>> Steve Langjahr: Today's October 18th. This is lecture 17 in physiology. We've embarked on this journey through the circulatory system. What's that organ that moves blood through this circuit?

[Inaudible]

>> Steve Langjahr: We consider the heart on Monday. Now we want to talk about the associated blood vessels, and you know those vessels that carry blood away from the heart are called?

>> Arteries.

>> Steve Langjahr: Arteries. Those that return blood back to the heart?

>> Veins.

>> Steve Langjahr: Those are called veins. The movement of blood through this circuit is obviously dependent on pressure. When you open the faucet at home water comes out. Not by gravity but by pressure. So the importance of blood pressure is without any possible doubt. It is the factor, the number one factor that propels blood. So let's start. Arterial blood pressure is just that. The pressure of blood which is created by blood pushing against the inside of arterial walls. Then this pressure, of course, is generated, it's created by the activity by the contraction of the heart, and it's also supported by the contraction of blood vessels. You might think a blood vessel is passive, it's just a tube, but as we'll see, arteries in particular can exert a force, which contributes remarkably to arterial blood pressure. The importance of this blood pressure is simply that it makes possible blood flow, and what matters in the circulatory system is not pressure, but flow. Once again at home you don't really care much about water pressure. You care about water flow, and so in the body we care more about blood flow, then blood pressure. Certainly blood pressure is important for this flow, but it is only one of many factors that we'll uncover today. So blood pressure is created by the contraction of the heart. It's also influenced by the condition of the blood, the condition of the blood vessels. And so there are other factors, but mainly this pressure is established by the contraction of the heart. Now with that said, the heart undergoes two phases in a normal cycle. The period when it's contracting is called systole. The period when it's filling with blood is called diastole. So arterial blood pressure is not a constant pressure, but it's a pulse style pressure. That means it rises and it falls with systole and diastole. So we really have to examine two phases, two periods during the contraction or activity of the heart, which create two very different blood pressures. So what's going on when the ventricle is contracting? When the ventricle contracts its putting a lot of blood into the aorta in one second, and the amount of blood that's being put into the aorta is called the stroke volume. It might amount to something like 80 millimeters, 80 milliliters. The interesting thing is though that doesn't sound like a lot of blood, but we're putting that blood into a vessel that's already filled with blood, and therefore there is some resistance to the introduction of this blood. And, in fact, this stroke volume cannot be moved forward during this brief period of time. Remember, systole

only lasts about a second. So during this period of contraction, only about one third of the stroke volume actually moves forward. There's too much inertia, there's too much drag, there's too much resistance to the movement of blood. And none of that blood can go back, because the aortic valve closes. So here's the question. If only one third of the stroke volume actually gets advanced forward, where is the rest of it go? It essentially has to be accommodated by the stretch of the aorta. The ballooning of the aorta, which is exaggerated in this image just to show that it is the aorta which absorbs or accommodates two thirds of the blood volume. And that's going to turn out to be important because what follows systole? What's that period of time after systole, it's called diastole. And is the heart pumping any blood outward during this time? No. It's filling with blood. So it's at this time that the aorta assumes a pumping role, because remember, it's been stretched during systole. So does it have a capacity to actually squeeze back now during diastole? And the answer's yes. So remember during diastole, no new blood is coming in because the ventricle is filling at this time, but now the aorta will squeeze back. We call this recoil, and essentially it's moving two thirds of the stroke volume forward during this more lengthy period of time called diastole. So these images remind us that blood pressure is not just a function of the heart, it's also a function of the contractal or recoil ability, especially of the aorta. And so pressures obviously rise and fall with systole and diastole. And I'm sure you know then that blood pressure is not reported as a number, but actually it's recorded as a fraction, systole over diastole. And so in a chart or in a report, what would the systolic pressure be? Normally it's 120. The diastolic pressure is 80, so the standard way of expressing or reporting normal adult blood pressure is systolic pressure over diastolic pressure. We can, and we should graph this. The vertical axis, the units for measuring blood pressure, M, m, H, g, what's that? Millimeters of mercury? Notice the peak is 120, the low end 80. And as you draw these curves in the space provided for you, be sure you make the systolic period short and the diastolic period longer. This is important. First, it's just a fact that diastole lasts longer than systole, and so the diastolic phase lasts longer than the systolic phase. The overall pressure then never falls to zero, because even though the heart is not adding more blood during systole, what picks up or otherwise creates pressure at that time is the recoil of the arterie. So blood pressure is reported then as a fraction, systolic over diastolic. There's another measurement that we can and sometimes refer to something called pulse pressure. Now, pulse pressure is not to be confused with pulse rate. Pulse rate is the same as heart rate. Pulse pressure is the difference between systolic and diastolic values, and what's a normal systolic pressure? One-twenty. What's a normal diastolic?

>> Eighty.

>> Steve Langjahr: Eighty. So what is pulse pressure? Forty. Now, why does that matter? Or when is that even useful? If this were a blood vessel filled with blood, and the pressure was constant, and we put our fingers on here, would we feel anything? No, the only time you feel anything when you put your hand on a vessel is when there's a difference between systolic and diastolic. So when you

take somebody's pulse, what you're feeling is the pulse pressure, the difference between systolic and diastolic. And normally that's about 40. In some settings you may hear the remark, "patient has a weak pulse pressure, a weak pulse pressure," or simply a weak pulse. Now, weak pulse doesn't mean the heart rate is slow, it simply means the difference between systolic and diastolic is much less than 40. Now think about it. What could cause a weak pulse pressure? Well, pretty easily. It would have to be anything that reduces systolic pressure or anything that might raise diastolic pressure. In usual practice, that means more often than not, when pulse pressure is weak, it's because systolic pressure is weak. And let's figure out why that's the case, and why that even matters. Systolic pressure is generated during systole, which is a function of SV. What's that?

