. You can see the word Oh to content, oh to our content or to your blood level, blah, blah, blah. So this number 20. It's actually a real number and there are units to it. But we're not getting worried about what the units are. We just established that oh two capacity in your arterial blood and your venous blood are going to be exactly the same because I'm adequacy exactly the same hemoglobin concentration is going to be exactly the same, which means that your arterial blood and venous blood have the capability of carrying the same amount of oxygen now You tell me this arterial blood? Are we saturate? Are we nearly 100%? Saturated in arterial blood? Yes or no? Yes, we've established that we established it with this curve. At a PO two of 90, we are about 100%. Saturated and arterial blood, we established it here with the pulse oximeter, arterial blood a normal essayhow to is about 95 to 100%. So what that means is, is that pretty much every single hemoglobin molecule in your arterial blood looks like this. So every seat is filled. Right? So Oh, two content, and oh, two capacity, and arterial blood is going to be exactly the same. But not in venous blood. What's our saturation and venous blood? 75%, right, the co2 in your venous blood is about 40 as co2 75%, I'm showing it to you in this chart. So what that means is that your venous blood has lost 20% of its oxygen, or there's 20%, less oxygen in venous blood. Where did he go? To the cells? arterial blood takes the oxygen to the cells. And what's left is what's left in your venous blood. So your venous blood has 25% Less? How do we know that because 75 minus 100 is 25%. And by the way, these are resting values, this is you just chilling out sitting in this room right now. That would be the what's called the extraction, the oxygen extraction ratio. When it comes to exercise, it's a different story. It's the numbers are different. But we're not going to talk about exercise, unfortunately. So this number now is 25%. Less, it's 15. It's again, it's a real number. So why is it less, it's less, because oxygen was given, I'm gonna say 25% of oxygen. 25% of oxygen was given away by hemoglobin was given cells by hemoglobin. And by the way, this is the way that it works. If we look at this picture over here, I obviously do not have in this picture hemoglobin. But if we're talking about oxygen, there's still the hemoglobin that's present here with all that action bound to it will let go, you'll get dissolved in the blood that it will not work its way into the interstitial fluid, and it makes way into the cell. So there's an equilibrium between the hemoglobin molecules and the dissolved oxygen here, we did not get into that kind of detail at all, because it is well beyond the scope of this course. All right. Are we good? Yes. All right. So just a couple of terms that I'd like you to know and to be aware of. That's all. Alright, so with that, let's take a break. When we come back, we'll keep on talking. Shall we continue? Alright, so we just got done talking about how oxygen is transported. Let's talk about how carbon dioxide is transported into the blood. Now, we're not going to get into this in a lot of detail. Here, I just want you to know the punch line of everything. So more stuff that's crossed out, you can see it here on the screen.

38:25

There are bigger differences between what we have in arterial blood and venous blood, whether we're talking about dissolve on hemoglobin and a third way within carbon, or I'm sorry, by carbon itself. Carbon dioxide is carried the same way ocean is dissolved on hemoglobin, but then distinctly in a molecule called bicarbonate. And so let's just go over this a little bit. There's clearly more dissolved co2. Why? Because it just dissolves better. Its solubility coefficient is way higher, not that you have to know what the solubility coefficient is. But I do want you to know that solubility coefficient for co2 is way higher than it is for oxygen, which means it's just going to dissolve better. In arterial blood, we're looking at about 5% dissolved in venous blood, it's 30% dissolved. So vast differences, which is why I'm presenting both of these, whereas with oxygen, it's about one and a half percent for both at about 98 and a half percent for both it's slightly different between venous and arterial blood, but I didn't think it was worth the trouble of just you know, putting those different values in the notes. Everything

holds true for co2 as a gas as a different option. So Henry's law holds true for carbon dioxide as it did for oxygen. So all the same stuff. As we discussed in the beginning. What about balance of hemoglobin 5% in arterial blood again, and now we have only 10% in the venous blood. Again, this is the percent that's being carried. This is not sa oh two This has nothing to do with sa Oh 200000. These percentages are the percent of carbon monoxide carry In the blood and these different blood vessels, veins versus arteries. Now something I want you to note and that is co2 bound to hemoglobin is called carb amino hemoglobin don't get it mixed up with carboxy. Hemoglobin as carbon monoxide bound to hemoglobin don't have to know about the holiday effect, we're not going to worry about it. By the way, you don't have to know about the Bohr effect either, which was over here. When we were talking about oxygen, and so we don't know the Bohr effect. All right. Again, it's autopilot. And then last but not least, we have this molecule called bicarbonate and within bicarbonate as a co2 molecule. In essence, co2 is converted into bicarbonate by hydrating it with water. And so we have this chemical reaction that we're going to be learning about when we get to the acid base chapter, which is why I'm comfortable crossing all this stuff off, we're going to talk about the stuff really in the acid base chapter, we're co2 and bicarbonate play a very large role as we're going to see 90% of co2 is carried as bicarbonate in the arterial blood, of course it is, if you have 5%, dissolved 5% on hemoglobin that's 10%, after it gets gotta be 190 is what's left 60% is carried as bicarbonate in the venous blood. Well, of course, 60% plus 10% plus 30% equals 100%, tests equal 100% venous blood has to equal 100% of the arterial blood, that's all you have to know, we're not going to get into a lot of the specifics. With carbon dioxide, it just need you to know these values. More and more on this when you get to the acid base chapter. Now what we're going to talk about a couple of terms that we're already familiar with ventilation and perfusion, I'm going to remind you of when we talked about ventilation. And this is specifically alveolar. Ventilation, right big as alveolar, I'll remind you as the one we talked about this, just had to remember what lecture was, I think it might have been this one. There it is. alveolar ventilation is what the amount of air going in and out of the respiratory zone and the amount of air available for gas exchange. That's alveolar ventilation. Again, we've already discussed it Now something I want to talk about before I move on to perfusion when it comes to ventilation. And this is going to come up a number of times in the next 35 minutes that we have left in lecture tonight. And that is this. So alveolar ventilation game, we already learned about it abbreviated capital V with a.on, top of it subscript A. Now, when alveolar ventilation goes up and down, it's going to affect the amount of oxygen and carbon dioxide in your lungs and your airways or I'm sorry, in your alveoli, which will then affect the amount of oxygen carbon dioxide in your blood such that

43:01

when we have an increase in alveolar ventilation, what does that mean? It means more air is getting into the conducting zone into your alveoli and that air is coming from the atmosphere which has oxygen in it. And you are breathing off more air to. And so what that means is this, what this is going to do is cause an increase in alveolar co2 Big A, you know what that is we learned about it in the end of the last lecture. And it's going to cause a decrease in the amount of co2 in your alveoli. Let me remind you what those two things are just to make sure that we're on the same page here. There was this picture, so P O two is going to go up when ventilation goes up. P A, that is big A co2 is going to go down when ventilation goes up because you're going to breathe off more co2. Alright. And when you affect the amount of co2 in the alveoli, you're going to affect the amount of co2 here at the venous end of the pulmonary capillary, right? Whatever this is, is what that's going to be correct. You're also going to affect the amount of oxygen in the blood because whatever this co2 is dictated by what this co2 is, therefore, when you increase P big a co2, you're going to increase P little a co2 which is partial pressure of oxygen in your arterial blood and oops you're going to decrease P little a co2 So there's a direct correlation between ventilation, oxygen in the blood, co2 in the blood. And I just put a hard line

here and we'll be thorough with it. decrease in ventilation, so you're not going to breathe in as much air. So not as much oxygenated air is going to get into the alveoli. So clearly, you're going to have a decrease in the amount of oxygen in the alveoli. And because you're not going to breathe in as much air, you're not going to breathe out as much air. So you're not going to get rid of as much co2 from the lungs. So you'll have an increase in the amount of co2 in the alveoli, and as a result of those two, well, then we're going to get a decrease in the amount of oxygen in your arterial blood. And you're going to get an increase in the amount of co2 in your arterial blood important points when we start to discuss pulmonary diseases, for example, and the relationship between ventilation and perfusion, which is now coming up. Before we do that, though, let's talk about perfusion. And let's remind ourselves what perfusion is by definition. And so we learned about that in the last lecture. And that was that now we'll get we'll learn about two lectures ago, here's perfusion, if you recall, it's cardiac output from the right ventricle. And we know the cardiac output from the right ventricle is blood going to the lungs to do what gas exchange to get oxygen into get rid of the excess co2. And the abbreviation is capital Q with a little dot above it. There's a relationship between perfusion and ventilation. And I'm going to show you what that relationship is. And it's a very intimate relationship, they are matched, they're not matched perfectly, but they trend in the same direction. And we'll talk about why. So this is our v Q relationship here. I'm going to draw a lung. And this is going to be an upright lung, I'll show you where this is in the notes. Now, what does an upright lung mean? It means that a lung position when you are standing or sitting, for example. So right now everybody in this room has an upright lung. And so this is all right here. So ventilation perfusion relation, I'm going to start with upright lung. So I'm going to draw a lung that's upright. And here it is.

47:04

Now, that's an upright long, I'm just going to say that this is the apex, this person isn't standing on their head, they're standing on their feet, while they're standing on their head, the same principles would hold true, it would just be in reverse. This is the base of the long, that's the bottom of the lungs. And I'm just going to put these three little imaginary lines in the lungs, although they're not that imaginary are these regions in the lung, that we're not going to get into. Now, when you breathe in air, when you ventilate the lungs, air is not going to get distributed the same throughout the lung, there are going to be regional differences. When you breathe air more than air is going to end up at the base of the lung, and less of the air is going to end up at the top the apex of the lung. And so I'm going to signify that with the size of the abbreviation for ventilation. So most of that air, or I should say most of it, more of that air is going to go toward the base of the lung, the least amount of that air that you're breathing in, is going to go to the apex of the lung, and then right in between is right about the middle of the lungs. Now, when we talk about this air going into the lungs, what does it have? It has oxygen, right. And we just established that as ventilation goes up and down to two levels go up and down such that when we have higher ventilation, we're going to have higher oxygen levels in the lungs, we have lower ventilation, we're going to have less oxygen in the lungs. And so the other thing that I'm going to do here is this is that I'm going to just say that our P O two levels over here at the top of the lung are one arrow up our co2 levels here in the middle of the lung are two arrows up in the PO two levels at the bottom of the lung at the base of the lung are three arrows up, they're all still going up, they're just going to go up more toward the base of the lung because we're getting more air there. It is in large fact because of the effects of gravity. You might think to yourself, well the lungs are about the size of a football, how much different can gravity be from, you know, an object that's this big? It is it has to do with the openness of the alveoli. So there's some physiology going on here that we're certainly not going to get into you want to go to med school I teach them now talking about perfusion, perfusion is what again, blood going to the lungs to pick up oxygen. There's going to be regional differences when it comes to perfusion as well. So this blood is going to the lungs to pick up oxygen in a bunch of different blood vessels. It's not one big huge blood vessel that goes to the

lungs a bunch of smaller ones. So you tell me, where do you is safe, we would want more blood to go to base of the lung, or apex of the lung, where do you want more blood to go to the base of the lung, that's where there's more oxygen. If you want more candy, go to the candy store with more candy in it, don't go to the candy store, there's only two pieces of candy go to the candy store with lots of candy. There's lots of candy over here for the blood. So there are regional differences when it comes to perfusion as well. And so I'll depict that the same way I did with ventilation the size of the queue. So we don't have as much perfusion at the top of the lung at the apex of the lung, why there's not as much oxygen there. We have way more down here. And we have a middle amount. Here it is matched. It's very, very efficient. I'll just give you another visual, these are blood vessels. And so we're a bit more basal constricted here, we're a bit more basal dilated here. And we're somewhere in between there. Greater oxygen, more blood flow to the lungs, such that P O two has the biggest effect on Q by far other things affect Q, but not as much as the amount of oxygen that is present in the alveoli in these areas of the lung. Now let me ask you this. Let's say and I'll just draw it over here smaller, here's a long. And I'm gonna say let me label that long. So we know that it's a long i gonna assume that you know, the other one over here is a long, but you know what the what the hell? Okay, that's a long.

52:04

Let's say that that area right there. We have a new Mo. Semi draw that I want to go into right in it. So there's our long, and we have a new Mo. Right there. So that little part of the lung for big part of the lung collapsed, how much oxygen is there? Tell me a big fat zero. There's no ventilation here. So with that pneumo there's zero ventilation. So how much oxygen is there? Not zero. Oxygen, how much blood do you think is going to go there? Nothing. That would be stupid. We're going to basal constrict the hell out of those blood vessels and make it go to other parts of the lung where we do have oxygen. So we'll be here. Zero Q. Just so you know. All right. Although, no, nevermind, nevermind. I don't want to go off on tangents. All right. So far, so good. This is all up right long. Now how about a horizontal line. And so that's going to be the next one. Now we have these regional differences when the lung is port, I'm sorry, upright. Horizontally, all those regional differences are going to disappear. And when we talk about a horizontal lung, we're talking about a patient who is either supine or prone. Supine, how's your patient laying down? on their back, right prone, they're laying on their front stomach. And there are ways to do it for the patient to be comfortable, by the way. So here's our lung. I'll put those three little regions again. And I'll just say that this is the AP, you know, it doesn't even matter. You don't have to label it Apex bait doesn't matter. They're horizontal depends on how they're laying on their on their stomach on their back doesn't matter. What's going to happen now is this ventilation is going to be consistent throughout as his perfusion, the regional differences disappeared. Now there is slight regional differences from the way that the lung is oriented here at the top over here to the bottom, slight regional differences, we're not getting into that kind of detail. So regional differences disappear. So we have more uniform ventilation and perfusion. Now you can take advantage of this fact. So let me ask you this. Let's say that this person has a pneumothorax at the base of their lungs. Would you want that patient to be upright? Let's think about this for a second, would you want that patient to be upright? No. Okay, the effects of gravity are causing more perfusion and ventilation to want to go to the bottom of the lung. Instead, what you would want to do is take advantage of those areas of the lung where you do have ventilation. And so certainly putting it off, putting them upright, would not be the best idea. Now, I mean, theoretically, if you had a pneumothorax at the base of the lung, you know, maybe one of the best things you can do is stand them on their head. That might sound kind of fight with their beds that have the ability to try to twist and patients all over the place. They're called roto prone beds, they're really look them up, they look like they're, it looks like a torture chamber, I'm actually gonna write it down, look them up. They're just the coolest looking things ever. roto prone beds. You can't even buy them. They rent them out to

hospitals for like 1000 bucks a day. There's one company that makes them I don't know, if other companies make these types of beds, they were very prevalent, with a condition called ARDS. And here's another thing. It's the prone position, especially prone position. Has gas exchange. Stored I want to use the value especially inpatients with a condition called ARDS, acute respiratory distress syndrome. One of the main reasons is that we have uniform perfusion and ventilation. That's one of the main reasons now you might ask yourself, Well, why not supine, because that's the horizontal lung.

