Okay, good morning. Let's see, how's that level? Not too bad. Okay. So good morning. So ahh, my name's Geoff Erikson. I'll be guiding you through the next part of the HUBS tutorial as we go through the ahh, HUBS192. So you've been working with Greg Jones, who's been teaching you cardiovascular anatomy. Now you're going to be working with me for the next six lectures, I believe it is, on cardiovascular physiology. So we're going to talk a lot about how the heart and various parts of the cardiovascular system work in a physiological sense. One thing you're probably picking up on right away is that you can tell from my accent, I'm not a native Kiwi. So I'm actually from the United States. That means that I'm going to say words just a little bit differently than what you're used to hearing. So Greg likes to talk about capillaries, I talk about capillaries. Hopefully we'll all be able to be on the same page about that and you won't have to double your learning by learning all the words again and ahh, we're, if anybody has any questions about the words I'm saying, hopefully there won't be too much of a problem. You'll pick up on my accent quickly. But before we get to today's lecture, I do want to introduce Jenny McDonald who is here to teach us just something very quickly about a kind of a new programme that's going to help those who are interested with their learning by doing sort of an interactive question and answer session that you can do online. I'm not going to spoil it any more. I'm going to let her take it from here. So she's going to take just a couple of minutes and then we'll switch back to me.

Jenny
Thanks Geoff. Can you hear me okay? Right. Umm, right, so good morning everyone. My name is Jenny and I'm part of a research team that's put together a, umm, an interactive tutorial, as luck would have it, on the cardiovascular system and really the purpose of, of our research is to explore different strategies that we can use in an online environment to help students like yourselves learn the concepts associated with the cardiovascular system. Umm, the system's been designed specifically to help you with HUBS192 umm, but the important thing to remember, it's an addition to your coursework, not a substitute for it. What will you be asked to do if you decide to click into this tutorial? First thing and the most important thing is to answer some questions and really, I guess that's the main benefit for you too, if you choose to use this tutorial, is it's going to give you lots and lots of practice and get your minds going and really get you thinking about the cardiovascular system. You'll be asked some short answer questions, you'll be asked some multi choice questions and you'll just be asked to read some stuff and think about it. Okay. Umm, so it's really good practice and it's a good opportunity for you to get your minds in gear all, all in good preparation for the terms test that I think's ahh, coming up towards the beginning of August. Umm, if you do the tutorial, we'd really like you to attempt all the questions, ahh, if you can. So really do try to get right through to the end. Umm, it shouldn't take you any longer than 30 minutes and some of you will get through quite a bit quicker than that. Umm, one week after you've done that tutorial, we'll send you an email and ask you to just answer a few more questions. Again, this is good practice

for you and there'll be a mix of multi choice and, and short answers. Ahh, finally, if you've still got any energy left, we'll ask you to complete an online questionnaire just about your experience of using the system and some of you may be willing to even come and talk to us about it as well. It's completely voluntary. All the, it doesn't matter if you go through and get every question wrong, Geoff is never going to know, all right. It's okay umm, but hopefully it'll be really good practice for you to answer questions. How do you get started? There's the ahh, web address. More information about the tutorial is ahh, all ahh, included at the end of your GLM, I think, for, for this section of the course. So you'll find the link and ahh, much more information about the ahh, research project itself there. To log into the system, just use your standard university username and password. Any questions? If you have any concerns about the tutorial or any bugs or whatever, send me feedback, send me an email. My email address again is included in the materials with the GLM. Okay. Thanks Geoff.

Geoff
Okay. Thanks Jenny. I just want to reiterate, I've actually ahh, used this tutorial a little bit myself and it is a great way, especially when you're revising this material, to ahh, kind of catch up and see exactly where your knowledge sits in terms of what you need to know for the cardiovascular portion of the course. So I'd definitely recommend checking that out when, especially when you're doing your revising. So let's get to today's lecture and I always like to start out by talking about what the objectives of the day are. This kind of gives you an idea of what the key points are that we're going to be concentrating on and again, this is the kind of thing that could hopefully help you when you're revising.