>> Stroke.

>> Steve Langjahr: And stroke volume is a function of end-diastolic volume and also venous return. So this takes us back to Monday. Venous return is the amount of blood that's coming back to the heart. Yes. What's the number one factor that can lower venous return? H word. B word. Bleeding, hemorrhage. So if you're bleeding, would venous return be down? Would end-diastolic volume be down? Would stroke volume be down? Would systolic pressure be down? Would pulse pressure be weak? Yes. So the number one cause for a weak pulse pressure is because you're bleeding, and therefore systolic pressure has suffered, and this number falls below 40. So pulse pressure has its biggest utility. That means it's most important contribution to diagnosing mainly hemorrhage. Finally though, we come to this value which is called MAP, mean arterial pressure. And what does mean mean?

>> Average.

>> Steve Langjahr: It means average. How do you average two numbers? What's the usual method?

>> Add them together.

>> Steve Langjahr: Add them together, divide by two. So what are the numbers we're adding together? Well, 120 plus 80. You can do that in your head. One-twenty plus 80 is 200, divide by 2?

>> One hundred.

>> Steve Langjahr: A hundred. So if you were asked to calculate the average or mean blood pressure, you would add 120 to 80 and divide by 2, and you'd come up with? A 100. But that's not actually the case, because it goes back to our previous remark. Do both of these pressures persist or last the same amount of time? Which of these two pressures last longer?

>> Diastolic.

>> Steve Langjahr: Diastolic. Therefore, it's not appropriate to add these numbers and divide by two. That would give them equal weight. And which of

these deserves more weight in the computation of this average value? Well, it's diastolic. So with that mind, with that in mind, the normal MAP is not 100, but closer to the diastolic value. It's normally about 93. So what? What's the importance of mean pressure? Well, mean pressure turns out to be the prevailing average pressure, which is key or important for blood flow. In other words, when it comes to blood flow, we don't really care about systolic or diastolic. We care mostly about MAP. What's MAP? Mean arterial pressure. Now, obviously that number will fluctuate or vary as these pressures might change, but when it comes to blood flow our focus will always be on MAP. MAP, mean arterial pressure. So before we leave this page, we can just summarize, blood pressure has two values. The [inaudible] value called?

>> Systolic.

>> Steve Langjahr: Systolic. That normally is about 120. The low value? Diastolic. Diastolic is created by the recoil of the great vessels, especially the aorta. Systolic pressure created mainly by stroke volume introduced into the aorta. Before we leave those two values let's focus even more closely on the individual influences to each of these two pressures. So let's start with systolic pressure.

>> One question. How did you come up with the 93 if it's not [inaudible]?

>> Steve Langjahr: Well, actually you can go to your book and get the calculation. I didn't want to dwell on the math, but it's a good question, nonetheless, because if we just add these numbers up, we get what?

>> Two hundred.

>> Steve Langjahr: Two hundred. Divide that by 2?

>> One hundred.

>> Steve Langjahr: One hundred. That would be true if each of these pressures lasted the same amount of time, but do they last the same amount of time? No. Systolics very brief. Diastolic is very long. So rather than being 100, it's much closer to the diastolic value, simply because that pressure persists or lasts longer. But if you want the math, it's in your book. There's a way, there's an actual formula for calculating MAP. Back to systolic pressure. Let's start with the definition. I'll get you started. Systolic pressure is the pressure that exists in arteries during?

>> Contraction.

>> Steve Langjahr: Contraction, during systole. And it's normally what value? Okay. We'll go back. The normal systolic pressure is?

>> One twenty.

>> Steve Langjahr: One twenty, okay. So systolic pressure is the pressure that exists in arteries during systole, during contraction. The question is, what are the factors that can change that? Well, as you look backwards, you see that

most of what creates the systolic pressure is the volume of blood that's coming out of the ventricle with one contraction. And that of course is stroke volume. And stroke volume is a function of contractility, meaning how hard the heart is contracting. So what do you think? If stroke volume is high, that means we're putting a lot of blood into a space which is already filled with blood. So what would that do to systolic pressure? If the stroke volume goes up, then the systolic pressure is going to go up too. Make perfect sense? It should. You're simply putting more blood into an already occupied space. So if the stroke volume goes up, SP will go up. Is the opposite true? If the stroke volume goes down, systolic pressure will suffer. Easy enough. Next, heart rate. Heart rate, of course, is the frequency, the number of contractions per minute, which is normally about what? Let's say 60. What if the heart rate goes up? What would that do to systolic pressure?

>> Raise it.

>> Steve Langjahr: Yeah, because now we're putting stroke volume into that space more frequently, right. Cram, cram, cram, cram, cram. C-R-A-M, cram, cram. We're just putting that blood in there, right. Giving very little opportunity for the blood to move forward, and so yes, intuitively high heart rate will tend to raise systolic pressure. And the opposite is true. That is low heart rate tends to reduce S-P. What's S-P?

>> Systolic pressure.

>> Steve Langjahr: Systolic pressure. The next factor, a concept called compliance, and the reference here is arterial compliance. Now, before we go into this we need to understand some words here. Big vessels are called what?

>> Arteries.

>> Steve Langjahr: Arteries. The branches thereof are called arterioles. We're not talking about arterials here. We're talking about arteriole. Arteriole what? Compliance. Now what does that even mean, compliance? We're going to compare it to, and distinguish it from a similar sounding concept, namely elasticity. Here's a rubber band. The word "compliance," means the ability to be distended. Can a rubber band be distended? Is it compliant? Yeah. Is this compliant? No. So a rubber band is very what?. What's the c word? Compliant? Is the aorta compliant? That is it capable of stretching or ballooning, especially during systole. Now what happens to compliance of the aorta with age you think? Does an artery get more compliant with age or less compliant with age? Less. And that's because of a condition we all suffer from or will, and that's right here. Arterial what?