56:53

When you're on your back, your liver can kind of tend to push in this direction on the diaphragm, for example, and not not allow you to ventilate quite as well. If you're prone, your liver will not have that same type of effect. So the effects of the liver and gravity pushing. It's just not as good Now certainly supine, okay, problems way better. COVID patients were put in the prone position all the time to help them with the gas exchange, that was absolutely terrible. Cuz a lot of a lot of COVID patients were in ARDS, unfortunately, that's what killed him. And yet is, you know, things that happened because of the virus itself, ammonia leading to other really, really bad things. And again, it takes advantage of how these regional differences just more or less disappeared. All right. Now what? So as I was talking about before somebody has a new mo someplace, you lay them in a position where you can take advantage of the area of the lung, where you'll have better perfusion and better ventilation. So you just kind of twist and turn them and those beds, the left, kind of rock them a little bit as well, again, look it up. They're really really cool beds. It looks like they're torturing the patient. They're not typically the patients aren't even conscious when they're in those beds. Typically, they're not. So anyway, now what? This should be crossed off and it is on pilot, we're not going to get into this kind of detail here shunts PQ defects, we're not going to get into that kind of detail. Okay, so these these words, right here is what I had. Put in those, those two pictures that picture that picture, along with some words over here is all that stuff in the notes. Now what, let's talk about some diseases. Let's talk about pulmonary diseases. There's two types of pulmonary diseases, obstructive diseases and nerve restrictive diseases. And so what's the difference between the two? Well, I'm going to present to you passageways law to help you kind of understand. And then we're going to get into some specifics about restrictive and obstructive diseases, and we're going to compare and contrast the two. So plus ways law airflow equals, as we know, a pressure gradient over the resistance to airflow. When it comes to an obstructive disease, that obstructive disease tells you exactly what's happening, there is an obstruction that is inhibiting the flow of air so there's greater resistance. So when it comes to obstructive disease, let's just say obstructive diseases, because there are many. We have an increase in resistance bless you, which means why we're going to get a decrease in airflow. Which means why we're going to get a decrease in ventilation, actually, I'm going to put that separate. Alright, so we get a decrease in airflow. Why? Because we have an increase in resistance. With restricted diseases. We don't have any obstructions We don't have any, any problems moving air, the problem is moving enough air. That's our problem. And so when it comes to a restrictive disease, restrictive diseases, I should say. And we'll talk about specific ones, we won't get to it tonight, restrictive diseases, we're going to have a decrease in pressure gradients, and therefore, a decrease in airflow. Now I'll put them together if we have a decrease in airflow because both of those things are going to occur whether we have an obstructive or restrictive disease, well, that means we're going to have a decrease in ventilation, which means we're going to have a decrease in the amount of oxygen in our alveoli and an increase in the amount of co2 within our alveoli, which means we're going to have a decrease in the amount of oxygen in our blood. And we're going to have an increase in the amount of co2 in our blood, things that we've already discussed. And so these are the kinds of things that are going to happen with these pulmonary diseases, which are obviously bad.

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So now what are we gonna do, we are going to compare and contrast restrictive and obstructive diseases. And then after that, we'll talk about specific obstructive and restrictive diseases. Now, we have a number of facts in your notes, right there. So all that right there, obstructive pulmonary diseases, and I give you some examples. And then I go to restricted diseases, and I talked about the same exact things underneath restricted disease. And then we have this now this was not in the original PowerPoint that was on pilot. I saw this last night when I was going over the lecture, and I said, You know what, and this is from a lecture I gave like two years ago. So you know, I'm gonna put this up there, which is a table that I put down, it just saves me time, because I'm kind of pressed for time. This is comparing and contrasting obstructive and restrictive diseases. And so the first thing that I talked about are volumes and capacities and the volumes, the capacities of those volumes of capacities that we learned in the first lecture, these over here. Volumes and capacities when it comes to obstructive diseases. Within obstructive disease, because we have problems moving here, one of the biggest issues with the restrict or obstructive disease, is you just trap more and more air in your lungs. What's that call? The air that's trapped in your lungs all the time, tell me what that's called, what volume is that? residual volume. residual volume goes up, up, up, up, up, up up with obstructive diseases as the disease process gets worse and worse and worse and worse. And so if residual volume goes up, up, up, up up, that means that FRC will go up to which means that TLC will go up. So with obstructive diseases RV goes up. And as a result, FRC goes up and TLC goes up. Well, you might say to yourself, well, that's fantastic. We have all this extra air in the lungs. No, it's crappy air. It's stale air. It's deoxygenated air. It's not fresh air, it's trapped, crappy air. That's not good. Those three will go up, everything else goes down. Because it crowds out the other volumes and capacities, ERP goes up, Irv goes down, it goes down, VC goes down, they all go down. Except for those three. And that's in the chart, by the way, this is on pilot. So RV goes up. And as a result, these two go up, although their volumes capacities go down. With restricted diseases, all of the volumes of capacities go down. Again, our issue is not being able to move enough air, these pressure gradients are small, because the pressures themselves are small, and the pressures are small, and the gradients are small, the lungs are just not going to move as much air, so they're just going to get a little smaller. So in that chart, again, which is on pilot, all volumes and capacities go down. And then the other thing that we talked about with spirometry was the FEV one FEC maneuver the forced vital capacity maneuver the one where you've taken as much air as you can and then blow the air out as hard as you can remember that I'll remind you, we're actually going to go through it again. And as we do, I'm actually going to put some real numbers in here. So this maneuver right here. So you go to total lung capacity, and you blow out as much air as you possibly can. And we established that under normal conditions healthy long. You should be able to after the first second which is FEV, one blowout about 70 to 80% of your vital capacity. And we should have normal values for FEV. One and vital capacity. That's going to change with obstructive and restrictive diseases but change in different ways. So there are already ways to distinguish a restrictive and an obstructive disease by doing what by looking at the billions of capacities but also this very, very powerful test that doesn't take very long at all. So this is what we're going to do, we're going to draw normal versus obstructed, then we're going to draw normal versus restrictive. And we're going to see what this test looks like when it's taken. So we have our little graph here, volume of air. And that's just time. And this is going to be normal. So that is normal. And then we will contrast it with no, we'll go red. And we're going to say that red is going to be obstructive. So we're going to do with obstructive disease first. So they're going to go to total lung capacity. And then they're going to, and that's when we start our test that's at time zero, and they're gonna blow out as much as you can as fast as they can. And after about, I don't know, 234, sorry, about three, four seconds, test is over. So that's one second, two seconds, three seconds, four seconds. And of course, this is residual volume. I'll just throw that in there for the hell of it. This is obviously total lung capacity up here.

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Here's our FTB. One, right? In our FEC is this whole stinking thing, from top to we get to this point, right? Now let's do an obstructive disease and what it would look like. Now we know with typical spirometry, we cannot measure total lung capacity, right? We can't measure residual volume, but I'm going to draw this in a way that shows what total lung capacity will be and what residual volume will be, which is what bigger for both correct. So I'm just going to draw it higher, okay, this total capacity would be higher. And now this person at time zero is going to blow that air out as hard as fast as they can. Now they haven't obstruction, do they? It's going to take them longer to get the air out of their lungs, or absolutely well. So this is not going to be very steep now depends on how severe the condition is, but I'll make it severe.

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It's gonna take them longer to get it out, residual volume is going to be higher, right, bigger. So we have a bigger residual volume. I'll just remind you of that, we have a bigger TLC. I'll remind you that now let's do our FTB. One, two FEC. So here's our one second mark. Does that look 70 to 80%, to you know, after the first second, if they're not getting 78% of the out of their lungs, they can't. They tried, they just can't because they have an obstruction. So our FEV, one went down a lot. FVC went down to just not quite as much, I don't know, I'll just go two arrows, or even one, it doesn't matter. So what does that mean? That means our ratio is not certainly going to be 70 to 80%. And so what's our ratio going to be our ratio is going to be something less than 70%. So FEV, one, two FVC is less than 70%, which means you cannot get that air out as fast as you normally would because of the obstruction. Okay, so far, so good. Now we're going to put real numbers in just a little bit, we'll compare normal to obstructive to restricted. And so now let's do restrictive. So we'll do a normal first. And again, normal is dark line, restrictive, we'll go with blue. And again, we'll talk about specific obstructive and restrictive diseases in my next lecture, which won't be until Tuesday, Siva is going to come here on Thursday, I don't want to eat into her lecture at all. I mean, it's okay, I'll just finish up on Tuesday and then start right into the kidneys. So again, normal. So you breathe into total lung capacity. That's where our test starts at times zero. And then under normal conditions, 70 to 80% of the air out within a few seconds. There's one second, two seconds, three seconds. Okay, there's our Fe v1. Our FPC is from top to bottom. Now let's see what happens with a restricted disease. There's no obstruction. There's absolutely no issue moving air through the airways, none whatsoever, the problem is not moving enough. So our total lung capacity certainly will not be as big but we'll certainly not have any issues moving here. So this the slope is going to look exactly the same. And we'll probably be done blowing out the air a bit quicker. So we have a lower total lung capacity. We have a smaller residual volume, everything smaller, let's do our FTP one sec.

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I'll say that we're down three. Now let's go down to could be three, but let's just do two

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and our MVC is also down to is proportionately going to be the same. So what do you think our ratio is going to be? If it's proportionately the same, what's our ratio going to be? Gonna be normal? FEV,

one? VC, it's gonna be normal. It will be between 70 and 80%. Well, Dr. Arhat, how you gonna know if they have a restricted diseases studied 80%. You got to look at the raw values, the raw numbers, and that's what we're about to do. So I'll do normal again, when it comes to FEV. One FEC, then we'll do obstructive, and then we're going to do restrictive and you're gonna see the differences between the three. So normal. And I'll do the same numbers that I did before in I don't know how many lectures that was one lecture ago, I think. So FTD, one to FEC. I did 4000 millliliters for FEV. One 5000 millliliters for FEC that equals 80%.

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What color do they use? Red? Yep. Obstructive disease? FEV, one FEC? Let's go 2500. So we're going to cut it in half. That is the FTC. And let's go.

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I'm picking this out of the air completely out of the air. Oh, no. 1000. I don't want to do that. I don't want you to think it's just half. Yeah, that's fine. It's fine. 40% doesn't necessarily have to be half of what normal is, it just happens to be easy math. Oh, you know what, let's do this, too. I'm sorry. I'm gonna put how far down each went, I want to go with the same amount of arrows down three, down to. So down three. For FEV. One. FPC doesn't go down quite as much. Okay. And now let's do restrictive. Again, the average or I'm sorry, the percents going to be exactly the same. So we went down to with FEV. One, we went down to with FVC. Well, that worked out perfectly, because that's 2500, the same exact number that we have over here down to FTCS to down to FTCS. But this is only down to over here. So are 2000. So 80 percents normal. So again, how do you know if it has a normal ratio? You have to look at the raw numbers. You have to know what normal is and what is normal, around 5000 for a vital capacity. And again, normal when we talk about respiratory fears is for an average sized adult male with healthy lungs. So you have to know what the normal is. And then you would realize that even though this is 80%, like a normal individual, these numbers are too stinking small. Alright, so that takes care of that chart that takes care or not. No, actually it doesn't. We already know this. We're going to have a decrease in ventilation when it comes to both restrictive and restorative diseases. So this part over here, we actually already wrote it down. Here, again, this is happening with our obstructive and restrictive diseases. So that's the third part of the chart. We already took care of it. When I come back on Tuesday we will talk about specific pulmonary diseases both obstructive and restrictive, and that will be done after won't take probably more than 1015 minutes and then right to urinary we go so again secret will be here on Thursday.

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Thu, 3/31 11:13PM 1:14:07

SUMMARY KEYWORDS

gfr, filtrate, kidneys, capillary, glomerulus, filtration, blood, blood pressure, draw, filtered, plasma, big, normal, cardiac output, efferent, bowman, mercury, emphysema, urine, called

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00:02

Okay, folks, it's about that time. So in the last lecture, we were finishing up the respiratory chapter. And we were comparing and contrasting obstructive and restrictive diseases. And now to finish it off, we're just going to talk about specific obstructive and restrictive diseases. This is not an exhaustive list, but these are the big three. As far as I'm concerned. The first two emphysema and chronic bronchitis are considered COPD. Asthma is not. And so we've already talked about emphysema, we talked about how it's due to allow some elastic fibers. There's some details that I actually that I actually didn't discuss when it comes to the recoil, the lung also being lost and how it's going to actually cause the obstruction itself. It's a whole level of detail that is beyond the scope of this course. So I'm just I didn't discuss it, I'm not going to discuss it. But do understand that emphysema is considered an obstructive condition, obstructive disease along with chronic bronchitis, chronic bronchitis a little bit easier to see why we have an obstruction in the airway. And so with chronic bronchitis, what's happening is that you have a lot of inflammation, a lot of mucus being produced, that inflammation of mucus is going to be in the airways. So now the airways are going to get smaller because of all the extra mucus, and the inflammation is occurring. So that's pretty straightforward and easy to see why there would be an obstruction within the airways itself. Now, people get bronchitis some people do occasionally. That's not this. This is chronic bronchitis, it doesn't go away. And that's the other thing when it comes to emphysema. It doesn't go away. Asthma might not go away. But the signs and symptoms of asthma can come and go, meaning somebody can walk around with asthma. And then this day have an asthma attack, where the airways are hypersensitive, and they constrict and at that particular point in time, they have an obstruction. Two hours later, they might be walking around completely normal do a spirit metric test. And their values would be like anybody else. And they can go like that for months and years and years and years. And then all of a sudden, maybe two, three years down the line, boom, another asthma attack. But under normal, you don't have an emphysema attack. You don't have a chronic bronchitis attack. You have emphysema and the signs and symptoms of emphysema are going to be there. Same thing with chronic bronchitis. And here's another thing. If you have one, when it comes to the COPD, you're going to get the other one. You might have one first and eventually the other one develops, but hardly ever you're gonna see a patient that has been treated that does not have both emphysema and chronic bronchitis almost never you will see that they're always together. Reason. The main cause anybody want to guess what the main cause of emphysema, chronic bronchitis are? Or is I should say, smoking. So eventually it's going to cause both of those things. So again, COPD is just these two. Where am I? COPD is just these two, asthma is not a COPD, although some websites will tell you it is people will tell you this, we are changing the structure of the lawn with emphysema and chronic bronchitis, not so much asthma unless in very rare cases, you have a severe severe, severe severe form of asthma, where it might start to change the structure of the airway, Senate can become a

chronic condition. But for the most part, that's just not the way that it works. So those are the obstructive diseases that I want you to know. When it comes to the restrictive I think I have four, I think three of which we've actually already discussed, but we'll touch upon them. fibrotic lung disease interstitial lung disease, when we talk about those we discuss those. Where is it over here? We're talking about compliance.

