Heart PM 2-15-22

Fri, 3/11 9:13AM 16:55

SUMMARY KEYWORDS

action potentials, conduction, cardiac muscle, myocardium, gap junction, draw, cells, atrial, fibers, pathways, cardiac conduction, remember, muscle, cardiac muscle cells, av node, contract, potential, atria, excite, heart

---

00:01

Yeah. The PowerPoint for this section if you haven't, I think so. Really? Okay. I'll put it up, I promise. Okay, but thanks. Thank you. So let's talk about the cardiac conduction system. What is it, that is the electrical system of the heart that's going to excite the heart. And it's made up of a number of different structures. And these are the major structures that are part of the cardiac conduction system. And so what this cardiac conduction system does, and here's kind of a pretty picture of it, I'm going to draw it myself is that it generates action potentials. And these action potentials are going to conduct along the cardiac conduction system. And those potentials are going to excite cause action potentials within the atrial myocardium in the ventricular myocardium. So let's draw the cardiac conduction system. And I'm just going to abbreviate it, well, I don't even have to abbreviate it, we're not gonna even do that. So I want as much room on this page as I possibly can get. So here's the heart. And I'm going to put the four chambers of the heart. And so let's draw the cardiac conduction system, I'll do it in green. It all starts right here. So there's a whole bunch of cells right here in the right atrium. And those kernel of cells together signle atrial node, I'm going to abbreviate it as a node, and I'll label it.

01:28

So there's a bunch of cells there. In front of those cells, we have these projections. We have these long arms that project out to the muscle of the atria.

01:46

We also have these long projections, going out to another node called the atrioventricular node, I'm going to draw that a different shape, right there. Now, what are these long projections called these long projections, they're called internodal. Pathways. I'm going to point to them, so these long projections in their synapses with the cardiac muscle, by the way, their synapses via gap junctions, electrical synapses, if you remember that from last semester. So inner neural pathways in a neural pathways, internal pathways. And then we have the AV node over here, I'll point it in, I'm going to label it. So there's the AV node has a very, very important job that I'm going to discuss in a little bit. We also have then from the AV node, something called the bundle of hiss, not his but his it's also known as the AV bundle. So I'll point it. So these are all structures, once again of the cardiac conduction system. And so that's the bundle of hits is how it's pronounced then from the bundle that

has it branches, right and left, we have what are called bundle branches, that's the right bundle branch. That's the left bundle branch, I will label them accordingly. So there's our left bundle branch. And that is, of course, going to the left ventricle or toward the left ventricle.

03:24

And this one's the right bundle branch. And the right and left bundle branches have branches from them that are smaller, that are now going to the ventricles.

03:45

And so the right bundle branch has these branches going to the right ventricle and left on a branch as branches go into the left ventricle, what are they called? They're called purkinje fibers. And the Purkinje fibers are going to directly synapse with the ventricles. So how does this cause the heart to be excited? I'm going to show you so I'm going to put little black dots in this picture. Now, these black dots are going to represent action potentials. We talked about action potentials last semester in excruciating detail, did we not? We get to talk about him again. We're gonna see two more action potentials. This semester, we saw X potentials last semester of what neurons skeletal muscle cells right, now we're gonna see two action potentials that are involved with the heart, then these little dots are going to be one of those action potentials action potentials of the cardiac conduction system. And it all starts right here at the SA node. So these represent action potentials that are being generated in these cells. And then these action potentials are going to do this along the inner neural pathways. So all these dots are action potentials and what are they doing? Tell me the fancy word? Not that it's a fancy word? Starts with the seat I heard it. Conduction. What time tell me. There's two kinds. Saltatory is where? Where do we see Saltatory? Conduction myelinated axons are these axons? What kind of conduction is continuous conduction? Right? So continuous conduction along these internal pathways. And then what's going to happen is, is that those action potentials are going to get to the very ends of these internal pathways. And those action potentials are going to excite the cardiac muscle. And then the cardiac muscle, they're going to generate their own action potentials. And when muscle does that was muscle do when muscle gets excited? What does it do? It contracts. So these x potentials are conducting, and we are going to excite the atrial muscle so that the atrial muscle will contract Well, what about the ventricles? Well, we need to get the action potentials to the Purkinje fibers, how are we going to do that? Well, these action potentials are also going to conduct a lot of these internal pathways, which will make their way to the AV node. Which will make their way to the bundle of hiss, which will make their way down the right bundle branch and left bundle branch and then finally to these purkinje fibers. And it's these purkinje fibers when we get these ash potentials to the very ends of them that are going to excite the ventricles, the ventricular myocardium. That is, and when that happens, they can track right we know that. me ask you this. Do we want the atrial and ventricular muscle to contract at the same time? Does that sound good? That sounds terrible to me. The atria are going to contract first, and then the ventricles are going to contract and what's going to ensure that is the AV node. And this is how it's going to do it. The AV node delays, action potential AP conduction, approximately 1/10 of a second, which is the same as 100 milliseconds, I'm pretty sure I have both of those in the notes. That's all you need. So what's gonna happen is this, these action potentials are certainly going to get to the atria first anyway, because they started here. But x potential conductions really fast. We remember that for last semester. And so they would, in essence, arrive at both of the myocardium at the same time, and they would contract at about the same time. But the atria are going to get excited first and contract first, because we're going to have this delay of conduction right here. And it's just I didn't say 10

seconds, I said a 10th of a second. That's all it takes. So the atria will contract, and then the ventricles get the contract. And so the AV node has this very important job. And it's very important that we understand what that job is. And that is just to delight conduction, a 10th of a second, how does it do it? It doesn't matter to us. Alright, so the internal pathways, what do they do? They directly excite atrial muscle, the atrial myocardium. The purkinje fibers directly excite the ventricular myocardium This is a story you already know. I told this story last semester, I just use different cells.

08:43

I'll remind you, I'm gonna I'm gonna draw this like in 30 seconds Am I 15 Right now 15 seconds remember, this action potentials along the lower motor neurons releases acetylcholine onto acetyl on to the nicotinic receptors nicotinic receptors or sodium channels, sodium channels open depolarize skeletal muscle to generate action potentials. And then the skeletal muscle contracts. Remember that these action potentials cause action potentials in skeletal muscle and then the skeletal muscle contracts, right? These action potentials cause action potentials in the cardiac muscle and then the cardiac muscle contracts. This is the same store he's this is just different cells. It's not skeletal muscles, cardiac muscle. It's not lower motor neurons. It's a little pathways and purkinje fibers. It's not releasing acetylcholine. It's a gap junctions. It's electrical synapses. You already know this story. It's just different cells. So I'm going to draw something similar to this. So I'm going to draw this in a little bit more detail when it comes to the cells themselves that are involved. Alright, so I'm going to draw this line or this tube here, it's in green. So as part of the cardiac conduction system, and we are going to gap junction couple that electrically coupled that to cardiac muscle cells. And by the way, that's what it's going to look like, it's going to look like a stringy tube. So as cardiac muscle, it's gonna look like a fiber. So that's cardiac muscle. That's cardiac muscle. This is the last picture we drew from last semester, this should look familiar to you. Well, maybe it does, maybe it doesn't. Remember when we are comparing and contrasting skeletal muscle, smooth muscle, cardiac muscle, remember that? I think this was the last picture we drew or maybe one of the last pictures. So striations, right, cardiac muscle straight it so there's a straight itself, straight itself. Remember this.

11:13

So these are all individual cardiac muscle cells that are coupled to each other, electrically. So there's one, there's one, there's one, there's a whole bunch more, we have a sensation, right? Remember that term from last semester sincere mean sheet. So let's see how this works. Now, the action potentials, the little dots are going to be represented by that action potential right there, you got to the very end of that, whatever that is. And you're going to actually tell me what that is in just a second. I'm going to give you the punch line, right now, I told you that we're going to go over two different action potentials, the X potential the cardiac conduction system and the action potentials of cardiac muscle, I'm just going to tell you that the ion involved with the fast depolarizations calcium, you're going to see it in the next lecture, positively charged ion is going to move through that gap junction right there. make its way to that membrane and stick to it, what are we going to do the membrane starts with a D, we're going to depolarize it. And we're going to get an action potential, the action potentials of cardiac muscle cells are pretty funky looking, they look like that. We just excited the cardiac muscle. This action potential caused this action potential. And then it conducts. So continuous conduction, that's what those dots are going to be. And I'm just going to draw one right here, another one. That's continuous conduction that occurred. And I'm just going to give you the punch line once again, the ion responsible for fast depolarization in cardiac muscle cells is sodium. So sodium comes in goes through this gap junction here. And then depolarizes, this cardiac muscle cell

to threshold, and then we get more action potentials. So this is how it works. On by the way, if this is atrial muscle tell me what this is. Tell me what that is. If this is atrial muscle, tell me what the green tube is. Tell me what the green tube is. It's an internal pathway is not internodal pathways directly excite atrial myocardium. I'm showing you how if this has ventricular muscle, what's the green tube? Perkins Purkinje fiber, right. So this is either an internal pathway or it's a Purkinje fiber. This is either atrial muscle or ventricular muscle. So this is a sequence of events that's going to occur. We have cardiac conduction system action potential. I'm going to draw one, they look like this. reach threshold boom. It looks like that I'm gonna show you in the next lecture

14:42

that leads to myocardial myocardium action potential, which again looks funky looks like that. Those action potentials cause more action potentials in other myocardium oops I just drew it so let me draw another so the second one I draw you know what? Let's do this 123123 All right, you with me? Tell me what happens when we excite muscle tell me what happens. You can try it there we go. That makes sense. Oh, by the way, what's gonna happen when the heart muscle contracts, what is the heart pumps blood. So this is going to lead to the heart pumps blood it better or you're going to die within a few minutes. So what we're going to do come Thursday is I'm going to show you in detail these action potentials and all the channels involved. I'm going to show you the action potential with the myocardium and all the channels involved. So two more action potentials to visit. Let me see if I can talk about anything else before we get out of here. No, we're done. Alright folks, I will see you on Thursday.

Heart PM 2-17-22

Fri, 3/11 9:13AM 1:13:24

SUMMARY KEYWORDS

ekg, depolarization, action potentials, cardiac conduction, heart, calcium, draw, repolarization, sa node, p wave, called, potential, cardiac muscle, atria, block, measuring, normal, ventricles, picture, normal sinus rhythm

---

00:02

Let us begin. So on Tuesday, we started the heart chapter. And we were discussing the cardiac conduction system and its importance and its importance is to excite the muscle of the heart so that the muscle of the heart can can track. So at the heart can pump blood. And the revolves all around these action potentials that are going to be generated and conduct along the cardiac conduction system. And what we did was show exactly how the cardiac conduction system is going to excite the heart, I'm not sure why that just happened, give me a second, I need to call that back up. So it's going to excite the heart. Go we are back now. And that was shown with this picture right here. So this is either the inner neural pathway, or the Purkinje fiber. And to remind you of what that is. And neural pathways come off at the SA node, and they project out to the atrial muscle and in the Purkinje fibers, they come off of the left and right bundle branches, and they project out to the ventricular muscle. And they're going to couple with them, electrically. So we're going to have gap junctions, which was shown here. And so the action potentials, so this action potential that you see here is one of the dots, the dots, again, the kinds of weird stuff going on up here, those dots are individual action potentials. And those action potentials in the cardiac conduction system are going to cause action potentials in the cardiac muscle cells. And that's where the story is going to begin tonight. So we're going to go through those details. So we're going to go over first, this action potential right here, of the cardiac conduction system. So what I'm going to do is that I'm going to take one single cell from the SA node. So the SA node is just a whole bunch of cells, I'm just going to yank one out of the SA node. And that's what we're going to draw up on the screen. And we're going to measure the membrane potential of that cell and see how we generate action potentials. And so let's draw the SA node. And we'll use the same color. So I'll do it in green.

02:15

So this is an SA node cell. And I will label it. So SA node cell, just one. And we're going to measure membrane potential of this cell.

02:35

By the way, that's what it looks like it does look like a fiber. And as we do that, we're going to track the membrane potential. So membrane potential versus time. membrane potential versus time. So let the story begin. So these cells, just like every other cell in the body have a resting membrane

potential, but resting membrane potential just for a split second. As soon as word resting membrane potential, this cell is going to do this, and you're going to tell me what this is please. What's that call, starts with the D depolarization. So we're gonna get to slow depolarization, the membrane potential is going to slowly get more positive. And I'm going to show you how that happens. That slow depolarization that we see there has a name, it's called pacemaker potential. I'm actually going to show you where we are in the notes just to make sure that we're on the same page here. It's this stuff right here. So what we're doing now is this cardiac conduction action potential, we are going to draw all that right now. That's what we're going to do. And we started with the pacemaker potential. Now, how does that happen? How do we slowly depolarize there are two channels that are going to be involved in this, one of those channels is called the HCN channel. So I'm just going to draw it right here. So there's our HC n channel, you don't have to know what HC N stands for. So h c n, we have to make it plural because there's a whole bunch of them. When that channel opens, it's going to gate a certain ion, it's going to transport a certain ion that is sodium. And we know from last semester, that when a positively charged ion is transported into a cell, some of the some of its going to get stuck to the inner membrane, and we're going to get a depolarization. So as sodium comes in we depolarize there's another channel involved and that channel is a calcium channel. Now I'm not going to tell you exactly what kind of a calcium channel it is because it will cause some confusion. So all I'm going to do is call it calcium channel.

04:37

And so here is a calcium channel. And it has to be plural, because there are many of them.

04:44

And when it opens Well, of course calcium is going to be transported into the cell, it's positively charged. So it's going to contribute to this slow depolarization. So what I'm going to do here is so sodium in. Yep. And calcium in is responsible for the slow depolarization. And we're talking about an action potential, what do we have to reach Tell me please? Threshold, right? Now we are going to hit threshold. And when we hit threshold, as we well know, we're going to get a rapid depolarization. Now what's responsible for the rapid depolarization? Another channel, of course, we're going to put that channel over here. This is also a calcium channel, I'm going to call this one a voltage gated calcium channel. And we know what that means. So voltage gated calcium channels, we have to make a plural because there's a whole bunch of them. And that's gonna bust open, and when that opens, we're gonna get a flood of calcium coming in. And that's why the membrane potential gets so positive, so fast. And so this too, is calcium and calcium in from a different calcium channel, we're gonna hit a peak, and we hit that peak, then this is going to happen, tell me what this is called, please. It's called repolarization. Right? Now, what's going to be responsible for that? Another channel. And what that channel is, is our old friend from last semester, the voltage gated potassium channel. And so we will label it as such. Oops. So voltage gated potassium channel is responsible for the repolarization, we will make it plural, of course, because there's a whole bunch of them. And we know that when these channels open, when potassium channels open, potassium is gated outward, is transported out. And as we remove that positive charge, our membrane potential gets more negative as we can clearly see it is. And so the repolarization is due to potassium out. And then we are going to eventually hit resting membrane potential. And as soon as we do starts all over again, for a split second, we are at rest. And then we are going to start to depolarize, the threshold, and boom, we're going to get another one. And these are being spontaneously generated, by the way the HCN channels and the calcium channels are spontaneously opening, they're controlled, their frequency is

going to be controlled. But it's spontaneous within the cell itself. And it just keeps on going it better. Because if it doesn't, well, then you can't excite the heart. If you don't excite the heart, or it's not getting getting try, you're not going to pump blood. And you're done. When it comes to the frequency of these action potentials that again, let's make sure that we understand that these actions potentials are these dots that we see here in this picture. Why does it keep doing that? Are the dots that we see? No, no, no, no no. Are the dots that we see in this picture here, so we just drew one of the dots. That's what we just did. And the frequency at which these action potentials is generated, is going to dictate what your heart rate is. So if the SA node is generating one of these action potentials, and then of course, they're going to conduct one action potentials every second, how many seconds or a minute 60, what's your heart rate going to be 60. Cardiac conduction system dictates your heart rate in a dictate your heart rate, because it generates these action potentials that are going to eventually stimulate cardiac muscle, which is going to allow the muscle to contract. How many beats per minute a beat is a contraction, the contraction is due to the excitation of the muscle, which was caused by these action potentials of the cardiac conduction system. And it all starts at the SA node, because the SA node and this is something that didn't put in this picture I wish I had. So I'm going to go to the notes here is the pacemaker the heart, that's what paces your heart. Under normal conditions, we're going to see abnormal conditions in a little bit. So we have these action potentials they're being generated, and then they're going to conduct along the cardiac conduction system to the internal pathways and eventually to the Purkinje fibers. And so we'll go back to this picture right here. Hopefully it doesn't shut down for the third time

09:33

is going to cause an action potential in the cardiac muscle. So now what are we going to do? Now we're going to go over this action potential. So this action potential causes this action potential and it's a completely separate action potential because it's in a separate cell. So now let us go over this action potential right here. So now what I have to do is is that I have to draw cardiac muscle cells. So let's do that. This is either an atrial muscle cell or it's a ventricular muscle cell, it doesn't matter. And so here's the cell, we're going to measure the membrane potential of this cell as well, we're going to track it over time. So cardiac muscle cell, take your pick as to which one, and we're going to measure membrane potential in this cell, and we're going to track it. So membrane potential versus time. Now, when it comes to cardiac muscle cells, they have a stable resting membrane potential. And that's what this is going to be representative of. That's resting membrane potential, something that we saw last semester, and I will put resting membrane potential underneath this stable line. So that's resting VM. Now, when it comes to what we're going to draw here, I'm going to show you where we are in the notes now. So we're here now, we'll get back to our topic, pacemaker, just a second, I want to do this first. This is what we're going to draw. So we have all these different phases, phase 0123, and four. Phase four is resting membrane potential. So what I'm going to do here is that I'm going to put above this, the number four, now what's responsible for resting membrane potential? What's what channels, I'm not going to tell you? It's not important for us. We're going to worry about the action potential. So now, what's going to happen is this. Once again, tell me what that is. That's depolarization is a slow depolarization. Where are we going to reach eventually, threshold? Now, what causes the slow depolarization here in an SA node cell was channels that are involved with the cell itself. Cardiac muscle, no different story. And I actually already told you what the story is in the last lecture. And what's the story, we'll go back to this picture. These action potentials, the action potential that we just drew is responsible for this slow depolarization. That's happening here. So the calcium that's coming in during fast depolarization, the calcium that is coming in during fast depolarization. This calcium is going to diffuse through the gap junction, it's going to end up right here, and it's going to stick in that word we use last semester is going to stick to this membrane, when these positive charges stick to the membrane, what happens to membrane potential, it gets more positive. That's

how these action potentials cause these action potentials. So what we have to write here is this is that that slow depolarization is due to the calcium from the cardiac conduction system, action potential, that's what depolarizes that or not going to tell you what the order is yet, I will in a little bit, but someone else does it as well, it can do it as well. But for now, we're just going to stick with this, we're going to hit threshold, when we hit threshold, boom, we're going to get our action potential. So finally, we get our action potential. And there it is, there's our fast depolarization. So now our first channel that we're going to draw here, what's responsible for the fast depolarization our friends from last semester, the voltage gated sodium channels that we talked about with neurons that we talked about with skeletal muscle cells. So right there is a voltage gated calcium channel, or I'm sorry, sodium channel. We have to make it plural, because there's a whole bunch of them. And when we hit threshold, those open, and we get a flood of sodium coming into the cell, some of that sodium gets stuck to the membrane, and that's what causes the depolarization. So what's responsible for this sodium in through those voltage gated sodium channels.

14:05

This is phase zero, so I'm going to put a zero right there. Again, resting membrane potentials, phase four, the fast depolarization is phase zero, we're going to hit a peak. And when we hit the peak, then we're going to repolarize. And we're going to repolarize really, really fast. And here we go, just as we saw with our action potentials last semester, and just as we saw just a second ago with the SA node, but something's gonna be different here. What's responsible for the repolarization the same exact thing that was responsible when it came to the SA node cell and that is voltage gated potassium channels. So voltage gated potassium channels are responsible for that fast repolarization and that's phase number one. So I'm going to put over here potassium out and above it the number one because That's phase number one. But then something happens immediately after this starts to occur at this point right here, and I'm going to point to it, I'm going to tell you what happens, another channel opens right at that point, that's going to counter this voltage gated potassium channel that's causing potassium, to be transported out of the cell. And what opens right there at that point, is a calcium channel, a voltage gated calcium channel. So voltage gated calcium channels open right there at that point. I'm going to put it right there. So that is a voltage gated calcium channel. Much the same as the voltage gated calcium channel that we saw in this picture over here. So voltage gated calcium channels. And when they open, we already saw the calcium is going to come in. So what's happening here is this. Potassium is leaving, and the calcium is coming in at the exact same time. So they're gonna cancel each other out. So what's going to happen the membrane potential, not a whole lot, it'll start to repolarize, just a teeny bit. So instead of its screaming down, as it was doing here, and as we saw last semester, it's going to do this. It's going to plat toe. And that's the way I drew it in the last picture from the last lecture tonight, I told you, they look kind of funky. There's our plateau right there, there's a reason for that, there's an incredibly important reason for that, I'll tell you when we get to it. So at that point, those calcium channels open, the potassium channels are still open. And so during this time, right here, potassium is still out. So potassium out in calcium. And once again, they're cancelling each other out, for the most part, potassium kind of wins. But it's pretty flat, we call that the plateau phase, I'm going to put the word plateau over here, a very important phase, and it's phase two, you're gonna see just how important that is, then we're going to get to a point. And that point, I'm just going to make it right there, those calcium channels close. So the voltage gated calcium channels closed right there at that point.

17:34

And when they do, because the potassium channels are still open, we're gonna repolarize fast again.

And so potassium out, still, that doesn't stop. And so that's phase three, and then we get back to resting membrane potential word phase four, and it just repeats itself. So what I'll do here is this will be here, the voltage gated sodium channels, phase zero potassium channels over here, I'm going to make the cardiac muscle cell just a little bit thicker, so that I can put numbers below there. So we can equate everything. So those voltage gated potassium channels, that's going to be involved in phase one, it's going to be involved in phase two, it's going to be involved in phase three, the calcium channels over here are only involved in phase two. And there's your action potential that will allow the cardiac muscle to contract. Because we need to first excite obviously muscle before the muscle is going to contract. Learn that last semester in detail. Now, I have to add one more thing, the or so what's the oral how else can we get this cardiac muscle cell to threshold, we're gonna go back to the picture that we drew on Tuesday. So that actually potential that I just drew is this one right here that I'm pointing at. And by the way, the numbers one, two, and three have absolutely zero to do with these numbers here, absolutely nothing, nothing, nothing, nothing whatsoever. All right, the reason that I numbered these, the way that I did is because I wanted to equate it to that right there. That's all this picture right here. And this picture right here is what I'm going to talk about right now. So we generate this first action potential and these action potentials are going to conduct along the membrane. We know what that means we learned about a last semester. And so now we get to this point. So we have the same action or we have an action potential like that one. And look what I have I have sodium coming in at this time, right? Is that what happens during fast depolarization? Of course it does. We just learned sodium in at this time. Sodium is flooding in these cardiac muscle cells. They're all interconnected to each other by these gap junctions. And so as the sodium comes flowing into the, into the cytosol of this cardiac muscle cell, what it's going to do is some of it is going to diffuse through the gap junction and end up right here stuck to the membrane of the next cardiac muscle cell, which is then going to depolarize it to threshold. So what's the or the or is or sodium from some other cardiac muscle cell action potentials. Initially, the cardiac conduction system gets the ball rolling. Initially, the cardiac conduction system is what's going to initially excite the cardiac muscle then after that the cardiac muscle is on its own cardiac muscle has to excite other cardiac muscle cells, but it needs to start somewhere and where does it start? It starts at the neural pathways, it starts at the Purkinje fibers, but then after that, the cardiac muscle cells have to excite each other. And that's why they're put together the way that they are as a sensation. So there's a reason for that. Plus, it's really, really fast. It's an electrical synapse, right? They're way faster than the chemical synapses that we talked about in detail last semester. And plus, they're all going to contract at about the same time. So we have coordination of all these muscle cells, because they are a sensation, they are all interconnected by these electrical synapses. And so there you go. And so then then what happens is if the cardiac muscle is going to contract, and the heart is going to pump blood, like the heart needs to pump blood. Are we good so far? Yes. Now, what else do we need to talk about? Oops. Well, you know what, let's go here really quick. Let's talk about this right there. Let's talk about the actual contraction of the muscle. So what I'm going to do now is I'm going to draw another cardiac muscle cell. And I'm going to draw up with a little bit more detail. I'm going to draw with the T to be I'm going to draw with SSR. So here's our cardiac muscle cell. Oops, strong red. There's our T tubule. And here's the SSR. What's so important about the ASR? What's the NSR remind me beautiful calcium. Here's our voltage gated calcium channels right there.