>> Sclerosis.

>> Steve Langjahr: Sclerosis. The common translation or definition for this word is hardening of arteries. In other words, as we age, arteries get not more compliant, but what?

>> Less compliant.

>> Steve Langjahr: Less complaint. More like a steel tube. Less like a rubber hose. So what? As we get older, what happens to the aorta? Does it become more compliant or less compliant?

>> Steve Langjahr: Therefore, we're putting blood into a tube which is not, c word? Not, what not?

[Inaudible]

>> Steve Langjahr: And does that affect pressure? What does it do to the pressure? If the vessel is less compliant, which is guaranteed to occur as we age, is that going to raise or lower systolic pressure?

[Inaudible]

>> Steve Langjahr: I hear both. Think about it. We're taking that stroke volume. We're putting it into a tube, which is not going to expand because it is not c word?

>> Compliant.

>> Steve Langjahr: And is that going to raise pressure?

>> Yes.

>> Steve Langjahr: You Bet. So with age the aorta becomes less compliant, and therefore systolic pressure will uniformly rise as a result of this condition. What condition? Arterial Sclerosis. So this should ring true with information or knowledge you already have. What happens to a person's blood pressure as they age? It goes up, especially the systolic because of this reduced c word. Reduced what?

>> Compliance.

>> Steve Langjahr: Compliance. And there's no way to change that. That is you can't say, well, I'll exercise. Well, go right ahead, but it's not going to diminish the fact that as you age, the Aorta becomes less compliant. Let's look at the other pressure. The other pressure is diastolic. That's the pressure that exists in arteries during?

>> Relaxing.

>> Steve Langjahr: During relaxation, during diastole. And that pressure is normally?

>> Eighty.

>> Steve Langjahr: Normally about eighty millimeters of mercury. What factors determine that? Remember, diastolic pressure is not a function of the heart as much it is the function of the recoil of the aorta, and the recoil of the aorta relies upon a different property. Not compliance, but what? Elasticity. Now, it's easy to think that compliance and elasticity are the same. Especially if you look at a rubber band. Is a rubber band compliant?

>> Yes.

>> Steve Langjahr: Yes. Is it elastic? Yes. But is everything that's compliant, also elastic? Think of bubblegum. I didn't have any, but, okay. Take the bubblegum out of your mouth. Is it compliant?

>> Yes.

>> Steve Langjahr: Yes. Is it elastic? No. So elasticity and compliance are two different properties. What happens, you think to the elasticity of the aorta as we age? Does it become more elastic or less?

>> Less.

>> Steve Langjahr: Less. And that means it's squeezing less during this period of time, this period of time called diastole. And if the aorta is not squeezing as hard during this diastolic phase, what would that do to the pressure that's otherwise created by that recoil?

>> It lowers.

>> Steve Langjahr: It lowers it. So with age the aorta becomes less compliant, it also becomes less elastic. And that would do what to DP? What's DP?

>> Diastolic pressure.

>> Steve Langjahr: Diastolic pressure. Now that might not match your expectation, especially with respect to age, because what is the usual expectation about blood pressure when we age? Well, the systolic does go up, and we explained that, but the diastolic pressure does not go down despite what? Despite the reduced arterial elasticity. So there must be another factor which intervenes or supersedes this loss of elasticity, and that's this. The resistance, the resistance to blood flow through the small vessels called arterials. Through the arterial network. The name for this resistance is PR, which is not public relations. It's peripheral resistance. It's essentially a factor of friction, and we speak of arterial rather than artery, because blood resistance through arteries is negligible because they're big. So where's the real bottleneck of the circulatory system? It's not arteries. They're very big. But rather these small guys. They're called what?

[Inaudible]

>> Steve Langjahr: So it turns out that the biggest factor in diastolic pressure is the resistance within the arterial network, something called PR, peripheral resistance. And that's a function of the size of the arterials, of course, but later in life it's also a function of a different pathology called atherosclerosis. Not to be confused with arteriosclerosis. Atherosclerosis is not hardening of arteries. It's lipid deposits in what?

>> Arterioles.

>> Steve Langjahr: Arterioles. That makes these vessels which are already small, smaller because they're filled or clogged with what?

>> Lipid.

>> Steve Langjahr: Lipid. And so think about it. If vessels become narrowed or obstructed by lipid deposits, what would that do to the ability of blood to move into these arterials during the diastolic phase? Would blood have easier or harder access as a result of atherosclerosis? Harder. And what would that do to pressure then? What would that do to pressure upstream? If those vessels that are narrow, the pressure upstream would be correspondingly higher. So when there's atherosclerosis, which raises PR or peripheral resistance, that's going to raise DP. What's DP?

>> Diastolic.

>> Steve Langjahr: Diastolic pressure. Now, this should correlate perfectly with your expectation. The expectation of what happens to pressure, blood pressure in the elderly? Does their diastolic pressure go up? Yes. Because of increased resistance within the arterial network. Does their systolic pressure go up? Yes, because of reduced, c word, reduced what?

>> Compliance.

>> Steve Langjahr: Compliance. And with that said, does their MAP go up? What's map?