So our objectives for today, first off, we're going to talk about the fact that the heart connection can be considered to be two pumps in series. We're going to talk about what that means and what it kind of means for overall cardiovascular function. We're going to talk about the anatomical and cellular basis of contraction. What that means is that we're going to talk about the contraction cycle and we're also going to talk about, at a cellular level exactly how contractions are occurring within the heart. We're then going to delve a little bit more deeply into the cardiac cycle. This is probably going to be one of the most important things that we learn today and one of the most complex. We're going to go over that in pretty good detail. We're going to learn two new terms called systole and diastole. Again, these are some of these terms that as an American, I say that way. You might hear these as systole or diastole from some people but never from me. And then, finally we're going to look at a blood pressure wave and identify all the different key features of a blood pressure wave, including all of the different types of data and other kinds of factors that we can get from one of those curves.

So let's start with that very first point, that very first objective and what we're making the argument here is that, in fact the heart,

while it is one cohesive unit, is actually two pumps in series and the reason that this is the case is because we have, essentially two halves to our heart. We have the left half and the right half and each half of the heart is pushing blood in a different direction. One side is pushing blood towards the pulmonary, pulmonary portion of your body, so this is towards the lungs to reoxygenated your blood and the other side of the heart is pushing blood out and into the rest of your body and so we have two pumps, two, the left half and the right half which are operating in series and what we see here is kind of a diagram of that and I actually want to concentrate on the right diagram first here. What you see is you have this right pump and we've coloured it blue to give this impression of de-oxygenated blood and what's happening is that as the right pump is pushing blood through the system, it's pushing blood towards the lungs. Meanwhile, at the same time, your left pump, the left half of your heart, is pushing blood towards the rest of your organs and you can see that this is red to indicate that you have oxygenated blood in this case. But what's critical to understand about this is that these two lie in series. These two pumps work in series and the reason that that's the case is because they form a continuous unit. So if we were to continue along this path, what would happen is that after the blood reached the organs, it would actually recycle back over to this portion of the figure. It would become de-oxygenated and would move back to the right side of the heart to be pushed back to the lungs.

So at all times the, the blood is being pumped continuously through the heart. So we have two pumps and they're lying in series and you can see a more detailed diagram of this in the left half of your figure here where, again you see the left ahh, the left side of the heart pushing blood down to the capillary, ahh, capillary beds that are embedded throughout your system and then when the blood comes back out it, it's coming through the right side of the heat and then back out to the lungs. So two, ahh two pumps that are existing in series.

What's important to understand though is that the left and the right side of the heart are two pumps, are contracting simultaneously. So they contract together with one another at all times. This makes sense as if they were mistimed from one another, you'd get more blood pumping in one part of the, or in one system, for example, down in the body than you would get into the lungs and then it would have to pool up and the whole system would become jammed up and not work properly. So you want that nice continuous flow of blood throughout the entire system which means that you need good accurate timing between both halves of the heart.

So the, ahh, the heart is made up of the atria and the ventricles. The atria umm, when we look at a typical picture of a heart, the atria are sitting up here on top of the heart and the ventricles down below and what you can see then is that there's a left atrium, a left ventricle, a right atrium and a right ventricle and each one is in charge of pushing blood in opposite directions,

So let's talk about what happens when these two pumps contract and to orient yourselves, these two pictures up here, and this figure is from your book, this is ahh, this is a picture of what it would look like if you cut the heart in half from top to bottom so that you're getting sort of a side on view of what the heart looks like. These pictures down here represent what it would look like if you did sort of a transverse cut, if you looked down on to the bottom half of the heart and the reason that we get these two different views is that the top view gives us a very good idea of what's happening in the atria and the ventricles and the bottom picture gives us a good idea of what's happening with the various valves that control where blood's moving throughout the heart and what you end up seeing here is that we have two sort of phases that were showing between what's happening in the heart. We have an atrial contraction and we have a ventricular contraction and this actually denoted. So here you can see that the atria are contracting in the left half up here, here and here and you can see that there's a sort of classical cartoonish, wavy line which indicate that there's a contracture or a movement of that portion of the heart. So what's happening here is that the atria are contracting, this is reducing the ahh, the size of the space in which the, the blood is, is occupying and this is forcing blood from the atria into the ventricles. As that's happening, if you look down here, what you can see is that the valves between the atria and the ventricles are open. This allows blood to flow from the atria to the ventricles but the valves that connect the ventricles themselves to the aorta and the pulmonary artery, the main vasculature of the body are closed. So what we're doing here is we're forcing blood, as much blood as possible, into the ventricles without letting it escape into the rest of the body and this is going to become important a little later.