22:37

And let's put that action potential here up on the screen. So here's that action potential that we just drew. Fast depolarization plateau phase, blah, blah, blah. Last semester, when we did skeletal muscle, how do we get the calcium release from the ESR? What did these action potentials need to do? Conduct down the T tubules. Remember that it causes release of calcium. It's not how it works in cardiac muscle. This is how it works in a cardiac muscle. During Phase Two, as that calcium comes

into the cell, I'm just gonna call it a and I want to go 1234. So a, that calcium is going to be gated inside of the cell. We already know how that happens. We just threw it a second ago. That has a special name, by the way, it's called trigger calcium. I mentioned it last semester. I just didn't talk about it much, because I knew I was going to talk about it this semester. So that calcium that comes in, is going to diffuse to the sarcoplasm and make its way to that calcium channel right there, and it's going to bind to it. That calcium channel is going to bind calcium. And when that calcium channel binds calcium is going to release the calcium. That's what releases the calcium in a cardiac muscle cell, not the action potentials that conduct down to T tubules. That doesn't do it. And it has a name to your notes. I'm going to abbreviate it on the screen. It's called calcium induced calcium release. That was in your notes last semester. I just didn't really talk about it. And then what happens is is that this calcium does what binds to troponin, troponin changes its conformation pulls on tropomyosin reveals the active sites, myosin heads, right forum across bridge, the ratchet, boom, we get contraction, all the same stuff that we talked about last semester in a lot of detail when it comes to skeletal muscle. The sliding filament model works for the cardiac muscle cells themselves, but getting the calcium released is what is different. So why is it that we have such a funky looking action potential? If you don't happen you don't contract the muscle you cannot get this calcium released unless this calcium comes in during Phase Two already happening. And you're dead in minutes. Are we good with this? Yes. So that picture right there are these words right here and then the contraction, calcium is pumped into the ASR. This all the stuff that we talked about that I'm not really going to mention again, because it's unnecessary. Yes. So that triggered counting. Yeah, this calcium right here is this calcium right here. That happens during this time, that calcium is just going to these calcium channels are going to grab it. And when they do, they open and they let go with the calcium, that's an ESR. And then we get the contract the muscle,

25:47

so that would be the calcium from the ESR. That's what this calcium is from the ASR. Thank you. You're welcome. Okay, now what? Go back a teeny bit. Let's talk about an ectopic pacemaker and what an ectopic pacemaker is. So, earlier on, I said that pacemaker the heart is the SA No, that's under normal conditions, what's going to paste the heart that's where the action potentials start, they started the SA node. Sometimes that's not always the case. If something other than the SA node paces the heart, it's called an ectopic pacemaker. Do you guys ever hear of an ectopic pregnancy? ectopic means outside of what's normal, where's the embryo supposed to implant in the wall of the uterus, right. But sometimes that doesn't happen in the embryo might implant in the fallopian tube, for example, that would then be called an ectopic pregnancy because the embryo implanted outside of what is normal. So in a topic, pacemaker is pacing the heart outside of what is normally going to place the heart, which is the essay No. And it can be anything. I give a couple of examples in the notes, but it can literally be anywhere in the heart, they can actually even be within the muscle itself, cardiac muscle can be an ectopic pacemaker, we're just in this little area, the muscle, all of a sudden, it just starts to generate action potentials all by itself. That's way not normal. By the way, big topic is outside of normal. So I've given a couple of examples in the notes. So if the SA node all of a sudden doesn't work. I've seen now that the AV node will start to paste the heart. The AV node has cells that can generate action potentials, just like the SA node does. Now we get these action potentials in the AV node, they will conduct along the bundle of His left and right bundle branches, purkinje fibers and off to the races we go, we're going to excite the ventricular muscle and boom vocal tract, the Purkinje fibers can paste the heart. So let's say none of the rest of the cardiac conduction system is working for whatever reason, it's not that important as to why but some damage of some kind. Now we have the Purkinje fibers, they can just start right there and paste the heart. And when these two different areas paste the heart, your resting heart rate will change. Now normal resting heart rate for the average person is somewhere between 70 and 80 beats per minute. If the AV node is pacing the

heart, study about 40 to 60 for Kinsey fiber 25 to 45, somewhere in there. Now this doesn't mean that if you have a low resting heart rate, and this is resting heart rate, doesn't mean that you have an ectopic pacemaker, most often it means that you're just in good shape. So if you're in good shape, and you train properly, your heart adjusts to that acclimates to that your heart rate is slower, but your heart pumps harder. That's what you want slowed heart rate pumping hard, more efficient, heart hearts gonna last longer. Most often if you have a slow heart rate, that's the issue, but not always. And so when the with these topic pacemakers, this is about what your heart rate would be and just know those. I don't want you to think, however, that every ectopic pacemaker slows down your heart because other topic pacemakers can speed the heart up. It just depends in that's what this line is saying right here. You have to slow down and speed up the heart. It just depends. And there's a lots of different ectopic pacemakers. But these are the two ones I'd like you to know.

29:27

Now, what else do we need to know here? was going to say something and it just left me.

29:35

I'm sure it'll come back at some point. Now what? Let's talk about EKGs. So what is an EKG I am forgetting something. Let me just make sure that I'm not forgetting something really, really important that I'm going to have to come back to at a later time during the lecture and it's going to screw things up. Nope, doesn't look like it. Alright, if it goes back to me that I'm sure I'll blurt it out. Question. Somebody read my mind. And you know what I was gonna say? I thought I heard some. All right. So what are we going to do now? We're going to talk about EKGs. So what is it he and by the way, EKG, ECG, same thing. What is an EKG? Well, they define it right here, recording of the electrical activity of the heart of, of cardiac muscle, let's make sure that we remember that it's measuring action potentials of cardiac muscle. And what cardiac or I'm sorry, what action potentials would those be? It would be these, these are the action potentials that the EKG is measuring. What the EKG is not measuring, are these action potentials of the cardiac conduction system. Why? Because they're the signal from the cardiac muscle cells is way bigger. So the EKG doesn't pick it up. The cardiac conduction system is not as vast as the cardiac muscle is there's just more muscle cells. And if there's more muscle cells, there's going to be more of these action potentials. And that's what will simply dominate when it comes to the electrical activity of the heart. So that's what's being picked up by the EKG. So once again, the EKG is measuring the action potentials of all the muscle by the way, atrial, ventricular, left and right, all the same time, we're gonna see something else that the EKG is not. And that's what this line is not a recording of the potentials of the cardiac conduction system, I just said that. It's not a recording of mechanical activity, what would mechanical activity be contraction, EKG is not measuring contraction. EKG is not measuring blood flow, that would be another mechanical event, it's electrical. Something else I want to point out as well, even though we're not recording the action potentials of the cardiac conduction system, doesn't mean that we can't infer things from the EKG itself. So even though the EKG is measuring the potential to cardiac muscle, it still tells us plenty about the cardiac conduction system, it can tell us plenty about the muscle itself of the heart and its health. So you can get an awful lot of information from an EKG just by measuring the actual potentials of the cardiac muscles themselves. Now, some stuff here, this, you're not going to have to know. I'll get back to this in just a second, I'm going to show you this picture right here. So when you do an EKG, you're going to be putting electrodes on different areas of the body, some people call them leads, and that would be incorrect. A lead is a measurement, there are 12 leads, but there are only 10 electrodes placed on the patient if we're talking about a full EKG, Now traditionally, where you're

going to put the leads, and they're just these sticky little leads that conduct electricity, so that you can measure the electrical activity on the wrists and on the ankles. But you might not have that you might not have the luxury of putting on the wrists and the ankles, because maybe this is somebody that doesn't have an arm, for example. Or maybe they have a condition where their arms are shaking, maybe they have Parkinson's, for example, and you can't put them on the wrists, there are other places you can put them. upper thigh, shoulder shoulders, I decided the belly button are other areas where you can put these electrodes and just going to stick there. And these are what are called Lin leads, because again, traditionally, you're putting them on the lips. So those are the four electrodes. And again, I said leads, that's a measurement.

33:59

And the machine is what's going to be doing the measuring by the way, we'll get into it. So we have Lin leads, and then we have what are called chest leads, otherwise known as precordial leads. And so once again, these are electrodes, there's going to be six of them. And they're put in very specific places. And so V one v two, I decided to start them and then again place in these very specific places and you want to get them as close to perfect, because if you don't, it could screw up the measurements, it could screw up the leads. So six chest leaves, six limb leads. And again, the leads, those are measurements, so there's going to be 12 measurements in total for a full EKG. Now, going to the notes. I define what the limb leads are, and again, there's six of them six measurements based on four electrodes that are placed. There's limb lead, one, two and three. We have our roman numerals again that you have to know for the upcoming exam right? So you have to know some more Roman numerals here. One, two, and three, very, very simple. And then we have other limb leads, called augmented limb leaves. And those are aVR, aVL ABF. And there's some extra stuff in here that you don't have to know. The only thing you don't need to know the positive and the negative stuff, I don't give a crap about that stuff. What I care about is that you know, what angle each of these leads is measured, and the plane. So the leads are measured in a frontal plane, you guys know what a frontal plane is, up and down. And we have the six different angles, the six different leads, and I need you to know what the angles are for each. So Lynley. One, what is zero degrees, Lindley two plus 60, Lindley three, plus 120, and so forth and so forth, I need you to know those please. For the chest leads, we don't have angles. For the chest leads, we simply have measuring the electrical activity in a transverse plane through the hearts, all six of them. And what I'll do is I'll explain what that means. Here's the picture that I have in pilot. That one right there, I thought it was going to be good enough. And I thought, You know what, I'm going to do something different here. And I'm gonna explain to you what all this means. Let me say something else. The EKG is just simply observing. It's not affecting the heart in any way whatsoever, it's not affecting the electrical activity in any way whatsoever, at least it better not because then it'll screw things up. So let's just observe it. And so when it comes to what these leads are, what the angles mean, what the plane means what, what all of it means I'm going to give you an analogy here. It should be enough time before the break. So this right here is going to be some event. What do I mean by an event, I don't know, you're going to go to a concert. Or you're gonna go to a basketball game or a soccer game, or I don't know, whatever the hell people go to something that you enjoy. And there's going to be seats all around this event. I'm not going to be able to go to the event. But I want to know what happens at the event. And I had the luxury of 12 different people that I know that can go to this event. And I want those 12 people to come back to me and tell me all about the event. The best thing for me to do is not put on, like all right there. Because everybody's gonna say or tell the same story pretty much because they're all sitting at the same place. If I wanted a thorough story of this event, what I would do is I would put somebody, maybe right here, and I might put somebody right there. And I might put somebody right there. And I might put somebody right there in there. And they're looking at the event all at these different angles. All of them having a different perspective on the event. Now that six people and by the way, they have

floor seats, they're looking at the event or like like this, like in a frontal plane. So they can see it right here at eye level. All these people different angles. Well, that six people, where am I gonna put the other six? Well, this is a cool event. We have seats above the event. And they're going to be looking down at the event in the transverse plane.

38:46

And so I'm going to put my friend v one over here and my friend v two over here and my friend V three, and V four v five and V six over here, spaced them out above the event, but far enough apart from each other that they're going to have just a slightly different vantage point. So they can tell a slightly different story. Now could I if I just had one person to take to this event or go to this event? Could they tell me the story of the event? Of course they could. They would tell me but it wouldn't be as thorough as 12 different people. And it certainly wouldn't be as thorough as 12 Different people seated in 12 different places to see the event. Everybody's going to give me a slightly different story but the story is pretty much going to be the same. So when it comes to a 12 lead a key EKG and do you have to do a 12 lead EKG you don't but that's the best way to do an EKG. So why a 12 lead and again a lead is a measurement EKG being answered in the I see fit that all in one line. It gives a thorough story. That's the reason Why a 12? Lead? EKG or ECG? Both are correct? Answer it gives a thorough story. You see everything. If if I just had, you know, people one, two and three there, they might miss something that AVR sees, because it's way on the other side of the auditorium, they're gonna see something different, they're gonna see something that person one would have missed. All right, let's take a break. When we come back, we will continue our story. Okay, folks, here we go. So this picture right here is the picture that I just kind of drew except this one's obviously a bit prettier than mine, showing again, all the angles of these limb leads, and then again, the the different plane of the chest leads. So what we're going to do now is, and actually, you know, what I'll do really quick is I'm just going to show you what an actual EKG trace looks like, right there. So when we 123, aVR, aVL, aVF, and then B, one through six are the chest leads. Now, I don't want you to think that this is all time on this scale right here. And it's time zero all the way to time, whatever. This is literally one for probably about three seconds, this is AVR, for that same three seconds v1 For that same three seconds, are you seeing that? And so we have these traces, I'm sorry, all these leads here. And you look at them, and you say, well, they look fairly similar. But then there's definitely differences between each of these. And this cartoonish picture, you can kind of actually see a little bit better when these 123, aVR, aVL, aVL. I mean, there's obviously similarities between, okay, we have this little bump, the sharp thing here and other bump, say little bump, sharp thing, but much bigger. This one's a little bit, you know, sharper than that one. And then this one's a little bit more blunted, and then what the hell is going on here, it's upside down, it's supposed to be because it's at a different angle is the angle that causes it to be upside down. And so although they have their similarities, they're just slight, slight differences, sometimes big differences between the three that put together the story. And you need all this to put together the proper story. And so again, this is what it will actually look like. And somebody who's very well trained, and very well versed will look at that and within seconds know exactly what's wrong with the patient. But it's an art. And the more you do it, the better you get. And someday, people in this room are going to be very artful in reading EKGs. So anyway, what we're going to do now is is that we're going to take Lin lead to, and we're going to draw a trace, and we're going to go over what each of those little bumps and spikes is and what they represent. So let's do that now. So we're going to draw this right here. And so let's do that. So here's our trace. So our first little bump is right there. Then there's going to be owned, by the way. So this is electrical activity. I'm not gonna I'm gonna reset just a second. So it's picking up electrical activity, and then this is time, I'm just not going to draw the x&y axis, it just makes it too busy, no reason for it.

43:29

So we have our first little bump. And then we have our spike. And then we're going to have another bump. So what do they all represent? This is called the P wave. This is the QRS complex.

43:49

And that's the T wave and good for us. Is the alphabet PQ RST. What do they represent? The P wave. Oh, and by the way, there's one other thing right before the P wave, and right at the at the queue. We call it the PQ interval interval. There's many different intervals. By the way, we're just going to do the PQ interval, it's just a, it's a length of time is what it is. So that is the PQ interval, right there. And so we're going to learn about what each of these is. So let's go P wave first. And so what is a P wave? A P wave is depolarization of the Atrium, both left and right, by the way, so depolarization of the atria. The QRS complex, is depolarization of the ventricles, both left and right. We're talking all the muscle cells, T wave. That's Repolarization of the atria and then the PQ airtable. I'm going to save that for just a little bit, we're going to get to it today. I'm going to save it for right now. Now what I'm going to do is I'm going to draw the action potentials and what action potentials am I going to draw to draw these action potentials right here, I'm gonna draw two of them going to draw an atrial action potential and then draw a ventricular action potential. I'll draw them in different colors. So I'll draw one in red one in blue. So I'm gonna draw one here. There it is, there's our depolarization. There's our plateau. And there's our repolarization. I'm gonna draw another one, I'll draw it in blue. Here is our depolarization. Here's our plateau. And here's our repolarization. Now, I'm going to label these.

45:50

The red one comes from what muscle Tell me. It's got to be atrial, right? So that's from atrial muscle. So that is atrial muscle.

46:04

action potential, that's the red one. And then the blue one is then of course, ventricular ventricular muscle action potential. I didn't randomly put them where they are. I put them according to the timescale that I have with the EKG trace. Is this depolarization? Right here? Yeah, it coincides with the keyway. Is this depolarization right here? Yes, it coincides with the QRS. This is repolarization right here coincides with the T wave. The T wave is Repolarization of the ventricles. QRS is depolarization of the ventricles, the P wave is depolarization of the Atrium. Now, this is something we have to make perfectly clear here. When we have drawn action potentials, we know that when we deflect upwards, it's depolarization. And when we deflect downwards from a change, it's repolarization. Right? It's not how you look at an EKG that like from the art of the S is not repolarization. So how it works, the entire P wave is depolarization. The entire QRS is depolarization. The entire T wave is repolarization. What the EKG does is pick up changes in electrical activity. So why is the P wave go up and down? Why is the QRS go up and down? This is the reason at the peak of the P wave. That's when there's the most of these action potentials of the atria occurring. Where we have depolarization. That's why remember area of the curve in math. Remember that? Yes, sir.

47:54

Oops, you're right. Thanks for catching that. But does that make sense? The entire P wave, I have one action potential here. How many action potentials you think are being generated? Millions and millions and millions. And they're not going to happen? Just like that. We're going to be spread at least a little bit. We're looking at about 100 milliseconds right here. There are timescales to this. So the entire QRS is depolarization. All of the T Wait, even this part right here is repolarization. Please make sure that you understand that all right. Yes. He says like you're just saying, like report like the whole the whole thing. Pulsar, not part of it, that from start to finish. It's repolarization. I just have one action potential up here. There are millions of them that are generating this trace, millions and millions and millions and millions of action potentials. Thank you. You don't have time to draw millions to is all I have time for tonight. Now, let me show you something before we move forward. Oh, by the way, what's missing here? Obviously, something's missing here. depolarization of the atrial depolarization of the ventricles Repolarization of ventricles. What's missing? Repolarization of the Atrium. Do you think the atria repolarize? Of course they do. I'm showing it to you right here. But when is it happening? At the same time, the ventricles are depolarized. If the ventricles and the atria got into a fight, who's going to win? The ventricles will kick the crap out of the atria. The ventricles. more muscular means there's more muscle cells means there's more action potentials. The reason we pick up depolarization of the ventricles over Repolarization of the atria is because this signal is bigger. If the ventricles didn't depolarize we would see Repolarization of the H for you. But under normal conditions, that's not what's gonna happen. So the repolarization of the atria is absolutely happening. The EKG just does not measure it, the signals completely drowned out by the signal of the ventricles. Before we move forward. Let me show you this picture right here. This one right here, make it a little bit bigger. I'm probably gonna screw something up. Not not, I'm not gonna mess with it, forget it. So what we have here is the heart at different points in time, same heart, just different points in time, we're starting here at time zero, and we get all the way over here. Yellow is depolarization. Green is repolarization. So we had here the right atrium, it's it's depolarizing. First, why would that be? By the way? Why do you think it's depolarized? Before the left atrium is? Yeah, yes, I noticed there in the the neural pathways coming off of the SA node, which is going to depolarize ventricles is closer to the SA, know where it all starts. So you're going to start to depolarize, the muscle of the of the left atrium before you can start to depolarize the right atrium, before you start to depolarize, the muscle of alethic left atrium. Again, it doesn't all happen at once, it's a way. Like if you drop a pebble in a pond, you get those waves that come out takes a little bit of time for us to come out here. Well, it's gonna take a little time to depolarize all the muscle. But it's gonna start here. But as soon as you start to depolarize, you're gonna start to generate the P wave. And that's hopefully you can see it, it's starting to come up. Now we're starting to depolarize the left atrium. And now we're getting more of the P wave. And when we have all of the muscle depolarize, the P wave is going to be complete, then one, well, now we're going to start to depolarize the ventricles, we can see that when we started to generate the QRS complex and look what's happening at the exact same time, we're repolarizing, the atria, we just don't see it. And then by the time it's all said and done, and we have depolarize, the ventricles fully, we have a full QRS complex. And then we're going to start to repolarize the ventricles. And as we do, we start to generate our T wave. And by the time we have we polarize all the muscle cells, boom, done with the trace.

52:17

And that's about a second, by the way, give or take, we have these times that you guys have to notice in your notes. So P wave, you're looking at about 100 milliseconds, give or take, that's a 10th of a second by the way. And then we have QRS complex a little bit shorter 60 to 100 milliseconds, give or take T wave, up to about two tenths of a second PQ interval, we're going to come to in just a

little bit, actually a little bit as now, let me explain what the PQ interval is, again, it's a time. So in order to do that, I'm going to draw a heart over here, and I'm going to draw the cardiac conduction system. And I'm going to do it really fast because we don't need to get the detail. All we need to do is understand. So here's the heart. I'm just going to put very, very quickly the cardiac conduction system, so SA node internal pathways, internal pathways, AV node bundle hiss, left bundle branch, right bundle branch purkinje fibers, alright. And again, what is the PQ interval, it's the time right before the P wave, which is depolarization of the atria to right before more or less the QRS complex, right before we are depolarizing the ventricles. So where are the action potentials? Do we have any action potentials in the atria? Right before the P wave? Yes or No? No. If we did, we'd be measuring those action potentials in the atrial muscle. So right before the P wave, our action potentials are still in the essay know, right before the P wave. Again, the P wave is action potentials in the atrial muscle, we're not there yet. Right before we're in the essay, no, then what happens is, over time, these action potentials are going to conduct along the neural pathways, and we are going to get our P wave. Okay, so I'm going to kind of trace here and make a little bit darker. So we are generating this P way because we are getting those action potentials into the muscle cells of cells because we just excited. While that's happening, these action potentials are conducting to the AV node where they are stopped for about a 10th of a second. So maybe we're right about there. Can you can you see where it start on darkening it. And as we're there we know these potentials are conducting along the left and right bundle branches and they're marching through the Purkinje fibers. And so we're right about there now. There's a PQ interval. Tell me what PQ interval this What is it measuring? The time it takes action potentials to conduct through the conduction system. That's what it measures. I told you that the EKG is not measuring electrical activity of the cardiac conduction system. But I also told you that it can tell us stuff about it. And the PQ interval tells us how well those action potentials are conducting within the cardiac conduction system. And sometimes they don't do that. Well. We call that a heart block. We're going to be talking about that in a little bit. So what is the PQ interval, it is a time time it takes action potentials to conduct through the cardiac conduction system, CCS. That's what it is. And that has a normal time as well, I'd like you to know that. Okay, so we're again, two tenths of a second is a high end normal. So 120 milliseconds, 200 milliseconds, I need you to know that. And these when these values change, we can infer some things about what's going on with the heart. Speaking of things that aren't normal, we're not going to do a main electrical axis takes it takes a long time to explain. And in the past, it's confused the hell out of students and I just don't think it's valid, and there's not a lot of value to it. It's something that's EKG also measures. If you need to know a Sunday, you are going to know it. What we're doing right now, just the basics of the EKG. And that's plenty. So now what, let's talk about arrhythmias, dysrhythmias, you can use the words interchangeably. It's just simply the electrical activity, the heart is not normal, that's all. And there's a bunch of different ones, we're gonna go over, I don't know, five or six of them. Nothing super complicated.

56:58

The first two are very straightforward and simple. The first one's called tachycardia. And tachycardia just simply means a heart rate above 100 beats per minute. And again, average 70 to 80. Give or take is considered normal all the way down, maybe almost a 60 is considered really normal. Now, why would somebody Oh, and by the way, let's look at these traces here. The one that we have up on the top is normal, and then we have the dysrhythmia, the arrhythmia underneath it, so we can compare them. So what we have at the top here is what we have a P wave, QRS, T wave, little space, P wave, QRS T wave, everything spaced perfectly. So from one QRS to the next, the spacing and looking good, the P waves look great T way everything looks fantastic. What we have here is what we call a normal sinus rhythm. You guys ever hear that term normal sinus rhythm, where you think you get the word sinus, from? What pace is the heart the sino atrial note, normal sinus rhythm, the SA node is pacing the heart, normally normal sinus rhythm. And so that's what we have here. What we have down here

is the tech party card. And what can we can see blainley is how many more QRS complexes we have. Over time, that's showing us that the heart is beating faster, how much faster well the machine is going to tell you. But when you guys go, you know to med school or PA school wherever you going to go someday you're probably going to be asked to do it by hand you're gonna have to count the little the little boxes and do it the old fashioned way. That's where it used to be done. Nowadays, everything's automated but it's good to do it the old fashioned way because it helps you really understand what's happening. Now why would somebody is hard I'm by the way, this is resting heart rate. Not I just ran a marathon and my heart rates above Of course, it's going to be above 100. This is resting. Now why would somebody is resting heart rate behind a few examples here high sympathetic tone, we talked about that last semester. your sympathetic nervous system stimulates the heart makes it be harder and faster, right. You might have autonomic dysfunction where you have high sympathetic tone. You have a high heart rate maybe this person might have hyperthyroidism give me a hyperthyroidism. Give me one of them. Under the G Graves disease, this person might have Graves disease and when we did hyperthyroidism in graves, that was one of the symptoms in the notes. For signs in the notes I should say high heart rate, a topic pacemaker right? Face being accepted. pacemakers can make the heart rate go up or down just depends on the type of pacemaker. So a few examples of why somebody's heart rate would be too high. Now we're gonna go the other direction. So once again, normal sinus rhythm versus a bradycardic heart. And we can clearly see way less QRS as a few ways. Tea weighs as compared to our normal, a bradycardic card is considered to be bradycardic. If it's below 60, what would cause this? Well, high parasympathetic tone, we know from last semester that parasympathetics, slow the heart down. What else, we're not hyperthyroidism, but hypothyroidism, maybe this person has Hashimotos disease, and the thyroid gland is destroyed. So they have a low heart rate, ectopic pacemaker that's already been established. And then athletes, so what's called an athletic heart. And I told you earlier, I mentioned it earlier that if you do have a low heart rate, it doesn't necessarily mean something bad. And more often than not, it means something good. When you train, your heart is going to adapt to that training, and it becomes more efficient. And an efficient heart is a heart that beats slow, but beats hard. It doesn't take as much energy when the heart does that the heart is trying to be as efficient as possible, because again, of the rigors that you put it through when you work out. So another benefit of working out, your heart is going to last longer. If you have a tactic card or card, it's pumping faster, probably not pumping as hard because it doesn't have time. And that hearts using hell of a lot more ATP generating way more metabolic products, that heart's not going to last as long. So the goal is slow heart rate, and it beats heart. That doesn't mean you're wishing for hypothyroidism. By the way, no, I'm talking about an athletic heart.