[Inaudible]

>> Steve Langjahr: All right. Mean arterial pressure. Also what is the condition, the single word which afflicts many elderly. It's hypertension. You've heard of that, hypertension? High blood pressure. And it's a disease process, which is the conspiracy between atherosclerosis and arterial sclerosis. In other words, it's basically created by hardening of arteries and lipid deposits in arterials. The next factor and the only final factor that has any say in diastolic pressure is heart rate and contractility. And incidentally, let's distinguish these two things. Are they different? Yep. Here's rate. What's contractility? Right. Contractility is the force of contraction. Rate is the frequency of contractions. And if both of these were to go up. If rate and contractility were to improve, you're not surprised to find that that would also raise DP. DP, diastolic pressure. So it was all that said, when the rate and contractility go up, naturally, systolic pressure will go up as we mentioned, as well as diastolic. So these are factors that have to be understood, not just memorized, because they each alone, and collectively determine MAP, mean arterial pressure. But now we're going to do what we said we'd do. We're going to transition away from pressure, because remember what matters is not pressure. It's the f word.

>> Flow.

>> Steve Langjahr: Flow. And pressure is only one element that determines blood flow, but here's the formula and this chapter and this unit is full of mathematical formulas. The formula is BF, which stands for blood flow this time, is Delta p over PR. Delta P, the symbol Delta or the triangle means the

difference in pressure, specifically the difference between arterial pressure and venous pressure. Remember, we're talking about a circuit, which is a circle, right. And blood leaves the heart through what vessels?

[Inaudible]

>> Steve Langjahr: Comes back through what?

[Inaudible]

>> Steve Langjahr: What if the pressure at the arterial side were the same as the venous side. If the pressure was the same, there'd be no difference in pressure. There'd be no pressure gradient, therefore there'd be no what? Circulation. So clearly in order for blood to move, there has to be a Delta P. There has to be a significant difference between the arterial pressure and the venous pressure. In most settings, especially as we talk about the vena cava, which are the largest veins returning blood back to the right atrium. Venous pressure is zero, practically zero. So for that reason, we don't even usually deal with venous pressure, because unless something's unusual, it's nothing. So this formula really reduces to just MAP over PR. So even though Delta P is technically the difference between arterial pressure in venous pressure, in most situations, venous pressure is negligible. Therefore, we're talking to just about MAP. So Delta P is mainly determined by MAP. But what's this peripheral resistance? We mentioned it just a moment ago. It's abbreviated PR. Peripheral resistance means the resistance to blood flow in the periphery, and what vessels impose the greatest resistance to blood flow? You might think it would be capillaries. I did when I was a student. I said, well, obviously the capillaries are where all the resistance are, because they're the small ones, right. But it turns out capillaries are not providing most of the resistance, because there's so many of them. The real bottlenecks of the circulatory system are these guys. The what? The arteriolesls. So PR, as we've already said, is mainly established by the resistance to blood flow through arterials, but there are other factors too. Peripheral resistance is also determined by blood viscosity, because peripheral resistance is really a function of friction. And does blood itself affect the movement of blood through any vessel? Sure. Blood can be thick or thin. Right. And that is a rough description of the V word. What's that? Viscosity. Now viscosity actually refers to how sticky something is, but for our purposes we'll simply refer to how thick it is. And with that said, what has more viscosity? Well, let's see, water or ketchup?

>> Ketchup.

>> Steve Langjahr: Ketchup, okay. So clearly viscosity when it comes to blood is a function of the concentration of blood cells, which is, you know, is a measurement we took in lab. Remember that ratio of cells to plasma or remember when we put the blood through the centrifuge? You might even remember the name for that. It was hermatocrit. So the viscosity of blood is a function of the concentration of blood cells, and specifically what blood cells do you think? What are the cells that make up most of blood?

>> Red.

>> Steve Langjahr: Red. So we're talking about the concentration of red blood cells. So simply put, if we have a high concentration of red blood cells, then the viscosity would be what?

[Inaudible]

>> Steve Langjahr: And if the viscosity is high, so will the peripheral resistance be high. And if the peripheral resistance is high, what would that do to blood flow? This is intuitive, but it's also laid out for you mathematically. If this number down here gets big, then obviously the blood flow will suffer. So if the concentration of blood cells high, what's it going to do to viscosity?

[Inaudible]

>> Steve Langjahr: What's that gonna do to resistance?

[Inaudible]

>> Steve Langjahr: What's that gonna do to blood flow?

>> Decrease.

>> Steve Langjahr: Decrease. Now, this is interesting in the context of a tactic or a maneuver that a lot of athletes engaged in. You may know it. It's called blood doping. Blood doping is when an athlete will donate his blood, that is, give a lot of blood, and then come back for it two weeks later. Why would they do that? Well, in the two weeks he's replaced those blood cells. Yes. And now he's getting those cells back again. What's that doing to the concentration of blood cells and why would they even do that? What's the logic? What's the parent reason? Blood cells contain the –

[Inaudible]

>> Steve Langjahr: Oxygen carrying molecule called hemoglobin. Would that deliver more oxygen?

>> Yeah.

>> Steve Langjahr: Sounds good, but let's follow this through. Blood doping will raise the concentration of red blood cells. What will that do to the viscosity?

[Inaudible]

>> Steve Langjahr: What will that do to the resistance?

[Inaudible]

>> Steve Langjahr: What will that do to the flow?

[Inaudible]

>> Steve Langjahr: So this notion of getting better oxygenation is true only to a degree, because if you raise the viscosity too much, then you're going to

adversely raise the peripheral resistance, and that's going to suffer. That's going to cause lower blood flow. But with all that said, blood viscosity is not a normal factor in blood flow, because blood viscosity doesn't change that much minute to minute. So even though it's a factor, it's not a more or less immediate factor in determining blood flow. What is, is number two. The overall status of arterials. Something called Vaso motor tone. Remember, arterials are made of smooth muscle, yes. And you know that smooth muscle is never fully contracted, nor is it never fully relaxed. So when the walls of an arterial, the smooth muscle maintains a kind of basic contraction at all times, and that contraction can be lessened or increased. Right. So if this smooth muscle relaxes, what happens to the size of this vessel? It gets bigger. What's that called when a vessel gets bigger?