Now in a ventricular contraction, the exact opposite is happening. So you can see that the left and right atria are no longer contracted and instead our wavy lines are now around the left and right ventricle. So this, when this is happening, we're getting this contraction of the ventricles which is, again reducing the volume that the blood is allowed to occupy and putting it under greater pressure and in this picture, what we're seeing is that now the valves that connect the atria and the ventricles are closed. This prevents blood from going backwards back into the atria and instead, the valves that connect the ventricles with the aorta and the pulmonary artery are open. This allows blood to flow out. Now this picture is a little bit misleading in that it really only shows two snapshots of what's happening during the cardiac cycle. We're actually going to go through the cardiac cycle in a much more detailed way a little later in the lecture but for now, I want you to understand that what's happening here is that we have two pumps that are working simultaneously and in series with one another to move blood throughout the system and that there's these, that the atria and the ventricles work in tandem along with these valves to make sure that blood is going in the right direction.

All right, so let's talk a little bit about the cellular mechanism of cardiac contraction and hopefully you've already done some

studying on how skeletal muscle works and so you have some idea of the process by which muscular contractions work. The heart itself is a muscle and so it works in pretty much the same way as the skeletal muscle, albeit with some subtle differences which we'll talk about later.

So when you think about the way that the heart is arranged, what's critical about the heart is that it works, that it exists as a series of fibres and what's, that's shown up here in these pictures but since these are far too small for anybody to be able to see, let's take a little bit closer look at that. So this is a cartoon of what the structure of a heart looks like and what you can see is there's lots and lots of little fibrous bundles that exist in parallel with one another. It's almost like a box of drinking straws that's been cut in half so that you can see all of these different fibres that make up the whole, the whole muscle tissue itself and as you can see, this muscle tissue is striated with different bands and we're going to talk about what those bands are in just a moment but you can also see that there are several other structures that exist alongside the muscle fibres themselves. So the fibres themselves are the main structural component of the heart but you also see that there's the mitochondria, the T tubules where calcium is entering the cells. You see the, ahh, they don't actually have the nuclei on here but we're going to see that in just a moment. So there are other kind of cellular components associated with this. But for right now, the part that we really want to concentrate on are these fibrous bundles and just to get an idea of what this actually looks like under a microscope, this is, instead of a cartoon version, an actual microscopic version of what cardiac tissue looks like and again, you can see that it looks pretty similar to the cartoon where you're seeing these long, fibrous strands with mitochondria breaking those up in different places and a nuclear space that's down here.

All right. So how does the cardiac tissue contract? Well cardiac tissue contracts because these long strands are actually made up of a couple of different types of molecules. One is actin which is just long structural elements. Again, these are like our straws that are running down the lengths of the muscle itself and then we have myosin which is the motor of the cardiac tissue and what the myosin does then is provide the power to allow contraction to occur but this can only happen when myosin's associated with actin and at baseline, when the muscle's relaxed, there is no interaction between myosin and actin and the reason for that is because there's a complex of proteins that includes troponin and tropomyosin which block the binding cyte where myosin and actin interact with one another. So when muscles are relaxed, there's no interaction between myosin and actin and therefore you can't generate any power. The only way that you can generate power is to expose that cyte and that only happens when calcium enters a myocyte. So when calcium enters the cell, it binds to troponin and then this troponin and tropomyosin complex moves out of the way. It goes through a conformational shift. It moves out of the way and this exposes the binding cyte on actin and this allows myosin to then associate with

that cyte and perform its function of a power stroke which pulls on the actin fibre and causes this shortening or contraction of the fibre. It's very much like a game of tug of war where a person would hold on to the rope and pull. So by, when calcium enter the cell, that allows the cyte to grab on to the rope to be available, then you can grab on and the myosin pulls on the rope and this puts force by shortening the rope or in this case, the actin fibre and this causes the contraction of the muscle which then causes the heartbeat.