04:03

Here where's our increases a decrease in compliance damage, scarring of elastin fibers there it is interstitial lung disease, the lung gets stiff, it gets scarred. Why would this happen? A ton of different reasons. Damage to the lung in such a way that the lung starts to get scar tissue. And also elastic like it normally is. It's it's hard. And so as we know, because we learned in the last lecture, the biggest problem with these restrictive diseases is that you have the difficulty moving enough air, it's not that there's an obstruction, you just don't move enough air in. In the case of the fibrotic lung disease, you just can't expand the lungs properly. And if we go back to the mechanics of breathing, and we're going to in this little flowchart that we did when we were talking about the mechanics of breathing and how we inhale and exhale, we had this series of events that needs to occur. And those respiratory muscles might be completely healthy and they might contract properly. But they're just not going to expand The thoracic cavity properly or they're not going to expand the lungs properly. Why? Because the lungs are scarred. So everything downstream is going to get affected because the lungs just simply cannot expand. So that would be a fibrotic lung disease. The other thing with fibrotic lung disease, and I'm going to add this over here. So I'm going to draw fibrotic lung disease for your interstitial lung disease. It's the same thing. So capillary, there's our polar, there's our pulmonary capillary, there's the alveoli, here's fibrotic lung disease, kind of in a nutshell. There's our fibrosis right there. So that scarred tissue. And this is ILD interstitial lung disease. And so another issue with fibrotic lung disease in official lung diseases is that we have poor gas exchange, we need this exchange membrane over here to be just one layer of endothelial cells that just one layer of the alveolar cells, the type one pneumocytes, mainly, well, if we have this thickened area right here, because we're scarred, we're going to have impaired gas exchange, that just makes the problem even worse. We already have a decrease in gas exchange, because our ventilation is all screwed up, we know that we have a decrease in ventilation, we discussed that in the last lecture when it comes to all these pulmonary diseases. But now on top of it, we have this problem as well, because this membrane is just not what it's supposed to be, it's thicken, it's going to be much different, much more difficult for the gases to diffuse across that membrane. So that's interstitial lung disease. And then we have some diseases that affect our respiratory muscles. And there's a bunch of and you can have some kind of a neuropathy like Ganbare, you have a muscular dystrophy, which is a my apathy. You can have ALS, we talked about this last semester destruction of the what the motor neurons, and now those muscles are weakened. Well, if the muscles are weak and the respiratory muscles are weakened, let's go back to our little flowchart. If these muscles right here, do not contract properly, everything else after it is not going to happen the way that it should. And you simply will not move enough air. If you took the X ray, of a lung of people with these kinds of that would look completely just like anybody else in this particular room. There's nothing wrong with the lungs of people with these diseases that affect the respiratory muscles. The problem is the muscles themselves, the diaphragm, the external intercostals, what even the accessory muscles that we actually didn't talk about. So understanding the mechanics of breathing helps you understand why these particular conditions are going to compromise the movement of air because we went over in some detail. Surfactant deficiency disorder, we already talked about this too, when we were talking about compliance. So I'll go back to it, we were just there. Right here. So we were talking about compliance, loss of surfactant. And then we do this little picture over here talking about what happens when we don't have surfactant alveoli flat as the stinking pancake. We have a stiff lung, now we know that our compliance is going to go

down. Certainly gas exchange is going to be impaired, right? You don't have any air in the alveoli, how you gonna have gas exchange. I know it's not specifically in the notes. But we'll have gas exchange issues with surfactant deficiency disorder. And so with that, again, we're going to have difficulty moving enough air.

08:34

It's also going hyaline membrane disease that used to be called infant respiratory distress syndrome, because most often we do see it in preterm infants. Because, again, the last thing that develops during gestation is the respiratory system. And so little babies just don't make enough to back again. So that's another restricted disease. And then last but not least ARDS, acute respiratory distress syndrome. I'll draw this one for you, too. We have diffuse alveolar and pulmonary capillary damage. So the entire gas exchange membranes are destroyed, or damaged, maybe completely destroyed. So here's ARDS for you. So we have a pulmonary capillaries, the alveoli. There, that's ARDS, it's all been reached. And so with that, we will also have impaired gas exchange. We will have a theory a stiff lung why we're gonna have a stiff lung with this. Because with the destruction of those membranes, we're going to now get infiltrates of like fluid within the lung itself, all that fluid will stiffen the lung up. So we're also going to have a stiff lung because of fluid excess fluid. So I have a decrease in compliance with this as well. Step one, because of the hell am I talking about here because of excess fluid in the lungs? Very difficult to treat arts, there can be a lot of people who had COVID developed arts, and it was very, very difficult to treat them. Because we have all kinds of problems with this particular condition. And so, I don't know if I have anything else. No, I don't. So that's pretty much what we need to know, when it comes to the restrictive forum. Are there more? Yeah, so they're more obstructive, yes. Don't have to know. So you don't have to know. So that's that for respiratory. Now let's move directly into the urinary system. So the next chapter, which is going to be the only other chapter for the next exam, which is why a week from today. So these two chapters are going to be on the next exam. So let's talk about the functions of the kidneys. We already did all these. We already talked about those talked about those over the last two semesters, talked about EPO earlier on this semester, with the blood talked about blood pressure regulation, we did that when we did the circulatory chapter, we talked about the production of vitamin d3, the most active form of it, made by the kidneys, if you remember, for last semester, when we were doing the skin, we included this. So really, this is what we're going to concentrate on in this chapter. And so let's talk about the functional unit of the kidney, which you will aware of is the is the nephron. There's about 2 million of them total, but a million in each kidney. What we're going to focus on in the beginning part of these lectures is a cortical nephron. We'll talk about the juxta medullary nephron in the next lecture on Thursday. Now what's the difference between the two, a cortical nephron, most of its structure lies within the cortex. So over here to the right, that's a cortical nephron, a juxta. Imaginary nephron has structures within the cortex as well. But they have really long loops of Hanley, and these loops of Hanley can go all the way down into the renal pelvis very, very deep to this area right here. And I'll show you another picture on Thursday, when we talk more specifically about these particular nephrons. But of these two, these are the majority of the cortical nephrons, 80% of them that are doing the bulk of the work the bulk of the function of the kidneys that I will be discussing. And that's all we really need to know for right now. More on especially the juxta mega Larry's on Thursday. Now, the main job of your kidneys is not to make urine. And that's what a lot of people will tell you as well books will say as well websites will say no, that's not the job of your kidneys. Your kidneys do produce urine, but they produce urine as a result of filtering your blood and specifically your plasma, your urine, every single bit of your urine used to be in your blood, your kidneys removed it all, turn it into this waste product that is going to get excreted from the body. And eventually it's in the bladder and eventually, probably in a toilet. So the main job is to filter I have blood here, but it's specifically the plasma that it is filtering. Because it's not going to filter the core puzzles, red blood cells, white blood cells, platelets,

too big to be filtered. There are three processes that are going to allow the filtering of the plasma and they're here glomerular filtration to be the reabsorption and secretion. So what I'm going to do is I'm just going to give you the big picture of those. And I'll refer to this picture a number of times and draw it in a little bit more detail at some other times. So what we're going to have here is Bowman's capsule, I'll label everything. So that's going to be Bowman's capsule, then we have a tube coming off of it.

13:36

So that's Bowman's capsule. So this right here, is going to represent let me just label this Bowman's capsule. So that is I'm just going to abbreviate a BC. So there's Bowman's capsule, and then we have a tube coming from Bowman's capsule, that's a pair to or the PCT proximal convoluted tubule, followed by what the loop of Henle. So LM H, then the distal convoluted tubule. And then we have our collecting duct. That's what that tube represents all of those, all those structures. Then inside of this cup, is a bunch of blood vessels. And those blood vessels are called what the glomerulus. So the glomerulus they're specifically capillaries. Now, they're coiled inside Bowman's capsule, I don't want to draw them like that, because number one, I suck at drawing. And number two, it's just not going to help me explain what's happening here. So what I'll do instead is I'm just going to draw one tube, kind of make it go in there. And we know that the tube starts out is what the efferent arteriole. Then we have the glomerulus. And then we have the efferent arteriole. You know that wasn't you know what, let's just label them really quick. So there's our efferent arteriole efferent E from a ferret. arteriole. And so that's that in between the two is a bunch of capillaries called the glomerulus. And that's what the rest of that is going to represent. So again, the glomerulus is capillary. So there's plasma, there's blood flowing through this too. And it's part of this nephron. And so what's going to happen initially is, is that we are going to squeeze some plasma out of those capillaries. And so that's what this is going to represent. This arrow is us squeezing fluid out of those capillaries. Tell me what that fluid is now going to be called now that has been caught in this cup filtrate. So I will write the word, I'm going to draw that just a little bit higher, because I'm going to tell you what's in the filtrate. A little bit higher. So filtrate used to be plasma? And how did it get there through the process of filtration, and we know what filtration is, at least I hope we do, because we learned about it in a crap ton of detail last semester, Chapter Three passive transport mechanism, we discussed it after we discussed osmosis, if you remember, that's glomerular filtration filtration that occurs at the glomerulus. And so I'll write it here. glomerular. Gonna make my Bowman's capsule just a little bit bigger. So glomerular filtration. And if you recall, last semester, filtration is driven by hydrostatic pressure. We'll actually talk about it in a little bit of detail shortly. And so the first thing we need to do is form this filtrate. So let me lower it a little bit. So what is filtrate made up? Well, filtrate was at one time plasma, we can't call it plasma anymore, because it's no longer inside of these blood vessels. And so it's mainly water plus small solutes. So filtrate is water plus small solutes. Now, what do I mean by small solutes, sodium, potassium, chloride, calcium, magnesium, amino acids, glucose, etc, etc, etc, etc. What it's not, it's protein, why proteins too big. We cannot squeeze the protein through the walls of the glomerulus. Too big can't squeeze red blood cells through it either can't squeeze white blood cells can't squeeze platelets through. And there's a number of things that you will not find in the filtrate why they're too big. So it excludes big things. What it doesn't exclude are these little itty bitty things water plus too small solutes. That's why if you see protein in your urine, and eventually there's going to be urine, right? Eventually, it's going to be so this filtrate is going to do what it's going to flow through all of these tubes. And eventually, we can call it urine. But not until it gets to the minor kala C's then we can call a urine. Why can't we call it urine over here, because it's going to get modified in the modification process is part of the filtering of the plasma. So what am I showing you here, the three processes that allow filtering of your plasma, the first one is glomerular, filtration. The others are what reabsorption and secretion so let me show you those. So we have this efferent arteriole. And it

is going to give rise to some more capillary. So we have a very, very specialized structure here, typically what happens, this is the efferent arteriole. Right, so typically, it's what arterial capillary, and then what

18:37

venue is about the way the circulatory system is put together. This is very different arterial capillary back to arterial. So we have very specialized structures here for very good reasons. Now, we have this now, when it comes to a cortical nephron, this blood vessel or these blood vessels here, and I'm drawing this very simply, I'll show you a pretty picture of it in just a second. I'm sure you've already seen the pretty pictures of these things. This is the peritubular capillary. So we go arterial capillary, arterial capillary. And so these are the arterial capillaries in the arterial capillaries are running alongside the proximal convoluted tubule, the loop of Henle, distal convoluted tubule in the collecting duct, whereas the glomerulus I want you to think of the Bowman's capsule as it's a cup, take a whole bunch of yarn and throw it in the cup, the yarn, that's what the glomerulus is, now put a hole at the bottom of the cup, and stick a tube on it. That would be the proximal convoluted tubule along with all the other structures. And what's happening is is that as this filtrate is produced as the plasma is squeezed out of the yarn, it falls into the cup, and then it kind of trickles down to the little hole at the bottom of the cup, when it goes into the proximal convoluted tubule. And then that fluid then flows through all of these tubes and acid Does it gets modified? Well, how are we going to modify it? Well, one of the ways that we modify it is we take most of this filtrate, most of the water, most of the solutes, and we transport it all right back into the blood. And we call that reabsorption. I'll write that here. So this is re absorption. Re absorption is taking the filtrate and transporting it right back into the blood. And most of its going to go back into the blood 99% Give or take is going right back into the blood, but very selectively, and when we get to reabsorption, we'll talk about what that means. There's another process that's going to occur, that's going to help with again, the filtering of the blood. And that's called secretion. Now, when it comes to the plasma, actually, I draw that in red, when it comes to the plasma. And again, that's what's getting filtered that all the plasma gets filtered here, it couldn't, right. If all the plants we got filtered here, it means that we'd have no plans would be on this point. And blood flow would stop right there. Because all you have left is white, red blood cells, white blood cells, platelets, you'd have no fluid left. So we cannot filter all the plasma in one run, only 20% is going to get filtered in one run, it'll eventually come back all of your blood, all your plasma is going to get filtered eventually, but not in one run. So when it comes to glomerular filtration, we're looking at about 20% of the plasma gets filtered at any one time as its float. And again, this is this is representing one nephron there's 2 million. So we'll get into this in a little bit more detail shortly. So again, we still have blood flowing through all these tubes and add blood has plasma and it has everything that blood has. And as it does, something else will happen. And that is we're going to take some solutes and water from the peritubular capillaries and we're going to transport them into the filtrate. We call that secretion. Now, the angle I've had so far is is that we are taking solutes and water from the filtrate and adding them to the blood and we're taking them from the bloating adding them to the filtrate. But another angle to look at this, and this is the angle that you should look at this is we're adding things to the blood taking things away from the blood. And in so doing, we are filtering the blood. We are modifying the blood the way that we need to modify it. So that we keep our sodium levels normal. Our potassium levels normal are chloride levels, overall calcium levels normal glucose the way it's supposed to be pH the way it's supposed to be, and so forth, and so forth and so forth. What we what we will remove from the blood is what needs to be removed from the blood and eventually become urine. So these three processes were mainly filtration, reabsorption, and secretion all work together to remove things from the blood that don't belong in the blood that

the body does not want. What we do want is going to stay and maintain homeostasis. And we'll get into these things in a lot more detail. But this is this chapter in a nutshell, this picture is, but we're going to talk about each of these things in more detail.

23:28

So let's start. So let's start with glomerular filtration, that'll be the first thing that we talked about. So with glomerular filtration, as we already know, we're taking plasma, and we are filtering it. And it ends up in Bowman's capsule, and then it ends up obviously into those, the tubing that we just discussed. It's made up of water and small solutes. What is it not made up of big things like those. That's what it's not made up of? So those are the kinds of things you will not see in your urine. Now, can you have trace amounts of protein, for example, in your urine and trace amounts of red blood cells? Of course you can. There are little itty bitty proteins out there. albumin is really really small, I can squeeze through a little bit of it, even hemoglobin, small enough to kind of squeeze through. So there are some proteins that can squeeze through some red blood cells that Mike made their way through trace amounts of these things as normal, elevated levels of these things is not normal. And that can happen for a number of different reasons. I've highlighted one and it's damage to the glomerulus glomerular nephritis. And so now these kinds of big things that you would not normally filter here are going to start to squeeze through. Why because the glomerulus is damaged. So you have big gaping holes in these blood vessels. Things are just coming through. And if they do, there's no way to get in the hell back out. There's no transporters for these big proteins and red blood cells and such no transport so they're gonna stay and eventually they will end up in the urine. So Sunday many of you are going to be physicians are going to be looking at your analysis, you're going to see what the hell's in the urine. Urine tells us a lot about what's going on in the body. And we'll explain why at a later time. So no glomerular nephritis know that, you know, when we have protein in the urine is called protein area of blood in the urine, we say blood in the urine, we're talking red blood cells, imati area, are there other reasons that these things can occur? Yes. But this is the one I'd like you to know. I might mention another one a one or two at a later time, they litter time as in next lecture. Now, it happens at a certain rate. So when it comes on, we can't go there yet, we got to do this first. So I can't tell you the normal numbers and bad numbers until we can really understand what this is. So again, we're concentrating on this right here. So just the production of the filtrate. So let us understand what's actually happening here. And so what's going to help us understand is a little bit of math, and hopefully, thankfully, a second grade math again. And so these are the mathematics of GFR. And so we're going to draw this right there, that's what we're going to draw. And when we're done drawing it, you should understand exactly what GFR is, and the filtrate, and what it means and how much is produced and all those good kinds of things. So let's do some math. And let's do some cardiovascular fears really quick, we're going to draw the heart, that's where it's all gonna start, it's gonna start with a heart. So this is the heart clearly. And here's our left ventricle. And we know that the left ventricle is going to pump out a certain amount of blood per minute. It's called cardiac output. And so CO that's not carbon monoxide. But that's cardiac output. And we know that in cardiovascular physiology, normal cardiac output at rest, is 5000 milliliters of blood per minute. So that's how much blood the left ventricle alone is pumping into the systemic circulation. How much of that blood is going to go to the kidneys? Well, not all of it, it better not because then the brain doesn't get any, and neither does any other tissue in the body. But the kidneys together are going to take up a lot of it 20%, your kidneys get as much blood as your brain does. Now when I say 20% of the blood is going to go to the kidneys not 20% to each kidneys. It's about 10% to each kidneys for a total of 20%. So 20% of this blood goes to the kidneys. And I'm going to write that here. 20% to the kidneys. That has a name. It's called the renal fraction, the fraction of blood that goes to the kidneys renal. So what does that mean? Out of that 5000 milliliters of blood that is pumped every minute at rest, 1000 milliliters of

it is going to go to the kidneys 500 To the left, and 500 to the right. So I'm going to write this down now. So 20% of 5000 is 1000. Simple math 5000 times point two. So 1000 milliliters of blood per minute