>> Vasodilation.

>> Steve Langjahr: Vasodilation. When it gets smaller, that's called vaso?

>> Constriction.

>> Steve Langjahr: Constriction. Does that affect resistance friction imposed by blood trying to navigate those tubes? Yeah. So the biggest factor in peripheral resistance is not viscosity, but rather the status, the overall condition of the arterials. Remember, we're not talking arteries. We're talking what? Arterials. And so as we know, arterials are essentially subject to change based upon the contraction of the smooth muscle, and that smooth muscle is under the influence of the sympathetic nervous system, not the parasympathetic, which is a notable exception to this rule, because we've come to expect that most visceral effectors received sympathetic and what?

[Inaudible]

>> Steve Langjahr: Not so here. Vaso motor tone is almost entirely determined by just what?

>> Sympathetic.

>> Steve Langjahr: Sympathetic. And/or, and/or the influence of hormones acting on an adrenergic receptors. So here's a standard cut through, let's say an arterial. It's neither fully relaxed, nor is it fully constricted. So this is called normal what?

[Inaudible]

>> Steve Langjahr: Can we go either way?

>> Yes.

>> Steve Langjahr: Yes. When we contract that muscle more, that's called Vaso?

[Inaudible]

>> Steve Langjahr: And when we relax it, that's called vaio dilation. Which of these would impose more resistance to blood flow? Vasoconstriction. And if we raised resistance, then we're going to do what to flow? If that number goes up, that number will go down, and that's intuitive because obviously it's harder to deliver blood through such a narrowed blood vessel. Vasodilation reduces PR. What's that?

[Inaudible]

>> Steve Langjahr: And therefore it raises BG, raises blood flow. Now the importance of the ability to change vasal motor tone can't be overstated, because are there times in life where we expect blood to flow more here to provide for the support of that tissue? And I'm talking of course primarily about exercise. When you exercise, do you want, do you expect more blood to go to those muscles that are contracting? And how's that going to be achieved, but by what? Vaso?

>> Dilation.

>> Steve Langjahr: Dilation. And you might think, oh, that's a simple matter. Let's just vasodilate. Let's vasodilate all the vessels, but that's not possible, because do you have a finite amount of blood in your body? Yes, let's say five liters. And if all vessels were to dilate, that would open up space, which was previously, previously not there. And now this five liters would go into these dialated vessels and there'd be no blood left to come back to the what? Does that sound like a good thing? If no blood's coming back to the heart, what does that do to venous return? What does that do to end diastolic volume? What does that do to stroke volume? What does that do to blood pressure? What does that do to blood flow? So the reality is even though there are occasions where certain organs must receive more blood, we can't just dialate all vessels. Some vessels have to be C word, constricted. Other vessels have to be and will be dialated, so that means the behavior of vessels certainly might and occasionally must be subject to change. And here's the story. Arterials may have adrenergic receptors of different types. Sorry, that's off the page a bit, but you have it in front of you. And so these are important vessels by name. First of all, coronary vessels. Where are they?

[Inaudible]

>> Steve Langjahr: They're on the surface of the heartm and these provide blood to the myocardium, to the heart muscle. And then skeletal muscles. Does skeletal muscles have arterials? Of course. And those are arterials which have a beta two adrenergic receptor. How do they respond to increase sympathetic action? It's The D word. What?

>> Dilation.

>> Steve Langjahr: Dilation and dilation. So during a sympathetic episode, what's going to happen to the status of coronary and skeletal muscle arterials?

During its sympathetic discharge they're going to go from this to this. What's that called?

[Inaudible]

>> Steve Langjahr: And what will that do to resistance through these vessels providing blood to these organs? The resistance would what?

[Inaudible]

>> Steve Langjahr: And what would that do to flow into those areas?

[Inaudible]

>> Steve Langjahr: Makes sense? But again, we can't tolerate dilating all vessels, and so what's listed down there that you can see, and I can't? You've got it on your page.

[Inaudible]

>> Steve Langjahr: GI tract, what else?

>> Kidneys.

>> Steve Langjahr: Kidneys.

>> Skin.

>> Steve Langjahr: Skin. These are also supplied by vessels, but they don't have beta two adrenergic receptors. They have a different kind of receptor called a?

>> Alpha.

>> Steve Langjahr: Alpha. And how do those receptors respond to the same increase in sympathetic action? Instead of dilating they what?

[Inaudible]

>> Steve Langjahr: Constrict. What does that do to the resistance through those vessels? It what?

>> Raises.

>> Steve Langjahr: Raises the resistance. What does that do to the flow through those vessels therefore? Decreases it. So in this kind of episode, we have managed to improve blood flow where, coronary arterials and skeletal arterials, but we've actually decreased blood flow where? Decrease through the skin. Decrease through the kidneys. Decrease through the Gi tract. And that might seem sad or unfortunate, but if it weren't for that, we would minimize or reduce venous return, and therefore we'd have a fatal lack of blood returning to the heart. So simply put, why are we compromising blood flow to these areas to allow better blood flow through these areas? And when and why would you expect more blood to flow through coronary and skeletal muscles? During fight or flight. During exercise, during any activity which involves the use of the

heart and skeletal muscles. Now, when you're not in a fight or flight situation, then we're going to have normal or reduced sympathetic activity, which will normalize vasal motor tone in the coronary and skeletal arterials, and at the same time it'll open up and provide for blood flow through the skin, the kidneys, and the Gi tract. So before we leave this page, which is complicated enough, it was mainly about BF. What's that?