But the important thing to understand is that as the muscle is beating, every fibre within the muscle itself or within the heart itself is actually beating. They're all beating at the same time. So they're signalling between all of the different muscle cells. We're going to talk about that a little bit more on Thursday. But for now, this actually raises kind of an interesting question. So let's look at this a little bit more closely. How can we increase the force of cardiac contraction? In other words, if every single fibre, if every single cell is contracting every time the heart beats, then when you need to dig down deep and have a stronger heartbeat, like if you're, if you're exercising or just nervous about a talk or something like that, how do you get that extra bit of energy out of your heart? How do you get that extra strength of the beat? Well the answer is because the strength of the heartbeat is not specifically associated with the number of cells that are beating but rather the number of cross bridges that are forming. So these cross bridges are these connections between myosin and actin. The more of those connections you have, the more power that you get out of each heartbeat. So because that's the case and because these connections, these cross bridges between myosin and actin are dependent on calcium release, the way to get a stronger heartbeat is to release more calcium and this is how your system determines whether or not these beats are very strong or very weak in your heart, be releasing more or less calcium for, to trigger these contractions. So more calcium release means more cross bridges which means greater force of contraction. This is actually, for those of you who are interested in the medical side of things, this is something that actually plays a very big role during heart failure.

So if you think about a patient that's going through heart failure, one of the key features of such a patient is that they've lost a lot of their calcium control. So just briefly, I want to show something that's a little bit more basic science. So the idea here is that a person who's undergoing heart failure loses the ability to control calcium. Now this picture won't be in your packets and that's okay. I'm just showing this for interest. So if you look at the calcium release from cells in the healthy heart, it's very tightly controlled and this makes a lot of sense because again, the force that you generate when your heart is contracting is closely tied to your ability to regulate calcium coming in and out of the cells. So the greater of a difference that you can create between relaxation and contraction, the stronger heartbeat you can have. So what we see here is, this is actually a microscopic version of what's

happening inside a cell and what's happening inside the cell and what's happened is these are myocytes that have been treated with a dye that glows fluorescent green whenever it binds calcium. So anywhere where you see green, that's calcium that's in the cell but what's happening with these cells is that they're not actually contracting. These are cells in isolation. So what you see is that in a healthy heart cell, you can hardly see any kind of green dots in here, just a few small and very weak green dots which means that the cell itself is doing a very good job of preserving calcium within its cellular stores, within the sarcoplasmic reticulum which means that this cell is primed and ready for good, strong heartbeats.

By comparison, a cell from a failing human heart, so this is a person who's going through heart failure, you can see these very dark green dots here. These are sometimes called calcium sparks, and this is because calcium is leaking out of the sarcoplasmic reticulum spontaneously, in between heartbeats and because they're losing that calcium and because it's going out into the cells, this means that the ability to go from a relax state to a contracted state is compromised. So a person who's undergoing heart failure is no longer able to react very well to periods in which they need exercise or that extra little bit of, of ahh, of ahh stimulation. So this person is actually having a weak heart which is forcing their heart then to work harder which puts it under stress and puts them at risk of an arrhythmia or a heart attack.

Now going along with these contractions is also relaxation. So what happens during relaxation? Well ATP binds to the myosin. So ATP is our energy molecule. It's what myosin uses to perform its motor function. We need ATP to rebind to myosin to then release from the actin head. So two things are happening simultaneously. ATP binds to myosin to reset the head and meanwhile the calcium is being pumped back out of the cell and into the sarcoplasmic reticulum which lowers the amount of calcium within the cells themselves. So calcium goes back out, ATP goes on to the myosin head and what these two things do is cause the cross bridges to separate. The troponin and tropomyocin complex goes back to where it was originally blocking off the site and now don't have this interaction between actin and myosin, so this cell can then go back to relaxing and this allows the heart to relax.

So from a molecular level, we've just sort of described how an entire contraction and relaxation cycle occur and again, just like all of the myocytes contract in one beat, they also all relax in one beat and, by the way, this particular reason, in skeletal muscle, this is one of the reasons why we see some of the things that we do when, after people die. So if you think about after death, your mitochondria are no longer producing ATP and because they're no longer producing ATP, you never get to that point at which the ATP can rebind to myosin. Eventually you use up your ATP stores and so what this means is that your muscles become stuck in a place where myosin and actin are interacting with one another, contract and what that means is, is that your muscles will stay contracted which is

why rigor mortis sets in when you die.