28:18

to the kidneys, and that's plural. I didn't say kidney. I said kidneys. Now, does blood get filtered? Or does plasma get filtered? plasma gets filtered. 1000 milliliters of blood or 5000, if we're talking about Cardiac output is whole blood. It's the plasma red blood cells, white blood cells, platelets, it's everything. But the only thing that's going to get filtered. And we already know and you just told me in unison is plasma gets filtered. So not all 1000. When it comes to the volume is going to get filtered. Just the plasma is and where is it going is going to the kidneys. So I'm going to draw the kidneys. Now, the kidneys are just going to be represented by a big huge rectangle. So these are the kidneys. And I'll label them. So we have our kidneys. And that's where that bloods going, yes, we want to filter the plasma. Now, again, only the plasma is going to get filtered well, when it comes to plasma, what's the concentration of the plasma? Well, in the notes, I use the concentration of 55% or 200. I get that because I'm using a hermetic rate of 45%. If I'm adequate 45% plasma concentration has to be 55 percents got equal 100. So I'm using 45% because I've used 60% Because I said hermetic. It's 40%. Go ahead. But real fear is typically 55% is what's used as a plasma concentration. So what does that mean to us? What that means is this is that 55% of the blood His plasma. And so what that means is is that only 55% of that 1000 is plasma. So what's 55% of 1551? By the way, this value right here, this 1000 milliliters of blood that goes to the kidneys is called renal blood flow rate. I'm going to abbreviate that. So renal blood flow rate is the amount of blood going to your kidneys per minute, easily calculated by taking cardiac output, multiplying it by 20%. Now 55% Of that total is plasma. So the renal plasma, that's p naught A B, renal plasma flow rate is simply going to be 55% of that 1000. So of this 1000 milliliters of blood, how much is plasma? 550? So 550 milliliters I'm not gonna put blood down what am I going to put instead? Plasma per minute, is going to your kidneys to do what to get filter. Now I asked you this is all of that plasma going to get filtered in one run yes or no? How much 20%. We already learned it, it's sitting right there. So of their 550 milliliters that is going to flow through both of the kidneys about 275 per kidney 20% 20% gets filtered, not 100% can't be 100%, or blood flow would stop in its tracks, that has a special name is called the filtration fraction, not renal flexion filtration fraction. Now, thankfully, it's the same percent is over here. complete coincidence. Okay, they have absolutely zero to do with each other nothing, it's just coincidental that they're both 20%. But fortunately, they are 20%, because it's going to be easy to remember, just remember, they're both 20, they'll remember, they're both 30 Because then you're going to get a rock. So this is called the filtration fraction. Now, to make this calculation, all we need to do is multiply 20% times 550. That gives us GFR. That gives us glomerular filtration rate that gives us the rate at which we are squeezing plasma out of their glomerulus. And so now we have GFR glomerular filtration rate. And what is it 110 milliliters now I have to put the word filtrate

32:56

filtrate per minute. So what does that mean? Your kidneys are producing 110 milliliters of filtrate every single minute, that's not one nephron. That's all the nephrons from both kidneys. So if we translate that out, that's per minute, or 60 minutes in an hour, and it's 24 hours in a day. That translates into and they have the number in the notes 160 liters of filtrate in a day. Now, what is filtrate eventually become urine. Who urinates at two liters of two liter bottles of urine in the day anybody know you urinate maybe 1% of that? Well, how the hell is that possible doctor are because

most of the filtrate that we produce goes right back into the blood through reabsorption, because we will talk 99% Give or take if the filtrate goes right back into the blood not randomly, very selectively. So we maintain normal values of all the things that are being filtered from the blood. things we'll talk about at a later time. So after we go through this, I'm hoping that you understand now exactly what this is. Now, can. Here's another thing. That's a normal value for GFR by the way, 110 beautiful number. But there's a wide range. And when you as physician someday if any of you become nephrologists, this is not how you're going to get GFR. We went through this so that you can see and learn what GFR is. In order to get a GFR you're going to do some blood work is going to be based on the amount of Creatinine in the blood and it's actually an estimate done with an equation two equations. They plugged the the amount of creatine in your blood into the equation boom. Again, it's a complete estimate. It's not even that accurate. It's kind of accurate, it's accurate enough. If you have a patient that has kidney issues, you will do it in a much more accurate way. We'll talk about these things on Thursday. So anyway, this is an exercise to help you learn what GFR is, it's not an exercise to show you how it's actually determined. Now, can GFR change changed a little bit? And it can change with certain conditions? Let me give you a couple of examples here. So let's say we have congestive heart failure, CHF, cardiac output upper down, tell me down, of course, heart's not pumping very well Correct. That's why the hearts failing, got a decrease in cardiac output, what do you think's going to happen to GFR? Lower Cardiac output is going to be a lower renal blood flow rate, it's going to be a smaller renal plasma flow rate going to have a lower GFR. So one of the things that happens with CHF is that you can have failing kidneys. So that can cause a lower GFR. It's one example. Exercise. What happens to cardiac output with exercise? In going go up a ton. Now, you might think yourself, well, of course GFR is going to go up, it actually doesn't know why is that. So with an increase in cardiac output? Most will go to what do you think is going to go? Your muscles less to the kidneys. So as a result of this, you will actually get a decrease in GFR. During exercise, they might think what the hell that's terrible. Why would anybody want to exercise if your GFR is going down, because GFR tells you how well your kidneys are working. It's temporary, it's not that big of a deal. Everything will be fine. After you're done exercising, if you're exercising literally 24/7 Then you know, seven days a week, then you'll start to have some issues. But that's not how people exercise. Exercise for sure bouts you recover from it exercise, your short bouts you recover from it. Doctors not telling you not to exercise Doctor hours, you know, for the last two semesters, it's told you to exercise. But this is just something that I just wanted to throw out there. So can it change? Yes. Oh, and by the way, can it go up? Absolutely, you can. And we'll talk about why in a little bit. Now, let's go to the numbers. And then we'll take a break. So what's considered a normal GFR, anything above 90 is considered normal classically. However, if you have a GFR 60 or above, it's still considered normal if you don't have any kidney pathologies.

37:37

No sign of kidney issues. So if you get the blood work of somebody, and it shows that it is always a little e next to the GFR, because the E stands for estimate, because it's done by an equation, literally, it's done by an equation, anything above 60 is typically okay. Once we start to get below 60, now we start to run into kidney issues, or the kidney issues are causing a lower GFR. Again, GFR is an indication of how well your kidneys are working. So below 60, is considered kidney damage. And then we have stage one, stage two, stage three, stage four, stage five kidney disease. And as we go from one to two to three to four to five, it's getting worse and worse and worse. Once we get to a GFR of about 15, you're probably in stage five. Now what can cause these, the zillions of different things, I just want you to know generically, what's considered normal, what's considered abnormal when it comes to GFR. All right, let's take a break. After we come back, I'm going to tell you why the glomerulus is so incredibly efficient when it comes to filtering. Let us continue. So we're still on glomerular filtration. Now, what I'm going to show you is why it's so efficient. And so I have three

things listed here. Number one, the glomerulus, which again is a capillary, the blood pressure within a glomerulus is really, really high, about two and a half times as high as it is in other capillaries. Why is that? I have it written down here. But I'm going to actually address this a little bit later, a little bit later, as in in this lecture. So I'm going to address why the blood pressure is so high. Number two, it has a huge surface area. Now when I draw the glomerulus I just draw it is one two going in and out of the Bowman's capsule. I mean, you guys know that that's not at all what it looks like I told you to just think about a whole bunch of yarn and just throw it in the glass. There's a lot of capillary there. Now I have just this one arrow going in this right? There's gazillions of arrows wherever we have surface area of the cap of the glomerulus. We have plasma getting filtered from it. The more capillary you have, the more membrane you have, the more membrane you have, the more filtrate that you can produce. So that's another reason why it's so efficient. Number three is that the glomerulus is very leaky. Now, it's not so leaky that it's letting protein through and white blood cells and red blood cells and platelets through. But it's a little leakier than most other capillaries are. And so those three things together allow a very efficient filtration to occur at the glomerulus. Now what we're going to concentrate on is this high pressure that we have here, the high hydrostatic pressure, which will in the end, give us a high, what's called net filtration pressure. And I'm going to explain what that means. Now, I think we probably all know what net is, like, if you work, you get your paycheck, it's got the gross amount. And then down here is your net amount, because the government took all your freaking money away. And that's all you have left. So net is the end result. And so let us draw this, again, not that kind of detail. But we're going to draw the glomerulus in Bowman's capsule is really all we need to draw here, this will be kind of the big picture. And then we'll get a little bit more detailed. So Bowman's capsule, you don't have to worry too much about the tubules coming off of it. And then I'm going to draw the glomerulus. So we know what all this is, is there a ferret? Notice that I'm drawing a ferret and efferent and drawn the efferent bigger than the efferent. A ferrets bigger than the efferent. Explain why in just a little bit has to do with the high blood pressure within the glomerulus itself. So this arrow that I'm about to draw is not filtration, it's a force. It's a pressure. Oops. And this pressure is called net filtration pressure. So it's a pressure that is going to favor filtration to occur. So I will label that net filtration. Pressure. Now it's made up of three different forces that in the end, create this one force. So I'm going to draw a Bowman's capsule just a little bit bigger, because I want to tell you what all these are. So made up of

42:15

glomerular blood pressure, that's one. So glomerular BP, that's one too glomerular osmotic pressure, specifically Cole Lloyd, osmotic pressure. And then Bowman's capsule BC hydrostatic pressure. So all three of these pressures are forces, and in the end, creates one big force. And that's net filtration pressure. And so I'm going to draw Bowman's capsule really, really big. So I'm just gonna kind of redraw this a little bit, because I wanted all these words to be included inside of Bowman's capsule. So all of this is Bowman's capsule. Again, this is, of course, the glomerulus. So now what I'm going to do is, we're going to draw another picture. And that picture is going to include all three of those forces, how the interplay between all the three of those forces creates this net force, net filtration pressure, which is going to favor obviously, filtration. So let's draw this again.

43:34

So Bowman's capsule, I'm going to draw kind of big. And then the glomerulus, I'm going to draw this kind of thick,

43:49

because I need to write some words inside of it. So might not look as proportioned as it did before, but it doesn't matter. So let's see how these three port forces interplay with each other and then create any end is net filtration pressures is the first one I'm going to put in this picture is the blood pressure in the glomerulus. So glomerular. Blood pressure, and it's high. It's 50 millimeters of mercury. That's ridiculously high for a capillary. Most other capillaries have blood pressures of about 20. And I'll explain again, why in just a little bit. Well, blood pressure is a type of hydrostatic pressure, right? We've learned that this hydrostatic pressure push or pull, reminder pushes. So this force is going to try to push plasma out of that capillary. So what I'm going to do is this arrow was a force. I'm going to make it nice and big. It's a big force that favors filtration. So when it comes to net filtration. What we're going to do here is a little bit of math. Again, thankfully, it's second grade math.

45:08

So net filtration pressure we already established. What it is, is the force that favors filtration. And so the first thing that it's made up of that I had that I'm going to discuss is glomerular blood pressure.

45:29

So glomerular blood pressure, and this is a force that favors filtration. It's trying to make filtration happen. It has a value, it's 50 millimeters of mercury. And because it favors I'm going to put a plus sign in front of it. So it's plus 50 millimeters of mercury, a big huge force that once filtration do occur. Then we have our other two forces. The first one colloid osmotic pressure, and it's a nickel MaryAlice. So glomerular colloid osmotic pressure. And this is 30 millimeters of mercury. So pretty darn big force. Now remind me osmotic pressure pushing force or pulling force. It pulls, right. And what compartment are we in? Well, we're in the glomerulus. So it's pulling into the glomerulus. So this arrow that I'm going to draw, which again, is the force is going to go in the opposite direction. They won't be as big as the Gomery of the blood pressure, because what 30 millimeters of mercury is smaller than 50 millimeters of mercury. So this is now glomerular. Osmotic pressure op. Now this opposes filtration, obviously it does, we're going in the opposite direction. So I need to put a minus sign here. 30 millimeters of mercury. So these two forces are fighting each other. Fortunately, blood pressure is winning. And then we have a third force. Bowman's capsule BC hydrostatic pressure. Now, is there fluid inside Bowman's capsule? Yes or no? What's it called? It's called filtrate. Right? That's the fluid a Bowman's capsule. Bowman's capsule is catching the filtrate to come. So there's fluid in that compartment. Anytime there's fluid in a compartment, there's a hydrostatic pressure we learned last semester. So we have Bowman's capsule hydrostatic pressure, I'll put it up here. So Bowman's capsule, hydro pressure. Now, is there a lot of fluid in Bowman's capsule? Not really, because it is catching on the fluid, but then immediately the filtrate, which is what the fluid is gonna start flowing through proximal convoluted tubule loop, and we discovered it to be so it's not just sitting there and building up. It's constantly flowing out. So it's not very big. It's about 10 millimeters of mercury, still sizable. Hydrostatic pressure, as you guys already told me, it's a pushing force. But we're in this compartment. And so which direction are we going to push, we're going to push in this direction. So we have another force that is opposing filtration. So I need to put another minus sign here, minus 10 millimeters of mercury. So these two oppose filtration. Well, let's just do some simple math. Plus 50. Minus 30. Minus 10. Is 10 millimeters of mercury. Where did I get this 10 millimeters of mercury over here? Oh, I didn't tell you what it was nevermind. But net filtration pressure, I'll actually put it here. It's 10 millimeters of mercury. Where they get it. These three forces are at play. And in the end, there

is a net in the net. Let's put a 10 there is 10 millimeters of mercury. Now, can these forces change? Of course again, by the way, when it comes to the word colloid, what's the word that you should immediately think of telling me? Cool Lloyd last semester, first lecture, first lecture of last semester? Didn't I tell you last semester that I would do that lecture that day, we will be talking about toward the end of this semester, and I tell you, I won't lie it starts with a P. Protein. Did you guys already say and I'm just rambling on and not even listening to you guys. Protein. Is there a lot of protein in this blood right here? Tell me why? Because it can't get filtered. Right. Are we altering water for example?