[Inaudible]

>> Steve Langjahr: That's a function of pressure, of course, but it's also a function of peripheral resistance. Peripheral resistance can be, can be influenced by viscosity, but this is not a big concern, because viscosity doesn't change that much moment to moment. What can change moment-to-moment is vasa motor tone, and that's because it responds immediately to sympathetic action or the presence of hormones that are acting on adrenergic receptors. And in case this is vague or unclear, what is the hormone we mentioned on Monday that can and does act on an adrenergic receptors?

>> Adrenaline.

>> Steve Langjahr: Adrenaline, and also known as epinephrine. So sympathetic action and or einephrin have this very interesting and less than consistent effect on blood vessels. Some vessels, D word, what?

>> Dilate.

>> Steve Langjahr: Dilate. Some vessels, C word, and it has nothing to do with the hormone or the sympathetic. It has to do with the receptors. The receptors that are found on coronary and skeletal muscle are called beta two. The others have a type of adenergic receptor called?

>> Alpha.

>> Steve Langjahr: Alpha. All right, so now we've established some facts, and basic relationships between pressure, resistance, and flow. But let's get down to the final topic for today, and that is what controls blood pressure. Certainly it's not the cerebral cortex. What do I mean? Do you think about that? Do you say, Oh, I'm going to have to run now. Let's get the blood pressure up. This is all automatically performed for you based upon circumstances, and that means a part of your central nervous system is monitoring and responding to circumstances appropriately, and therefore regulating blood flow, and certainly blood pressure. So where is the control center in your central nervous system? It's actually in the medulla, a portion of your hindbrain. And we're going to call it the CVC, which is an acronym for cardio vascular center. This is the area which monitors and determines heart rate and blood pressure 24/7. Obviously if it's going to do this, it has to receive information, which is useful in deciding what to do. That is how to change or alter the blood pressure. And so essentially the CVC controls or exerts autonomic influence on both the heart and these vessels called arterials. And so what are these receptors that give information to the CVC? We're going to call them peripheral receptors,

because they're located throughout the body, responding to various changes, which should and do influence blood pressure. And let's divide these receptors into two groups. Those that inhibit the CVC, and those that excite the CVC. So before going into that, if we inhibit the CVC, that's going to lower heart rate and lower blood pressure. If we excite the CVC, that's going to raise heart rate and raise blood pressure. So what are these so called peripheral receptors? The first are either called baroreceptors or presoreceptors, depending on which textbook you open up. Wwe're going to call them presoreceptors, but the same, the same word bororeceptor is sometimes used. These are essentially [inaudible] corpuscles, which are embedded in the walls of arteries, which normally sustain rather high pressure. And the arteries which have and sustain the highest pressure are the carotid arteries, and the aortic artery. These receptors then are located in the aortic arch, which is just beyond the aortic valve, and the carotid sinus. You know the carotid artery goes up the neck, and it splits into the internal carotid and the external carotid. Right there at the junction there is a large bulb, B-U-L-B, which is called the carotid sinus. And in that sinus we have a number of these pressoreceptors. Now obviously the name tells you what they do. They respond to pressure. Not air pressure, obviously blood pressure. And actually they respond to increased pressure. And so if there is an episode of inappropriate sudden increase in blood pressure, what would you expect or assume that should do to the elements of the CVC? Let me say it again. If pressure is suddenly surging, these are going to respond to the high pressure. Would you want that to lower pressure or raise it higher still?

>> Lower it.

>> Steve Langjahr: Lower it. So it's not surprising to find that these exert a negative influence on the CVC. Their job, you could say, is to watch out for high blood pressure and therefore bring it down as a result of inhibiting the activity of the CVC. Great. Now, on the other side of the story, we have receptors that actually stimulate the cardiovascular center, and there are two that we'll care to discuss. One are chemoreceptors. Now the word is generic. What's a chemoreceptor? A receptor that responds to a chemical, so that's not very helpful, but what chemical, what thing in the blood of a chemical nature is perhaps the most important to keep an eye on or otherwise be aware of? Oxygen. Does that make sense? So even though these are called chemoreceptors, they're really oxygen receptors. And as you read on they're also found in the aortic arch, and in the area of the carotid sinus, an area called the carotid bodies. Their action, even though it's not revealed here, is fairly easy to predict. Remember, these are going to have what effect on the cardiovascular center? They're going to stimulate it. So would you expect they respond to high oxygen or low oxygen?

>>Low.

>> Steve Langjahr: Low oxygen. And indeed they do. They react to low levels of oxygen in the arterial blood, and therefore bring about what change? They bring about a positive or excitatory influence on the CVC, which raises heart rate and with it blood pressure. So this is a logical response. Indeed. It's

a reflex. It's a reflex working through these receptors, and the neurons in the cardiovascular center. Now, finally, there are receptors called mechanoreceptors, which obviously respond to what? Mechano movement, and these are found where you'd expect movement to be occurring. In other words, they're found in joints, and in the tendons, and connective tissue of skeletal muscles. Obviously they respond to movement or contraction. That is contraction of muscles or movement of joints, and you can think ahead, you can be way ahead of me here. If these respond to movement, what effect would you hope they'd have on the activity of the CVC? Would you want that to be stimulated or inhibited? Stimulated to bring about what? Increased heart rate, increased blood pressure, and with that increased BF. What's that?

>> Blood flow.

>> Steve Langjahr: So this should make sense. It's what you would naturally expect. So these mechanoreceptors are sensitive to movement, and bring about excitation of the CVC, and with that and increase in blood pressure, and therefore blood flow. So these are the so called peripheral receptors, which report to or have an effect on the cardiovascular center. In other words, the cardiovascular center depends upon these to know what's going on and to act appropriately. In summary then, what would be the expected response to a dominance of inhibitory input? How would the CVC respond to negative or inhibitory input? First, it would raise parasympathetic activity, and decrease sympathetic activity, and that would have its first and obvious effect on HR. What's that?