1:01:34

One of the things I need to mention here is this, I typically don't have pictures on my exams, right? I did last semester for the skin. If you remember, I'm going to put EKG traces on this exam, it's only going to be one question. And you're going to identify the dysrhythmia. All right, I won't even have the normal long one on there, you're just going to identify it. And is it going to be these? No, it will not be these exact traces that you see on pilot net you're going to see here up on the screen. So those are tachycardia bradycardia. Now we're going to do heart blocks. Now what is a heart block a heart block is when the action potential conduction is slowed down or blocked, it can happen anywhere can happen within the cardiac conduction system that can happen within the muscle itself. Well, we're going to concentrate on his block at the AV node. Now, let me remind you that it's completely normal for action potentials to be blocked, slowed down at the AV node by a 10th of a second, that's normal. I'm talking beyond that. I'm talking a longer delay at the AV node because something has gone wrong with the AV node. And how much longer is the block it depends on the severity of the block. We have first degree second degree third degree blocks, as we go from first to second to third is getting worse and worse and worse. So our blocks are going to be concentrated right here, could we have a block in

the left bundle branch or the right bundle branch we can or anywhere, I'll actually show you an example actually, I'm going to show you an extra one of a block at the right bundle branch. But it can be anywhere. But again, we're going to concentrate at the AV node. And so let's do that. So we're going to first do a first degree heart block, and it's going to be a first degree AV node heart block. So what I'm going to do first is I'm going to draw a normal trace. So there's our P wave, QRS and then our T wave. So that's going to be normal. So there's our normal sinus rhythm, just a second of it. Now what we're going to do underneath that is a first degree heart block and a first degree AV heart block. Now try and draw as close to it as I can to the one up top that just doesn't look the same. I just want to make it look as close to the same as possible. When it comes to that P wave. Not too bad. Actually, it sucks, dammit. Whether it sucks or not, I don't care right now. That's good enough. Alright, so this is going to be our first degree. heart block, first degree AV heart block. So make sure that we understand that because again, these hard blocks can be anywhere and it would make the trace look to make it look completely different. So let's think about this for a second. So we have our AV heart block. We're gonna go back to this picture. So we have our AV heart block right here. It's not a 10th of a second, maybe it's two tenths of a second twice as long as it's supposed to be. And when it comes to these action potentials, where are they eventually going to start to conduct to the bundle his left and right bundle branches, purkinje fibers, right. And if we're blocked here for a longer period of time, and by the way, the sad note already did its thing, the internal pathways already did their thing, which is why the P waves look exactly the same. Because we're depolarizing, the atria just fine. It's beyond that now. So we're at the AV node, it's blocked for a longer period of time. It's blocked for a longer period of time, it's blocked for a longer period of time. And eventually, we're going to get to the Purkinje fibers and depolarize the ventricles. And then we'll repolarize the ventricles. So what's going to happen? We are going to have, I'm going to draw our P queues. There's a PQ interval, we're going to have a longer PQ. Again, if it's an AV hard block. So longer, pq interval is going to take longer for the action potentials to conduct through the cardiac conduction system, simply because they get blocked here for a little little longer period of time.

1:06:25

What kind of symptoms is this person gonna have? None. With first degree hard blocks, whether it's at the AV node, the bundle his wherever, doesn't matter. The person isn't going to have any symptoms. Because it's not that big of a deal. It's minor. That is first degree second degree is a different story. Third degree way different story. So no symptoms. So then why on earth would the person have an EKG, if they didn't have any symptoms, they were having an EKG for a different reason. When it comes to a first degree hard block, it is found out by accident just could be some routine EKG that's done, or something else happened to the person. And they found out that the person had a first degree heart block, like me, 15 years ago. I have a right bundle branch block, and I never knew it. And so let's think about that. A right bundle branch block. So here's normal. Here's a QRS complex. Never had a symptom, life still don't have any symptoms from this. And it was found out completely by accident. So there's normal. So here's gonna be our right bundle branch block. So right bundle branch block, we'll call it the Tucci block. So what is it going to look like? Well, the P wave going to be completely fine. PQ interval completely fine. Should we start the QRS complex? Right when we're supposed to? Sure. Wrongful? Let's run the branch? It's conducting X potentials to the left ventricle just fine. So I'm going to start to depolarize my left ventricle just fine right on time.

1:08:25

What's gonna be different? Is it going to take longer to depolarize? All the right ventricle? Yes, yeah, because we're blocked here. So we're going to take a little bit longer for the potentials to get to these

purkinje fibers to depolarize all of these but in the meantime, left ventricle depolarizes fine. So what should the QRS look like? Well make it dramatic. It's not quite this dramatic. It's wider.

1:09:00

So it takes a long period of time. Never knowing why did I get an EKG I watched my son wrestle. And he broke his leg while he was wrestling, and it disturbed the crap out of me. And Dan went to sleep. So I went into search, I went, I went into shock. And I went to sleep. And so because I came to about 30 seconds later hit my head really hard. Got a concussion. Really cool in front of all the other wrestling dads, by the way. So when I came to I had the medics and if you've ever been to wrestling matches, there's it's just a freakin Zoo. And there's medics already there. And they're evaluating me and I said, I go home and said, Sir, you're not going anywhere. So my son and I drove together in the ambulance to the hospital. And when I got to the hospital because I passed out, it's just routine. They're gonna they're gonna EKG. And when they did, they got all excited because they found out what I did. I taught at Wright State blah, blah, blah. When he said, you know, you have a right bundle branch block, and I said I had no idea and they showed me the trace, I still have to trace I just can't remember where it is, since somewhere in my office. And again, had 00 symptoms my entire life. And that happened I got that done when I was 40. And up into 4015 years later, I'm still completely symptom free of any of it. Now, why do I have it might be a question. I can tell you exactly when this happened. This happened in the eighth grade when Claire Hartman I swear to God, that's her name. I swear to God broke my heart. All that for a joke. She asked me out the prom in high school by the way, guess what I said? I said no, she was hot. I said yes. No, I'm kidding. I turned her. I turned her down. You only get one chance with her Tucci. It didn't happen in the eighth grade, I was probably born with it. Because I didn't have anything happened to me when I was a child that would have caused any issues like that no viral effects into my heart. So this was something congenital with me. And it's very minor. But again, I had no idea I had. So first degrees again, and it's the first degree mine's a first degree. You're not going to know you have it. It just gets picked up again. on accident. My was completely accident. Yes.

1:11:39

So you just said that for you? It was?

1:11:47

Oh, yeah, you could have some type of infection of the pericardial cavity. And that can cause damage to certain places of the heart neck can certainly cause that heart attacks can cause these kinds of blocks all kinds of different things. Oh, yeah. Yeah. can be acquired later on in life. Yes.

1:12:03

On the right side, how do they tell them the right side of

1:12:07

it? I'm certainly no expert. When it comes to that there's this is just one of the leaves. I'm sure. You

it? I'm certainly no expert. When it comes to that there's this is just one of the leaves. I'm sure. You know, some of the other elevens probably indicated that it was right as opposed to left. I am absolutely not an expert when it comes to EKG, but they knew it. And they told me and I believed them. So that was that. All right. Yes.

1:12:30

Did you slow it down enough

1:12:32

to piece just say again?

1:12:36

So you know, that's a maximum amount of tax credentials and what? Not one moment, right. So like, if you have the left side, it's like the other side does the same thing. Did you get to these?

1:12:53

Oh, yeah, you can get all kinds of weird stuff with EKGs. Yeah, you, you Google EKG online, you'll see all kinds of weird stuff going on. And again, they're interpreted by these experts and they can look at it. They just know. I'm not an expert. I'm far from an expert when it comes to EKG. I know the basics. And that's about as far as it goes. So you don't want 618 Let us stop and I will see you guys on Tuesday.

Heart PM 2-22-22

Fri, 3/11 9:13AM 1:13:48

SUMMARY KEYWORDS

ventricles, valves, atria, murmur, cardiac cycle, heart, diastole, blood, ventricular pressure, systolic volume, arteries, closed, called, normal sinus rhythm, systole, ejection fraction, contraction, qrs, draw, stenosis

---

00:03

Alright folks, here we go. So what were we doing in the last lecture, we were talking about hard blocks, and specifically at the AV node. And so we're gonna stick with, except for my right bundle branch block, but everything else is going to be a B Block we already did first degree, we know what's not serious, don't even know you have, it's usually found out by accident. Now we're gonna go to second degree and second degree is worse, there's two kinds of second degrees is type one and type two. So what I'm going to do to demonstrate this, and again, these are easy blocks, all that means is that you're slowing the conduction of the action potential. That's all it means. So what I'm going to do here is I'm going to do a normal sinus rhythm. And so we're going to have P wave QRS, a number of times and a T way, of course, and then a little time will pass now the P wave, QRS T wave, I'll do two more a space amount, the same T wave and then we're going to do one more. There's a P, QRS, and a T way. Now, what does the second degree type one gonna look like? So the next trace is going to be a second degree type one. So we're going to have a trace here P wave. Now just like our first degree, we're going to have an elongated PQ interval because again, we're at the the AV node, that's where we're getting the block. So it's going to take a little bit longer to get to our QRS.

01:51

And then we're gonna have a normal T wave and then another P. And then something's gonna happen here. And then P wave, of course, we're going to have an elongated PQ.

02:09

There is our QRS. And that's good enough. So obviously, something's missing here. And what's missing here is a QRS. Again, this is a v block. So we have a missing QRS. So we missed QRS. Now if you miss a QRS, of course, you're not going to have a T wave either because a QRS is depolarization of the ventricles. If you don't depolarize you can't repolarize so we have a miss QRS. Now why does that happen? I don't have time to get into it. If you want to talk about it in my office, I will, it won't be on the exam. point is is that you're going to have occasional missed CQRS is with a type one second degree at the AV node. Now, what kind of symptoms are you going to have here? Well, if you don't have a CQRS, you're not depolarizing the ventricles, what are the ventricles not going to do? They're not going to contract. That's how we get muscle to contract we excited depolarization is excitation. So if the ventricles don't contract, that means the ventricles don't pump blood, we're not going to get

blood pumped to the pulmonary circulation, systemic circulation. So you might get a little dizzy every once in a while. When this happens, all it takes is one time with a ventricle stoke contract, and you're going to get dizzy. And again, it's going to happen occasionally it might happen when this occurs, it might not happen when this occurs, but that is a common symptom of this particular condition. You might ignore it depends on how old you are, you might just chalk it up to old age, being tired, whatever. Now we're going to get to a type two, now we're gonna get a bit more severe. It's still a second degree heart block, though. So both of these are second degrees. But this one is a worse one. You're gonna see why. And again, at the AV No, let's make sure that we understand that. So here's the type two. So once again, we're going to have a prologue to PQ because we are blocking at the AV node. And here's our T wave. Here's a P. We're going to miss a QRS. Here's a P, we're going to miss another QRS. And what the hell let's just go three in a row. There's another P with a miss QRS. So missed QRS is and so the difference is clear. Here, you're going to have more frequent miscue RS and you can have multiple QRS is missed in a row. So we missed one there, we missed one there and I had one missed right there. If it's the way that it is that I have drawn up on the screen here, this person is probably going to go to sleep, they're probably going to pass out. Once the QRS has come back they'll come to but there could be a temporary time where they just fall asleep or at least fall over because of this, and that's something that can be very, very dangerous if you're doing that while you're driving a car, or whatever walking up a flight of stairs. Without a flight of steps and you go to sleep, you end up getting into a car accident hurting somebody else falling down a flight of steps hitting your head that could potentially kill you. So this is not something that you want to mess with. And so because of it, again, depending on how severe the type one or the type two is, you are probably going to need an artificial pacemaker. Now with a type one. Now, what is the pacemaker we're not going to get into the details of a pacemaker at all. In the end, the pacemaker, in essence is just a couple of wires. Those wires are then fed into the muscle of the heart. And then from those wires as an apparatus that causes current within those wires that's going to excite the heart muscle, allowing the heart muscle to contract the way that it's supposed to. Again, and there's there's different kinds of pacemakers that just paste the ventricles that are pacemakers that face the atria and the ventricles. There are pacemakers that have a defibrillator. And there's all kinds of pacemakers, we're not going to get into it. All I need you to know is that with a second degree type to AV block, you're probably going to need one why? Well, because again, we're missing all these QRS is, you need the QRS as you're not going to pump blood to pacemaker is going to ensure that that occurs. So that's the second degree now let's go to third degree, this one's worse. So third degree. And what this one going to look like, there is no relationship between the P, the QRS t no relationship whatsoever. And so what might be happening here is, is that you might have a p right here. And then a QRS happens. And while the QRS is happening, this is not Repolarization of the atria, by the way, this right here is another P wave. So the SA node is just kind of pacing itself, the Purkinje fibers are kind of pacing themselves, you might have another P wave will be here during the T wave, you might then all of a sudden have a QRS before the P wave. And here's your T wave. And now all of a sudden, now you have a P wave. So that's P, that's Q, that's our s, t, q, r s t, but then we have a P wave that's immediately after that, or it might be during it, I mean, all kinds of crazy stuff are going to occur. So I'm just going to put it right next to it like that. And then there might be a time when there's just nothing. And then all of a sudden, we may have another CQRS that happens again, before the P wave all just just complete disarray of the cardiac conduction system in the heart. And so we have no coordination whatsoever, everybody is just kind of doing their own thing they've gone rogue, you are going to have it's not a probably you are going to have a pacemaker if you have a third degree heart block. So you will require a pacemaker. If not, that's you're done is rock.

08:10

So what you're trying to clarify with a third degree only goes to the second and third or the third

degree, so it doesn't go like PKR like that, as it goes through like it may get distorted.

08:30

It is gonna get distorted. It's happening. There is no coordination at all whatsoever. Just nothing, it just complete. Chaos is what's happening here. So you're going to need a pacemaker. Alright, thank you. You're welcome. So, let's move forward. Now a couple more arrhythmias, a fib. There's a flutter, there's a fib we're going to talk about a fib. So what we have going here if you look at the trace that I'm not going to draw this one we can clearly see QRS. Now when you do see these QRS is they're not spaced like they are with a normal sinus rhythm. Notice over here with these normal sinus rhythm, the QRS is are spaced perfectly apart from each other. That's a typical normal sinus rhythm here, we have changes in spacing, we have a large space here, then we have a really short one here and this one's kind of medium, it's just all over the place. So that's one thing just going to see what they fit the other thing to see what they fit or I should say not see what they fit me I can see P waves were blocking the action potentials from getting into the atria. So on this trace, there is no P wave. The atria because of that are not going to fully contract. And because of that means they're not going to pump blood into the ventricles, which believe it or not, is okay. So let's talk about this often presents with no symptoms. So this is about a fit I'm going to draw the heart here. Now we're going to learn later on today, when we talk about the cardiac cycle, which are all the events that happen in the heart, that during the cardiac cycle, let's put a couple of valves over here. That most of your blood that is delivered from the atria to the ventricles happens without the atria even contracting. So most of the blood most blood is delivered from the atria to the ventricles. Without the atria contracting, how much about 80%? Actually, without the atria contracting, how does it happen changes in pressure, I'm going to show you in detail in just a little bit. And so because of that, because we have enough blood in the ventricles, for the ventricles to pump blood into the pulmonary and systemic circulations, you might not present with any symptoms. Now, you're not running marathons, you're not going to present with symptoms if you lead a fairly sedentary life. And let's face it, most people do. And the other thing too, is a fib, you're going to see these kinds of conditions more so let's say, in older people, or they might just chalk it up to I'm just getting old, or again, they're just not active to begin with, and just aren't going to realize that they have this condition. Now, if you are an active person, you're going to know because you're not going to have as much blood pumping from the ventricles because you don't have all of the blood going to the ventricles that should be going to the ventricles. So it might not present with symptoms. So this is the reason it might not present with symptoms might not present with symptoms. And then the question is Well, why? The answer is that that's the reason why now, doesn't mean it's not dangerous, just because it doesn't present with symptoms. This is dangerous. And this is the reason why it's dangerous. So this could lead to well, let's just let me say something here be a little review for your exam today. So if we're delivering most of the blood, that means we're not delivering all the blood, right? Which means that there's going to be excess blood in the atria just sitting there. And so as a result of that excess blood in atria when the aborted another way, excess blood sits in the atria. So it's not moving. What do we call that? stasis? What we what do we increase the risk of with stasis?

12:52

Blood clots can lead to blood clots in the atria. If somebody has a fib, what do you think they're going to be prescribed?

13:09

Give me some Plavix Xarelto Coumadin, also known as warfarin, you're going to be giving them anticoagulants. Why? Because if you have blood clots in the atria, those stinking blood clots can end up anywhere can end up in the pulmonary circulation, systemic circulation. They're going everywhere. So a very dangerous condition, even though it might not present with symptoms, it could one day that person is walking around with no symptoms, and then all of a sudden, they dropped it without any idea that they even had it. All right. So that's a fit what we just drew their addresses. This right here often presents with no symptoms, boom, there we go. Last but not least, when it comes to arrhythmias, we're going to talk about the worst one that you can possibly have. And that's V fit. This is what's called cardiac arrest. People say cardiac arrest, and, and heart attack and use those two terms interchangeably. They are not the same thing at all, cardiac arrest is the hardest failing because we have a problem with again, the electrical activity within the heart, the conduction system is completely disordered. If you look at that, it looks like I tried to draw an EKG trace with my foot. There's nothing discernible there. We don't see QRS, we don't see P wave LCT we don't see anything. At least we still do have some electrical activity, which means this person can be shocked into a normal sinus rhythm. Now, because the ventricles are not depolarizing clearly we don't see a QRS. Well, the ventricles are not going to be pumping blood at all, this person will die within minutes. If intervention is not done, and that could certainly be paddling them. I think you probably at least seen that on the TV shows when they say clear. They're literally objecting Correct. into that person's body hoping to God that they get a normal sinus where there really something close to it to where the ventricles will start to pump. But let's say that somebody just kind of drops right here because they gone into V fib and we don't have a defibrillator. What can we do for that? Are they done? Chest compressions? What are you doing with chest compressions? By the way, what's happening with chest compressions? Anybody know how, why it works? What are you doing? You are, you're literally squeezing the heart without squeezing the heart, you're pressing on the chest cavity so hard that you increase pressure within the pericardial cavity, that you're squeezing the heart. And it's literally just squirting blood out of the ventricles, and getting blood to where it's supposed to go. Because it'll also help with venous return, and you're not doing it like they do on the TV, you know, doing this, you got to, you got to straddle that person, and you're going to press as hard as you can, you're probably going to break their ribs, there'll be thankful that the ribs are broken, because while they're alive. So that's what you can do until you can get that normal sinus rhythm. And actually, sometimes the CPR just all of a sudden, the heart will start to go into a normal sinus rhythm. So this is the worst of the bunch, you're going to lose consciousness and you're going to die. If this isn't fixed, within probably two or three minutes, the person's done brain damage, at the very least, because they're losing oxygen to the brain. So worst of the bunch right there. And again, I'm going to put these EKG traces on the exam and you are going to tell me what arrhythmia it is. Now, why don't have to know that because we didn't do the normal, we can't do the abnormal. So that won't be on the exam, I will put on pilot those things that you don't have to know as I always do. Now, what are we going to do, we're going to switch gears, we're going to talk about how valves are opened and closed in the heart. Now I know you guys talked about this a little bit already, we're going to talk about it a bit more, because it's going to be very important to the discussions that we have today. So the first files that we're going to open and close are the AV valves tricuspid bicuspid, also known as the what valve bicuspid mitral valve. So opening of, we're just going to put them together AV valves. And then we're going to talk about how they close. So the closing of the AV valves. And we'll just take them in order and draw the heart. There are four chambers right there. We'll do the same on this side. And then we're going to put our valves in the picture. So here are our valves.

17:35

And we're going to open them, we're going to close those valves. Now how are we going to open and

close those valves, it's simply a change in pressure between the two chambers involved in this, that would be the ventricles in the atrium. So how can we pop open those valves, all we need to do is this atrial pressure. So the pressure in the atria, the P is for pressure is greater than ventricular pressure, I'm going to show you when and how that happens. So if you have greater pressure in the atria and the ventricles, you're just going to pop them open. And when you do that, blood is going to flow from the atria to the ventricles. And by the way, at the exact same time, whatever happens on the left side of the heart happens to have has to happen on the right side of the heart, it needs to be coordinated perfectly. Now I know when you guys do blood flow, you go what right atrium, right ventricle, pulmonary trog, pulmonary arteries, pulmonary arterioles, and blah, blah, blah, the lungs, pulmonary vein, left atrium, left ventricle, aorta, systemic circulation, right? If we put a camera on top of a red blood cell, that would be the journey of that one red blood cell. But when it actually comes to blood flow within the heart out of the heart, blood is going from the right atrium to the right ventricle at the exact same time that blood is going from the left atrium to the left ventricle. It is coordinated perfectly. It has to be. Alright. So anyway, that's how we open those valves. And we're going to come back to these pictures a number of times during the lecture. How are we going to close the valves and actually, let me give you a visual that as well. Let's put a nice big piece in here for pressure, a nice little piece in the ventricles for pressure showing that there's a difference in pressure. To close the valves, the complete opposite is going to occur. So we're going to have little piece here, and now we're going to have big pressures within the ventricles. And that's going to and that's going to be my little thing for closing the vowels, little v's, boom, the valves are going to snap shut. Well, how did that happen? Well, the complete opposite ventricular pressure is now greater than atrial pressure. How does that happen? I'm going to show you later in this lecture. Now when those valves snapshot, it is a violent event so violent that you can hear it and that's going to be our first heart sound. The first hard sound is the closing of the AV vowels and they close at the exact same time. So this is the first heart sound, an important point it's abbreviated s One. So now let's go to the semilunar valves, we're going to open and close those, it all has to do with differences in pressure just like these particular valves. So opening of the semilunar valves, that would be pulmonary semilunar, aortic semilunar semilunar valves. Now when I draw the heart and the great arteries, I'm not going to draw them anatomically correct. And I'll tell you why when we get to it, so closing of the semilunar valves. Now, you guys well know being that the great arteries and the great arteries are the aorta in the pulmonary trunk, why do they call them the great arteries because they're the biggest arteries in the body, you guys know that they criss cross, right? Hopefully, you remember that from anatomy, I don't want to criss cross them, because it makes the picture look ugly. That's all on the crappy drawer to begin with, it's going to look even crappier if I actually crisscross those arteries. So I'm just going to draw them straight up, coming out of each of those ventricles. I'll label them now. So there are four chambers, that's going to be the pulmonary trunk. And again, I'm just going to draw it straight up as opposed to going to the right, this is the order. Actually, I'm going to move it over just a little bit.

21:18

So again, pulmonary trunk. And I'm just going to abbreviate it pt. And then the Ord is going to be over here on this side.

21:28

And I'll just write that out. So how are we going to get the semilunar valves to close and let's put the semilunar valves in here. So pulmonary semilunar valve aortic semilunar valve, so we're going to get those to open, because the pressures in the ventricles are going to be high compared to the pressure and this P is not within the atria, the P is within the great arteries themselves. So ventricular pressure,

greater than great artery pressures will open those valves. And when we open those valves, we are going to inject blood from the ventricles and in to the great arteries. To close them, the complete opposite is going to occur. So let's draw the heart. We're going to put those great arteries in this picture as well, again, not anatomically correct. So there's our pulmonary trunk. There's a orda. label that one last time, pulmonary trunk. And now we want to close those valves. And how are we going to close the valves and again, this P is within the great arteries, not within the atria. So the pressures in the great arteries are greater than the pressures in the ventricles. And so great artery pressure exceeds ventricular pressure. And again, I'm going to show you how all this happens. Now, once again, our valves are closing a violent event that makes noise. Second heart sound otherwise known as S two. So there we go. So this is all normal stuff, valves opening valves closing. Now let's talk about some abnormal stuff when it comes to the opening and closing of valves. So we're going to talk about some valve disorders. So what did we just draw? We drew all this. So we just drew. Now what? Let's talk about murmurs. What is a murmur? A murmur is a sound, it is a sign that a valve might not be functioning properly or though there are other types of murmurs as well. In your notes, I have what's called and by the way, the murmur sounds like a swoosh. When it comes to heart sounds. It's lub dub, lub dub, lub dub, both love first start sound dub, second heart sound. Somewhere in between all of that, you might hear this swishing sound what the swishing sound is, is turbulent blood flow. And the turbulent blood flow makes noise. And again, that's what a murmur is. Now, you might have what's called a physiologic or innocent murmur, meaning that there's nothing wrong. Not too uncommon, and kids and athletic heart could have an innocent murmur. Because an athletic heart is a strong heart that pumps blood really hard, meaning that the blood is going to its velocity is going to be high, and it just might make some noise. We're not going to get into the physiologic or innocent murmurs, we want to talk about the pathophysiologic stuff. I know I'm pretty sure that in your notes, I also have septal defect, do I or do I not? Alright, I'm not going to get too much into that, but I'm going to explain it really quick. So you guys know what the septum is. It's it's separates the left and right side, the heart. There's an atrial septum and as a particular step, the defect would be a hole, a hole right here, or a hole right there. And as blood is moving through the heart, some of the blood might go through one of those holes. It creates turbulent blood flow, it creates a noise. So a septal defect might create a murmur, which is once again, just a sound. Alright, but we're gonna concentrate on valves. That's what we're gonna concentrate on. So let's talk about disorders. When it comes to valves. There's two kinds, there's a stenosis, and there's an insufficiency insufficiency can also be said to be a regurgitation, you can use those terms interchangeably. So I'm going to show you the difference between the two. So I'm going to draw a normal valve on the left hand side and an abnormal valve on the right hand side. So I'm going to put normal up here. And it'll be here on this side, I'm going to put stenosis and so my valve looks like a V. And so we are going to have a closed valve. So that's closed. And again, it's a normal valve. And then that's an open valve. Nice wide opening so that we can easily get the blood from point A to point B. And so that's open. With the synoptic vial, they closed just fine. So they're going to look like that closed. But when they open, I'll give you two different scenarios, it could be that they just simply do not open wide enough. So we have a stenosis and a stenosis. stenosis just simply means a narrowing. That's all stenosis means. Or it could open just fine.