50:02

Yeah. So are we concentrating the protein here? For sure are? That's why this colloid osmotic pressure is so stinking high because concentration of protein is pretty stinking hot here. Can the concentration of protein in your blood change? Of course it can. Can it go up? Can it go down? Of course it can. Can these values change? Of course they can. Do you think I'll change them on the exam? Of course I will. And when I do, you're just gonna do some simple math. And you're gonna tell me just filtration happen? Or doesn't it happen? That's all. So, again, this is going to in the end, under normal conditions, favor filtration favor. This, this isn't happening. Unless the interplay between these three forces is not what it is what I have up here on the screen, could it be plus five and still happen? Yeah. But if it's minus 10, if for whatever reason, Plumeria blood pressure gets too low, or these others get too high? It ain't happening. What does that mean? Your kidneys are failing, you are not filtering your blood. The first thing that has to happen when you filter your blood is we need to make the filtrate or you're not going to filter your blood properly. GFR. Again, it tells you how well the kidneys are working, which is why it's a measurement made with routine bloodwork. Again, it's an estimate, but the estimate is accurate enough. So anyway, now what GFR in all conditions is kept constant. How, because we can have all kinds of changes. The reason we want to try to keep it constant is this. If it's too high, it means the kidneys are working too hard. And if the kidneys work too hard over time, they're going to start to fail. So we don't want the kidneys to work too hard. But if GFR gets too low, well, what does that mean? Well, it means we're not filtering the blood well, and then that's going to lead to problems because then we don't keep the things that are normal in the blood. Well, normal in the blood, like what sodium concentration and pH and potassium concentration and all those things that are normal in the blood do and main part to the kids. So we want to keep GFR right where it needs to be within its normal range. But there are a lot of things that can change GFR. And know the angle that we're going to tackle is blood pressure. So let's do that. Now. Now I'm going to tell you why blood pressure is so high within a glomerulus. And here's another little picture showing, normally, what's the blood pressure in capillaries. A little bit right around 20 millimeters of mercury, the blood pressure in your, in your in your glomerulus, which is capillaries way up here, which is right around where the arterioles are. So why is that blood pressure so high? How can that be and it needs to be right? If the blood pressure in your capillaries isn't so high, what happened? filtrate filtrations not happening, this number has to be freaking huge, because it needs to be bigger than these two combined. How does that happen? It's about anatomy. And it's about the afferent arteriole and the efferent arteriole and how there's a difference in their diameter. So let me show you. So I'm just going to draw this very, very simply. This is going to be the efferent. This is the glomerulus and this is going to be the efferent. A pharyngeal Marisol label everything efferent so a ferentz is here efferent is here. And then in between the two is the glomerulus. And clearly, we have a bigger diameter here versus the efferent, where we have a smaller diameter. And it's the difference between those two, that's going to cause back pressure. And we're going to get a blood pressure here once again about 15 millimeters of mercury where normally a capillary would be around 20 ish. And so because we had this bottleneck over here, on this side, we have again what's called back pressure, I'm going to give you an analogy, a garden hose. So here's a garden hose. I'm going to draw this garden hose nice and uniform. And so we're going to take that garden hose, we're going to turn the

hose bib on. And now water is going to flow out of that garden hose and it's going to flow just going to kind of spilled out of the garden hose. It's going to flow nice and smoothly out of the garden hose. Why? Well because the pressure within the garden hose is relatively low. Because the size of the garden hose throughout is uniform the diameter here and the diameter here exactly the same.

54:58

But if you took your thumb and put your thumb on the end of the garden hose. What's going to happen to the water is it just going to spill out what happens to that water? It sprays out, it squirts out really hard, does it not? Why? Because what you've just done is you've made the opening here, the direction in which the water is flowing smaller, versus the diameter of the hose itself, which is bigger. And you've created back pressure, there's now higher pressure within that hose. And that's what's forcing that water to squirt out of that hose, the premises the same here, the smaller diameter, the efferent, causes back pressure within the glomerulus itself, it causes the pressure to go up. So we can control the blood pressure in the glomerulus by controlling the efferent arteriole, the efferent as well. But our story is going to be the a fair, we're going to keep it simple can both affected Yes. But the effect is going to play a bigger role. So again, keep in mind that the reason his pressure is high, not 20 millimeters of mercury like other capillaries, but 50 Because this is so much bigger than that. And so with that, let us talk about autoregulation. And so what is autoregulation? Again, the kidneys themselves keeping GFR normal, how do we keep GFR normal, we keep the blood pressure in the glomerulus. Around 50, we don't let it change, even if blood pressure in the systemic circulation goes up or down. So what we're going to do first is we're going to talk about what what if your blood pressure goes up. But if somebody has hypertension, if somebody has hypertension, their blood pressure is going to be high everywhere, including in the kittens. And so theoretically speaking, that should make the glomerular blood pressure go up even higher than 50, which means what GFRC go up. So actually, let's draw that, we're going to draw the kidneys without autoregulation, which is not normal, we'll do that first. So here's our efferent.

57:17

There's a glomerulus. And there's our efferent. And so what's going to happen here is this is that we're going to have an increase in systemic

57:32

blood pressure, so he's gonna have hypertension, blood pressure is gonna be high everywhere, when you're measuring the blood pressure in that person's arm, you put that cuff around their arm, that blood pressure that you're measuring is going to be the same blood pressure close to it, at least in their legs, and their left arm and your kidneys and their lungs. So it's going to go up everywhere. So if this is the case, what should happen is, is the blood pressure in the glomerulus should get bigger than 50 millimeters of mercury. That is if autoregulation is not happening. But that's not normal. This is what's normal. So we're going to draw that again. So what's going to happen here, despite the higher blood pressure, is we are going to keep the blood pressure in the glomerulus at 50 millimeters of mercury. So again, we're gonna have high systemic blood pressure. We're gonna keep this at 50. Well, how can we do that? Well, tell me why this blood pressure is so high to begin with. Because the a fair arterial is so much bigger in diameter than the efferent arteriole. Right? That's the reason. So do you think that if we had got the size of the a fairing in the efferent, closer together, this value

would go down? Wouldn't the pressure in the glomerulus go down? If the diameter of the a fair and efferent were closer together? Yes, that's exactly what's gonna happen. So with this higher blood pressure, the a parent arterial is going to constrict. So we are going to get constriction of the a ferret. And what's going to happen? It's not going to let this go up, like it did over here without autoregulation because we had auto regulation. And so up here, we're going to have a higher GFR without the auto regulation. But because we're keeping blood pressure, the same GFR stays the same. Now, I know that some of you are thinking right now. Well, Dr. Otto What about mA Pete was cardiac output types little peripheral resistance, that equation that you told us to memorize and never ever, ever forget. And when total peripheral resistance goes up, blood pressure goes up. You told us that Dr. Heart and part of total peripheral resistance is what? Size blood vessels. And when you constrict, what happens total peripheral resistance goes up, should that make blood pressure go up? It should. But do we not have different anatomy here? Absolutely we do. For this story, you need to get that equation the heck out of your head. Because that's why I asked you. The reason this pressure is so high is because this is so much bigger than that. And I asked you right before we did this, if we can get these diameters closer together, witness pressure go down. I asked you that. And you guys said, Oh, of course, Dr. Ark. So the diameter of this is now closer to that that should make this pressure go down. We don't have as much back pressure. Well, how much? Is this going to basil constrict just enough to keep it at 50? All right. So the vasoconstriction story does not work when we talk about this, because it's very different anatomy than we've discussed previously. So we have a very special circumstance here. Again, the anatomy is way different. Arterial capillary arterial, and then it turns into a freakin capillary again, so we got kind of all kinds of cool stuff going on here. Now, how high can the blood pressure get and still keep a blood pressure in the glorious 50 can get about as high as 180 millimeters of mercury. Okay, so we can auto regulate up to 180 millimeters of mercury. Now, that's not mean arterial pressure, that's systolic pressure. That 180 millimeters of mercury is kind of a magical number, isn't it? Remember when we did afterload. And I said, once we get blown up beyond 180 millimeters of mercury, that our stroke line will start to go down. Remember that? Fortunately, it's 180 millimeters of mercury for autoregulation, too. So it's kind of an easy number to remember, because it's a number that's kind of already in your brain. It's a different story. But it's the same number. Once your blood pressure gets beyond 180, let's say the systolic is 200. This is going to go up and anyone can he's going to start the work hell a lot harder. And we don't want that. Why is this happening to keep GFR? The same? Why? Because we don't want the kidneys to work too hard, because they'll wear out too quickly.

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So this is high blood pressure. Let's do low blood pressure. I know you guys know what the story is, but we're going to draw it anyway. So we'll do without autoregulation first. So a Ferrant, arterial glomerulus efferent. A ferret glomerulus efferent. And so now we're going to have low systemic blood pressure. Well, what should happen is that this blood pressure should go below while rule 50, which means well, then we get a decrease in GFR. That's what should happen. If we didn't have autoregulation, fortunately, we do. Let's draw what really happens. So here's our A Farren again. glomerulus II fair, a fair, the Mariela C fair, blood pressure is going to go down. And what's gonna happen to the efferent arteriole, you guys already know, we want this pressure to go up because otherwise it would have gone down. And so the A Ferren is going to basal dilate. Now the difference in diameter is going to be even bigger. Now we're going to get even more back pressure. So instead of this value going down to let's say 45, stays 50. And so we get dilation. Of the a fair enough to get us to where we need to be and that's around 50. And so GFR

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may use the same words they did before stays the same. Why? Because we do have autoregulation. It is working the way that it's supposed to up to a point or I should say down to a point

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down to about 90 millimeters of mercury getting that's the systolic pressure, not a mean arterial pressure. That was mean 90 would be perfect. Okay, up to 180 down to 90. That's pretty much a range where you can keep GFR where it's supposed to be that's too high and not too low. Now, in your notes. There's one other thing included that you don't have to know And that is when we change the rate of filtrate flow which can happen. We don't get into it. I don't even think you guys talked about macula densa adjust to glide glomerular cells and with anatomy, so I'm not getting into it. So you don't have to notice. It'll be on pilot. But you don't have to know I think I might have already put it at least I did for the respiratory system, I'll certainly do this soon. So anyway, that's that for GFR, at least for now. Now, let's explore the other two, reabsorption and secretion. And so what we're going to do now is that we're going to draw it again, and talk a little bit more about reabsorption or what occurs. And what I'll do here is, here's Bowman's capsule. And I'm actually going to label each of those as opposed to just making it all one tube and being the same thing. So I'm going to say this is the proximal convoluted tubule. This is the loop of handling. This is the distal convoluted tubules, just putting them in the order at which they are anatomically. And so we have all those structures. Now, of course, they're you know, twisting and turning, I'm not doing that. And of course, we have the glomerulus here, which then gives rise to the peritubular capillary. And it's what to take home that reabsorption is occurring everywhere here. So just a little bit more of a visual that we have, where we're seeing reabsorption. Now, as I've stated, about 99% of the filtrate is reabsorbed, approximately, it might be 99.1 89.9 88.9, whatever, but it's always going to be around 99%. So about 99% of the filtrate is reabsorbed. So the vast majority of it is going right back into the blood. They might ask yourself or be thinking to yourself, What the hell? Why the hell would we waste our time, if almost all of its going right back into the blood, why even bother to create a filtrate? The reason is that we need all this filtrate within these tubes so that we can selectively pick and choose exactly how much we want in the blood. It's just a more efficient way to do it. And it doesn't take big changes, like I said, 99%, but it might be 99.1. Or it might be 89.9. Just those small changes can make big differences in the concentrations of sodium and potassium and chloride, magnesium and calcium and glucose and so forth and so forth. All those things that the kidneys are controlling. Now, if we look at this, I think we can logically see that those things that are being reabsorbed that is taken back into the blood are because we want them in need that. But I'm going to state that anyway. Why reabsorb? Why do we do it? The reason the body wants needs those things. And what are the things water and small solutes. Those things that are reabsorbed. That's the reason the kidneys certainly would not bother doing this if the body didn't need them. Because if the body didn't need them, it would be extra stuff in the body. sodium level would be too high, calcium would be too high potassium levels will be too high chloride levels be too high, and so forth, and so forth, and so forth. Something else that you could probably logically see is is that as we vary the amount of things that we reabsorb, we're going to control the level of those things in the blood. And so with that, let us write this down as well to statements as we increase the reabsorption of x. Now, what's x x is water, x's, small solutes. So it can be almost anything you want it to be except well protein and wet breast cells. And likewise, it's not those things because we don't reabsorb. As we increase the reabsorption of x, well, we're going to increase the level of x in the blood, of course. And this is how we're controlling the level of these things in the blood by controlling reabsorption. And certainly, if you have a decrease in reabsorption, well then the opposite is going to hold true.

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You're going to have less of those things in the blood. So a decreased level of x in the blood. And so I'll give you a couple of examples. So this line right here is going to represent Let me see what I do at blood sodium, the level of sodium in your blood we needed to be a certain level, right? Right around 140 plus or minus about five. We learn that last semester, when we did the nervous system chapter. So we are normal here. So we're right around 140. But now all of a sudden, you decided to go on, I don't know, you just decide this day to say to crap ton of salty foods. So you're going to eat lots of salty foods? Well, a lot of that salt is going to be sodium. So the sodium is going to be in your digestive tract get absorbed into your blood, what's gonna happen, your sodium levels in your blood up, up, up, up, up, up up? How far it doesn't matter, it's gonna go up. That's no good, high sodium levels in your blood, absolutely not good in the kidneys not going to stand for that. What do we need to do? Well, negative feedback tells us what we're gonna do this, well, how do we do that? We decrease the reabsorption of sodium, we just don't reabsorb as much of it. And when you do, the level comes down.

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Let's go the other direction. You're not gonna eat a whole bunch of salty foods, maybe you're not gonna eat any food.

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So do you very little. Or you drink lots of water? So you kind of dilute your blood down a little bit?

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Well, we need to do this need to get our sodium sodium levels can't be too low, either. That's terrible. So now what are we going to do? Or what are the kidneys going to do? We're going to increase the reabsorption of sodium. And so the level of sodium will go up and how much it could be as little like point 1% Or point 2% Or point three or whatever, it's going to be exactly what it needs to be the kidneys, we'll know exactly because the level of sodium is going to be detected within the blood. We're going to have the homeostatic mechanism occurring with regulators and effectors and reset all that stuff that we talked about last semester, like the very first picture we drew, I think. So these are the kinds of things your kidneys are doing. You can throw in all kinds of stuff at your body, and yet you do bloodwork and almost always your blood works gonna look just fine. Because you have these beautiful organs that can change their transport mechanisms, both reabsorption and secretion, to keep things normal within your blood, which is good, because if they're not bad things can start to happen. All right. Now what? Let me see if I missed anything. Now there's some things I don't need you to know. Again, I will put it on pilot as to what you do. And once you don't have to know I had the word highly selected here, I want to make sure that we understand what that means. And I want to compare it and contrast to Liberty lift filtration. This process here are done by specific transporters. So it's very selective. When it comes to glomerular filtration, when we were talking about squeezing that plasma through the glomerular walls, it's not selective at all. Other than if it's small enough, it's getting through. There are no transporters, doing glomerular filtration, we have these little holes in the glomeruli and things are just going to get pushed through which is why I don't have the word selective with glomerular filtration I do with secretion and I do with reabsorption because there are

very specific transporters that are doing and so when we come back on Thursday, we're going to talk a little bit more about reabsorption and and get right into secretion. So I'm going to start right into secretion and then we'll get into the specific things that are reabsorbed and secreted. And then we'll finish this chapter. Alright folks, I will see you guys on Thursday.