[Inaudible]

>> Steve Langjahr: So think about it. What does an increase in parasympathetic coupled with a decrease in sympathetic, what does that do to heart rate?

>> Lowers.

>> Steve Langjahr: Lowers it. Aand what would that by itself due to MAP? What's map?

[Inaudible]

>> Steve Langjahr: So it's pretty logical. If heart rate is down, so would MAP beap down, and if MAP is down, what's going to happen to BF? BF is?

>> Blood flow.

>> Steve Langjahr: Blood flow. So you can easily see lowered heart rate would translate to lower MAP, which would contribute to lower blood flow. But remember, the heart is not the only element in this story, blood vessels are too. But remember this, blood vessels are not supplied with, they're not influenced by parasympathetic. Only what?

[Inaudible]

>> Steve Langjahr: And a decrease in sympathetic would cause vasodilation, especially in those vessels that have alpha adrenergic receptors. And if we vasodilate, what's that going to do to PR?

[Inaudible]

>> Steve Langjahr: Vasodilate. Peripheral resistance would be down. And that would further lower MAP, which would in the end lower blood flow. You can memorize those, but it's much better to simply understand it.. So let's start over. Anything that inhibits the CVC will immediately raise parasympathetic and decrease sympathetic. This will lower HR. That will lower MAP. And that will lower blood flow. At the same time, vessels, especially those with alpha adenergic receptors are not going to constrict, they're going to dilate. That would lower PR. What's that?

[Inaudible]

>> Steve Langjahr: Peripheral resistance, which would further help to lower MAP. What's the flip side of inhibitory input? Excitatory input. And obviously everything would be turned the opposite way. When there's excitatory input, there's going to be a decrease in parasympathetic action, an increase in sympathetic action. And what would those two by themselves due to heart rate?

>> Raise it.

>> Steve Langjahr: Raise it. What would that do by itself to MAP?

>> Raise it?

>> Steve Langjahr: What would that do to BF?

>> Raise it.

>> Steve Langjahr: Raise it. And remember, vessels are only innervated by sympathetic, and if we increase sympathetic activity, those with beta two will be dilated, so there'll be some vasodilation, but off the chart here you'll see also what?

[Inaudible]

>> Steve Langjahr: Some vaso constriction. So the question is what's going to prevail here, vasodilation or vasoconstriction? They'll actually be more vasoconstriction, which will raise PR, and therefore raise MAP and raise blood flow. Now, this is going to take you some time to come to terms with this, to become comfortable with this, but to set you at least on course, let's do a kind of exercise, which is this view on the next page, which essentially starts with some circumstance, which would bring about excitatory input to the CVC. And rather than being abstract, let's be quite precise. What do you think? What activity that you can think of would probably stimulate some of the receptors we spoke of, and therefore bring about an excitatory effect on the CVC?

>> Running.

>> Steve Langjahr: Running. I was thinking of exercise, but that's the same thing, isn't it? So let's think about it. You exercising. What receptor of the group that we mentioned would be immediately stimulated by the act of running.

[Inaudible]

>> Steve Langjahr: What were the receptors? They're back on the previous page. There's –

[Inaudible]

>> Steve Langjahr: Pressoreceptors. There were chemoreceptors, and what?

[Inaudible]

>> Steve Langjahr: Now, which of those three is going to be first to respond to movement?

[Inaudible]

>> Steve Langjahr: All right. You might say, well, what about the chemo receptors? They respond to what?

[Inaudible]

>> Steve Langjahr: And believe it or not, there's not going to be low oxygen. Certainly not in the beginning. Maybe not ever. That's another story for another time. So of those three receptors, which would be the first to respond to an activity such as running? Mechanoreceptors. And they would have what effect on the CVC? Excitatory. Now we know what's going to happen. That is the conclusion or the outcome is a foregone conclusion. I mean, what happens to blood pressure? What happens to heart rate when we exercise? Everybody knows that, right? It goes up, and it's tempting to say, well, it goes up because the body needs more oxygen, but that's not physiology. That's teleology. That's saying something happens because it needs to happen, so we're not about teleology, we're about physiology. What are the mechanisms that bring that about? We know that it happens. We know that it should happen. We're not after a justification. We're after an explanation, so this is the explanation. When you begin to run what receptors respond?

[Inaudible]

>> Steve Langjahr: And they have what effect on the CVC?

>> Excite.

>> Steve Langjahr: Excite. What does that do to parasympathetic action, which comes from that location? Decreases it. What happens at the same time to sympathetic output? Up. And what happens to heart rate and force of contraction as a result of those two changes?

[Inaudible]

>> Steve Langjahr: Heart rate goes up, force of contraction goes up. Again, let's make that clear. If heart rate goes from [banging sound] this to what, [banging sound], and not only does it become faster, it becomes what?

>> Force of contraction.

>> Steve Langjahr: Force of contraction as well as rate. And what do these together due to stroke volume?

[Inaudible]

>> Steve Langjahr: Well, obviously they raise it. Yeah, because now we're putting more blood into the aorta with every single contraction, and of course a higher rate or frequency of contraction. And let's just follow that. If stroke volume is up, what happens to systolic blood pressure?

>> It raises.

>> Steve Langjahr: It goes up. There's no mystery there. We'll come back to it though. Remember, sympathetic, don't just affect the heart. The sympathetic nerves also go to the adrenal glands, and what do the adrenal glands produce and release as a result?

>> Epinephrine.