26:32

But they're shorts on it. Now what could the shorts be? calcium deposits are a very common thing on valves, especially in older people. Like mama Tucci, I remember to She just got her valves replaced about two months ago, didn't even need open heart surgery, they got some really, really cool techniques. She was out of the hospital, like in a day, and she was walking around completely normal within two weeks, and she got her aortic valve in place, and she's at freakin three years old. So let's talk a little bit more about that later. So that would be an example of a stenosis. And what the

problem here is that because the opening is narrower, don't you think the heart is gonna have to work a little harder to get the blood through it? Yes. And so part must work harder heart works harder. We don't want that. Lama Tucci sorry, it was working harder. Now it doesn't have to because she has a brand new spankin vial. So heart works harder because of this stenosis. And because we have a narrowed opening, as the blood travels through these valves, it creates turbulence, which makes a noise, a murmur, a swishing sound. Alright, now let's go and talk about an insufficiency or regurgitation. So once again, on the left hand side normal. And over here, I'm just going to abbreviate insufficiency, regurgitation, you can use those terms interchangeably. Here, we're going to start the valves open to begin with. So we have a nice, beautiful open valve. And then the valve is going to close. So now we're closed, we're going to have an open valve over here, everything is good. The problem with an insufficiency or regurgitation is it does not close properly. So the valve kind of becomes floppy. And so I'm going to draw that just by making the bottom of it just looks like crap. And so it's not going to close properly. And so that's close. What's going to happen because of that, we'll get a little back pressure and blood will start to go in the wrong direction. bloods not supposed to go in that direction is supposed to go in the other direction. And then it's going to run into blood that's coming in the right direction. Now the heart's got to work harder once again. So heart works harder here as well. Now, when it comes to Valve disorders, the valve that we see the most disorders with Dizzy aortic valve. Does that make sense? When we talk about the aorta, once I heard of you talking about the left side were the pressures of the heart higher. The left side tell me why. Like the ventricle the left ventricle has to pump blood were freaking everywhere, up to your head all the way down to your feet. So the ventricles have to correct the left Medical Institute trek really freaking hard. Where's the blood go from the right ventricle. The lungs were the lungs, sitting right next to the heart. The pressures in or the pressure in the left ventricle is 10 times higher than it is in the right ventricle. So that aortic valve gets pounded hard during a lifetime. And because of that, that is and that's in your notes. I hope it is because I'm talking about Yeah, if you were to fail disorders of the most Got it. That's the valve that Mama Ritchie got replaced, it was a aortic valve. Okay, that calcium deposits on it, it was a little worn out. She's good as new now. Yes.

30:20

He just doesn't close properly. That's all it's like all floppy, like the BIOS should have some integrity to it, boom. And if it doesn't, it just, it doesn't close properly, that's all. And I get one therapy like. That's what this is talking about right here. This is indicating that the blood is flowing in the wrong direction, you get a little back pressure, and it just kind of gets pulled in the wrong direction. And then it hits blood coming in the right direction. And now the heart is pumping blood against blood that's coming in the wrong direction, making it work hard. We're coming back to this. Especially because we have this over here that I'm not going to talk about right now. I'm going to talk about that when we go through the cardiac cycle, you're going to find out why. If you know a systole while you know, I'm just going to tell you what systole a systole is when the heart contracts. diastole is when the hearts relaxing. That's what systolic means and diastolic mean. We have valve disorders during that that are indicative during systole. We have valve disorders that are indicative during diastole. I'm going to show you, Okay, we're coming back to this four times. As I go through the cardiac cycle you'll see now, when it comes to hearing a murmur, how do we hear it? Well, we're going to hear what the stethoscope because we're going to be what's called all skull trading the heart when you asked rotate it means listen, it's a fancy word for Listen, that's all. And you will listen to many things you will listen to bowel sounds, you will listen to chest sounds you will listen to heart sounds, you'll listen to all kinds of things with the stethoscope, we're going to concentrate on all skull tating the heart. In these two pictures, we see these circles. And what these circles are indicative of is where you put the stethoscope to best hear a specific bow. So this yellow circle that we see over here is where you would put the stethoscope which is at about the second intercostal space just to the right of midline is

the best place to hear what's going on with the aortic valve on the other side have been lined at the second intercostal space is the best place to hear the pulmonary valve. Now, let's remember the order comes off of the why the left ventricle right. And the pulmonary trunk comes off of the right ventricle correct. But we have this criss cross we have this on the right side of the heart to hear the aortic valve. And this on the left side of the heart to hear the pulmonary valve but again, the great arteries crisscross right you guys know that. So this is not a typo. This is the way that it's supposed to be the aortic valve is to the right of the pulmonary valve, the pulmonary valve is to the left of the aortic valve because the great arteries crisscross. I didn't draw it like that in my drawings because it would look ugly. And I'm all about pretty drawings, of course. So what about the green circle tricuspid valve just to the left of midline at about the 52 causal space and then around the mid the nipple line at about that same place, we have the mitral valve the bicuspid valve, that's the best place to put it. So you move that stethoscope and you're listening, maybe all of a sudden, maybe you put the stethoscope right here at the yellow circle. And yet you think you might hear some bushings like is that a murmur. And so what you're going to start to do is is that you're going to start you're going to take the stethoscope that was here, and you think you might hear the murmur. And then all of a sudden you move it over here. And it's still kind of sounds faint. Now you take that stethoscope and you stick it where the green circle is. And now it sounds much more clear. And so what that's telling you is, especially if you put it on the orange one over here, and it doesn't sound that clear anymore, wherever you hear it the loudest in the clearest is going to be indicative of where the vowel disorder is present. So that's why you're going to just move it all around until you hear again, most people don't have murmurs. But if they do in order to pinpoint what valve is the problem, you move that stethoscope around you do it normally anyway. On the exam, there's going to be a bare chested man, there's going to be circles on this person's chest. I'm going to ask you, where would you best put the stethoscope to hear in a I don't know a pulmonary semi semilunar, a pulmonary semilunar insufficiency, or stenosis or whatever. I'm just going to name some bell disorder with a specific valve. You just pick the right circle. It's money. It's vomit stuff on the exam. All right. Are we good? Yes, so there's going to be two pictures on this exam. This one and then we have going to have an AKA key arrhythmias, EKG strips.

35:11

Now what, let's get to the cardiac cycle. Now, what is a cardiac cycle the events in the heart with one heartbeat, a cardiac cycle takes about one second. So what we're going to spend about 40 minutes on, takes one second. It's the heart contracting and relaxing. And what we're going to do, as we talked about the cardiac cycle, is we're going to put everything together now. We're gonna go back to the EKG, opening and closing valves, murmurs, blood flow, we're putting everything together now, in the story, contraction, relaxation of the heart, the whole nine yards. Now, systole, I define it now, specifically systole is contraction. That's what systole is. diastole is relaxation. Your heart is relaxed twice as long as it is contracted during a cardiac cycle. So your heart is relaxed more than its contracted 1/3 of the time is spent systole, two thirds of the time is spent diastole under normal conditions. And that's going to be an important point in the next chapter. When we talk about something called mean arterial pressure. I'm going to remind you of this. There's atrial systole and ventricular systole, atrial systole, the atrial contraction, ventricular systole, the ventricles are contracting, atrial diastole, the atria relaxing ventricular diastole, the ventricles are relaxing. And then I have two lines here. If systole is used without specifying a chamber, ventricular systole is applied. diastole is used without specifying a chamber ventricular diastole is implied. What does that mean? It means this. If I say systole, you assume I'm talking about the ventricles. That's what you assumed because I did not specify a chamber, all I said was systole. If I say just the word diastole, you assume it's ventricular diastole. Because I did not specify a chamber. The only time I'm talking about atrial diastole, or atrial systole, is when I say atrial diastole, atrial systole. Otherwise, it's ventricular systole

or diastole. All right, are we straight with that? Yes. Now, before we take a break, these are the five periods of the cardiac cycle, and I'm going to write them down. So cardiac cycle, again, this is just one heartbeat. It's going to take about one second. And we have these five different periods that are going to occur, we have ISO volume, and we're just going to take these one at a time contraction. We have period of ejection. And then we have our three left over here we have isovolumic. Again, now it's relaxation. Then we have passive ventricular filling and active ventricular filling. Two of these are systole. And three of these are diastole. The first two are systole. The last three are diastole. And I'll put that down here as well. So I'll just do this. Those are systolic phases. And those are diastolic phases. And what we're going to do is talk about each and every one of those in order and go through lots of detail. And again, EKG opening and closing valves, blood flow, contraction related, just all of it is going to come all together after we take a break. So let's take a break. Alright, let's finish this cardiac cycle discussion. Well, it started and finishes. So what we're going to do is we're just going to take these one at a time until we're done with them. We're going to start with isovolumic contraction. And I'm going to put that right here at the top. And I'm going to remind you that this is systole. Now I just said systole. What's contracting tell me the ventricles, again. When I don't specify a chamber, you assume ventricles. Let's draw the heart and we're going to have our four chambers here. Now, what is ISO mean? Means the same, ISO means same. Let's keep that in mind. Now this is Sicily that means the vector They're contracting. What do we need to do to contract the ventricles? What do we need to do the muscle first?

40:07

Need to excited, right? And when you excite cell, what's the membrane potential doing is becoming more positive. And what's that call depolarization. What's depolarization of the ventricles called an EKG? The QRS complex depolarization of the ventricles. So again, we're putting everything together now. So in order for the cardiac cycle to be initiated, we have to depolarize the ventricles so initiated by the QRS complex, which is depolarization of the ventricles. Now, if you depolarize, the ventricles, ventricles are going to contract. So from this, I'm going to draw an arrow. And I'm going to say that the ventricles contract because well, they do. Because they were just excited. How do we know that? Because the QRS tells us now if the ventricles contract, that means the chambers are getting squeezed. Like if I took a balloon and I squeezed it, my hands are the cardiac muscle, the balloon is the chamber, what's going to happen to the pressure inside the balloon? If I squeeze it? It's going to go up, what do you think's gonna happen to the pressure in the ventricles when I when we squeeze, it's going to go up. So as the ventricles contract, we are going to get an increase in ventricular pressure, such that ventricular pressure is going to exceed atrial pressure, tell me what happens when ventricular pressure exceeds atrial pressure, tell me I'll give you a hint, tell me, we're going to close the AV valves, we're going to get our first start sound. That's why we started isovolumic contraction, by the way, because that's what happened. That's we got our first heart sound, our lob, so let's go back to the picture. So as a result of this, AV valves, close S one. So let's put our AV valves over here. AV valve AV valve, we're going to pop shut, I'm not going to put the pressures in here because I already did it in another picture. All right. Now, I also have to put in this picture the great arteries, because they are going to play a part in this particular story. We're going to put our valves over here. And this is something you just have to take my word for it. You're going to see later on in this discussion tonight. The AV valves, I'm sorry, the semilunar valves were already closed. So the happenings during isovolumic contraction do not cause the closing of the semilunar valves, they were already closed during another phase. So I'm going to write that down semilunar valves were already closed before this phase occurred. And so what does that mean? It means all the valves are closed. Now, if all of the valves are closed, are we going to be able to get any blood into the ventricles? Yes or No? If the AV valves are closed, can you get blood into the ventricles? No. If the semilunar valves are closed, can you get any blood out of the ventricles? No, what does ISO mean? Same same volume

of blood in the ventricles. Why? Because all the valves are closed. The ventricles are contracting and yet the volume of blood in the ventricles remains the same because we've closed all of the valves. Now, the AV valves just closed. What valvular disorder? Could we hear at this particular time via our murmur? If that's the case, if there is one, the valves just closed, AV valves just closed and they didn't close properly. What's that called when a valve doesn't close properly? And insufficiency or regurgitation? Right, improper closing of valves. I added that by the way during the break and proper closing. So I'm going to add here, a murmur due to an AV insufficiency, or regurgitation, could be hurt, of course, if it's present. Now what we're going to do is we're going to go back on by the way, what's this? What's this called again? isovolumic contraction is what part of the cardiac cycle systole AV insufficiency or regurgitation. AV regurgitation is right there. It is a systolic murmur. What does it mean? It means a murmur that happens during systole that's why I saved this until I went through the cardiac cycle so that you understand it fully Now, AV regurgitation, is it tricuspid or bicuspid?

45:05

How you gonna know, you also take the heart. Now here's the other thing too. I didn't mention it. That's not the way that you're going to diagnose that murmur for the AVR, or the valve EULA disorder by by Oskol tating the heart, that's going to tell you that there's a murmur. But now you have to image the heart. That's the way you're going to actually make the diagnosis. But the murmur is going to be a sign that you have valvular disorder. Now you have to do an echo, for example, to see whether or not it is the tricuspid or is it the bicuspid? All right. So again, AB regurgitation and that's insufficiency it's the same thing that is a systolic murmur Are we good with this? Yes. I'm sorry, you gonna do what

45:58

I was gonna copy the murmur due to the AV, I forgot to write that down.

46:07

You guys know I put these pictures on Pilar. Right? Okay. So, what we have here up on the screen is everything that we have to know for us the volume and contraction. So it's all these words, right? Here, we just drew that. Now let's go on to the period of projection. 3d projection is what we're going to spend most of our time on, because this is where most of the action is occurring as far as I'm concerned. So we're going to put at the top here period of ejection, I'm going to remind you that this is also systole.

46:47

Let's draw the heart. Now, it systole. So we are still contracting. The AV valves, as we know, are closed from the last period. There's our pulmonary trunk, there's our aorta, I'm not going to label them. I'm going to put over here, ventricles continue to contract, they're not done contracting. And if they're still contracting, while certainly that means the pressure is going to go up even more. So let's do this. I'm going to put two arrows now when it comes to ventricular pressure. And we're going to get to the point to where now ventricular pressure is now going to exceed great artery pressure. Tell me what happens when ventricular pressure exceeds great artery pressure, tell me what happens. I

heard it good. Ventricular pressure exceeding great artery pressure opens the semilunar valves, we already learned it. And so let's open those semilunar valves. So let's actually put them in the picture. And if those valves open, what's going to happen, we're going to eject blood period of ejection. And so we are going to Egypt a certain fraction of blood into these great arteries. And that has a special name, it's called stroke volume, I'm going to abbreviate it SV. Now the amount of blood that was in the ventricles prior to this is called end diastolic volume II d v. Now that's gonna make more sense when we actually get to diastole. And diastolic volume is the volume of blood at the end of diastole. We haven't even started diastole yet, there is an average value for that for an average sized adult healthy male, which are values used in cardiovascular physiology for a female on average, it will be about 20% less because well guys are just on average bigger than females. These values, though, can vary widely, depending on age, how tall you are, what kind of shape you're all kinds of differences when it comes to these values, as we will see in the next chapter. Now stroke volume, on average, is about 70 milliliters. This can vary widely, can be upwards of well over 100. I'll tell you why in the next chapter. So clearly, oh, something else that I have to do here, actually, I'm not going to put I'm going to write PDB differently here, I have to do something else. I'm still going to put those average values though in the picture. I'm just going to put it above EDB. So we started out with an end diastolic volume. And as we got rid of the stroke volume, the volume that's going to be left in the ventricles is called end systolic volume, the volume at the end of it systole. So I'm going to do that the both sides, whatever's happening on the right side of the heart is what's happening on the left side of the heart. I'm going to put that average value for end diastolic volume above and diastolic volume, which is around 120 milliliters. So why don't you go ahead and tell me what end systolic volume is? Tell me, what is it 50. We started out with 120, we got rid of 70, what are we left with 50. And so that is an average end systolic volume. And so you're going to have to do some math on this exam. I'm going to give you the equations that you're going to need, we're going to write three equations, they're going to be the exact same equation, they're just going to be rearranged. So n systolic volume, for example, equals how much you had to begin with and diastolic volume minus how much you got rid of which is systolic volume, we can also write it as the stroke volume, the volume that you ejected is equal to how much you had to begin with minus how much you were left within the ventricles, we can also write this as end diastolic volume equals stroke volume, or N systolic volume, plus stroke volume, those equations are exactly the same. And on the exam, you're going to have to be able to do on this exam coming not going tonight.

51:21

But the third exam, you got to do man, add, subtract, multiply, divide, which you learned how to do in a second. So if you're smarter than a second grader, you can do the math on this exam. Now, am I just going to say, end diastolic volume? Is this and stroke volume? Is that tell me what ends to solid volume? Do you think I'm going to do something like that? Of course, I'm not. That's too easy, it's not a math class, you're going to have to critically think you're gonna have to determine what stroke volume is, or whatever it is, and this is, and this is, I'll give you two variables, kinda. And then you have to give me what the third variable is, when I say kinda, you'll see. All right, so I'm going to make you think still, of course I am. Now before we move forward, you know what we are going to we're going to, we're going to get to more math here. Oh, no, we're not. So the great artery pressure is exceeded by ventricular pressure. And what that's going to do is it's going to open the semilunar valves, of course. That's how we get the blood from the ventricles into the great arteries. Of course, the AV valves are still closed, nothing's happening to them. So we're all good here. Now we just open the semilunar valves. If they don't open wide enough, what do we have? A stenosis, so we could at this particular point in time hear a murmur due to a semilunar stenosis. Once again, this is systole systole semilunar stenosis, let's go back to the notes, systole. semilunar stenosis, so semilunar. Psygnosis, is a systolic murmur, which of the two semilunar valves is the problem Oskol, take the

heart, and then try to figure it out. And then of course, then image the heart, do an echo, and then you're gonna know for sure. So that systole now we're not done with systole. Because we have to talk about ejection fraction. So some more math now. So let's go back to this. Clearly, not all of the blood is being ejected from the ventricle into the great arteries, it's just a fraction of the blood, we started with 120. And on average, we're going to get 70 injected into the great artery. So that's not 100% That is a fraction. That's what we call the ejection fraction. Now ejection fraction is typically expressed as a percentage. So let's go through some more math here. So I'm going to abbreviate ejection fraction as E, F. And so here's the equation for ejection fraction. It's how much blood you injected in the numerator divided by how much you had there to begin with, which is in diastolic volume. And again, we don't really know what end diastolic volume is yet, but we're going to shortly. Again, it's expressed as a percentage, so we have to multiply by 100. Now, anything above 50% is considered normal. I know in the notes, I think I have 55 to 70%, something like that. We don't have to get quite that technical. So and ejection fraction and E f greater than 50%. is considered normal, which means what how low could this stroke volume be to be considered normal? Defarge ETV is 120. what's half of 120 60? Right So it can be as low as 60 and still be considered normal. If we do the math with 70, we're looking at about 58%, give or take somewhere in there, which is certainly above 50%. Now, ejection fraction is a very, very powerful, powerful value. ejection fraction is one indicator is an indicator of how healthy the heart is. Again, I said anything above 50% is considered normal, let's hit the parts not healthy. So let's go with an unhealthy heart. Somebody has heart failure, ie, heart failure. Now the reason the heart is failing is because the muscle is weak. Now why would the muscle be weak could be lifestyle, could that person had a heart attack, there could be all different reasons why we weaken the muscle like some of it might be dead because of the heart attack. You know, we don't have as much muscle to contract and pump blood. So unhealthy heart, ie we have weak heart muscle.

56:16

When it comes to a stroke volume, in order to have a normal stroke volume, certainly the ventricular muscle has to contract hard enough to eject that volume into the great arteries, right? If the heart muscle is weak, do you think that our stroke volume is going to be high or low? It's going to go down. So let's just do some simple math. So again, ejection fraction stroke volume over EDP, so ejection fraction, or EDB, we're just going to say 120. Instead of 70. I don't know, how about 30. times 100. We'll do the math. Anybody really quick. Are You Smarter Than a second grader, actually, this might be third grade math,

57:04

I take it back. We're doing fractions now. 25%. Right. So 1/4 We have an ejection fraction of 25%. That shows a pretty stinking, weak heart.

57:20

When it comes to ejection fraction, we can track the health of the heart over time. So maybe it started out a 40% a few years ago. And then over time, maybe you got down to 30, and then 25. And then 20 just keeps on going down. That'll help us track how it's progressing. But it also helps us determine whether or not the treatment is good. So let's say we caught it at 30%. And you start treatment, and there's some drugs that you can get that I'll mention in the next chapter. And let's say

over time, it stays at 30%. Treatments working, let's say you do the treatment, and it just keeps on falling, that treatment sucks. Got to do something else. So bottom line is is a very, very powerful, powerful tool that can be used to determine the health of the heart and track the progression of the disease itself. And sometimes when it comes to the heart failing, the way that it is, sometimes it happens so slowly over time that the person doesn't even realize it, or might chalk it up to just getting old. Most people with congestive heart failure are older. And so they might walk up a flight of steps. And they're pretty tired said Well, I'm just getting old. That might be the case. But it could also be a case to where well the heart is becoming unhealthy. And then by the time they get evaluated because boy, I'm really not feeling that great. Their ejection fraction is 20% the first time that they ever get evaluated. Like popper Tucci Popper, he was having some issues, he was finally convinced to go to the cardiologist, he had an ejection fraction of 20%. And that was the first time he was ever evaluated. We know that he has symptoms he probably doesn't say anything, but at the same time it happens so slowly over time. Now why was popular to choose heart failing, he didn't have a heart attack. And I think I've made mention of this a number of times over the last two semesters. Poverty, she didn't live a healthy lifestyle. He was he wasn't fat, and poverty. She was skinny as hell, like five, eight above 25. So he thought because he was thinking he was healthy. I said, That's not the way it works, pop, ate like crap, didn't exercise, smoked, and over time, develop harfield. Now what actually killed him was lung cancer because of all the smoking he did over his lifetime. But by the time he was evaluated, he had ejection fraction of about 20%. His meds actually got it up a little bit. I think they got to between 25 and 30%. Not that that's fantastic. But there are ways to treat this. You're not going to stop the progression but you can certainly slow the progression down. So no one Egytian fraction is and know what a powerful tool it is and no values that are normal and values that are abnormal and it's the low values that indicate Again, heart failure. So that is that for the period of objection. Now what at least I think I've covered everything with a period of objection, did we get the Yep, we got the murmur due to a semilunar Psygnosis. Okay. Now what? Now the diastolic periods. And so we're going to start with isovolumic, kinder, I'm sorry, relaxation. So let's do that now. So isovolumic relaxation, I am going to remind you that this is diastole. So let's draw the heart. All in. There's our word ISO again. So we know what's going to happen here. We're going to have the same volume of blood in the ventricles during this entire phase this entire period. So here's the heart. There's our four chambers, great arteries. Here are the AV valves, the AV valves are still closed. When did the AV valves closed, they closed during isovolumic contraction, they're still closed, they were closed during isovolumic contraction period of ejection, and they're still closed during isovolumic. Relaxation. Let's put the semilunar valves in the picture as well. Now, the ventricles are relaxed now. So I'm going to write that down. It's diastole. Of course, the ventricles are relaxed, because I said diastole. Right. And so I am you imply ventricles. So ventricles are relaxed. Now what else do we need to know about the ventricles So and there's less blood in the ventricles now? Why? Well, because we got rid of a stroke volume, we are at end systolic volume. Now, this period of ejection was the last systolic period. And so because we're at the end of it, we've actually reached in systolic volume. So I'm going to put ESV here, and I'm going to remind you, that is that is around 50 milliliters, again, on average, not for everybody. And again, I'm going to show you big differences in certain kinds of people.

1:02:22

So we're in systolic volume, so we have much less blood in the ventricles than we did during isovolumic contraction, for example. And actually, let me put here, and diastolic volume, because we're going to visit it very, very quickly. So during isovolumic contraction, we're still at end diastolic volume, and I'm actually going to put here the average value of 120. Alright, so let's get back to what we need to talk about. And that is diastole. So we're in systolic volume, so there's less blood in the heart. So we have a decrease in ventricular blood volume. Yeah, by over half, most times. So the

ventricles are relaxed, and there's less blood there. What do you think's gonna happen to the pressure gonna freakin plummet? It's gonna go way down. So we're going to have a decrease in ventricular pressure at the same time, what do you think the pressures in the great arteries are? Do you think they went up or down? At this time right here, they went up, we just smacked them with 70 milliliters of blood, we hit their walls, heart. So the pressure in the great arteries went up, the ventricular pressure went down. So I'll actually put that over here. Great artery pressure. I don't have to put that in parentheses. This is a major point, great artery pressure increases. So we have an increase in great artery pressure. Because of the stroke volume that was introduced into that, there's more, there's more blood there now. So now what, well, great artery pressure. Oops, let's do it this way. Great artery pressure is bigger than ventricular pressure. What happens when great artery pressure is greater than ventricular pressure? Tell me. Beautiful. We just closed the semilunar valves. We have our we now have our second heart SAP. So let's close those valves. closed, closed. And so ventricular pressure oops, put it over here like this. So we are going to close semilunar valves let me do the same right at the same way I did for the other one just to be consistent. Open saw just go Close close semilunar valves and the AV valves were already closed, right at valves already closed. They closed during isovolumic contraction. And so all valves are closed whenever you see isovolumic you know all the valves are closed isovolumic contraction, AV valves closed semilunar valves were already closed isovolumic relaxation, maybe valves were already closed semilunar valves are going to close, all valves are closed and if all the valves are closed, you can't get any blood into the ventricles and you can't get any bloody out. I saw volume it now the semilunar valves just closed. What if they didn't close properly? What can we have? And I say semilunar insufficiency or regurgitation, so a murmur due to a semilunar insufficiency, regurgitation. Let's go back to the notes diastole semilunar insufficiency, or regurgitation? diastole semilunar. regurgitation, or insufficiency is a diastolic murmur murmur happening during diastole. What else do we need to know here? Actually, I think we covered everything when it comes to isovolumic relaxation, second heart sound that I put that there I don't think I did. I didn't close semilunar valves. There's es two second heart sound. There you go. Second diastolic phase, passive ventricular filling. So passive ventricular filling, the name tells you exactly what's going to happen, we are going to fill the ventricles with blood. This is also diastole. And of course, it's ventricular diastole.