Urin PM 3-31-22

Thu, 3/31 11:13PM 1:14:56

SUMMARY KEYWORDS

urine, osmolarity, filtrate, blood, kidneys, reabsorbed, gfr, secrete, nephrons, concentration, adh, molecule, dehydrated, distal convoluted tubule, picture, proximal convoluted tubule, collecting duct, diuretics, capillary, glucose

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Alright, let's finish up this chapter. So last thing we were doing on Tuesday where we were talking about reabsorption, which is one of the processes involved in filtering of the plasma. And now we're going to go and move on to secretion, I've actually already got a little head start here. And I've drawn secretion. And so we have Bowman's capsule, glomerulus peritubular, capillary. And then this big long tube is going to represent proximal convoluted tubules, for Haley all the way to the collecting duct, as we did in the last picture. And secretion, as we know, because we already discussed it very, very briefly, is we are removing things from the peritubular capillary and transporting them into the filtrate. Why do we do this? Why do we secrete because we want to get rid of what we are secreting. Now, when I say here body does not want or need what is being secreted, it doesn't mean it's getting rid of it completely. It might get rid of some of it just so that we have normal levels of it. And so this is going to be part of trying to keep things within homeostasis, trying to keep our potassium concentration normal in the blood, for example. So now there are some things that we do completely secrete. And then there are other things where we just secrete some, he just depends on what it is. And we'll talk a little bit more about that in just a second. So to parallel what we did in the last lecture, let's talk about what happens when we increase in decrease secretion. So if we increase secretion of x and x can be whatever, well, then, actually, what that's going to lead to is a decrease in the level of x in the blood, of course it is. If you're doing more of this, you're going to be removing more from the blood. And then conversely, and of course, we want to be thorough here. So if we decrease secretion of x, well, then we're going to increase the level of x in the blood. So even though we are secreting to get rid of things from the body, if we decrease the amount that we secrete, the level will go up. So again, this is a way of controlling the level of x and s can be potassium, for example, it can be things that we do actually need, but we just don't need as much of it. And so we do blood work and the blood works normal. A lot of it has to do with the kidneys, the kidneys doing its job, or the kidneys doing their job, I should say. Now, we'll do the same thing that we did. When it comes to reabsorption, we'll do a homeostatic line, we'll give a couple of examples of increasing and decreasing secretion and how that's going to maintain something normal. And so this will be blood concentration of x, or not X but H plus. So blood concentration of H plus DHI we have a normal concentration of H in the eye and the blood why? Because that's going to help us maintain pH. And so we have a normal level. And let's say all of a sudden that the level of H plus is high, which would equate to more acid. And let's give an example of why this would happen. An example of which we already know ketoacidosis. So we have a type one diabetic doesn't get their insulin when they go into ketoacidosis. And so now a lot of age plus in the blood, we need to do well, we need to lower the amount of H plus. So again, ketoacidosis, more H plus ketones are acidic, that equates to a low pH. Of course it does. And so how are we going to do that? Well, we want to secrete more H plus. So we

would increase the secretion of H ions, which the kidneys can do. And as the kidneys do that, we lower H plus concentration and our pH gets back to where it's supposed to be. Conversely, there are going to be conditions where we have a lower concentration of H plus I'll give you one right now. And we'll understand this when we actually get to the digestive chapter. Vomiting. If you vomit a lot, you will actually increase the pH in your blood, you'll decrease H plus concentration, you'll understand why when we get to the digestive chapter. Just take my word for it. Let's put an h there let's arrow down here, which is going to equate to a higher pH. and So conversely, we're going to decrease the secretion of H plus if we don't secrete as much, well, then the level will go up. And we are chronically secreting, and we are chronically reabsorbing. And so if we can change the amount that we secrete and reabsorb, we're going to change the concentrations of these things in the blood So a couple of examples. Now, what are we gonna do?

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We're gonna go to a couple of pictures. So we talked about reabsorption. And we talked about secretion. Let's start, let's go back to reabsorption here. So in your notes, there's a page, maybe it's a page, I don't really remember, we have all these different structures. And then we have a whole string of things that are being reabsorbed at these particular places within the renal tubules. So at the proximal convoluted tubule, these are the things that are being reabsorbed that you guys have to know about. So all these words on this page are actually this picture right here that's on file. So what I've done is that I've taken this arrow here and here and here. And here, I want you to understand that this arrow is talking about transport transporting out of the renal tubule into blood vessels, blood vessels are not in this picture. So these arrows in the transport that's occurring here is reabsorption. So to remind you, we're talking about oops, these arrows over here. So again, this is reabsorption from the last lecture at the very end. So transport from the filtrate into the blood. So what's not shown in this picture are the blood vessels, but they're there. And so what you have to know is, these are the things that are reabsorbed at the proximal convoluted tubules, you have to know that the only thing you have to know what the descending limb of the loop of Henle is, is that water is reabsorbed there, and then the ascending limb of the loop of Henle. These are the things that are reabsorbed and then the distal convoluted tubule and into the collecting duct as well. I want you to notice something proximal convoluted tubule waters included descending limb of the loop of Henle water is included distal convoluted tubule waters included as is at the collecting duct, you don't see it at the ascending limb of the loop handling. That's going to be an important point, when we discuss something in about, I don't know half hour. So when we get to just imaginary nephrons, this is a very, very important point. The ascending limb of the loop of Henle is impermeable to water, there are just a handful of places in the body. We have cells that are impermeable to water. And we're going to have an example here. And we'll give you I'll give you another one next semester actually later on this semester. So please know those things that are reabsorbed in where and then same thing for secretion. secretion, the story is a little easier. Stuff that much stuff. So I have everything listed that you have to know at these different places. Notice the lupa handling, no action. So you have to know anything when it comes to the lupa handling. But these other areas, yes, we do, we actually just did H plus secretion there it is possible convoluted tubule discovered a tubular collecting duct. And so just make note of all those things that are being secreted and reabsorbed in in their very specific places. Now these are actually done by very specific transporters. And back when I used to teach just a physiology course on campus, I had the students know what the transporters are. So I'm kind of giving you guys a little bit of a break. All you need to know is the punch line. You do have to know one transporter though and it's the one that's bolded in your notes, and I'm going to point it out to you it's descretion one, it's called the organic ion transport. It's located the proximal convoluted tubule. And what they transport are a number of things here that are listed, some of which you might know about. If you don't, it's not that big of a deal, but I'm going to highlight a couple of them. bile acids we'll talk

about later on this semester. But antibiotics I'm going to assume you know antibiotics are drugs given to treat bacterial infection. This transport this is secretion. We're getting rid of stuff Why the hell would we want to get rid of antibiotics? Well, antibiotics I mean, they don't antibiotics don't belong in the body as far as the kidneys know, are concerned. It's kidding What the hell is this? I need to get rid of it. It doesn't mean that antibiotics are not beneficial and not telling you not to take antibiotics. But when you do, they do their job and then the kidney says it's time to go. So the kidneys secrete them. And then creatine as well as something that we're going to touch upon in a little bit. Guys remember creatine from last semester, the phosphagen system and muscle is one of the ways muscles make ATP for very short, intense bursts for about 10 to 15 seconds. Remember that? Well, that phosphagen system has a waste product that no waste product is created. It's toxic to the body. We need to secrete it. And this transporter is going to do that for us. And then I have in parentheses air quotes your drugs, what kind of drugs that can be a drug like ibuprofen, ibuprofen, it's Motrin. Ibuprofen doesn't belong in the body as far as kidneys are concerned. So the kidneys are going to excrete it or secreted doesn't mean that ibuprofen doesn't have benefits when you take it. Of course it does. But the body is going to secrete it eventually. That could be illicit drugs as well.

09:58

Math we Whatever, when you guys get a job someday you're peeing in a cop, because I want to know what the hell you been up to. And in that cop is going to be illicit drugs if you're taking illicit drugs, because your kidneys are going to secrete those illicit drugs, because they don't belong in the body. So again, things that are secreted don't belong in the body, or we want to get rid of a certain amount of it to keep the level in the body normal. So the organic ion transport, or that's the one that you have to know. That's it. Now what don't have to know this doesn't mean we don't have to know plasma clearance, you absolutely have to know renal plasma clearance, what you don't have to know is how to do the calculation. So we're not going to do any math, when it comes to renal plasma clearance. I actually talked about it a little bit, that is this equation. But no, no math when it comes to this on this particular exam. So let's talk about renal plasma clearance. And what it is, the name tells you what it is. So I'm going to draw this simply, this is kind of the big picture of renal plasma clearance. So this is the blood. This will be the kidneys. I'll label everything. And then we're going to have, we'll do a circle over here, that's going to be the bladder. Obviously, bladder contains urine, blood vessels. And I've already labeled the kidneys. So renal kidneys, plasma, well, that's obviously the liquid part of blood clearance. So we're they clear something from the blood, what's going to do it, the kidneys are. So we're going to take x as can be a lot of different things. And the kidneys are going to take x and it's going to make its way in the bladder, boom, we cleared it. This is by definition what renal plasma clearance is. Again, the name tells you exactly what it is. So renal plasma clearance. Now, you can use certain molecules, and they're clearance to determine, for example, GFR. So I'm going to talk about that now. So if you had the perfect molecule to measure GFR, it would be freely filtered, not secreted and not reabsorbed. So what does that so let's do this. So this is a nephron. We'll put the glomerulus in there, peritubular, capillaries, everything else that we've we've drawn, we're gonna put the bladder in this picture. So this is the picture that I just drew just not the entire kidney, but just one single nephron. And we're going to put a little circle over here to represent the bladder. Now, what we can do is determine the concentration of some substances in the blood. There is a perfect molecule that can measure GFR based on its clearance and the name of that molecule is insulin, you're actually not going to have to know it, it's actually crossed off in the notes. It's right there crosstalk we're going to talk about this anyway. It's a carbohydrate, and what you do, you're going to inject it into your patient. So you're going to know exactly what the concentration is in that patient because well, you injected it into them. So you know what the concentration is, you're going to determine what the concentration of the molecule is in that patient. And so we're just going to call this molecule molecule x. So molecule x is in the blood because you injected it into their blood. And there it is. And this is a

molecule that's going to be freely filtered. That's what we mean by freely filtered, it's filtered at the glomerulus. Freely means very easily. And eventually we're going to clear this molecule. And so this molecule x is going to flow through the renal tubules. And eventually it's going to make its way into the bladder. So there's going to be a certain concentration of x in the urine over a certain amount of time, let's just say 24 hours. So you know how long, how long it took 24 hours, you're going to measure the amount of urine that is produced in those 24 hours, you're going to know what the concentration of that molecule was to begin with. You throw it into this equation that you don't have to know. It'll give you the clear answer that x. So you're going to know how much X is taken into the bladder over 24 hours. You need to know what the concentration is because you're going to be able to measure it. We're just going to take urine and send it to a lab tell me what the concentration of X's

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you can measure and by the way, this is GFR right. GFR is glomerular filtration rate, the rate at which this is happening right here. Now if you had a molecule that was freely filtered and not reabsorbed and not secreted, how is the only way this molecule is going to get into the bladder, the only way that molecule is going to get into the bladder is why? Through glomerular filtration, and nothing will pollute the value, we're not going to get rid of any, we're not going to add any. So the rate at which x is leaving the glomerulus, and entering Bowman's capsule, and the renal tubules will be exactly the rate at which x is going to make its way into the bladder. So if you have a molecule, so to measure GFR, you need a molecule that is filtered, not reabsorbed and not secreted. And again, there is a perfect molecule that does that and that molecules and even though this is the most accurate way to get GFR, it is not routinely done in clinical settings. The way that GFR is actually obtained from a patient is to take bloodwork and measure the level of Creatinine we just mentioned created a second ago in their blood. And then the level of Korean and in their blood is put into a couple of equations, not the one that I showed you. GFR is estimated, and it's done with routine bloodwork. And it's based on the concentration of Creatinine in the blood. Why not use any lint if it's so stinking perfect, because it's tedious and expensive. creatinine is something that we make naturally. And so because it's already in our blood, because we know what the concentration of it is, in our blood, somebody devised a couple of equations, where they derived it in such a way that it is at least close to what GFR is, is it? Is it perfectly accurate? It is not? Is it good enough for routine bloodwork? It absolutely is because that's what's done. And if anybody's ever seen blood work, and this is just normal blood work, you see a little e next to GFR. The E stands for estimated GFR, because it's not a completely accurate value. It's estimated with these two equations, there might even be three equations. I don't even know not that anybody's doing any calculations computer does it. That's all. So what do I want you to know? Well, I want you to know what the perfect molecule would need to be to measure GFR accurately. And it's here. I also would like you to know that creatine concentration in the blood is what is routinely used to estimate what GFR Yes, that's what I'd like you to know. There's another molecule pH that's used to measure renal plasma flow rate. Well, once renal plasma flow rate, let me remind you, we did it in the last lecture. It was that math stuff that we did right in the beginning when I was explaining to you what GFR is this renal plasma flow rate Sumana plasma per minute that the kidneys are filtering, that's what it is. And so that is a very important value to know, when assessing somebody's kidney function. There's a lot of things that we need to know when we assess kidney function. GFR is incredibly important, but so is renal plasma flow rate. And so putting all these variables together will help us tease out what's going on with that patient's kidneys. and pH is a molecule the clearance of pH is a molecule used to measure our PFR. I'd like you to know that. All right. And that's that, Oh, I like it is not one more thing I want to talk about. There are some things that we don't clear for the clearance of a molecule is zero. And so I'm going to give you two examples of how that can happen. And two specific examples of molecules where the clearance is zero. So the first one is here. So we'll draw the same thing that we draw over and over again. So we know tubule.

Here's our glomerulus peritubular capillary. Now this first molecule and of course we have to have our bladder over here. Make sure that we know that that's the bladder that I did in the last picture I did. So we're gonna have a molecule, let's call it molecule y. This time, let's use a different letter. Now this molecule y is freely filtered. Small enough, it can get through. But

19:49

we're going to have zero y in the bladder, which means we're not going to clear it. Tell me how that can be. If y has been freely filtered, how is it that we're not going to Have any wine in the bladder tell me, every stinking bit of its reabsorbed all of it. It's going right back in the blood, every molecule of it is, all of it is reabsorbed. If you reabsorb all of it, none of its gonna end up in the bladder. Doesn't matter how much we filtered over there at the glomerulus. I'll give you one example. For example, glucose. Glucose is freely filtered, so anybody molecule, but we want to reabsorb every single bit of it, and we do under normal conditions. Now, of course, if we are a diabetic, we're not treating our diabetes properly diabetes mellitus, that is, well, then we will start to see glucose in the urine, we will start to see glucose being cleared. But that's not normal. That's one example. I'll give you another. So hard line, same picture. So all the same structures. In this particular case, this particular molecule won't get filtered. So we'll make it z. That ain't happening. Neither resists. That's not happening. So certainly, if we don't freely filter, and if we don't secrete, reabsorption isn't gonna matter when it comes to this story. We're gonna have zero this molecule in the bladder, tell me what this molecule is. Give me one example. Starts with the P. Protein is protein filter. Tell me why. It's too stinking big. It's one of the first things that we talked about in the last lecture on this chapter was introduced. Protein is not clear. Now can you have trace amounts of glucose and trace amounts of protein in your urine? Of course you can. That's that's not that abnormal. But to have elevated levels, something's going wrong. So I'd like you to know how we can have a clearance of zero and what specific molecules we do not clear again, under normal conditions. Now what? Now what we're going to do is change yours. Talk about this, the counter current exchanger. What is the counter current exchanger? Well, it was already introduced.

22:33

Actually, before I do that, let me let me say something really quick. Can you give me a scenario where glucose is cleared? I think I just mentioned it a second ago. Untreated diabetes mellitus, right? Tell me why. How is it that we are clearing glucose with untreated diabetes mellitus?