>> Steve Langjahr: And does epinephrin enter the bloodstream? And does epinephrin find its way to the heart? And does epinephrin act on beta one adenergic receptors there? Does epinephrin support, that means further add to an increase in heart rate and force the contraction? Yeah, so that's all good. But let's look beyond the heart, because remember the heart is just part of the story. What about blood vessels? Some blood vessels, and by that I mean arterials. Some blood vessels have alpha and some have beta adrenergic receptors. Those that have the beta have beta two, and those include these guys, arterials that are found in and deliver blood to skeletal muscles and coronary arterials. You don't necessarily have to remember this, because it's almost intuitively logical. What would you expect or hope would happen to these arterials that supply blood to the heart muscle and to the skeletal muscles that are moving you through a treadmill or whatever? They're gonna not constrict, they're going to what?

>> Dilate.

>> Steve Langjahr: Dilate. And what will that do to the resistance of blood flow, the resistance of blood moving through those vessels?

>> Lower.

>> Steve Langjahr: Lowered resistance, and when you have lowered resistance, what are you going to expect in terms of flow?

[Inaudible]

>> Steve Langjahr: Is this making sense? Is this logical? Is this what you would assume? Naturally, because after all, you're exercising and you need to

support the activity of the muscle, namely the skeletal muscle and the heart muscle itself. But as true as that is, can we afford to dilate all blood vessels? We discuss this. Is it possible to dialate all blood vessels and make everybody happy? No. Because if we dilated all blood vessels, then our blood volume would be lost in all of these open vessels, and there would be no blood left to come back to the?

>> Heart.

>> Steve Langjahr: Heart. Venous return would be zero, and EDD would be zero. Stroke volume would be zero. Systolic pressure would be zero. Blood flow would be zero. So if we're going to give some areas more blood flow, we've got to cut back on blood flow to other areas. Other areas that can be compromised in the short term. Can we afford to cut back blood flow in the skin, and in the kidneys, and even in the Gi track? Can that be accommodated or tolerated? Yes. And the way that happens is those vessels are not endowed with beta two. They have instead alpha. So instead of, D word?

>> Dilation.

>> Steve Langjahr: Instead of dilation, they?

>> Constrict.

>> Steve Langjahr: Constrict. What happens to resistance through those vessels then?

[Inaudible]

>> Steve Langjahr: Up. And if resistance is up, what happens to blood flow into the skin, into the kidneys, into the Gi tract? That goes actually down. Before you start regretting that, that's actually good, because by denying blood flow into these areas, we allow and ensure there'll be enough blood coming back where?

[Inaudible]

>> Steve Langjahr: In order to maintain pressure and flow elsewhere. So as we return to this, let's start. We had exercise. That stimulated what receptors?

[Inaudible]

>> Steve Langjahr: And the mechanoreceptors brought about an excitatory effect on the CVC. That did what immediately to parasympathetic action? Brought it down. Sympathetic what?

[Inaudible]

>> Steve Langjahr: Up. What happened to heart rate? Up. Force of contraction? Up. What happens to stroke volume? Up. And stroke volume is the biggest factor on SP. What's SP?

>> Systolic pressure.

>> Steve Langjahr: Systolic pressure. So of course that will be up, but as we look around the page, we see also that we're going to have increased resistance through these areas, because they are constricted, and as a result there's going to be increased resistance there, which is going to do what to diastolic pressure? Remember, diastolic pressure is mainly a function of peripheral resistance, so by supporting and maintaining PR diastolic pressure will be up too. And what is the obvious conclusion? If you had systolic up, and diastolic up, then obviously what happens to MAP? That's up to, and that's going to provide for more blood flow through those vessels that are dilated. And what vessels have been have been dilated in this case? Obviously the coronary and skeletal muscle arterials. Therefore what happens to blood flow there? Up. Now, if you step back and look at this, it makes perfect sense. It's certainly what we expected, and no is going to occur, but we haven't given you the justification. We've given you the explanation. So we could go over it again, but I think it's self evident. Let's finish with this though. What happens to inhibitory input? Let's just say you stopped exercising. If you stopped exercising, you take away what?

[Inaudible]

>> Steve Langjahr: Take away the excitatory input, so what happens to the parasympathetic activity that was down?

>> Goes up.

>> Steve Langjahr: Goes up. What happens to the sympathetic activity which was up, it goes?

>> Down.

>> Steve Langjahr: What happens to heart rate as a result, it goes down. What happens to stroke volume? Down. What happens to systolic blood pressure? Down. Everything's reversed of course, and that restores blood flow to what areas that have previously been short changed. We now restore blood flow to the skin, and the kidneys, and the Gi tract, and necessarily we cut back on the generous supply of blood, which was going to the skeletal muscles and coronary arterials. So this is a rather logical, impressive, and immediate effect, a cooperative effect between receptors, sympathetic, parasympathetic, and even hormones. What's the hormone that's part of this story? It's epinephrin, which acts on beta one, beta two, and even alpha adrenergic receptors. So now we know not just what happens with exercise, not just why things happen, but how they happen. This is the mechanism that would be triggered by any excitatory input. And what would be another excitatory input? Not to belabor this, but we spoke of exercise, which would trigger what receptor?

[Inaudible]

>> Steve Langjahr: But what's another receptor that also has an excitatory input on these cells? It was the chemoreceptors. They respond to what?

>> Low oxygen.

>> Steve Langjahr: Low Oxygen. What would cause low oxygen?

[Inaudible]

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>> Steve Langjahr: High altitude. Did somebody say that? Okay. High altitude. And what would that do? It would bring about what? Increased heart rate, increased blood pressure, increased blood flow, and all of these things are quite logical. So study this, make sure it makes sense to you. Please don't memorize it. Get to the point where it's natural and logical to fill in the blanks here. Questions? To much information? Okay. Have a great weekend. We'll see you on Monday, and Tuesday, and Wednesday.