1:07:10

Let us draw our heart. So there's the heart, we'll put the great arteries in the picture as well, of course, we'll put those valves that are closed semilunar valves just close. So let's close them. Now, ventricular pressure is still low. We haven't added any blood in the ventricles or relaxing. But now what's gonna start to happen? Actually, it's already started to happen before this, but this is the only time I'm gonna mention it. Is this so those arrows are indicating we're getting blood into the atria, what that call was that call? What kind of return? That's what the V venous return. As we add blood to the atria, what do you think's going to happen to the pressure in the atrium? Go up. So with venous return and I'll just be thorough with venous return to the atria. There's going to be an increase in atrial pressure, where the pressure in the ventricles doesn't change. So as a result of this atrial pressure, will exceed ventricular pressure. Tell me what happens when atrial pressure exceeds ventricular pressure, please. I think I just heard it. We just opened the AV valves. atrial pressure exceeds ventricular pressure, AV valves open, of course they're going to open. This is ventricular filling, how else are you going to get the ventricles to fill the blood? If you don't open up those valves. And we're going to fill it with a lot of blood right? Did I already tell you what they did? That most blood is delivered from the atria to the ventricles without the atria contracting. That's why they call this passive ventricular filling. The atria are relaxed. During passive ventricular filling. Atria are relaxed. How much of the blood goes from the atria to the ventricles? About 80%? Not 80% of the total but 80% of what's going to be delivered. So 80% of the total, both sides and that's enough of the

ventricles to pump to the pulmonary and systemic circulation to where if somebody is not very active, are not going to know what the hell is happening if we are in a fifth. Now, so let me put this here, AV valves open. So the AV valves just open. What if they open wide enough? We have a stenosis. So we could have a murmur. Due to a oops, and AV stenosis, so AV stenosis diastole, I'm just going to prove it to you. AB stenosis diastole. So diastolic murmur, it's sitting right there. Alright. Going back to our picture, what else do we have to know here? Nothing, I think we covered it all. Now, the last diastolic phase, the last phase of the cardiac cycle is now active ventricular filling. And so I think you already know where this is going. This is also diastole, I will remind you, we will draw the heart. So there is our hearts for chambers, great arteries, those valves are closed, still. Oh, and by the way, there is something I need to add over here, we have to make the heart just a teeny bit bigger, because there's something that we have to add. Here, I'm gonna make the bottom of the heart just a little bit bigger. And what we need to add here is is that we are going from n systolic volume to end diastolic volume, that's what we have to add. So I'm just going to make the heart just a little bit broader on the on the bottom here, the ventricle a little bit broader. Like so. So we are going from and systolic volume and systolic volume towards and diastolic volume, because we're adding blood. And we are going to hit and diastolic volume during active ventricular filling, because that is the end of diastole. And so with active ventricular filling, those valves are closed, these valves are still open. Now something else I want to do as a visual, I'm going to make these arrows bigger,

1:12:13

showing that this is where we're going to get most of the feeling of the ventricles. And then the arrows that I put on the next one that is ventricular, or I'm sorry, active ventricular feeling, I'm going to make the arrows smaller. So we're going to get our last 20%. And again, it's not 20% of the total of all the blood in the event of the atria. It's just 20% of what we're going to be delivering. So our last 20% of blood during active ventricular filling in so we are going to actually hit ETV because we are going to be at the end of diastole and again to remind you that is 120 milliliters of blood. Now this is active ventricular filling. The atria are contracting. Well Dr. Tucci This is diastole. What the hell are you talking about? It's diastole when I'm What am I talking about? The ventricles, that's what I'm talking about. The ventricles are relaxed. The ATR certainly contracting. That's not what we're talking about when we talk about all these five periods of the cardiac cycle. Am I missing anything? Let me make sure I'm not missing anything. I'm not, we're done. Good luck on that exam tonight. Those of you that still have to take it. I will see you next Tuesday, or probably before because I have your exams for you and you're going to come during my office hours and look at the exam. Good luck, guys.

HeartCirc PM 2-24-22

Fri, 3/11 9:14AM 1:13:52

SUMMARY KEYWORDS

blood flow, blood vessels, stroke volume, contractility, blood, heart, blood pressure, cardiac output, decrease, increase, pressure, heart rate, harder, ventricle, preload, neuron, viscosity, draw, dilate, bigger

---

00:01

Here we go, folks. So in the last lecture, we talked about how the heart does its thing. Now we are going to talk about how the heart is regulated. And when we talk about the regulation of the heart, it's going to revolve around cardiac output. So let us define what Cardiac output is. So I'm going to draw heart. And during the period of ejection, which we know is when the ventricles are contracting enough to eject the stroke volume into the great arteries. Cardiac output is going to be how much blood is injected over a minute's time, whereas stroke volume is the amount of blood that is injected with each contraction. And so what I'm going to do here is I'm going to give an average value for cardiac output both out of the left ventricle in the right ventricle, and it is approximately five liters of blood per minute. That's per ventricle, by the way.

01:10

Now, how did I arrive at that value, what those values are for an average size adult healthy male. And so again, that's cardiac output, I will put above it cardiac output. Cardiac output is going to be abbreviated see, oh, I know I've used the abbreviation CO for carbon monoxide in the past, but that's not what this is going to be. So cardiac output co equals heart rate, how many times the heart contracts in a minute's time? How many times beats per minute? Times stroke volume. And so now let's do some math. average heart rate somewhere around 70 beats per minute. So 70 beats every minute. And an average stroke volume, can you just give me that? Because you know what it is? What's What, what's the value? 70. Right, when do we learn that we learned in the last lecture when we were doing creative projection, we know the stroke from on average is around 70 milliliters, so I'm going to use that value. So that is 70 milliliters of blood per contraction per beat. Simple math, those cross out, what are we left with milliliters of blood per minute, what 70 times 7070 times 70 is 4900 milliliters of blood per minute, which equals about five liters of blood is pumped per minute by each ventricle, not the whole heart, each ventricle. So think about that for a second, every single minute, your heart, take five, two liter bottles and fill them with blood. That's what your heart's doing every stinking minute, that's what it's pumping every minute, two and a half liters, or I'm sorry, five liters, every single minute out of each ventricle. It's a tremendous amount of work that your heart has to do every single day of your life now. And what we just did is all this stuff. One thing I want to add here is your heart is most efficient. I've mentioned this before, when heart rate is low and stroke volume is high. So I'm going to repeat that. If your heart doesn't have to contract as many times it doesn't have to expend as much energy, even though it beats harder when it is more efficient. So an efficient heart, we're going to talk about inefficient hearts to an efficient heart, what do we want, we want a

high stroke volume and a low heart rate I'll do I'll do it in the same order that I have it in the equation, we want a low heart rate and a high stroke volume. So I'll give you an example. So cardiac output. Again, heart rate times stroke, fine. I'm just going to use me as an example my heart rate at rest is owned by the way. These are resting values. This is you guys sitting in your seats right now. If you exercise that can be as high as 35 liters per minute, per ventricle. 70 Freaking liters of blood per minute coming out of the heart every single minute. I'm talking vigorous, vigorous, vigorous, vigorous exercise, but that's how high you can get. My heart rate at rest is 50. So 50 beats per minute.

04:42

Give or take. Now, what's every stroke line? 70 Do you think my cardiac outputs 3500? It's not because my stroke volume is not 70 What do you think my stroke volume is about? It's about 100 So 100 milliliters of blood for beat

05:08

five liters per minute, give or take. How do I get my heart rate to slow? I work out? That's all I got to do. I'm not doing anything special. There's nothing special about me. I just work out there probably people in this room with heart rates that low, maybe even lower. There are people in the world that have heart rates, Lance Armstrong, do you know Lance? at his peak, his heart rate? Yes, it was it was at rest 29 He had a stroke volume of about 150 to 180. That was his stroke volume. And I'm sure because he still works out, it's probably not much different than that right now. An incredibly efficient heart, his heart is going to last a really, really, really long time. So I'm going to wear out as quickly. And again, we're going to talk about inefficient hearts. And all you got to do is work out especially you got to do your cardio, that's what you got to do. All right. So anyway, we'll come back to this point a couple of times, before we move forward is a couple of terms that we need to be very familiar with inotropic chronotropic ionotropic, something that affects heart contractility, chronotropic, something that affects heart rate. So both of these effects are going to obviously influence your cardiac output because those are the two variables that determine cardiac output. But we have to add some stuff. And we're going to add towards positive and negative. So a positive Crono trope. Now I'm going to do is drill first, I know trope. And I'm going to mention what these are going to mention what they are in this lecture, I'm going to mention what they are in the next lecture. There are drugs that are positive and negative binary tropes and Crono tropes, there are conditions that cause positive and negative effects when it comes to these two terms. So a positive I know trope is something and it could be many some things it can be a drug, it can be things that we'll talk about is something that causes an increase in heart contractility, it makes the Heart Contract harder. That's all that saying. So heart contractility. A negative CRI inotrope is the complete opposite. That's something that's going to cause the heart to contract less hard or decreases contractility.

07:40

So that causes a decrease in heart contractility. And then coronal trope has to do with heart rate.

07:51

And so the words positive and negative are going to be the same exact thing increasing and decreasing. So a positive Crono trope, or a positive chronotropic effect is something that causes an

decreasing. So a positive Chrono trope, or a positive chronotropic effect is something that causes an increase in heart rate. And then, of course, negative is the complete opposite of that. And again, these are terms that I'm going to use a number of times in the lecture today and the lecture I'm going to give on the Tuesday when we come back from our nice spring break. As we shoveled snow, something that causes a decrease in heart rate. Now something else I'm going to add to this. And it's about I know trope, most of the time, when you increase the contractility of the heart, what that is going to probably lead to and I actually put this in parentheses, because there's going to be an exception I'm going to show you today. But most times, if you increase contractility, you'll increase stroke volume, of course you will, the heart contracts harder, it's going to inject more blood, of course. And then if you decrease contractility, you will decrease stroke volume. But there are exceptions to this. I'm going to give you an exception today. So terms that we have to be familiar with. So please, let's be familiar with them. Now what? Well, let's talk about what regulates a heart. Yes, sir. Well, he doesn't have increased output lancearmstrong, even though he has a heart rate of about 30 and a stroke line between 151 at his cardiac output still about between five and 5.5 liters. It's no different than anybody else. changes but it's a change. Because like, is this larger? Yeah. chambers are a little bit bigger. Yeah, that's exactly what it is. So he has an enlarged heart, but not a bad, enlarged heart. My heart's a little enlarged to just not like him because he's, you know, he's a freak. Just just because he's a You know, the way he trains and so yeah, his heart his chambers are a little bit bigger. Absolutely they are, the muscle is bigger is contracting harder. But yeah, that's exactly what it is I'm sorry. No, not much, it's still going to be within a normal range. So the normal range is anything above 50 up to about 70 His ejection is probably a little on the higher side in normal, because it's hard to contracting so hard, but it's not going to be you know, 90% of the of the volume of the of the ventricles questions yesterday

10:39

you're saying they're like, What, like blood addiction, you know, from the heart, like, you know, when you're talking about working out like doing cardio, one that like, one day like, you were doing, like cardio versus like the heart attack. And even though you know, your heart is like,

11:00

you will increase your cardiac output when you lift weights, how you lift it, but if you have a heavy set, you'll increase your cardiac output while you're lifting,

11:07

but does not like is that very similar to cardio? That's a completely different?

11:11

No, it's going to be different cardio is going to be bigger. Yeah, you're spending more energy? Yeah, yeah. Yeah. Yeah. Yes. You can't, you can actually work out too much. And your heart can get enlarged in a bad way. And the bad way is, is when the muscle gets too thick, and now the chamber gets too small, where it can't accommodate as much blood that's when it's in a bad way. His still has enough elasticity and compliance to where it can stretch enough to where you're not impinging on the size of the ventricle, where the blood is actually. So that's, that's what I mean, by a good way in a

bad way, without getting into a lot of detail. All right. So now let's talk about how we regulate cardiac output when we regulate cardiac output. We are influencing stroke, flying, and heart rate. And so we're going to have intrinsic versus extrinsic regulation. So interest aggressively, like regulation means something inherent within the heart is going to affect cardiac output. And so let's talk about them in there's three things. First one is how healthy is your heart. If you have a healthy heart, you have strong muscle, well, then you're going to have normal contractility. But if you have an unhealthy heart, if you're in heart failure, congestive heart failure, well, then you're going to have decreased contractility. So we're actually going to write this one out really quick. So the healthy heart that would make sense healthy heart contracts, fine, unhealthy heart. And again, this is an intrinsic mechanism. IE, heart failure. So low ejection fraction, for example, that we talked about in the last lecture, unhealthy heart, you have a decrease in contractility. Well, why? Well, the muscles weak heart failure, you have weak muscle, weak heart muscle, it's not going to contract as hard. If it doesn't contract as hard. Well, then what are we going to have a low stroke volume, even though somebody is in heart failure, and they have a low stroke, fine, the heart is still or the system is still going to try to maintain a normal cardiac output. So how is the body going to respond? What are we going to do to heart rate to keep our cardiac output the same? Our rates gonna go up body responds by increasing heart rate, what do we have now? Now we got an additional problem, right? What's it? What's inefficient heart? low heart rate, high stroke, fine. What do we have here? The complete opposite, we have a high heart rate and a low stroke volume. So we have a failing heart, that's going to fail even faster now, because it's more inefficient. So now we have an inefficient heart. Which means it has to work even harder. So it's just going to go downhill. There are ways to treat it to slow the progression down. But obviously not a good thing. So that's an intrinsic mechanism right there. And what are we affecting, we're affecting stroke five, and then indirectly heartbreak, but in the opposite directions that we want to. Now let's go on to another intrinsic mechanism. And that's something called preload. We actually already know what preload is, I just didn't tell you at the time. Although we're gonna simplify it a little bit. We're not gonna I'm not gonna tell you exactly what preload is, but for us, it's going to be good enough. So let's draw a picture what preload is I'm gonna draw heart here. And I'm going to put our chambers we're going to put a term in here and abbreviation that you're familiar with, and diastolic volume. And diastolic volume is The volume of blood in the ventricle before the ventricle ejection stroke volume, right, you know that. I'll remind you, we did it in the last lecture. So there's in diastolic volume, we first saw isovolumic contraction and diastolic volume. As far as we're concerned, preload and end diastolic volume are the same thing. As far as we're concerned, that's not exactly the way it works. But it's close enough. So I've got to put in parentheses over EDB, the word preload, same thing. So preload is the amount of blood in the ventricles before the ventricles contract. The amount of blood in the ventricles will dictate how hard the ventricles contract. So, with preload as we have an increase in preload, what that's going to lead to is an increase in contractility which will lead to an increase in stroke volume. Whereas if we have a decrease in preload, we're going to have the exact opposite occur. We have an increase in Cairo sorry, a decrease in contractility

16:11

and therefore, a decrease in stroke volume. So what kind of effect is an increase in preload? A positive one. I know tropic effect. So that's a positive inotropic. Whereas a decrease in preload is a negative either true, it decreases contractility. I'm going to show you a chart should be in your notes. Here it is. So we're going to concentrate on the left side to the left of the line. Here I have a column with preload values. Here I have a column with striploin values. What's average end diastolic volume, remind me what the value is? 120, right. We learned that in the last lecture. Well, here's 120. Right here, what's our average stroke volume again? 70. So it matches up perfectly. Smaller preload, 90. Well, now we have a straw, smaller stroke volume, bigger preload, look what happens bigger preload,

bigger stroke volume, bigger preload, bigger stroke, fine. As the preload goes up, up up to a point, by the way, you can get too big to the point to where it'll go the other direction, but we're not going to get into that kind of detail. Point is pre load up stroke volume up. So when it comes to me, for example, I have a stroke line of about 100. That means that my preload, my end diastolic volume is probably I don't know, 161 70, somewhere in there, Lance Armstrong, who has a stroke volume at a preload a stroke volume of between 150 milliliters, and 180 milliliters, he's off the charts, he's not even on here, I would have to keep on going over here to 151 6171 82, where his preloads would be close to 300. So again, his heart is much different than most people walking on the planet, because well, he's Lance Armstrong. The bottom line is this. Understand that as preload goes up, and this is the way that you should visualize it, you're just stretching the heart more, and it just kind of rebounds a little harder. That's the way I want you to visualize it. More blood in the ventricles means it gets stretched a bit more. And it's just going to be a harder contraction. That's what I want you to visualize with preload. Now, again, the complete opposite is going to hold true when it gets smaller. So anyway, that's pretty low. Now let's talk about afterload. This isn't exactly what afterload is, but it's going to be good enough for us. So we're going to draw another picture here.

18:48

So here's our heart. And I'm going to draw the great arteries in this picture.

18:55

And so there's our pulmonary trunk. And there's our aorta. And as you know, we're going to eject a stroke volume into each of these great arteries during the period of ejection. And there it is, so there's our stroke volume. And there's our stroke volume. And you know that the pressure in the ventricle has to be higher than the pressure in the great artery, we know that we learned it, that's going to pop open these valves, the semilunar valves allowing the ejection of blood into the great arteries. So there's resistance to that there is a force working in the opposite direction. So that black line there is not blood flow is the resistance against ejection of a stroke volume. And that's what afterload is. And so I'm going to write afterload above each of those lines. afterload has resistance against pumping blood. And as far as we're concerned, after load His blood pressure in those great arteries. And so after load is going to be blood pressure in the pulmonary trunk. afterload is the blood pressure in the aorta. And the pressure in the ventricle, the pressure in the in the, the left ventricle and the right ventricle have to exceed those blood pressures. And again, we learned it in the last lecture, I'll show you really quick. We're talking about period of ejection. Look at that ventricular pressure had to be bigger than great artery pressure. So the great artery pressure, what I'm circling right now, that's afterload, as far as we're concerned. All right. Now, let's go back to that chart that we had up on the screen a little bit ago, except what we're going to do now is we're going to concentrate on the right side of the line and look at the afterload versus stroke volume. After load is obviously not a volume, it is a pressure it's blood pressure, which is why the units are in millimeters of mercury. They're not in milliliters. So preload is a volume afterload is a pressure. Now the values that I have here are the pressures in the aorta, these are big. If these were pressures in the pulmonary trunk, they'd be about 10 times less than this. So we're going to stick with this story right here. So I have a pressure of the aorta of 100 millimeters of mercury. And I have a stroke volume of 70. And we go from 100 to 120 to 150 to 180. And yet we have 70 milliliters of, of a stroke volume at each and every one of those particular pressure values until we start to get above 190. And I have 200 as an example, now, stroke volume is starting to go down. So this is what we need to get out of this. Let me ask you this. Gonna go back to our picture. And this is where we are because we're talking about aortic pressures. If the

pressure of the aorta, let's say, is 100 versus 150, do you think the heart has to contract harder against an afterload? Of 150? Yes or no? Of course it does. When you have stroke volume is the same contract Kelly's going up. But stroke volume is staying the same. Why is that? Because the resistance is higher. I told you that when it came to an I know trope, let's go to it. Oops, over here. I told you that when you increase the the contractility of the heart, you should always not always will routinely make stroke line go up, but then there'd be an exception to the rule. Here's the exception to the rule. I'm going to give you an analogy. Typically this works. I'm going to look at this chart as I talk. So let's say I put 100 pounds on this chair.

22:52

And I want to push this chair 70 inches, I have to contract the muscles, the muscles in my body to push it hard enough to push it 70 inches, right? If I put 120 pounds in this chair, and I want to push it 70 inches, do I have to push it harder? Yes or no? From my muscles contracting harder? Yes or no? Of course they are. Am I working harder? Yes, I am. Why am I working harder because I want to push the chair 70 inches, I'll put 180 pounds on that chair. And I'm going to push it really really hard. I'm going to manage to get it 70 inches, but then put 200 pounds on that chair, I'm going to push it as hard as I freakin can. But I can't push it 70 inches because I'm not strong enough. But I'm still working hard harder than I was at 180 and 150. And then 220 To get it even 50 inches. I'm pushing it as hard as I can. But I can't push it 15 inches. That doesn't mean I'm not working hard. contractility is still going up and it's going up to maintain a normal stroke volume. That's why if it doesn't bat, right, because how do we have to make up for it. Heart rate needs to go up. So when it comes to afterload, an increase in afterload is going to cause an increase in heart contractility. Even though most of the time, you're not going to get a change in stroke volume, not because the hearts not working harder. It's working really hard. So an increase in afterload

24:28

is a positive inotropic effect. And then a decrease in afterload will just be thorough

24:36

will cause a decrease in our contractility. And I'm not going to mess with stroke volume over here because it's going to depend on what our afterload is. That will have a negative inotropic effect. So, going back to the chart there are many reasons Why high blood pressure is bad. This is one of them. If your blood pressure is high, you have to make your heart work harder. Why? Because it has to contract harder to maintain a normal stroke flowing. And then if we get to this point to our blood pressure is really high and there are people walking around with this kind of blood pressure. And by the way that don't even know it. Now look at stroke filling went down, high blood pressure stroke volume went down, how are we going to make up for it? What needs to go up to maintain cardiac output? Alright, so now your heart's becoming more inefficient. You got to hardest work in its butt off. And it's becoming more and more efficient. How long do you think that part's gonna last? Not very long. All right. Are we good? Yes, rock?

25:59

80 versus 200?

60 versus 200?

26:09

I'm try pushing the Chair Harder at 200 pounds that I wasn't 180. And yet, I can't push it. 70 inches. That's as far as I can push him. Because I'm just not strong enough. higher intensity assistance contract and harder to implement? No, no, it's got nothing to do with the heart. That was that was an analogy. No, me pushing the chair has nothing to do with the heart. Zero. I'm just trying to tell you that I have to work harder to push that. When there's more weight on it. That's all I was saying. Just like the heart has to pump harder, when that's the pressure it works against. So. Yeah. Yep. That's what I was talking about with the Chair. Thank you. You're welcome. Alright, guys, here we go. So those were intrinsic mechanisms. Now let's talk about extrinsic mechanisms. So these are going to be things outside the heart that are going to affect cardiac output, and stroke volume and therefore, and heart rate as well. So the first thing we're gonna talk about is the autonomic nervous system, the cardiac control center. So here's a picture that we are familiar with that we've drawn a number of times brainstem spinal cord, I will label everything. cervical, thoracic, lumbar sacral, of course, we have to put an X through the sea. And that's going to be our cardiac control center. I'm just going to abbreviate a CCC. It's just a bunch of neurons in the brainstem. And their job is to control the heart, control how hard it contracts, control how fast it contracts. And so let's put the heart in the picture. And I have to put the cardiac conduction system in this patient, I'm going to draw it really fast. So you know, there are no pathways and neural pathways AB node bundle hiss right bundle branch left on the branch purkinje fibers. So there's our cardiac conduction system. And we know that we control heart rate via the cardiac conduction system. And we know that we control contractility visa versus or I'm sorry, via the ventricular muscle. So these neurons in the cardiac control center are going to synapse with neurons in the brainstem are going to synapse with neurons in the thoracic region, and then this neuron coming out of the brainstem is going to have a long preganglionic neuron, right? We know this from last semester. Short postganglionic neuron tell me what that's from. What nerve is that part of darker the V vagus nerve correct. So it's parasympathetic nervous system releasing acetylcholine onto the muscarinic receptors. Remember that? What's the parasympathetic what's the vagus do to the heart does it stimulate it or does it inhibit it inhibits so we have a negative effects so we have a lot a negative chronotropic effect. When we really sad acetylcholine onto the heart, now, we have coming out of thoracic region a short preganglionic neuron along postganglionic neuron they are going to synapse with the cardiac conduction or I'm sorry, the sad note. We also going to via the sympathetic control the ventricles so it's going to send us with a muscle. What kind of effect is the sympathetic had on the heart? positive effect stimulatory effect. And so what is it going to do? Well, we are going to have a positive Let's just say, Yeah, Corona tropic effect and a positive. I know tropic effect. So parasympathetic slows the heart down sympathetic speeds it up makes it pump harder contract harder. Well, I will put the stroke line because that depends on situations. One of the things I want to add here, and this is something we learned last semester, is that your vagus, the parasympathetic nervous system has the greatest effect of these two things. On resting heart rate, of course it does. Parasympathetic is what rest and digest is most active most of the time, so of course, it has the greatest effect on resting heart rate. So, again, this is the cardiac control center and how it's controlling cardiac output by obviously controlling Oh, negative tone lighting and put it over here. So we have a decrease in heart rate. We have an increase in heart rate when it comes to the sympathetic. We have an increasing contractility over here. Yeah, certainly had to add all that stuff.

31:12

Okay. What else moving forward. So we just drew this. And we drew that that's what's in those pictures. Last two things, hormones, we already talked about this, we're not going to get into it in

pictures. Last two things, hormones, we already talked about this, we're not going to get into it in detail at all. All you have to know is epinephrine and norepinephrine from the adrenal medulla, increases contractility and increases heart rate. That's all. We already learned it anyway. Same thing with T three and T four, increases heart rate increases car contractility. So they're both positive, I know tropes and positive coronal drops, that's all. Very straightforward. And then body temperature, your body temperature can affect your heart rate, you increase your body temperature, one degree Celsius, you're going to roughly increase your heart rate, 10 beats per minute, decrease your body temperature, one degree Celsius, you will roughly decrease your heart rate 10 beats per minute. So body temperature has a coronal tropic effect, either positive or negative, depending on whether you increase the temperature or decrease temperature, very straightforward stuff. That's all we have to know. All right, done with that chapter onto the next one circulation. Now, the first page of your notes and that circulatory chapter, you do not have to know it's about a lot of anatomy. I know that Seaver went over a lot of that with you guys. It's got a little bit of venous return in there, she talked about venous return with you guys in blood flow, the first page is out. I don't know if it's the whole first page. But where it starts where you guys care is where we talked about blood flow and the two types. That's where our discussion is going to begin. So two types of blood flow. We have laminar flow and turbulent flows, show you pretty pictures of each of those and draw one in just a second. So laminar flows nice and smooth. Turbulent blood flow is well when it says turbulent, much easier to pump blood, that's laminar versus turbulent. But I don't want you to think that turbulent blood flow is abnormal, it's 100%. Normal, it's unavoidable within the body for a couple of reasons. So let's talk about laminar nice and smooth, not much resistance to blood flow. So that's why it's easier for the heart to pump laminar flow turbulent, we have an increase in resistance. So it's harder for the book for the heart to pump that blood. But again, it's unavoidable. One of the reasons that it's unavoidable is because of the way that the circulatory system is put together. So let's draw this really quick. I'm just going to draw a blood vessel here, actually, I'm gonna draw three of them. There's one, and then I'm going to have a branch. So obviously, this is the arterial system, big blood vessels becoming smaller ones at the branches. And so this is going to be nice, smooth blood flow. So I'm going to draw the lines nice and straight, happy and smooth. But then we have that branch right there. And what's going to happen is as as that blood is flowing, it's going to hit that branch. And then it's going to double back just a little bit. And it's going to cause some turbulence right here in this area, how much that not that much but enough to where it's turbulent. There are branches within the circulatory system. So it's completely unavoidable when it comes to turbulent blood flow. So branching causes turbulence, but it's okay. Something else that's going to cause turbulence is high blood flow. And so I'll draw another blood vessel over here and I'm actually going to name it this is the aorta. He already has very high blood flow. Velocity of blood through the aorta is very high. Why? Well, it's coming right out of the left ventricle where the ventricles contracting very hard. And so you have high velocity. It's kind of like a car driving a car too fast, you can kind of maybe get out of control, maybe hit the guardrail. And so this will contribute to turbulence. Again, it's okay. It's normal. What's not normal is when you have extra turbulence, that's not normal. And that's not what you want. Because it's going to make it even harder for the heart to pump blood because again, turbulent blood flow is harder for the heart to pump. And that's in the notes. And it's sitting right here. So I can treat increase turbulence. High Velocity highest higher so than normal. We'll talk about why that would happen with an increase in resistance. We'll talk about it later.