23:02

So what we do under normal conditions, is all that glucose is what? reabsorbed right? Well, with untreated diabetes mellitus, the level of glucose in the blood

23:20

is going to be really high. Which means we're going to have a heck of a lot of glucose in the filtrate. Right. And what we're supposed to do is we absorb all of it. But if we have a ton of glucose over here, we have transporters for this, right. So there's our glucose transporter. So what's going to happen to the transporter is going to be what dies with an S, it's going to get saturated. And so we saturate this

transporter. And so because of that, we'll maybe we only have this much glucose, but it's still some. So there you go. We've just cleared glucose, not normal, supposed to have elevated levels of glucose in the blood. This is a picture that we drew last semester. If you recall, we do it in a much simpler way. We didn't have these terms, reabsorption and all that when we were talking about it, but all we were doing at the time was talking about transport mechanisms and the phenomenon of saturation. So I just wanted to remind you of that. All right. Now what are we going to do? So now let's talk about counter current exchange. I already introduced this to you guys. Want to make sure I didn't skip anything before we move on to it. Nope. I think we're good. All right. So moving on to kind of current exchange and then show you where we introduced to it. That was maybe the second slide. Yep, the two different kinds of nephrons. cortical nephrons, just imaginary nephrons. The ones that we've been talking about up to this point have been the cortical nephrons because we've talked about peritubular capillaries coming off of the efferent arteriole. The juxta imaginary nephrons are the ones we're going to talk about now. The just imaginary nephrons had those really long loops of handling. And I showed you a picture of it, although the ones I'm going to show you now another picture, I'm going to show you that the loose of Henle are actually a bit longer than this. They go deep, deep, deep all the way down to the renal pelvis over here. And so a just imaginary nephron is responsible for regulating the osmolarity of the blood accomplished via the counter current exchanger. And the countercurrent exchanger is made up of two anatomical structures number one, it's the really long loops of Henle of a just imaginary nephron along with capillaries that are wrapped around these really long loose of handling. Now the cortical nephrons have short loops of Henle in comparison to the juxta imaginary nephrons. The loops of Henle of the cortical nephrons have wrapped around them peritubular capillary right you guys know that and I said it about 20 times over the last two, two lectures. The just imaginary nephrons because they have longer loops of Henle. They need extra blood vessels. They need extra capillary beyond the peritubular capillaries. And so what they have are what are called VAs Erecta. And that's the new notes and it's actually already here to begin with. Vas erector is capillary just like the peritubular capillaries are capillaries. And it's extra capillary wrapped around the loops of Henle have just imaginary nephrons, because they're so stinking long. So let me show you another picture of a juxta imaginary nephron. And this is more what they look like. The reason I have this picture up here is just I mean, you can see that the loop of Henle is a little bit longer and it compared cortical to just imaginary but this picture over here kind of does the Justice more here, so this is one and so look at that lupa handling all the way to the renal pelvis, it traverses the entire medulla. So very, very long. So we need more capillary. And so Vassar rakta is only wrapped around the loops of family of just imaginary nephrons, not cortical nephrons. Now I want you to notice something else. These numbers over here, these numbers are representing osmolarity. And I beat it into your head for two semesters now that normal osmolarity is what 280 to 300. And I usually you just use the word or the number 300 Because it just nice round number. And I said that's normal everywhere. Although last semester, when I first introduced it, I said, well, there are a couple of exceptions in the body. I'm showing you one right now.

27:55

Look at these numbers 369 12 That's all fluid that's interstitial fluid within the medulla of the kids. It's getting incredibly concentrated. And this is 100% completely normal, this must happen. And the just imaginary nephrons, those long loops of handling along with Vassar rakta, which together are the contractor and exchanger, create this humongous gradient. And it is huge. It's four times when we get to the deepest part of the medulla four times what is considered normal everyplace else in the body. But this is 100%. Normal, very important that this occur, why it's right here. Large osmotic gradient, the osmotic gradient I'm talking about is 369 12, is used to regulate blood osmolarity. And so let's remind ourselves I know I just mentioned it, but I'm going to do it again anyway. So this is blood osmolarity. And we know not that I had to even mention it to you, I'm sure you remember these

values. 280 to 300, is normal. The reason we can do that is because of that big, huge gradient within the medulla, the kidneys. And I'm going to show you how before we do that, we're going to get into some anatomy. So let's do that. So I'm going to draw the counter current exchanger. So let's do that. Now. Now I'm not going to put all the details of the nephron just what we need to know. So proximal convoluted tubule, and I'll label everything. So this is the proximal convoluted tubule. I'm not going to label the loop of Henle because I'm going to assume you know what the loop of Henle looks like. I'll label everything else though. So proximal convoluted tubule. lupa, hanly. distal convoluted tubule. I make that a bit longer. And then let's make this give us in three dimensions. And here are two dimension I should say. So lupa Hanley ascending limit the loop of Henle, distal convoluted tubule, distal convoluted tubule, proximal convoluted tubule. And then of course over here collecting duct I'll label it. Now the BAS Erecta. Vas erect, I'm going to draw as a horseshoe, just like I did the loop of Henle. Although it's wrapped around it like a snake, I don't have the ability to draw that well. So this is as good as it's going to get when it comes to a Tucci drawing. This is going to be the BAS Erecta. Alright, I'm actually I can label it down here, Bas rakta. And let's make sure that we understand it's just capital there. What else we need to put in this picture, I'm going to put this little dotted line across the screen. And that dotted line above it is going to be the cortex below, it's going to be the medulla. And so I'll label it, cortex medulla. And understand that that entire screen is fluid. Now it's interstitial fluid. And we know that there's a huge gradient at the cortex, the concentration of the fluid is 300. Right, where we start the medulla, the concentration is also 300, I'm going to lower this just a little bit to give myself a teeny bit more room. And then we go from three to six to nine to 12. And I'm going to put those values here 300 600 900 1200. And understand. Again, it's the entire screen were 300. Here, were 300 Here 600 912. But I got to do it over here as well. 369 and 12.

32:17

How do we do that? That's what I'm going to show you. So this is the anatomy of the countercurrent exchanger. Now let's do the physiology of the countercurrent exchanger. So let's start our story. Now when I explain this, I'm actually going to explain it backwards. But in the 20 years that I've been teaching this, I feel that this is the best way to do it. And easiest way for the students to understand here's something else that I need to make sure that you understand is that everything that you're going to see on this screen, even though I'm going to do it in a chronological order, backwards, it's all happening at the same time. Alright, so understand that as well. So this fluid out here is 369 and 12. But the fluid within the renal tubule. And the fluid within the resurrected the blood also has to be 369 12. So what I'm going to do first is I'm going to show you how the concentration of the filtrate is going to go up up, up, up, up, up, up, up up as we descend, and then it's going to go down, down, down, down, down, down, down as we ascend, then I'm going to show you how the concentration of the blood goes up, up up up up as we descend it to the medulla. And then it goes down, down, down, down down. As we ascend up toward the cortex, we'll show you how that happens. So let's start with the filtrate. At the proximal convoluted tubule, it is 300. When we start to descend into the medulla, it's still 300. But then as that fluid flows in this direction, it's going to go to 600 900. And eventually 1200. So it's going to get concentrated. Well, how is that going to happen? We are going to remove water, we are going to transport water, you know I'm going to make that a different color. I'm going to make water green, because we have a few things that are going to get transported here. And I'll just use color just so you can see it better. We are going to transport water out of the filtrate in into the innocence of fluid. As we remove water from a fluid well what happens to the fluid that gets concentrated. So why is the filtrate getting concentrated? We're removing water. We're removing water, we're removing water, we're removing water until we get it as concentrated as it's going to get around 1200. So that's the descending limb. So the descending limb, we are removing water, then what's going to happen is obviously our filtrate is now going to flow in the opposite direction. So the ascending limb that's why they call it ascending because it filtrate is ascending it's going to get

diluted again. 900 600 300 Now you might think to yourself, well obviously we're going to add water. We can't do that, can we? Why? I'm going to remind you. It was a point that I made a little bit ago when we're talking about reabsorption.

35:21

Remember, water isn't over here. So proximal convoluted tubule permeable to water descending limit the lupa handling permeable to water I just showed you that ascending limit the lupa handling. You don't see water written down here. Why? Because it's in permeable to water. distal convoluted tubules permeable the water collecting duct is permeable to water, you are not transporting water at the ascending limit a loop Paheli it's not possible there is no permeability. So how the hell are we going to dilute this filtrate? Well, if we're not going to add water, what can we do instead remove what solute both of those things will dilute a fluid and water or remove the solute. And that's exactly what's going to happen. So we'll do this in blue, we're going to remove solute. And what are the soul you said we're going to we're going to we're going to remove sodium and chloride. And as we remove sodium and chloride, we will dilute the filtrate until we get back to 300. And then the filtrate will enter the distal convoluted tubule at 300. But by the time it exits the distal convoluted tubule and enters the collecting duct, we are going to be actually at 100. Because we're going to remove a little bit more sodium and chloride. And so let's remove some more sodium and chloride. There's a very important reason that that occurs, I'm just not going to tell you yet. Now, I've explained to you how we keep the filtrate concentrated as we descend into the medulla. I'm also now going to tell you how we concentrate the fluid that is the main goal here and that is the interstitial fluid. How do we get this in three to six to nine to 12. The sodium and the chloride as the sodium and chloride is removed from the filtrate, where does it end up in the interstitial fluid, and just the perfect amount. So what we're doing is is that we're adding solute to the interstitial fluid. And in so doing we concentrated 369 and 12. So we've just killed two birds with one stone. Actually, we're gonna kill three birds with one stone, because the blood has to go from 300 to 600 to 900 to 1200. And it's going to flow in this direction. And we're going to concentrate the blood because some of that sodium and chloride is going to be transported into the blood. And as you add solute to a fluid, what happens to the fluid it gets concentrated so that sodium chloride is removal dilutes the filtrate, its removal is going to concentrate the interstitial fluid, and then it's addition to the blood is going to concentrate the blood. And by the way, look at the arrows. So the filtrate we have it's flowing down the descending limb, and the filtrate is flowing up the ascending limb look at the blood. It's going in the opposite direction. counter current exchange. Current is what can be the flow of fluid. Well, we have fluids flowing here, and they're flowing in the opposite direction counter to each other. And they're exchanging, exchanging what they're exchanging sodium and chloride over here. They're not done exchanging, by the way. Because now as the blood ascends up towards the medulla, well, now the bloods got to go back to 900 600 300. Well, how do you think that's gonna happen? I'm pretty sure you can already see it. The water that we removed from the filtrate some of it is going to make its way into the blood. And as you add water to a fluid, what do you do to the fluid you diluted? By the way, water is going from the filtrate into the blood tell me what that's call that's within our reabsorption don't forget those words. So we reabsorb water. The basil Rector does as it ascends up to the cortex and in so doing, it is diluted. By the way, sodium and chloride being reabsorbed over here. Yep, it sure is, are they sure our I should say. So we reabsorb sodium and chloride as the blood descends into the medulla. So I will write the words for the abbreviation reabsorbed here.

40:20

Don't forget these terms. All right? I've just explained to you how all this happens. Oh, no, I didn't.

One more thing, urea. So we have urea in the collecting duct. This is going to help contribute to concentrating both the filtrate and oops, that's an E, the filtrate and the interstitial fluid. So urea is going to be removed, the collecting ducts going to make its way into the interstitial fluid is going to help concentrated it's not going to go into the blood. But it'll be picked up by the filtrate. Again, helping to concentrate a URI is actually recycled a lot of his recycle. And it helps with this particular process, Uri also helps with something else that we're not going to talk about. So there you go, we've created our gradient. And we're maintaining the gradient. Well, because all these three compartments are going to have roughly the same osmolarity. And that's important because if they didn't, we'd have osmotic gradients and water would move where it's not supposed to move, and it would wipe out the gradient. That's not what happens. All right, let's take a break when we come back, we can see our story.

41:52

Shall we finish, I added something to this picture, the word excretion, clearance and excretion are the same thing. And so I'm just telling you that it's obviously the picture is going to be a pilot, but I can use those words interchangeably. Now what? Well, let me show you how this big huge gradient is going to allow us to keep blood osmolality, between 280 and 300. So what I'm going to do is I'm just going to draw a little bit of this story just to collecting Dr. distal convoluted tubule and some blood, and then show you exactly how this is going to happen. So distal convoluted tubule. collecting duct, we'll label everything again. And we'll put some blood over here we'll just put blood that's good enough distal convoluted tubule. collecting duct. And what we'll also do is we'll put a homeostatic line here for blood osmolarity. And the first story that we'll talk about is when blood osmolarity is too high.

43:14

So normal is 280 to 300. And so let's say we're way up here, so the blood is too concentrated. If the blood is too concentrated, we can add water to it. And it'll lower the osmolarity it will dilute it. And so we have high osmolarity. Now why would that be? Let's say that this person is dehydrated. They just didn't drink enough water. And because of that less water in the blood, more concentrated blood. That's no good. What we need to do is this. And our kidneys are going to do that for us. Now how are the kidneys going to do that for us? Well, because we have this hormone that's going to regulate osmolarity tell me what the hormone is please. ADH antidiuretic hormone. So let's talk about antidiuretic hormone. So with dehydration. What's going to happen is is that we are going to and I'll just put the word dehydration up here. So number one dehydration. Actually, number one is going to be high osmolarity we'll do it that way. So we have a high osmolarity blood osmolarity let me make sure that that's in there. So we have high blood osmolarity. Now, ADH is chronically released, we know that but it certainly can be acutely released and it's going to get acutely released because well our blood osmolarity is too high. So what's going to happen as a result of that is we're going to have an increase in ADH release moreso than we did prior to that. Oh the other thing I have to do here is put our gradient three Six 912. I'm going to show you why that's so important. And of course, the filtrate as it enters the collecting duct is at 100. Now, what we want to do is, is that we want to get water out of the collecting duct and we want to get that water into the blood. At this point in time that cannot happen. And this is the reason the collecting duct is impermeable to water. Why? Because it doesn't have any water channels in it at this particular point in time. And that's what ADH is going to tell the collecting duct cells to do. So this tube is made up of a bunch of epithelial cells that targeted ADH are those epithelial cells. So ADH is going to bind to the ADH receptors of these epithelial cells. And when ADH does that. It's going to tell those cells to do something, and what is it going to tell the

cells to do to incorporate these water channels, they're called Aquaporins. Without these channels, the collecting duck cannot transport water into the blood. The collecting duck is impermeable just as impermeable as the ascending limit of lupa handling. Those Aquaporins are not in those, the membranes have those cells unless ADH tells those cells put Aquaporins in your membrane. So when we talked about ADH earlier on this semester, and I showed you kind of generically, what ADH tells the kidneys to do. And that is to transport water into the blood. It was a very generic little picture. Well, this is more detail when it comes to it. And so now that these Aquaporins are in the membrane, now we have permeability, do we have a reason for water to move? Do we have a gradient? Yes, it's a very big gradient. So we're 300 here and we're only 100 here. That's why it's so important to dilute the filtrate. So that we create an osmotic gradients so that we can transport water into the blood, if we still need to, when we always need to. So now water, which is in the filtrate is going to lead the filtrate and get reabsorbed into the blood and as it does, what else molarity is going to decrease now, as we do this, and this filtrate flows, what's going to happen to the filtrate? It's obviously going to get concentrated, right? Why is it going to get concentrated? Because we're removing water from it? So let's say it gets to 400. Do we still have a gradient? Yeah, it's 600 Over here. What if this was 300? Are we screwed? Yep, we're completely screwed. If that 600 was 300, at that 900 was 300. If that 1200 was 300, we're ft,

48:10

we can only regulate our the osmolarity in the blood probably till about right there, we're screwed. The reason we need this big graded is just in case we need to transport a lot of water doesn't have to be a lot of water, more water to get our osmolarity where it needs to be. So what's going to happen here is this will release more ADH until we release enough, it will just go right down the line. So now it's going to be these cells that are moving the water down here. And we'll put more Aquaporins into the membranes of the cells. And we're going to reabsorb more and more water. And if we need more ADH to be released because the osmolarity of the blood is so stinking high. So I don't know I'm making up a number here 700. But that's pretty real. Well, then what are we going to do? We're going to put more Aquaporins in the membrane and reabsorb even more water into the blood. We're gonna concentrate the filtrate even more, I don't know I'll go to 900 we could still move water if we needed to because we still have a gradient. And 900 is very, very, very concentrated by the way. Under normal conditions that's about as concentrated as your urine is going to be because eventually this is going to be urine right. So in this picture now is we increase the reabsorption of water because we have our Aquaporins incorporate into the membranes without them the same happen and you'll have constant elevated Blood osmolarity no good. So now let me ask you this, let's go back to this picture. And so 900 is pretty stinking concentrated, right? And that's eventually going to be urine. So we're going to have concentrated urine. In this story. We're going to have a lot of air anything. Not removing a heck of a lot of water from that filtrate. So concentrated urine, low volume. And the whole story started when when we're dehydrated, right? So when you're dehydrated, you pee a lot. No, what's it look like? It's dark. I've just explained to you why. Why do you have dark pee, because you're removing a hell a lot of water from it because you're dehydrated. And you need to bring once again, the osmolarity back to where it's supposed to be, you're not going to be producing a lot of urine. Why? Well, because water is going into the blood. So I just explained to you how now let's go the other direction. Let's go over hydration, you drink too much water. So now we're going to be down here. So I don't know you drank a few gallons of water this day. So we'll draw the same picture. So distal convoluted tubule. collecting duct we'll label everything. We know we start out at 100. We know we have a gradient 369. Now in this particular case, we don't need the gradient. We don't need it like we needed is over here. But it's still important that it's present. And so there's our blood in this the blood that we need to worry about when it comes to the osmolarity. So now we have a decrease in blood osmolarity.