35:59

The branching and that's just again, normal, but it has to be included in the discussion center blood, lower viscosity blood, we're going to talk about the viscosity of your blood, the thinning of blood, the thickening of blood has nothing to do with blood thinners. By the way, I think I've made that perfectly clear. Here's something else. When we have turbulent blood flow, it can damage blood vessel walls,

why it causes a shear stress against the walls, if we have blood flow that's nice and smooth. It's not going to damage the blood, the blood vessel walls too much. But all of a sudden, if we have this chaos here, and we have these little whirlpools, called Eddie's and the blood is knocking against the wall pretty hard, as opposed to just going through it nice and smoothly. You're going to start to inflame the blood vessel walls when you inflate blood vessel walls, what can you develop Downey, please start with a T thrombus. So when you have turbulence, you increase the risk of a thrombus. And that's another reason you don't want to make turbulent blood flow even worse than it already is, is because then you increase your risk even more, it can also increase the risk of developing plaque. And that would be a condition called atherosclerosis, which I'll talk a bit more about at a later time. And so, obviously, not good things. Turbulent blood flow, normal, extra turbulence, not normal. You don't want that. Now what? Let's move on, and look at this equation here. passageways law. Now pulseway, very smart guy from way back when derived an equation that obey the laws of physics, when it comes to fluid flowing through a tube and air flowing through fluid through a tube. This is what we need to know from passageways law, fluid flow equals a pressure gradient. And that's what we have here. That's a gradient the difference right? Now what you think of when you think of gradient, you think of difference. So we have a difference between these two pressures. So that's the pressure gradient.

38:21

And there's resistance to fluid flow fluid flowing through it, too. There's resistance to it. And so because blood vessels are tubes, and because blood is a fluid, we can use passageways law to help us understand cardiovascular physiology. And so what are we going to take from passageways law? Well, instead of fluid flow is going to be blood flow. We're going to be specific about the pressures. And they're going to be blood pressures, blood pressure at point one versus blood pressure at point two, so a blood pressure gradient, then there's going to be some resistance to blood flow. Well, what's the resistance to blood flow? Well, I'm going to show you passageways law and then we're going to tease some things out a plus ways law. So resistance are equals eight times viscosity times the length of the two times pi times are to the fourth power. By the way, you're not gonna have to do calculations with passageways law. Alright, I'm going to do calculations on the exam, but not with this, we're going to use this particular equation qualitatively. We're going to tease out a couple of things from resistance, viscosity, and radius. So this is what you need to know what is our, our does not equal, but it's going to be proportional to viscosity, over radius to the fourth power. Now viscosity is a fancy word for thickness. Well, the viscosity of what the viscosity of the blood the thickness of the blood, so V equals viscosity of blood. And our little r is going to be equal to the radius of a blood vessel.

40:06

And with that, let's take a break when we come back, we'll continue our story Okay, folks, Sally pointed something out at the break, I forgot to put that this is a sympathetic nervous system, and specifically the cardiac nervous in the picture, it'll be on pilot. Now what, let's keep on talking about blood flow and what affects it. And so now we're going to concentrate on the blood pressure gradient. And when it comes to the blood pressure gradient, just by doing simple math, we can see what kind of effect we're going to have if the gradient is bigger or smaller. But what I'm going to do first is this. So I'm going to draw the circulatory system specifically systemic circulation, so aorta, and then we have arteries arterioles capillaries which opened up into venules. veins. And then VENA cave i because there's two of them and as plural and then right atrium, of course. Now, let's talk about VP

one and VP two. So again, we're doing breath blood pressure gradients, right now I can make BP one the aorta, and I'm gonna. And I can make the arterioles BP two or I can make the capillaries BP two, let's just do the arterioles. Let me stick it in the equation. I can make the arterioles BP one. So I'm just going to kind of maybe put a square around those two, to see that they're matched together, I can put arterioles can be VP one. Does that mean that the order is going to be BP two? No, it doesn't. BP two has to be downstream. And so arterioles is BP one and maybe I make the capillaries BP two. And I'll put a circle around both of those. Or I could start it in the venules. I can make the venules BP one

42:08

and the right atrium BP two. And I'll just underline both of those to show that they go together.

42:18

So when you're doing this on an exam, when I when I had these structures on the exam, how do you know what's going to be what? When it comes to Pathways logging, you're not doing calculations. But you still have to understand this, you have to know the flow of blood within the circulatory system. And hopefully you know it by now. BP water BP two is always downstream of BP one. And so we throw some numbers in here. That could be a viable number for your attic pressure and arterioles are no 30 viable for that. That's a pretty big pressure gradient right? Difference between those two numbers is 60. That's big. If we did it for the venules versus the right atrium, this would be I don't know somewhere around 10 millimeters of mercury. And anywhere from zero to five, I'll just put somewhere in between I will go three millimeters of mercury there. That's an itty bitty blood pressure gradient. That's why blood flow in your veins is less than what it is in your arteries. Do you need valves in your veins? Valves in your veins, right? Why do you need valves in your veins? Why do the muscles have to squeeze your veins to get blood flow? good blood flow because the gradient so stinking small? Do you have vials in your arteries? No, you don't need them. Why? Because look how stinking huge the gradient is it's humongous. That's an a very, very important point. Now, another point here. And before we move forward to that other point, I'm going to just basically just do BP one making it bigger BP one versus BP two is certainly going to increase blood flow. Of course it is. If you decrease the blood pressure gradient, well then of course, you're going to have less blood flow. And that's just simple math. That's in the numerator, you make the numerator big. Well, then this is big, you make the numerator small, well, then that small, third grade math, we're just doing fractions here. That's the other thing to look at that that's just a fraction. This is four equals eight over two. That's I mean, literally, it's a fraction. Don't let any of these variables intimidate you in any way. It's just a fraction and you learn fractions. I don't know second or third grade isn't that when we learned how to do our fractions, so it's no different here. Now something else important is that if blood pressure in arteries, I should say the arterial system because it can be anyway arterioles system is too low then your blood pressure gradient is too small. BP one versus BP two equals? Or is too small? What was that gonna do? Well, you got to decrease your blood flow a lot. Well, where's this blood going in the arterial system? Where's it going? It's going to these capillaries. What are the capillaries feeding yourselves. That's where you get exchange, you get exchange at the capillaries. That's how you get oxygen yourself. So you get nutrients to yourselves. What's gonna happen to the south? You're gonna die, tissues die, organs die. So what do you risk here? You risk organ failure. I've mentioned this to you guys a number of times over the last two semesters. For blood flow, you risk organ failure, I did it the first time in skin Janardhan. Even before that, I did it like in the transport chapter last semester, I did it in the skin chapter of last semester, we talked about third degree burns. Remember that fluid shifts. One of the dangers of third degree burns is why blood pressure gets too low, which then can cause

death. Remember that from last semester. So I'm not sharing anything new, I'm just putting a little bit more detail into the story here. So anyway, very important that we maintain a certain blood pressure or bad things are going to happen. And it goes back and forth. So as long. So that's the numerator. Now let's talk about the denominator. Let's talk about these two variables viscosity and radius, we're gonna take radius first. So what I'm going to do here is I'm going to draw a blood vessel right here in the middle of the screen. So that's the blood vessel. And what we're going to do to this blood vessel is we're going to make it dilate, we're going to make a constrict. So we're going to go in this direction, and we're going to vaso dilate. What that means is, is that the radius is going to get bigger. And so let's make the radius of the blood vessel bigger. How am I doing?

47:14

So the radius of that blood vessel is now bigger. So we have an increase in radius. And if we have an increase in radius, let's go back to our math, we have an increase in radius, if this gets bigger, what happens to this? It goes down, this is the denominator, the denominator gets bigger, this gets smaller, right? So little r going up is going to cause big our resistance to go down. And if big art goes down, if big art goes down, what happens to that? Blood flow goes again, third grade math, I'm doing third grade math right now. You get an increase in blood flow? Of course you do. Logically, that should make sense. If you have a bigger tube, you want to get more fluid through it. That's all we go the other direction. On by the way, how are we doing this? How are we constricting? How are we dilating we're controlling the smooth muscle that's wrapped around these blood vessels. And we've talked about that a number of times over the last two semesters. Now we're gonna go the other direction, and we're going to make the blood vessel constrict. So now we're going to have a decrease in radius, which means now we're going to have a bigger big R and increase in resistance, which means we're going to have a decrease in blood flow. Again, of course, mathematically, you can see it logically, you should be able to think about that. If you have an itty bitty straw, it's going to be harder to suck the milkshake through the straw. If you have a bigger straw, it's easier to suck the milkshake through the straw, because there's less resistance. No different here. So that's all about radius. Now something else I need to mention is that changes in radius. And when I'm talking about radius, club radius of the blood vessel changes in radius have huge effects on blood flow. Now, why is that? Well, we should be able to see it mathematically. So let's go back to our equation. It's to the fourth power. Why did fosway make it to the fourth power? Because if he didn't, it wouldn't obey the laws of physics. He didn't say you know what? I'm going to make radius to the fourth power. Or, you know what, I'm going to put this cost to the in the numerator here, and length of the blood vessel here and I'm going to put pi in the denominator just because I want to do it. No, it took him a very long time to figure out the equation that explains how fluids flow through a tube again, the guy was a genius and I don't know how long it took him to drive this equation but a very long time. And he figured out that radius had to be to the fourth power not to the second power not to the first power, not to the third power, but to the fourth power. So what does that mean? Why does it play such a huge role? Let's think about this for a second, I'm just going to give you an example. Let's say our radius double. So we've just doubled the size of the radius of the blood vessel, you're not going to double blood flow, you're going to increase blood flow 16 fold by doubling the radius, well, where the hell did I get 16? Double it. That's the number two, what's two to the fourth power 16 humungous change changes in blood flow with relatively small changes in the radius of a blood vessel. Alright, remember that you don't have to do calculations but do understand huge, huge influence when it comes to changing the radius of a blood vessel. Now we go on to the next variable, and the last one is going to be viscosity. And that's just a fancy word for thickness of the blood. And so we will say that an increase in viscosity and again, it's viscosity of the blood will cause an increase in resistance, of course, it will mathematically we should be able to see that if the numerator gets bigger, that gets bigger. And if that gets bigger, that goes down. So if you have more resistance, you have a decrease in blood flow.

And then the opposite is going to hold true. Of course, a decrease in viscosity will cause a decrease in resistance and an increase in blood flow. And logically, that should make sense. Increase in viscosity again, let's make sure that we remember what viscosity is. Viscosity is just a fancy word for thickness. So what's going to flow easier have milkshake or water? Water? Milkshake has a higher viscosity, it's a thicker fluid, it's not going to flow as easy. higher viscosity, lower blood flow, lower viscosity, higher blood flow. Now what affects viscosity of the blood? The most? Again, it's not blood thinners because blood is not in the blood. That's like the 20th time I've said it.

52:15

What affects the viscosity the most? What affects blood viscosity? I'm going to put blood there viscosity the most. And it's not even close. I know it's in your notes, but I'm going to write it out anyway. Um adequate answer, hum adequate and adequate is what concentration of white red blood cells such that you have an increase in red blood cell concentration, you have an increase in viscosity you have a decrease in automatic rate, you have a decrease in viscosity. Now there are a few other things that will affect discuss the the blood cholesterol level, but very little dehydration, very little. Nothing is going to compare to your red blood cell concentration. Tell me what it's called when you're when you're dramatic. Chris Hi. Starts with a pee while you say fimea. Who gets probably Cynthia? Give me patient? What kind of condition would that patient had? Give me one? Cardiovascular disease? Give me another one? pulmonary diseases or a person who does what? Actually, I guess you would hold it like this smoke I did. You can tell I've never smoked anything. Smoking. So those people had these particular conditions or smoke, and now they're going to make things worse. Why? Well, because you have a higher viscosity, you're going to have less blood flow, it's gonna be harder for the Do you think it's harder for the heart to pump blood that's thicker? Of course it is. So anyway, that's viscosity and the points that I'd like you to know about it. Now what? Let's talk about how the body is going to control blood flow. So we have a couple of it, we have three variables here. When it comes to blood flow, right, our variables are our numerator, blood pressure gradient, our resistance made up of viscosity and radius. The way that the body is going to control blood flow is through the radius of the blood vessel, we're not going to change viscosity. Can't do that very fast, we can change the radius of a blood vessel like that, right. So let's talk about that now. So we're going to now see how we regulate blood flow. At the beginning of this lecture, I talked about how we regulate cardiac output. Now I'm going to talk about how we regulate blood flow. And so we're going to start with just as we did with the heart, the nervous system. So we have something called the basal motor centers. And so let's draw them There's going to be a picture that looked very, very familiar brainstem spinal cord again. And I tell you last semester, when we do this 80 million times that we would draw a heck of a lot more this semester to, and I wasn't lying, draw us out to see, of course, there's no cervical or there's no motor output from the cervical region, from the autonomic. So now we have this region here that might look very familiar to what I drew earlier with the cardiac control center, in that same area are groups of neurons, whose responsibility it is to control blood flow. And so that's the vasomotor center. And actually, in the next lecture, we're going to have both the basal motor Center and the cardiac control center together when we talked about regulating blood pressure. So there's our basal motor center. Now in this particular picture, we're going to draw two blood vessels. Here's one. And here's another. And these are different blood vessels that I'm going to label what they are, these are blood vessels of your skin and viscera. So they're the blood vessels that are feeding your skin, and that are feeding the organs of your body. The blood vessels represented down here are blood vessels of skeletal muscle. So it's not a skeletal muscle, but blood vessels that are feeding the skeletal muscle. And around those blood vessels, of course, they're smooth muscle cells. That's how we're controlling the tone of these blood vessels, right? That's how we basal dilate, basal constrict, we sweep cause the smooth muscle wrapped around the blood vessels to contract, we constrict, we relaxed, the smooth muscles around these blood vessels, we dilate. Nothing new there. Now, these vasomotor

centers are just a bunch of neurons whose job it is to control blood flow, and how are we going to control blood flow, we are going to basal dilate, we are going to basal constrict, so we're going to control blood flow. And how are we going to do that, I'm going to put this neuron right in the middle of the thoracic in the lumbar region

57:08

to symbolize that is the sympathetic nervous system. And these neurons are going to come out of the thoracic in the lumbar region. But I'm just going to have one because I'm going to clean the picture up. So we're going to have this neuron we're going to have a branch, so short preganglionic neuron. And then a long postganglionic neuron. Same thing here long postganglionic neuron releasing norepinephrine on to these adrenergic receptors, if you remember from last semester. So the sympathetic nervous system is what's going to control blood flow. For the most part, almost always, there are some exceptions, I actually gave you one earlier on in the semester when we talked about erection and how the parasympathetic controls that if you remember, point shoot, remember that. So anyway, almost always a sympathetic couple of exceptions here and there. So we had the sympathetic nervous system. And so this is how it's going to work. If we have an increase in sympathetic tone, and we know what that means. So we're stimulating the sympathetic tones going up? Well, what is that going to do? Well, what that's going to lead to is we are going to get vasoconstriction of these blood vessels, ie, we're going to get a decrease in little r. And if we get a decrease in little r, while we're going to get a increase in bigger resistance, and if we get an increase in big resistance, while we get a decrease in blood flow. And again, logically, that should make sense. If you can strip the tube, you're going to have less flow through it. And the opposite is going to hold true. If we decrease sympathetic tone, well, then we're going to dilate. We're gonna make the radius of the blood vessel go up, which is then going to lead to of course, a decrease in the resistance, which of course is going to lead to an increase in blood flow. Of course, if the tube gets bigger, oops, more blood is going to flow through it. That's just plain old logic. So those are those blood vessels. And that's most of the blood vessels in the body, of course, skins the biggest organ in the body, we have lots of organs as well. Now we're going to do the same thing down here, we're going to increase sympathetic tone. In this particular case, we're going to do the opposite. We're going to vasodilation we're going to make these blood vessels go up complete opposite of what we saw above. Which means we're going to decrease resistance, which means we're going to increase blood flow. But those are blood vessels going to our muscles. sympathetic nervous system when we call it by flight, fight, run. It's active during exercise as well. Don't you want to dilate your blood vessels of your of your skeletal muscles during those times what's going to help you fight run exercise? Skeletal muscles? Don't they need more oxygen? Don't they need more nutrients to make ATP? Of course, we learned this last semester, Nothing is new here. We do a very similar picture to what I have up on the screen right now. And then the opposite is gonna hold true. Make me happy. Tell me why that what the difference is between these two blood vessels? Tell me what receptors are responsible for each of these stories here. Alpha once beta twos, remember the odd numbers, excite the even number inhibits remember that? You don't have to know for the exam. I was just hoping that I don't know. Maybe somebody would have gotten it. All right. That's okay, I'm not too sad. Alright, let's move on. Now, what do we call local factors. So what's a local factor? When it comes to the nervous system, and we're affecting the sympathetic tone, for example, that's going to be global. Because it's coming from the vasomotor Center, which is going to be synapses with many, many, many different different sympathetic neurons here and affecting the body globally. A local factor can affect the body globally, because it can be released into the blood circulate everywhere, but it can also be local. The effects I'm about to show you can happen just in a little part of the body, which is why they call it a local

factor. And these factors are things that are going to affect again, the smooth muscle that's wrapped around the blood vessels. And I just have a short list of those particular local factors that dilate those local factors that are going to constrict. And we're familiar with many of these.

1:02:02

Nitric oxide, I actually mentioned this one earlier on in the semester when we talked about erection if you remember, the two neurotransmitters are one Cetyl choline, the nitroxide. Remember that it's not the only place nitric oxide is used in the body. Nitric oxide is used everywhere. Nitric oxide plays a huge role when it comes to blood vessel tell it's made by endothelial cells. We also use nitric oxide clinically, especially to treat people with pulmonary hypertension, because it relaxes blood vessels. So nitric oxide, a huge player when it comes to blood vessel tone, and it causes vasodilation. Histamine is a something I've talked about a number of times. And that's something that also causes vasodilation. And it's part of the allergic response. So when you have allergies, and you have a stuffy runny nose and watery eyes is to me plays a big part in that because it dilates the blood vessels to your mucous membranes, your nose, you make more mucus, and it swells the mucous membrane. Same thing with the eyes, increases blood flow there and you just make more tears. That's why your eyes get running. We're gonna mention histamine again. Later on. In the next lecture, when we talk about circulatory shock, and the effects of histamine, you'll see. Tell me what thromboxane is from what makes her unboxing tell me platelets make from boxing, right. From boxing, we've already learned constricts blood vessels we learned last semester, we learned earlier on this semester. And now we're learning it for the third time. Anti diarrhea. antidiuretic hormone is a hormone that we learned about earlier on in this semester. And I told you at the time, his main job is to do what Regulate Blood osmolarity. But it will also regulate blood pressure and blood flow. We're going to talk about it later. So that's why it's here. It's coming in the next lecture. And then we have pa co2 and oxygen. And we're going to take these together, at least the pH and the the co2 together in this little story. So this is going to be a cell. Pick yourself doesn't matter which one, and this cell is going to have an increase in metabolic rates. So as metabolism is going to go up, part of that is going to incur an increase in the production of ATP anaerobically and aerobically. And you guys notice we learned it, that acids are produced anaerobically like pyruvate, and lactate, so that's going to cause an increase in acid production aerobically, we're going to get an increase in the production of co2. Now that cell is not going to hold on to all of that extra acid and co2. If it did, it would die. cells don't want to die. And so what the cell is going to do is transport the acid out of the cell and eventually that acid is going to be taken up by the blood. So now we're going to have more acid in the blood More co2 in the blood. So number one, we have an increase in metabolism. Number two, as a result, we make more acid in co2 and number three that we're going to have an increase in acid and co2 in the blood. Now, we don't want that to sit there. It's normal to have acid and co2 in the blood. But it has to be a normal level, we need to keep on homeostatic level. If we have extra co2 and acid here, we need to wash the excess away. So number four, what's going to happen is we're going to vaso dilate. And when we do that, well, obviously we are going to That's an increase in radius. So let's just put everything here that we normally have increase in R, which causes a decrease in big R, which leads to an increase in blood flow. So that's all for why is our question? Our answer is to wash away more acid and co2 to keep the levels normal around the cell. That's the reason the opposite is gonna hold true if we have a decrease in metabolism, and have less acid and less co2. So we'll draw that one as well. So here's our cell, we're gonna have a decrease in metabolism now. So that'll be number one. And as a result of that, well, we won't make quite as much acid, we won't produce quite as much co2. Here's our blood. Again, we need to have normal levels of these things in the blood, acid and co2. And so they are going to be transported into the blood where now we're going to have less acid, that'll be number three and less co2.

1:06:56

Once again, we need normal levels. So you don't want to dilate, wash a whole bunch of way, because then the levels will get way too low around itself. So you'll constrict so you'll wash less away, so that you'll keep more of it here. And so yes, is going to lead to number four vasoconstriction, which is obviously a decrease in little r, which means an increase in big R, which means a decrease in blood flow. And what's the why to that? To wash away less acid, and co2, so you keep the levels normal. And this again, this can happen locally just in a small part of the body or it can happen globally, it just depends. Now what about oxygen, I'm actually just going to put these two on the same page. So a cell, the cell, and this cell is going to have low oxygen levels around it, this cell is going to have high oxygen levels around it. So this cell over here on the left hand side is going to be in need of more oxygen, will this blood carry oxygen? It sure does. So number one, we have low oxygen levels in the cell, it needs more, the low level of oxygen in this area will relax the smooth muscle. And so as a result number two, we will vasodilation which means we're going to get a bigger R, which means we're going to have small r that is which means we're going to have less resistance, which means we're going to have an increase in blood flow good. That's going to give us more oxygen because our blood is carrying it. And we're talking about the arterial system here, by the way, because it's carrying it to the cell. And we know that then the opposite is going to happen over here. If we have cells are not greedy. So if there's a whole bunch of oxygen around here, this cell is going to be very charitable, and let other cells in the body have more oxygen. And so as a result of this number one, number two, we're going to constrict, we won't have as much blood flow to this area. As a result of that, we're going to have a higher resistance and as a result of that, once again, a decrease in blood flow. And that blood will go someplace else. It's called shunting. The blood will be shunted from this particular area so that the oxygen can go where it's more needed. The cells are needed. So high oxygen, constrict low oxygen dilate. Why? Get more oxygen to the cell. It gets more oxygen to the cell.

1:10:01

What's the why are we here? Blood, which has oxygen in it goes to other places that need it more. That's seldom needed. The level of oxygen is high. So those are the local factors, there's just a short list of them, I could have made the list of page long each. The reason I highlighted these is because we actually were already familiar with all these.

1:10:34

Now what you don't have to know how to measure a blood pressure I used to go over it, it just takes a long time and the yields not very high. Those of you that need to learn how to do a blood pressure will learn how to do a blood pressure, it literally takes two minutes to do a blood pressure. The reason that I talked about it is because it revolves around laminar blood flow, and turbulent blood flow, the little sounds that you hear those of you who've ever done a blood pressure those core cough sounds, you can actually hear the sound thump, thump, thump, thump, you hear the sound, there's no sound, then you hear the sound, and then the sound goes away. It's all about laminar blood flow, turbulent blood flow, and it dictates whether it's systolic or diastolic pressure, don't worry about it. So blood pressure, I have said the word blood pressure the two words blood pressure many times over the last two semesters, but I've never defined it. The name tells you exactly what it is, is the pressure that blood exerts against blood vessel walls and the walls of the heart as well. And when you measure a

blood pressure by the waist measured in millimeters of mercury, when you measure a blood pressure, there's a top number and there's a bottom number one systolic pressure and one is diastolic pressure. And we know what that means. systolic pressure is the pressure during systole. And we know a systole is right. So when the heart contracts, and when I say heart, what am I talking about? I'm talking about the ventricles, right? diastolic pressure. That's the pressure during diastole when the hearts relaxed. So we have a top number and we have a bottom number. And so if we did a blood pressure. So BP equals 110. And it's measured in millimeters of mercury, over 70. I picked this because this is my blood pressure and has been my blood pressure. As long as I can remember, it's just always this I don't know, it's like, it just it just won't change, which is good. I'm happy about that. So anyway, that's a blood pressure, the top number is systole. And the bottom number is diastole. The top number is always going to be bigger, the pressures are always going to be higher when the heart is contracting. Of course it is then we have something called a pulse pressure. A pulse pressure is not something that you really measure, but it's actually a calculation that's done in a pulse pressure simply equals systolic pressure. Oops, minus diastolic pressure. That's all it is. So given the numbers that I have here, what is my pulse pressure? 40 110 minus 70. So 110 millimeters of mercury, minus 70 millimeters of mercury equals four, which is good because that's dead. What is supposed to be 40 is normal. Anywhere from 30 to 60 is considered normal. And we're going to talk about pulse pressure and all these things, obviously in more detail when we come back from our spring break and I hope you guys enjoy

Circ PM 3-8-22

Fri, 3/11 9:13AM 1:15:01

SUMMARY KEYWORDS

blood pressure, blood vessels, receptors, blood, decrease, equation, arteries, increase, release, systolic pressure, heart rate, basal, called, pressure, aldosterone, hypertension, total peripheral resistance, inhibit, angiotensin, diastolic pressure

---

00:02

Alright, folks, we're just going to hit it the quickly tonight, because we have a lot to talk about. So to remind you last lecture, we started to talk about blood pressure, we finally define what it is, it's just the pressure that blood exerts against the walls of blood vessels in the heart. So just what it says, we define systolic and diastolic pressure, we also defined pulse pressure. And these are things that we're going to talk about in the lecture today. So we're more or less up to speed as to what we started in the last lecture. Now, let's start and talk about something called mean arterial pressure. So mean means average. And so the name tells you exactly what this is, this is the average blood pressure in your arteries. That's what this is. Now, when it comes to me, that's the average meaning that straight averages, you take numbers, you add them up, and you divide by how many numbers you've added up. That's why you get average. If we take mean arterial pressure, we can't do it that simply. And the reason is, is because the heart is not in systole, for as long as it's in diastole. And I'm going to remind you that. So in the heart lecture that we did a couple of lectures ago, we talked about the cardiac cycle. And I told you at the time that the heart is insistently a third of the time, it's in diastole, two thirds of the time. So we're going to have a systolic pressure for half the amount of time that we're going to have a diastolic pressure. And so because of that, we can't just take a straight average of the diastolic and systolic pressure, we have to do it in a different way. And so the example that I have in the notes is a blood pressure of 110 over seven. Now, the average of 110 70 Is somebody do that quickly in their head. 90, right, the average of 70 and 110 is 90, you add 70 and 110. Together, you get 180 divided by two, you can't do meaner to your pressure, that way, it's going to be more skewed towards the diastolic pressure. So we have a couple of equations here, these are the exact same equations, this equation right here. This one right here is the exact same equation as this is, you're going to have to calculate mean arterial pressure on this exam, I couldn't care less which of these equations you use, but what I would encourage you to do is to use them both. Why? Because if you get different answers, something went wrong, because these are the exact same equations just written algebraically in a different way. And so this one, you take a 30 systolic pressure, and you add it to two thirds of diastolic pressure. So obviously, it's going to be skewed towards diastolic pressure. Same here, again, skewed towards diastolic pressure. So if we put the numbers in, and again, using a blood pressure of 100, or 110, over 70, I should say, which is what I have here. So using a blood pressure of 110 over 70. Plugging the numbers in, our mean arterial pressure is 84. So 1/3 times 110, plus two thirds times 70, you're going to get any for using the other equation. This one, plug the numbers in 110 plus two times 70 divided by three, you get 84. At least you better get 84. Because it's the same exact equation. So this is one of the ways that you can check yourself during the exam to make sure that you've made the calculation properly. Use both of the equations. It's going to be a vomit question on the exam, I'm just simply going to ask you calculate mean arterial pressure, bring

your calculators if you're not comfortable with math. So any second grade, this is just second grade math, it's still second grade math. Now, there's actually a third equation, but you're not going to use this equation to do calculations, but you will use this equation a lot on the exam, this equation is going to help you understand what these variables that we're going to discuss how they affect blood pressure. So I'm going to actually write this one out kind of the long way. So mean arterial pressure. Make sure that you understand mean arterial pressure is just blood pressure. That's all. Keep that in mind. So mean arterial pressure equals cardiac output, we know what that is, times total peripheral resistance. Now we know what resistance is, but there's going to be one additional variable to this resistance here. And I'll remind you when we discussed resistance. So now what I'm going to do is Cardiac output is what? Heart rate times stroke fine. We learned that in the heart chapter. And then we're going to multiply by resistance. Now this is going to have three variables when we first learned about resistance. Let me remind you when we did that, we were talking about fossilize law. Where the hell is it? I think it all is still in this chapter. So let's go to passageways law really quick. There it is.

04:56

Where's my boss? There it is. No, it's not. The hell is it? There's so pathways law, there's a pressure gradient divided by resistance. And once resistance, well, this is resistance right here. But there were only two variables that we were concerned with, right? And what were those two variables discuss it, viscosity, the blood, the thickness of the blood, right. And then radius radius of the blood vessel, that's included with this new resistance that we see here. And that's total peripheral resistance as part of this new equation. Let me just write it out. So this right here, so now I'm going to put those same variables here. So Cardiac output is these two things, total peripheral resistance are these three things, one of which is radius, but it's got to be one over radius, we already know why we discussed that with possiblys law, times viscosity of the blood. And now there's a third variable. And this third variable is something that we have yet to discuss, we're going to discuss it now it's friction. So I'm going to tell you what friction means when it comes to this particular equation and how it's going to affect blood pressure. So we're going to draw two blood vessels, here's one. And it's going to be a versus another one over here. Now, the inner wall of this blood vessel has endothelial cells, right, so those are going to represent endothelial cells. Nice and smooth, very little friction between the blood in the walls, because those endothelial cells are smooth as glass. And so in this particular blood vessel over here, because we just have these endothelial cells, and that's it. We have low friction. It's like us sliding on ice in the wintertime, not too much friction between your shoes and the ice nice and smooth. Now we'll be hearing this other blood vessel, we're going to introduce some things on the wall. There's still endothelial cells there. But now what we can have is this. Remember, if these are the right colors, can you tell me what that is? Beautiful, it's a thrombus. Now we're introducing friction, as the blood is flowing along those blood vessel walls, it introduces for like rocks on the ground, try to slide against it, there's more friction, it's harder for the for you to move on that ground. So that's a thrombus. I can also put here, some inflammation. So that's going to represent inflammation of the walls more friction, I'm going to introduce one more thing. And I'll just make it black. That's going to be plaque, atherosclerosis. And so I'll label that atherosclerosis. So we've introduced these things on the walls that well, we don't want there. And what it's going to do is it's going to increase friction, because of these things. And so if we look at this equation mathematically, again, it's second grade math. If we increase any of these things to the right, we're going to increase blood pressure. If you increase cardiac output, blood pressure goes up. If you increase total peripheral resistance, blood pressure goes up. And that's something that's already in your notes.

08:22

And so that's here.

08:26

So I'll talk about it as we look at this. So what are the things that increase your blood pressure based on that equation? This equation right here? increased cardiac output? Well, how do we do that? We can increase heart rate and or increase stroke volume? How else do we increase blood pressure? Well decrease the radius that would make total peripheral resistance go up, make the blood thicker, cause more friction between the blood and the walls of the blood vessel. And then obviously, the complete opposite is going to hold true. How do you decrease blood pressure, he decreased cardiac output by doing one or both of these decrease stroke volume, decrease heart rate, and then again, the opposite when it comes to decreasing total peripheral resistance. You are not going to use this equation once again, to do any math on the exam. Although clinically, they actually use this equation, we're just not going to do in this class, we're going to use the two previous equations to calculate mean arterial pressure. But you will use this equation over and over and over and over and over and over and over again on the exam. I'd say about a dozen questions are going to come from this equation right here. We have to understand what this equation says. Fortunately, it couldn't be any easier. Make this stuff get bigger. Blood pressure gets bigger, make this stuff get smaller, blood pressure goes down. It really is that simple. We are going to refer back to this equation probably a dozen times tonight. Because I want to just bring it home over and over and over again. You'll see

10:00

So now what are we going to do?

10:03

And again, I want you to make sure that you understand mean arterial pressure is just blood pressure. That's all it is. How do we regulate blood pressure? The two main ways that we regulate blood pressure are these first two, the bare receptor reflex and right and angiotensin aldosterone system. So we're going to take the baroreceptor reflex first, this is probably the fourth time we've done this. And this will be the time where there's more detail in it. And so we have the barycenter reflex, this is the quickest way that you're going to regulate blood pressure. Second, the second minute to minute, it's the nervous system, it's the autonomic nervous system that we're going to be discussing again, if your blood pressure gets too high, we need to lower it. If your blood pressure gets too low, we need to raise it negative feedback. And the bare receptor reflex is going to be one of the things that ensures that that occurs that keeps your blood pressure where it's supposed to be. And so we'll just do this one right here. So that's the story right there when your blood pressure gets too high. How do we lower it? This story right here, if your blood pressure gets too low, how do we raise it? Negative feedback, I'm just going to do one of them, you're going to do the other one angle, I mean, obviously, you're going to do both them when you study. But let's do that right there. And so in order to do that, we have to put a number of anatomical structures in this picture that we've seen a number of times over the last two semesters. So brainstem spinal cord, I'll label everything. So brainstem, cervical, thoracic, lumbar sacral region got to put an extra to see, of course, then we have this area in the brainstem. It's a bunch of neurons. And we're going to give it two names, because there's neurons in that area that are part of the cardiac control center. We did that in the last chapter in the heart chapter, if you remember, I'm going to abbreviate it CCC. And in this chapter in the last

lecture, we call that the vasomotor centers. Well, if you remember, so that is both the vasomotor and the cardiac control center together, the neurons are intermingle. So that's a player in this story, we have to put another player in the story, and that's going to be the heart. So let's put the heart over here. Cardiac conduction system is also important. But I'm just going to put the essay note because that's what's going to be affected when it comes to the story. So that's the SA node, we got to put blood vessels in this story as well. And these are going to be specifically blood vessels of your skin and viscera, not the blood vessels of your skeletal muscles, but of the skin and viscera. So these are the most abundant blood vessels in your body. Obviously, the skin is the biggest organ in the body by far, and your viscera as well. So blood vessels of the skin, and the viscera,

13:02

your organs.

13:04

One more player involved here. And that's going to be obviously the bearer receptor. So let's put the bear receptors in this picture, and we'll make it blue. Now, this is not anatomically correct, the bearer receptors are in very specific places within the cardiovascular system, but it's not too terribly important for us to know, I need you to know how they work. And I will label that bearer receptors.

13:27

See, I'll make it blue.

13:31

And the job of the bearer receptors, as we learned a number of times is to detect blood pressure constantly never stops. And so our story is once again going to be and is going to start with an increase in blood pressure. Again, we're going to draw this. So number one, there's an increase in blood pressure. Oops. That's no good. We don't want our blood pressure to be too high. Oh, and by the way, let's do this too. Let's do our homeostatic line. So that's blood pressure. We're normal, we're normal or normal. And we're not normal anymore. Our blood pressure is high. What do we need to do? We need to do this. We need to bring our blood pressure down. That's the story I'm going to show you. When blood pressure is too high. How do we get it down? The barrier receptor reflex is going to help us do that. And so what's going to happen here is this. Number one, high blood pressure. Number two, what that's going to do is it's going to stimulate the barrier receptors. Now, blood pressure went down it would inhibit the barrier receptors. That's something that we learned last semester. So number two, we're going to stimulate the barrier receptors. So they're going to depolarize. They're going to have axons coming from them that are going to synapse with the neurons in the cardiac control center in the basal motor Center. And what's going to happen along those axons is the conduction of action potentials, and the conduction is going to go up. So number three, we're going to get an increase in action potential frequency, of course we are, the frequency of those action potentials is directly related to what your blood pressure is, directly and precisely. So whatever action potential frequency is, is going to tell these neurons right here, exactly what blood pressure is. And when I say exactly, exactly. So now these neurons know that blood pressure is high.

And now these neurons know that they must lower blood pressure. And so how are they going to do that? Well, they're going to synapse with neurons of the autonomic nervous system, and to make the spinal cord just a little bit thicker. So we're gonna synapse with neurons in the brainstem. We're gonna synapse with neurons in the thoracic region, and the lumbar region. And what are they going to do to those neurons, that is the neurons of the cardiac control center in the basal motor Center, where they're going to inhibit some of them, and they're going to stimulate others. Now, our blood pressure is high. How are we going to lower blood pressure? What should we do to the heart stimulated or inhibited? What are we going to do the heart

16:28

stimulator inhibited? We want to lower blood pressure.

16:33

We're gonna inhibit the heart, we're going to decrease stroke volume, we need to decrease heart rate, why blood pressure is too high. And we need to lower it MMP was cardiac output times total peripheral resistance. So now we've come to this equation once and again, it's going to happen another 10 times. So how are we going to do that? Well, this is our vagus nerve, right? We know that coming out of the brainstem long postganglionic neuron. Short, or I'm sorry, long preganglionic neuron short postganglionic neuron. We're going to stimulate it. So number four, we are going to stimulate the vagus nerve. And when you stimulate the vagus nerve, what happens your heart rate goes down because the vagus nerve inhibits the heart. We're increasing parasympathetic tone. And so as a result of this, we're going to decrease heart rate one, by the way, what's that called a negative Crona, Tropic effect, you learned that last lecture. Or maybe it was two lectures ago, I don't even remember. So the parasympathetics are going to be stimulated, what's going to happen to the sympathetic nervous system, which is here in the thoracic, thoracic and lumbar region, we're going to inhibit and so let's put a minus sign over here, when it comes to this preganglionic neuron. Here's our postganglionic neuron, which will also synapse with the SA node will branch off of that, because we're also going to synapse with the ventricles. I'll actually send offs off of that as well, or branch off of that I should say, thoracic region is going to be controlling some blood vessels, the lumbar region is going to be controlling some blood vessels. And we're going to inhibit them all. We're going to inhibit inhibit, inhibit, inhibit. And that's still number four, because this is all happening at the same time. And so when you inhibit the sympathetics, what is that going to do to the heart, it's going to inhibit the heart. And so this two, we're going to get a decrease in heart rate. So the parasympathetic and the sympathetic are going to work together to inhibit the heart. Why? Because the neurons in the basal motor in the cardiac control center are dictating that. So once again, negative chronotropic effect. Over here, we're going to get a decrease in contractility. The heart is not going to contract as hard. So the heart is going to slow down and not contract as hard. And as a result, the decrease in contractility, it's going to decrease stroke volume, which is a negative. I know tropic effect. Right? This is exactly what we have to have happen to lower blood pressure. Now, what are we going to do to these blood vessels? Well, what we want to do is that we want to lower total peripheral resistance, of course we do, going back to our very simple equation. So we've just made cardiac output go down. Now we want to make total peripheral resistance go down. And how do we make total peripheral resistance go down? Well, we control this blood or the blood vessel tone, we control the radius, we can make the Radius bigger, you make little r big. That gets smaller. So what are we going to do here? Here's my picture. So we're inhibiting the smooth muscle of this blood vessel. There's the negative sign. And what's going to happen as a result number five, oh, and by the way, a decrease in

heart rate is five, a decrease in heart rate is five, a decrease in contractility is five. And so two is vasodilation. Which is, as we know, an increase in little r, which means we have a decrease in total peripheral resistance. And when you do that, you dilate the blood vessels. I'm going to put one more thing, actually two more things in this picture, because it's going to come into play when we talk about some drugs that are used to treat hypotension and hypertension. I'm going to remind you that those are beta one receptors in our Member beta one heart, as soon as you hear beta one, I want you to think heart that's from last semester, beta ones over here, too. And these alpha ones, if you remember from last semester, so this is a bear receptor reflex. So now let's take this stuff that we have here up on the screen, and I'll write here baroreceptor reflex, just to remind you that this is what this is. This is the baroreceptor reflex when blood pressure gets too high, not too low. So now looking at this, what's happening to cause us to come down to where we're supposed to be. What's happening here is this is that we are stimulating the parasympathetics, which leads to a decrease in heart rate, what we're doing is inhibiting

21:36

the sympathetics, which also decreases heart rate, but it also decreases stroke volume, and it also increases radius, which decreases total peripheral resistance, there's a couple more things we're going to add to this because there's more stuff we're going to talk about. And if blood pressure was too low, the complete opposite is going to happen. I just want you to draw it all out. But we'll throw it in here. We're going to inhibit the parasympathetics. We're going to stimulate the sympathetics. And when you inhibit the parasympathetics, you increase heart rate, when you stimulate the sympathetics, you're going to increase heart rate, you're going to increase stroke volume, you're going to basal constrict, and therefore increase TPR. So what I just did right there, is this part of the notes

22:33

right? Here,

22:38

which I didn't draw, but I wanted to throw it just to be thorough in the blood pressure homeostatic line. All right. So Bear receptor reflex happens. Just like that really, really, really, really fast. And it's happening on us right now. This doesn't stop. Your bear receptors are never done. Detecting blood pressure, at least they better not Yes. Stimulate the vagus nerve,

23:05

vagus nerve. So one of the things

23:11

that is the only thing that you need to be aware of when it comes to the vagus yet the pecs heart rate, pretty tractability. That's sympathetic. Yeah, that's all this stuff will be here from the thoracic region

region.

23:28

Yep, makes him do that dilates. Those were inhibiting. Here, the sympathetic inhibit the sympathetic to the specific blood vessels. They dilate. So the radius gets bigger. And so the resistance goes down, which is what we want. We want result, we want to lower blood pressure. So we need to make this smaller, and this smaller. And that's exactly what's happening here. All right. Thank you. You're welcome, sir.

23:56

Now what? Well, that's not the only thing is regulating our blood pressure. vagus

24:07

nerve stimulator all simultaneously, simultaneously inhibiting the sympathetic. So happening same time. Yeah. So what else is regulating your blood pressure? So the first two are the big dogs, the varicella reflex. And now this. So let's talk about this. This is actually something that we've already discussed, but not the kind of detail we were allowed to discuss it and we discuss this in the endocrine chapter, when we're talking about dosterone. And I told you at the time, that reading causes the release of aldosterone, but does it indirectly and it was stuff that you needed to cross out. And I told you because we're going to talk about it later. This is later. So what we're going to do now is we're going to go over the renin angiotensin aldosterone system. And so let's put a kidney here and I'll label it kidneys on top of the kidney, adrenal gland We're going to put a blood vessel in this patient. And I only want to draw a blood vessel, I draw it vertically, I'm going to draw it horizontally and nice and thick, because there's stuff that we have to draw within the blood vessel itself. So here's a blood vessel. You know, label it. Now in those blood vessels is a plasma protein, you guys know what plasma proteins are proteins that are in the plasma, it's got a special name angiotensinogen. That is just a protein floating around in the blood. And that protein is going to be converted into another protein. And that other protein has been is going to be called or is called angiotensin one. Now we need an enzyme to catalyze this reaction. And that enzyme just happens to be rennet. And that's actually going to be number one, the release of random red is released into the blood. And what red is going to do is it's going to catalyze that reaction, Reddit is responsible for turning angiotensinogen into angiotensin one, which is going to be step number two. And then angiotensin one is going to be converted into another molecule called angiotensin two. Now we need an enzyme to do that. But rennen is not the enzyme that's going to do that. The enzyme that's going to do this is called angiotensin converting enzyme otherwise known as ace. So angiotensin converting, for lack of a better name, enzyme, otherwise known as angiotensin converting enzyme, eights, you guys ever heard of an ACE inhibitor? It's used to treat high blood pressure, we're going to talk about it later when we talk about high blood pressure. Now, angiotensin one, it's in the blood, its target. And this is going to be number Oops, got an eight. This is number three. This is going to be number four. Its target is the adrenal gland. And so that's going to be number four, where it's going to stimulate it. It's going to stimulate the adrenal gland to release you guessed it dosterone. So at the beginning of the semester, when we were talking about the endocrine system, I told you reading causes the release of aldosterone. And I said it does it indirectly, when I'm showing you exactly how it happens. We need these reactions to occur in the blood. Now, what does this have to do with blood pressure going to show you so angiotensin two is actually what it's going to revolve around. So that's what I'm gonna

put here is angiotensin two does a number of things, that's going to allow us angiotensin two to regulate blood pressure. Now, when angiotensin two is released, it does a number of things. One obviously is it causes the release of aldosterone, so I'll throw that in here. So it causes the release of aldosterone, we already see how that happens. It also causes vasoconstriction. It also causes

28:34

the release

28:37

of at ADH. Now we know how aldosterone works. We've talked about earlier on in this semester, aldosterone is going to work in a couple of different ways. We know one way that it works. And that is it increases blood volume by causing the transport of water because it follows the sodium that aldosterone facilitates the kidneys to transport into the into the blood and it also basal constricts. So we can see here that as all these things are working together, oh, and by the way, ADH does the same thing as aldosterone, but in a slightly different way increases blood volume and also causes vasoconstriction. So we see that we have these effects when it comes to blood pressure, and that is to raise blood pressure, although angiotensin two is chronically being produced. Aldosterone is chronically being released. And ADH not that it plays a huge role in this, but it'll also be chronically released because of this. And so this is what I want you to get out of this now. So that's how those players are interrelated to each other. This is what I want you to get out of this as well. And this this, it's not even so much all this stuff. It's this So

30:04

as we increase the production of angiotensin two and therefore, of course, the release of aldosterone, as you increase in both, what that's going to do is

30:28

increase your blood pressure. How does that happen? increasing blood volume vasoconstriction. All right,

30:37

as we decrease the production of angiotensin two, and therefore, decrease the release, let me put an arrow over here, decrease, that should be going up. So this should be increased production, which means we would increase the release over here, it would be decreased the release of aldosterone, the complete opposite is going to happen. We're going to decrease blood pressure, again, they're being chronically released, if you release more, you produce more blood pressure goes up, if you release less produce less blood pressure goes down. So that's how this is going to regulate blood pressure. So now what we'll do is based on this, based on this, we're going to add to the story right here. So over here, our blood pressure was high, we need to lower so what are we going to do, we're going to decrease the production of angio tensin. Two, and therefore decrease the release

31:47
of aldosterone.

31:52
This system right here that we just went over, not anywhere near as fast as the autonomic nervous system, of course now, but boy, is it powerful, it's slower, but very, very powerful when it comes to regulating your blood pressure. Now we'll be thorough, certainly, then, if your blood pressure is too low, and you need to raise it, well, then you're going to produce more angiotensin two, so increase the production of angiotensin two that will raise your blood pressure, of course it will, which would then cause an increase in the release of aldosterone. So there you go. So they're working together to maintain a normal blood pressure. And again, those are the two big dogs when it comes to regulating your blood pressure. Now there is one other thing that I'm going to add to the story. And that is atrial natriuretic factor. Sometimes instead of factor, we call it a peptide. Sometimes, instead of that we call it a hormone most often is called atrial natriuretic factor, which is why I'm going to abbreviate it. And this is something that's produced given the name by the atrium in response to high blood pressure. So to add this to the story, I'm not too terribly concerned about if you know the exact mechanism by which BNF works, I just want you to know the punch line of the punch line is going to be added to this picture. When your blood pressure is high, we will produce and release more atrial nature erratic factor. And when your blood pressure is low, you're going to decrease the release the production, the release of atrial natriuretic factor. So these three things are going to be working together again, the first two are the big ones, very receptor reflux renin angiotensin aldosterone system, those are the two big ones and f is going to contribute. This is how you're keeping your blood pressure right here. That's how it's happening. minutes a minute, second, second hour to hour day to day. This is how it's going on. Now, over here, we have a couple of other things that I'm going to mention. These are only going to be coming into play during emergency situations. Even though ADH is released by this system over here, it's playing an itty bitty role. The ADH is main job is what tell me regulate one ADH, blood, plasma clarity. That's his main job. But if we have an emergency situation where blood pressure is too low, we don't give two craps about osmolarity at that time, we care about we care about the blood pressure. Why? Because you don't take care of the blood pressure, you're dead. It's okay that your blood osmolarity is going to be off for a little bit as long as you're alive. And so the body is going to respond when your blood pressure is too low. It's called hypotension by releasing a crap ton of ADH to try to raise your blood pressure also releasing epinephrine and norepinephrine. So these are hormones that we talked about beginning of the semester when we did the endocrine system. And at the time, we talked about how not specifically with ADH, because I told you don't worry about the blood pressure story. We talked about it later. Well, this is later, these two things are not regulating your blood pressure on a normal basis. These things are trying to desperately raise your blood pressure when somebody is about to die because their blood pressure is too stinking low. And so how does epinephrine and norepinephrine raise your blood pressure increases your heart rate increases contractility so increases your cardiac output, and increases your total peripheral resistance. It goes back to how many times is this now four. It goes back to this equation right here. Math people, cardiac output types, total peripheral resistance again, over and over and over again, when it comes to blood pressure, that equation is going to help you understand why. Last thing before we take a break. And that is to talk about a factor that affects your blood pressure. This is not regulating blood pressure in any way. But it will affect your blood pressure. And it's called compliance. When you see or hear the word compliance, I want you to think of a couple of words. And I'll actually write it down. So compliance. It's a measurement, we're gonna see this again, when we

get to the respiratory system. So compliance, and I'm talking about compliance of blood vessels by the way, specifically arteries, but I'll just say blood vessels because veins have compliance as well. So compliance is how stretchy, that's one word, I want you to think of how stretchy how stiff. The other word. So the two words I want you to think of is stiffness stretchiness

36:53

a blood vessel list that's what it is. And there's a normal compliance. Compliance can be too high. Compliance can be too low, we're gonna we're gonna focus on the to low compliance. That's the story I'm going to tell you. high compliance bad low compliance, bad, normal compliance. Good. And when it comes to normal compliance, this is what I want you to know. During systole, and diastole, the compliance and again, we're talking about normal complaints. So with normal compliance, and before I move forward, I'm going to give you an analogy when it comes to compliance, we're going to talk about a rubber band. Rubber bands are compliant. They're stretchy, but there's a certain stiffness to them. I can take a rubber band and I can stretch it. And if I let go of that rubber band, what's it going to do? A snapback right, it's going to recoil. So that word recoil is an important word for you to to understand. It's just a rubber band. It snaps back, that's what recoil is. So with normal compliance during systole.

38:07

Again, because of compliance during systole arteries that I'm going to stick with the artery story here arteries.

38:18

They stretch.