52:00

And so again, ADH is chronically released, but now we're going to release less of it. So this is going to lead to a decrease in ADH release. It's still going to be present. I'm not saying it's going away. And so now what's going to happen? Let's get let's get us at 100 here. And now ADH is going to stimulate those cells. Oh, yeah, 123, we're going to incorporate the Aquaporins, we're still going to reabsorb some water. But we don't have to reabsorb very much it's gone down, we're certainly not going to reabsorb this much. You're just going to reabsorb that much. And so I don't know maybe this gets to about, I don't know 300 ish, somewhere in there. And then stays that way. We don't have to reabsorb any more water we've gotten to where we need to be. Oops. And so what's happening here is that we're going to decrease the reabsorption of water. And in so doing, we raise osmolarity. So now I asked you this. So eventually, obviously, this is going to be here. And so it's going to be dilute 300 It's fairly dilute urine. So dilute urine. Is there going to be a lot of urine. Yeah. Why? Well, because we're not removing that much water from so the volume is going to be high. So we're not going to have low volume, we're going to have high volume. And so that explains why it is that when you drink a ton of water during the day, you pee more when you do fairly clear. It has to do with this. Now one more example, an extreme example. What if somebody had zero ADH? Tell me what that's called, by the way. Nobody has that somebody doesn't really say th did I hear diabetes insipidus remember that we learned it. And this is a severe form of diabetes and Syphilis is you're going to get no ADH. Well, if you don't have ADH, what are you not going to have? Aquaporins so no Aquaporins and if you don't have any Aqua pawrents collecting duct is impermeable to water, you cannot move otter out of the collecting duct.

54:49

So what's gonna happen as a result of this? Lots of dilute urine Certainly your blood osmolarity will be one you know, moving any water into the blood, your osmolarity is gonna go

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up, up, up, up, up, up, up, up, up, right? So lots of dilute urine, high blood osmolarity. In this extreme case, tell me what the osmolarity of the urine would be. Give me the number 100 started out as 100. And it's not going to change one little bit, it's going to go all the way through. So lots of dilute urine. Because molarity equals 100. So diabetes insipidus. I told you at the beginning of the semester, when we're talking about the endocrine system, and specifically ADH in this particular condition, that somebody with an extreme severe case of diabetes insipidus, could urinate gallons, and gallons and gallons and gallons and gallons and gallons and gallons of urine, very dilute urine in a day. And now we know why. And certainly, we know how much filtrates produce in a day, right? I mean, when we went over it, our number was around 160 110 milliliters per minute translated out to about 160 liters in a day. A liter is about a quart, that's 40 gallons. So theoretically, that's how much somebody could use either long dead before that. But this is something that can dehydrate somebody very quickly, unless it is treated properly. So anyway, please know that. Now what, so what we just did. So all these words, here is this kind of current exchanger and how its comes about. And then what we just did is regulation of blood osmolarity. That's those words, plus, I added the diabetes insipidus thing in there just because well, I wanted to because we'd already learned it, why not revisit it. Now what? Let's talk about some drugs. Let's talk about diuretics. And this is something that I forgot to mention. But when

we talk about reabsorption, let's go back to it really quick, reabsorption and secretion. These are physiological when it comes to the kidneys trying to get us back to where we are supposed to be when it comes to the concentration of H plus. And when we did our little story about reabsorption, the concentration of what was it sodium. But sometimes the kidneys are completely overwhelmed. And they can't secrete enough of whatever, or reabsorb enough of whatever. And now the concentrations of those whatever's like sodium, like ah, plus, like potassium, like creatinine are not within their physiologic range. And so then bad things can start to happen. And so you don't want to actually I'm just gonna, I'm gonna write that down for you guys. So patho physiologic. So what we've done up to this point is physiologic even though we've talked about diabetes insipidus, and a couple of other things that ketoacidosis patho. physiologic changes in secretion, let's do reabsorption. First, that's what we talked about first, reabsorption. And secretion can be or secretion lead to changes in blood concentration. I'm not going to put this in quotes things. What kind of things potassium, H plus sodium, those things that we have in the blood that we want normal. Now how would this happen? Well, one thing would be kidney disease. If the kidneys are diseased, they're not going to be able to secrete reabsorb properly. And obviously filter at the glomerulus as well, that should be included in this story too. Another way that this can happen is certain medications. Certain medications can change the way that the kidneys secrete and reabsorb as side effects, for example. And the reason this came to mind is because we're going to talk about some medications that cause this to occur, and their diuretics. And so let's talk about diuretics now. Now, we're not going to get into the specifics as to how the diuretics work. I need you to know what I'm going to tell you here. Now what is the diuretic people call it direct water pills. I hate that name. A diuretic is a diuretic as far as we're concerned in this room.

1:00:08

Three kinds of diuretics. So what diuretics do is they cause the excretion of fluid from the body. And we've talked about them a little bit, we did it in the circulatory chapter, they are used to treat high blood pressure. So they were lower blood volume. So the cause the excretion of fluid from the blood, it's up in the urine, they can be used to treat a Deema. edema is excess fluid in the interstitial compartment, we know that. So that's what these diuretics are used for. And the three different kinds of diuretics or loop diuretics, that's number one, that's the most powerful of the diuretics. Now, why is the loop diuretic more powerful than the others, because they work at the loop of Henle. And so if we look at our story, here, where's most of the action happening? That's the loop. And if you effect where most of the action is occurring, you're going to have the most profound effect when it comes to what these diuretics do. Now, the distal convoluted tubule also has some action, just not as much. So the loop diuretics are the most powerful, I need you to know that lasix is an example, they're the most prescribed to the diuretics, by the way, they have a side effect, though, they will cause your kidneys to secrete too much potassium, which then will lower your blood potassium levels, which can become dangerous, as we talked about last semester, in the nervous system chapter, when we were talking about membrane potential, and how important potassium is at rest, and how important potassium is with the action potentials. And a lot of other things. So if you're going to give somebody a diuretic, especially a loop, you have to be very, very cognizant of what's happening with their blood potassium levels, you're gonna measure it a lot, it's going to lower it, there are ways to increase the blood potassium levels, but you have to be very, very sure that is around where normal is supposed to be. thiazides will also lower blood potassium levels by causing the kidneys to secrete too much potassium. They're not as powerful. Why because they work at the distal convoluted tubule. Just not as much action there. We do have one diuretic that's what's called potassium sparing, it doesn't cause the kidneys to excrete too much potassium, they're called aldosterone blockers. They also work at the distal convoluted tubule. So why don't we just prescribe these? Why would we prescribe something with this type of side effect? Because the Adastral blockers just aren't as powerful. They're

not as strong. And so you deal with these side effects. And it's not like you know, you can't deal with low potassium you can't, you just have to be very careful of it. You have to measure it, you know, routinely when people are on these kinds of diuretics. So know what's on the screen, and what's not crossed out, again, up with this on pilot as to what you have to know what you don't have to know. All right. Now what? Let's finally talk about urine. We've talked about filtrate. up into this point, let's talk about urine can't call urine urine until it's no longer being modified. In urine, the filtrate stops being modified once it passes through the collecting ducts. So once it enters the minor kala C's, now we can call it urine. Because at that point, no longer are we reabsorbing are secreting anything in that urine. Now when it comes to urine, I think I already mentioned this, but a liter and a half a day, about 1% of that 160 liters is what eventually will become urine. Most of it is water. And these percentages can change a little bit depending on whether the person's drank a little bit more water, less water that day and fluids and such. But on average, this is about what you're going to see when it comes to urine. So most of it is water. There's some nitrogenous wastes in it, there's obviously salts, there's sodium, there's chloride, potassium, etc, etc, etc. pH is going to vary widely. Here's something else that I know I've mentioned this is that you can tell a lot about what's going on in a person's body by looking at their urine. So let me ask you this. Just think about this logically. Let's say that somebody's pH is little on the low side, let's say it's around five. Tell me that that person's body was that person's body a little bit more acidic or less acidic? What does the urine tell us? It was more acidic. Why is the urine more acidic because the kidneys secreted more H plus, because the body was a little bit too acidic. And so that excess acid that was in the body causing the pH to be a little bit lower is now in the urine. It was a waste product of the blood. Remember the urine used to be in your blood. And so again, your urine can tell you a lot about what's going on in your patient's body.

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Honest polarity of between three and 900. So this would be somebody who drank a pretty decent amount of water, this would be somebody who's probably pretty dehydrated, not very dehydrated, but pretty dehydrated with the concentration at night, still considered normal, we know it can go as low as 100 with severe diabetes insipidus, and then can certainly be as high as 1200, because that's as high as the gradient goes within you to social fluid, the medulla. Now, here's something that I had yet to mention. And that is the term specific gravity. Specific gravity has to determines concentration of a fluid and the size of the solutes within the fluid. And so let's do this. So with specific gravity, you can get an increase, and I'm just going to now write it up. You know what, I'll do this. So specific gravity, little abbreviated SG you can get an increase in specific gravity with an increase in osmolarity. And specifically, now we're talking about the year and or bigger solutes. So the size of the solute will determine in part what specific gravity is, has to do with weight in the end. And then a decrease in specific gravity is just the complete opposite. We have a more dilute fluid and or smaller solutes. And then urine healthy urine is sterile. And so now what are we going to do? Oh, and by the way, you need to know the normal value for specific gravity 1.012 1.03. Now, before we move forward, I'm going to give you two different people their urine, and we're going to compare them and you're going to tell me what's going on with these people. That's what I'll do on the exam. On the exam, I'll say that this person had this much urine, the volume was this much in the pH? Is this E osmolarity. Is this specific gravity? Is this tell me what the person has? Or tell me what's going on with the person or the over hydrated, dehydrated? Do they have this condition or that condition? And we're going to do an example in just a second. But before we do that last page, here are these terms, these micturition terms. So let's say you have a patient and they're catheterized. And there's just nothing in the bag. That's what that's in urea. So there's no urine output. Two reasons that can happen. Number one, the kidneys aren't making any urine. And so that would be urinary suppression. Now, why would the kidneys make any urine? Well, because they're not working. And Stage Kidney Disease, they'd have to be on dialysis, their kidneys are not filtering the blood. And so they need a

machine to do it. So people who are on dialysis, they don't pee, because they don't make urine because they're not removing anything from the blood. Urinary retention is while the kidneys are making plenty of urine or dude is making urine, but they can't get rid of it. Why? Well, because it's being blocked somewhere. Maybe they have a big tumor within the bladder, and there's just no way to avoid it. So that would be an example of urinary retention. oliguria is you don't make as much urine or not as much urine is being excreted from the body. Certainly D hide something as simple as dehydration can cause oliguria. And we just talked about that a second ago. So in this picture over here, I can put the word oliguria with low volume. Over here, I could have put the word polyuria high volume of urine, which we'll get to in just a second. So there are a number of reasons why somebody can have a decrease in urine output. Don't make as much urine there might be a blockage just not a complete blockage like you get with urinary retention, renal disease, maybe it's stage two kidney disease instead of stage five. And then part of the area the complete opposite you drink a heck of a lot of water, diabetes and separatists. There it is. We already talked about why you'd have a lot of urine. That's the second time we did it. So people with diabetes and syphilis polyuria dilute urine when it comes to diabetes mellitus. So when we have a glucose problem type one or type two, we can also have a high urine output but for a completely different reason. So let me ask you this. So and we're talking untreated diabetes mellitus, by the way. So you have somebody with untreated diabetes mellitus, that means that their glucose levels in the blood are going to be high, right? So that means their glucose or their blood glucose or their blood will be concentrated. You buy that?

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When you're dehydrated, blood osmolarity aside like it would be if you're an untreated diabetic high glucose, high osmolarity. dehydration. High osmolarity. Are you with me so far? When you're dehydrated? Are you thirsty? Yes, it says. So the reason that you're thirsty is because you have a high blood osmolarity your hypothalamus senses that you have a high blood osmolarity and your hypothalamus makes you feel thirsty. Why? So you'll drink some stinking water? Because the hypothalamus is under the impression that you're dehydrated? Well, the hypothalamus isn't that smart. If your blood has a high osmolarity, whether it's because of dehydration, or because you have high glucose levels, the hypothalamus doesn't know. All it knows is that your osmolarity is high. So somebody with diabetes is not treating it properly. With a high blood osmolarity. We'll be thirsty. Now because they're dehydrated. But because the glucose levels are high, so people with untreated diabetes mellitus will drink a lot of fluids a lot of water, well, what do you do when you drink a lot of water? You pee a lot. But that urine will not be dilute. Like it is with diabetes insipidus it will have a higher osmolarity that is the urine because there's a lot of glucose. So in both cases, there will be lots of urine. But the difference is diabetes insipidus. It'll be dilute diabetes mellitus, it'll be concentrated, again, completely different reasons. dysuria, it just hurts when you go. That's all it means. All right, doesn't really say anything about the volume, it just hurts when you go for various different reasons. So now that we have these particular terms out of the way, let's just do a patient a versus the patient B. And then this will be the last picture of the evening. So patient a versus patient B. And they're going to look pretty close to each other. So let's say that patient a has a urine volume equal to 600 milliliters, that higher low. That's the bit on the low side, right about one and a one to one and a half is good. So what are we gonna call this? Poly urea and urea? dysuria? oliguria? Yeah. Let's say patient B urine volume is the same. 600 mils little on the low side. Just like this person over here. We are going to say that the osmolarity of the urine let's go about 800. That's high end a normal right. And let's do SG specific gravity. Let's say that this person is 1.025. Is that normal? It's a higher normal, but it's normal. That's GOP here. That normal? It's a bit on the high side 1.03 Is the high end the normal patient a I'm just going to tell you probably maybe they're just dehydrated. Now patient B could certainly be dehydrated, but boy, that SG is awfully high. And there's no reason for it to be higher than normal. Because certainly 800 is still within our normal range of three to 900. So it should really

be about 1.0 to five ish, maybe even 1.03 Surely not 1.04. So what do you think we might find in this person's urine? Why do you think it might be 1.04? Let's go back to SG and let's find out why SG would be high protein maybe? What could this person have? Starts with a G? Big long word ends with itis glomerular nephritis talked about lining things on the first page of the notes. Can that where the hell is it? Maybe second page of the notes. There it is. So maybe this person has that based on what you're looking at in the urine so they might be dehydrated. But maybe they have this doesn't mean they do but certainly means they can. So I'll do something like this on the exam. I'll give you some type of a urinalysis with a bunch of values on it and you're gonna tell me what's going on with the patient. Alright folks, we're all done.