38:21

So there's that word stretch. Now, is there a benefit to this? Absolutely there is. So what's the benefit? It's going to diminish the force on the walls. So it decreases force against the walls of the blood vessel. And what that's going to do is it's going to lower the pressure a little bit. So it decreases blood pressure just a little bit, I'll give you an analogy. If I came up to you and I want to punch you in the face, the last thing you want to do is walk into my punch, what do you want to do, you want to back away from the punch. Because if I hit you, the force of my fist is not going to be as hard against your face. Every time blood is ejected into the arteries during systole. The walls of the arteries are getting punched with blood. The arteries themselves are going to dissipate some of the force by stretching a little bit. So that's the benefit. So it lowers the pressure during systole. When you take a blood like the blood pressure, I talked about 110 over 70 It's 110 because of the normal compliance, and we'll talk about when compliance isn't normal what'll happen to blood pressure because during diastole and so let's recall as we just talked about it is that we get stretch during systole like I just stretched a rubber band what happens when I let go and over band recoils, so during diastole. The arteries, recoil, they snap back. Well, is there a benefit to that? Yes, there is. And what this is going to do is it's going to increase the force against the walls of the blood vessel. Obviously, diastolic pressure is lower than systolic pressure during systole, blood flow is better than it is during diastole, because the pressures

during diastole are lower. So this is going to increase blood pressure just a little bit during diastole. So the 110 over 70 value that I gave you wouldn't be 70. Maybe it would be 60. If it didn't snap back hard enough, if it didn't squeeze the way that it does. So that's the benefit to this. That is when we have normal compliance. All right. Now, what about one compliance isn't normal, and the story we're gonna go over is a decreasing compliance. And if you have a decrease in compliance, what that means is, is that we're too stiff. If we're too stiff, now during systole.

41:34

We're going to have a

41:35

increase in systolic pressure, it's not going to diminish the force the pressure on the walls as well, it's not going to be a stretch as well, it's too stiff. It's like you walked into my punch, you're not backing away from it during diastole. Because the arteries are now too stiff. Not gonna snap back as hard because they didn't stretch as far.

42:05

And so now you're going to get a decrease in diastolic pressure. And as a result of this, you're going to get an increase in pulse pressure. But the hell's pulse pressure doctor or

42:23

pulse pressure is systolic pressure might have cytosolic pressure. If this number gets bigger, and this number gets smaller, pulse pressure is going to get bigger. So if you have a patient with a high pulse pressure, it could indicate that their arteries are too stiff. Now what would cause this? Cause arterial sclerosis Well, what's that? That sounds kind of like atherosclerosis. Well, Atheros, this is hardening of the arteries, that is, by definition, what it is. Atherosclerosis is hardening of the arteries because of plaque. arteriosclerosis as hardening of the arteries, for whatever reason, clack, you got old, you do some stupid like smoke, that kind of stuff. All right, let's take a break. When we come back, we will continue our story.

43:25

Okay, folks, here we go. Yeah, before we move off of compliance, I want to talk about one more thing. And that is your pulse. So when you take somebody's pulse, now there was my boxing analogy that I was going to use. So when you take your pulse, what do you do is that you take and feel a part of the body where you have a very superficial artery, like a radial artery, for example. So you take some of these blocks, you might grab by the wrist, and put your middle finger there or whatever finger not your thumb, but one of your fingers, and you feel something you feel a pulse. What are you feeling? You're feeling the stretch of the arteries? During systole? Right? So during systole is when you have that stretch. And during systole is when the heart contracts, well, what is the pulse, it's measuring your heart rate, how many times your heart contracts in a minute. And so that beautiful property that

we have where the artery stretch allows you to very easily measure somebody's heart rate. So if you could know that, please, that would be fantastic. So again, superficial arteries, is how you can easily measure a pulse. Now, let me ask you this really quick. We'll actually add this to the story. If somebody has a weak pulse, you ever hear that term? Couple of reasons. I mean, there's a number of reasons we're going to mention too. One of them is and we're just going to stick with our compliance story is, is that we have a decrease in compliance. The arteries are too stiff. They won't stretch us So you might not feel it as well, this person could have arterial sclerosis could be an indication of that. Also, low blood pressure, if the pressures aren't that high, we're not going to stretch the arteries as much, because they don't have to be stretched as much, there's not going to be as much force on the arterial walls. So it can certainly indicate a very low blood pressure. And that's a nice segue into the next thing we're going to discuss and that is low blood pressure. Now, low blood pressure is good. Really low blood pressure is bad. And so that's what hypotension is. And so when you hear the word hypotension, I want you to think, abnormally low blood pressure. And so why is it so bad? Well, there's going to be two reasons. One we've discussed when we haven't discussed yet. One is your blood pressure gradient is going to be too low. And so what's our blood pressure gradient about? Well, that goes back to Pathways law. And so that's here. So we have blood pressure gradient, right? If that's too small, if the pressures are too small, well, then that's going to be too small. So you're going to have poor blood flow. And if you have poor blood flow, you're not going to be able to, to feed the organs of the body what they need oxygen and nutrients, they don't get enough of that they don't make enough ATP, they die, cells die, tissues die, organs die, people die. So for lead organ failure in death, if your blood pressure gets too low, and it goes back to possui, as well, because this is too small. So that's one thing that we've discussed. And actually, I mentioned that when we first talked about plus weight as well. The other reason is, is that we read something called critical closing pressure. We haven't discussed that yet. And all that is and it's defined in the notes, right here, blood pressure at which your blood vessels collapse, you need a certain pressure within your blood vessels to keep them open. And if your blood pressure gets too low, flat is pancake. So we don't only have no low blood flow, we have zero blood flow, we have ischemia. So we run two risks here, obviously low blood pressure gradients into even worse, we reach critical closing pressure. Now, we have replaces law here we have another equation, I don't care about any of it. All I care about is you know what critical closing pressure is and that is blood pressure, which blood vessels collapse, which will then cause ischemia. In other words, right there. That's the most important thing I'd like you to know. There's a certain pressure which we need to be within certain arteries and arterioles. It depends on a lot of different things, which is why I'm not going to give you what the pressure is, it doesn't matter. Just know that if your blood pressure gets too low blood vessels can start to do this, and I'm talking arteries arterioles. And not veins, talking about getting the blood to the to the blood vessels. Now why on earth would we have hypertension,

48:02

we're going to talk about shock, circulatory shock, so circulatory shock, severe decrease in blood pressure, which leads to obviously inadequate blood flow or no blood flow, three types that we're going to discuss hypovolemic, basal directory and cardiogenic. So I'll just define and quickly hypovolemic means we're losing too much blood. This goes to hydrostatic pressure. If you don't have enough blood in your circulatory system, your pressure is too low. And we started to learn that last semester in chapter three, if you remember, if you dilate your blood vessels too much, your total peripheral resistance goes down.

48:42

Going back to it.

48:44

Map equals cardiac output times total peripheral resistance if this number gets too small, because the radius is too big, blood pressure is going to plummet. And then there's cardiogenic shock, where the heart is inhibited so much that heart rate is too low. stroke volume is too low cardiac outputs too low, blood pressure is too low. So now we're at a half a dozen times referring back to this particular equation. So it almost always goes back to this equation when it comes to what your blood pressure is, because of these variables right here. And the most the the variables that you aren't going to be most concerned about heart rate so fine. And the radius. Okay, not discussing that friction so much. So now getting back to the story. Let's talk about the reasons why these shock types of shocks will have shock will happen. And so here's our list. hemorrhage is a fancy word for bleed. So hemorrhagic shock is shot due to somebody bleeding too much to be any kind of bleeding from a gunshot wound trauma, whatever doesn't matter. And so that would be a type of hypovolemic shock and so bleeding that causes a loss of blood blood. Very straightforward. anaphylactic shock. We're dilating the blood vessels too much. And this is a severe allergic response to something. And I'm not talking about the tree pollen that's in the air right now, that's causing you to have itchy eyes and a runny nose. That is an allergic reaction. But that's not a severe allergic reaction like your how bad your allergies are. You're not an anaphylaxis at that time. These are allergies to things like peanuts, for example, where we have a severe allergy to peanuts, shellfish, bee stings, to where you release a ton of inflammatory chemicals in the body, one of which we talked about not much at the time. Histamine it's just to me part of the allergic response. Don't you take antihistamines when you have allergies like Desertec and Claritin, and Allegra and Benadryl, those are all antihistamines. But the amount of histamine that's being released because of tree pollen, for example, is nowhere near the amount of histamine that's released if you have a severe allergic reaction to peanuts, and bee stings and whatever. So you get a ton of histamine release. So what does this mean? Do you already know, it causes basal dilation, which would then decrease total peripheral resistance immensely. And so once again, I know you're sick of me showing you this.

51:19

Well, you know where the hell it is. That's right there map equals cardiac output times current times total peripheral resistance. So with this to mean, that gets to stick in small, which means this gets to stick and small. So now you go into circulatory shock. What else? I think the next one is septic shock. We've hit the trifecta here. This is a hypovolemic shock. This is a cardiogenic shock. This is a basal dilatory shock, sepsis is you have some infection in your blood, bacterial infection, whatever COVID, for example, cause sepsis to occur in a number of people. And so it is going to be a hypovolemic shock because we start damaging capillaries, and they just start to spew blood. So we're losing blood from the blood vessels from the circulatory system, it causes your blood vessels to dilate, it relaxes the smooth muscle, it causes an inhibition of the heart where your heart rate goes down, and your stroke volume goes down because contractility is going down. So we've hit our Trifecta here, we know we have all three at the same time. Very dangerous when it comes to sepsis, because not only are we dealing with the decrease in blood pressure, but we're dealing with a systemic infection, that's wreaking havoc all over the place within the body. So very dangerous when it comes to sepsis. And emotional shock. You see something traumatic, and this is a defense mechanism, your brain shuts off because it wants to remove remove you from that emotional trauma. And I shared, I went into emotional shock when I saw my son break his leg when he was wrestling, I've doctor or went to sleep

for about 30 seconds. What happened to me, you might think that there'd be a strong sympathetic response at that time that it was a fight or flight situation, no, it was a go to sleep situation, because I don't want to see my son's leg bent in the way that it's not supposed to be bad. And so what happened there was I had a very strong parasympathetic response. And we know that your parasympathetic nervous system decreases heart rate. There's also a strong inhibition of the sympathetic, it's a complete this is the complete opposite of a fight or flight situation, literally the complete opposite. And so once again, when it comes to your sympathetic if we inhibit them, we're gonna basal dilate, decrease in total peripheral resistance. And we're going to get a decrease in heart rate, decreasing contractility decrease in stroke volume, decrease in cardiac output, Dr. arting, get enough blood to his brain, anyone to sleep for about 30 seconds or so and then it came to. So that's an emotional shock. And then we have a Neurogenic shock. And we're going to be affecting the controller of, of the heart and the blood vessels of blood pressure. And that's going to cause a strong inhibition of the sympathetics. There might be a strong inhibition of the parasympathetics. But the sympathetics, when it comes to change, because it's affecting the heart and the blood vessels, there's going to be a bigger response from the sympathetics, which is why just have the sympathetics. Here, we're inhibiting the heck out of the sympathetics big basal dilation, big decrease in cardiac output. Blood pressure plummets, once again, math equals cardiac output.

54:32

We go

54:36

this equate over and over this is eight times now I think I will start to lose count. Very, very important equation to understand. Now, if somebody is in shock, and they have really, really low blood pressure, how is the body going to respond to it? A body's going to desperately try to raise blood pressure. Of course it is. Well how do we raise blood pressure number nine. We increase heart rate we increase stroke volume, we increase total peripheral resistance, we basal constrict. That's how the body has a means by which to increase your blood pressure. Will it do that? Of course it will. Absolutely, it's going to if those weren't the problems. So if the heart is not the cause, if it's not a cardiogenic shock, in the heart has the ability to increase its rate and increase his contractility. It will absolutely do that to increase blood pressure. Again, if the heart is not the cause. Basal constriction, if the blood vessels are not the cause, we'll get a basal constrict the heck out of the blood vessels to increase total peripheral resistance. So the body is going to respond the way it needs to respond for survival, if it has the means by which to do that. So if somebody is bleeding to death, for example, you can be guaranteed that their heart rate is going to be through the roof, you can be guaranteed that their blood vessels are going to be vasoconstrictive. Oh, then why doctor? Or does it not work? Because you can only ask the body to do so much. It's losing too much blood. And the high heart rate. I contractility. The basal constriction just isn't enough to raise blood pressure. So are there times when patients are hypotensive? In circulatory shock, of course, go to what I see you. That's all they freakin do in ICUs, is treat patients with circulatory shock. Well, I wouldn't say all they do. But boy, a bunch of patients in the ICU is have really, really low blood pressure. So how do you treat it? There's a number of ways to treat it. So how can we raise blood pressure? Well, if somebody is bleeding to death, give them as much blood as you can. You can also give IV fluids, so increase blood volume, of course. What else can you do? You can give drugs that are called pressors. And what our presser is going to do pressors are going to work on blood vessels and the heart. And how are they going to work on blood vessels in the heart? Number 10. Increased heart rate, increased stroke volume, decrease

radius, which means increased total peripheral resistance. So how do we increase blood pressure, we increase cardiac output. How do we increase blood pressure, we increase total peripheral resistance by basal constricting blood vessels. So going back to our list, the first one one of the most powerful is epinephrine. And we already know how epinephrine works. What it does, I just showed you a little bit ago, it sitting right there. That for an effort increases blood pressure, how increases heart rate increases stroke volume, basal constricts. So we've hit the trifecta with this one. That's why it's so powerful, it works on everything. Dopamine is another very, very powerful one in high doses that will work just like epinephrine, it will cause an increase in stroke find increase in heart rate and it'll basal constrict it medium doses, it's just going to work in the heart. phenol Afrin this is going to work by Faisal constricting works on the alcohol. You know what I have beta agonist here. That's the reason I put the beta receptors in this picture. See that right there? Beta receptor, beta receptor. So epinephrine when it causes your heart rate to go up, works on the beta ones, when it causes the stroke line to go up, or some of the big ones when it causes vasoconstriction works on the Alpha ones. Alright, so that's what I mean by beta. Beta agonist, alpha agonist, that's what I'm talking about. Alright, dopamine, same thing, beta agonist and an alpha agonist at high doses, phenol efferent that works on our blood vessels at the Alpha One receptor. vasopressin, what's another name for vasopressin, tawny, antidiuretic hormone, it's the same freakin thing. vasopressin workstyle? Well, I already told you, ADH can cause vasoconstriction, it doesn't do the Alpha One receptor on the smooth bustle of those blood vessels and increases your blood volume. Now, which of these are going to use for your patient just depends. I'm not a clinician. So I'm not going to know exactly which ones to use. But if the blood pressure is really low, I'm going to assume that boom, you go right after that one right there to desperately try to cause the blood pressure to go up. And you might be using a bunch of these at the same time and giving blood and giving IV fluids you might be doing everything desperately to get that blood pressure up. Because if you don't, the patient's going to die. And it's not going to take long if their blood pressure is really, really low.

59:35

Now, let's go the other direction. Let's talk about high blood pressure. Now. Hypertension. There used to be a term called borderline hypertension, there used to be a term called pre hypertension. Those terms no longer exist. We don't use those terms anymore. Why? Because the American medica American Heart Association said we're not using those terms anymore. I think I've been teaching here at Wright State for 15 years, I've had to change all this stuff at least five times in those 15 years, I just keep on changing what's considered stage one, stage two, what the names are just keeps on changing all the time. And these are the latest things. And this changes right before COVID. So newest terms and and criteria, and when it comes for criteria when it comes to hypertension, now we have the word elevated, that has replaced borderline and pre hypertension. This is not hypertension. And that's probably one of the reasons that they changed the word to elevate it, because it used to be borderline hypertension and pre hypertension and might have fooled people in thinking Oh shit, I have hypertension. No, you don't. If you have elevated blood pressure, you do not have hypertension, that will not be your diagnosis, although this thing is is that creeping a little high right now where you might become hypertensive. And it's based on just the systolic pressure anywhere between 120 and 129. When it comes to your systolic pressure, you are said to have elevated blood pressure. If you have a patient, they have a systolic blood pressure 124, they're fine. They don't have anything to worry about, let's just kind of keep an eye on things. It's more or less what this means. diastolic pressure, they don't even take it into account when we use the term elevate. Now we get to stage one, this is true hypertension, a systolic pressure between 131 39 and or a diastolic pressure of 80 to 89. What's that? And orbing? Well, I'll tell you. So I'm going to put stage one here, I'm going to put just a number of blood pressures that would be indicative of a stage one diagnosis. So stage one, hypertension. Stage one hypertension would be a blood pressure of 132 over I don't know 7272 is

fantastic for a diastolic. But because that is within the systolic range that dictates a stage one hypertensive diagnosis. In stage one, both what I'm saying is both the numbers don't have to be high. Just one does. All right. Or we can go 126, which is still elevated, hypertensive over 86, the 86 that's the diastolic pressure that makes that stage one, and then certainly both numbers are high. 101 34 over 82. Well, that's certainly stage one, because both of those are within the range when it comes to start and diastolic. So that person has stage one hypertension, that's the diagnosis, stage two hypertension. Same story, just higher numbers. And so we'll put a few over here. So we'll go I don't know 148 over I can make the number 70. I can make the number 80. Something I can make it whatever the hell I want. You know what, what I'm actually gonna make it is 8282. diastolic is that stage 182, diastolic stage one. Yep. 148 systolic stage do. That trumps it. That's not stage one, that stage two, because the systolic is within the stage two range. And that's going to trump the stage one of the diastolic, or this could be 72 or 60. And then, as soon as that number is 148, for systolic and it's within the stage two range, it's stage two automatically. And I can go here, I don't know, we'll go stage. It doesn't matter. 9192. There. Now we're stage two. All right. So again, the andorre is telling us that only one of the two has to be within the range. And then we'll just do one more just for sake of thoroughness. No, no. 152 over 96. They're both definitely stage two.

1:04:05

Right. Now, once we get above 160. Well, now we're said to be in a hypertensive crisis, or I'm sorry, 180. Now we're in what's called a hypertensive crisis, or a diastolic Above 120 is considered a diastolic or I'm sorry, a hypertensive crisis. That's a brand new term, by the way, I never used to use that term before a few years ago. It's still stage two, by the way. hypertensive crisis is still stage two hypertensive crisis is not it's not another diagnosis. It's just saying, Damn, that's high right now, is all it's saying. We're in a crisis. Now. When somebody does have hypertension, over 90% of the time, you're not going to know why. That's what we call essential or primary hypertension, the majority of cases over 90% You don't have a clue why they're high why their blood pressure is high. Now you might have a guess theory as to why it's high. But you don't definitively know why it's high, because there's so many stinking things that can affect blood pressure. The times that you do know what the cause is, it's called secondary hypertension. Like if somebody has a certain type of kidney disease where the kidneys are not regulating blood volume properly, and blood pressure goes up, okay, it's due to the kidneys and you treat the kidneys to see if you can lower the blood pressure, while you might still treat the high blood pressure because while high blood pressure back, one of the things I want to add here is this. You guys ever hear white coat hypertension? Who wears white coats? You guys, someday, I'm going to wear white coats. You guys are going to be scary to patients in those white coats, especially when you walk up to them. If you ever take a blood pressure, you take that sphygmomanometer and that it's got the Velcro that makes that ripping sound, all of a sudden, patient's gonna start to get a little bit nervous. Then you put it around your arm, and you pump up as you just push a button nowadays, and now it starts to squeeze there, our patient gets even more nervous. What are they going to start to release?

1:06:05

What kind of hormones

1:06:07

stress hormones, cortisol, epinephrine, norepinephrine, ADH, what are those things that your blood

pressure may can go up. So it might be artificially high, the patient is nervous. That's what white coat hypertension is. So you got to be a little bit careful when you're measuring the blood pressure the patient has Are you a little bit nervous right now it's okay, you might have to measure it a few times wait for them to relax, and don't measure their blood pressure. As soon as they walk into the room. Let them sit there for at least Oh, actually nowadays, when you go into a doctor's office, you do sit there for about a freakin hour and a half before anybody does come in. But you want to make sure that they're as relaxed as possible. So it's a true true reading. You might also want to ask them, What is your blood pressure normally, because a lot of people know what their blood pressure is, especially because you go to buy right now and buy a stick an automatic blood pressure, cuff and whatever for like 20 bucks. So it's easy to do at home. And you might even ask your patient, you know, let's say it's kind of a 132 over in the at somewhere acid, can you measure your blood pressure every day for a couple of weeks at the exact same time and tell us what it is because again, they might best be so stinking nervous, that it's high because they're nervous and and it's not really normally highlight that it doesn't matter. In the end, when it comes to what you need to know on the exam. That's what you need to know on the exam, I decided throw that stuff out there. Now. Everybody knows high blood pressure is bad, why? These are the kinds of things that high blood pressure do to your body. It's going to make your heart work harder. So it's going to fail quicker, your afterload is too high, we talked about afterload you make your heart work harder, it's not going to last as long. So increase your risk of heart failure. It's gonna damage your blood vessels, those high pressures against the walls stress caused inflammation. We know that blood clots develop with inflammation, we know that atherosclerosis the plaque and develop because of inflammation. And now we have another problem. The number one cause of an aneurysm and all an aneurysm is is a blister on a blood vessel. That's just waiting to pop. The number one cause of aneurysms is high blood pressure. The aneurysm itself is not dangerous. But if they pop, that's when they're dangerous because now you got a blood vessel that spilling blood. And if you have a blood vessel, like an aorta, where you have a ruptured aneurysm, well, you might bleed out within a couple of minutes. Or one of the brain you might stroke out or die. Very very dangerous. What else you can actually rupture blood vessels now because of an aneurysm but you just freakin rupture the blood vessels, like smaller ones in your eye, for example, can lead to blindness. organ failure, because you're not getting good blood flowing out of the organs. Why? Because blood vessels just spewing blood where it's not supposed to be going to go into the cells, not the interstitial compartment. Now, when I was just about to say some Oh, yeah, this so when it comes to high blood pressure, let me ask you this. symptoms. This is not the notes of hypertension. What are they?

1:09:26

There are none.

1:09:29

There are no symptoms of hypertension. People will say I got a headache today. I think my blood pressure say no, it's not the way it works. You know, I feel tired today. I think my blood pressure is I know that's not the way it works. This is why they call hypertension. You ever hear this term? The silent killer. A ton of people are walking around with high blood pressure and they don't know it. Most people know not most everybody who has hypertension who's been diagnosed with it, I should say know it because it was measured. Not because they had some symptom. It's not the way that it works. And again, that's why they call it the silent killer. And again, there's a lot of people walking around. They don't have a clue that they have hypertension. And it's been going on for years, and

years and years and years. And what kind of things happen? Well, these kinds of things happen. And so then they go, you know, they have some big, catastrophic, catastrophic event. Why did you know your blood pressure is, you know, 180 over, you're screwed, you know, high and said, No, I had no idea because they had no symptoms. And so I want you to know this, please know that there are no symptoms, there just aren't any. All right. Now, last but not least, how do you treat it? I'm not a big drug guy. I think you probably know that I like to see if we can treat things with changing lifestyle. And that is a very powerful method at lowering your blood pressure. These are studies that were done some time ago that showed that nearly three quarters of the people who were overweight or obese that last week to a more healthy weight, who had high blood pressure, it went away. I actually the person who used to teach here before Siebert one day he had to go, he had to, he had to pee all the time. I find that strange. I just finally asked him what the hell you just drink that much? Why? I said, No. I'm on this blood pressure medication that makes me pee all the time. And I was very surprised because he was young, like young, like Seaver, like in his 30s at the time, and he was a bit overweight. And we were good enough friends that I could say, You know what, I bet you if you just lost weight, you wouldn't have to be on those meds anymore. So I helped him with a workout plan and an eating plan, he lost about 25 pounds, he's no longer on those blood pressure medications anymore. That's all he needed to do was lose weight. Now, that doesn't always work. And that's not to say that people who are thin and in shape, don't have high blood pressure, because they absolutely can. And so what do you do for those people? Do you do the complete opposite that you did for people with hypertension? What are you going to do for those people who have hypertension? Well, I think we've hit it now a dozen times lower heart rate, lower stroke volume, increased or decreased total peripheral resistance by basal dilating. Well, what kind of drugs can do this? Well, how can you lower your heart rate and your stroke volume?

1:12:25

Beta Blocker

1:12:27

decreases the contractility of the heart and heart rate. How can you basal dilate? alpha blockers, decrease peripheral resistance you vasodilation calcium channel blockers, what are those going to do? Now we go back to the chapter that we started this whole cardiovascular hard circulation material. And that is those calcium channels are part of the what the action potential of the cardiac conduction system remember that that's responsible for the fast depolarization. It's also responsible for the slow depolarization of pacemaker potential. You don't get as much calcium it, it takes us longer to get the threshold. As long as you get the threshold. You're not gonna have as many action potentials, you're gonna slow your heartbeat down calcium during the plateau phase, how important is that? Very, you need that calcium because muscle to contract. If not as much calcium is coming in. The muscle won't contract as hard a decrease in stroke volume. A diuretic? We haven't talked about diuretics. Yeah, a lot of people got diuretics, water pills. We'll talk about it in kidney chapter. A diuretic is a is a medication that causes you to excrete fluid from the body. And so then you'll have fluid shifts, less blood volume, blood pressure goes down, by the way that that professor that taught with me was on a diuretic. That's why he was peeing all the time. He was he had drugs make him do that ace inhibitor. Let's go back to a picture that we drew. This one right here, inhibit this enzyme right here. You don't make as much angiotensin two, you don't release as much aldosterone. So you decrease your production of angiotensin two, you decrease the release of aldosterone, you lower blood pressure, so that would be an ACE inhibitor. What else angio block the receptors for angiotensin

two where are the receptors the receptors are? well beyond the adrenal gland block the receptor, you don't release as much aldosterone. Last but not least, don't release as much rent, you don't release as much rent and you will make as much angiotensin to your release as much Adastra. There are other blood pressure medications by the way. These are the ones that I want you to know. And it all goes back to map blood pressure equals cardiac output, times total peripheral resistance. All right. Okay, guys, all done. I will see you on Tuesday. By the way once our exam. It's a week from today. All right. Hello