Okay. So let's talk about the holistic version of the heart and what I just want to point out here is one key structural feature which is that when we think about the heart and the way that it beats, it isn't all beating inward. So we kind of get that image of the heart beating inward and outward, kind of like that but in fact, the fibres of the heart actually wrap around and around the heart in this sort of circular pattern with this sort of contorted or torsion sort of angle and there's actually a reason for that. It's because the heart beats more efficiently and pumps out blood more efficiently when you get that torsional angle of contraction and when you think about this, it's best to think about trying to get water out of a wet towel. So if you take a wet, a very wet towel and you just smash it down all straight in kind of one big clump, you'll get quite a bit of the water out of the towel but if you twist the towel at the same time that you're crushing it down, you get much more water out because that torsional force puts more force on the liquid within the towel and so I just wanted to show a video of what this looks like.

So when you look at the way that this heart is beating up here, what you're seeing is that not only are you getting this contraction, this reduction in the volume of the heart itself but you're also getting this twisting motion of the heart which is helping to twist blood up and out of, through the aorta and pulmonary artery. You're getting this nice torsional force along with the contraction force. So these two things actually work in tandem to make the ejection of the blood from the heart as efficient as possible.

Okay. So that brings us to our focus break and I'm definitely a believer in focus breaks but I like them to be as valuable for the students as possible. So some people do crosswords or little things like that. What I like to do during focus breaks instead, is go over potential exam questions and so that's what I'll typically do as we go through this. So what I like to do is show an exam question like this and then I'll give you a couple of minutes to break and kind of, you know, get a drink of water, talk to your neighbour. But also, what I want you to do is think about what you would give as the answer to this type of question on the terms test that's coming up because like was mention, it's coming right up in August and then after the break, I'm going to go over what the answer is and kind of talk you through a little bit about the different types of questions that I tend to ask on the terms tests. So just for the benefit of those who are only listening through the audio podcast, I am going to read this out loud and then I'm going let you be.

So blood flow can be calculated by dividing change in blood pressure by what value? It's patient age, pulmonary pressure, capillary wall thickness, vascular resistance or oxygen diffusion rate. So just take a minute or two to think about it. I'm sure you all know the answer already but take a moment and then we'll get right back to this.

Alright, okay. I don't know what happened in the rest of the rooms where I'm lecturing, but in this room it got really quiet all of a sudden so I'm going to assume that that means that you're done resting and ready for the rest of the lecture.

So let's move on. So blood flow can be calculated by dividing change in blood pressure by what value? Anybody want to wager a guess here. Shout something out. D. I'm hearing lots of Ds and that is correct, yes. So blood flow can be calculated by dividing change in blood pressure by vascular resistance or just resistance. This actually calls back to this slide from lecture one. So I'm going all the way back to when Pete Jones was here talking to you, I guess it was last week that that happened, maybe last Monday. So this slide is showing you that flow is equal to change in pressure divided by resistance and this is a way of describing some of the molecular, or sorry some of the haemodynamics of how liquids move through blood vessels.

So what's going on with this question, and if we just hop back up to this question for a second? So this is a type of question that I call a recall question and the reason I call it that is because this is, this question tests pretty much straight your ability to recall one specific fact from a lecture. This is my least favourite type of question and the kind that I ask the least on the terms test. There's usually one of these from my section on your terms test and then another one in your final but for the most part, this is not the type of question that I do like. The reason I don't like these type of questions is because there's no way through context to be able to get this. It's either you straight up remember that that's the way that that equation is set up or else you don't. So I prefer some different types of questions and we're going to go through what those look like in subsequent lectures. One thing I will also say about this though, so we're not really very equation heavy with this particular segment. I never have been but there are a couple of key equations that are important to know. What you'll find is that I go over those multiple times over the course of multiple lectures and this is one that it is important to know. So this idea that flow of blood is directly proportional to both the pressure and the resistance of the vessels is very important. It's something we're going to talk about a lot on Friday.

Okay. So let's get back to this lecture. So I talked about the cardiac cycle and its many phases and so this is something that we're going to cover in quite a bit of detail. So we're going to be looking at this slide for a while unfortunately ahh, but I'm hoping that by the time we're done that we'll all have this down completely pat. So the first thing to notice about this is that there are five different phases of the cardiac cycle listed here. This is from your textbook. I personally find this picture to be a little misleading specifically because they've numbered these different phases. Now I understand why they do this. It's because they want to be able to refer back to the picture within the text but the thing, one of the things that you want to understand about this is

that there's no real specific nomenclature or numbering to these different phases. They're actually all continuous within the heart. So there's no beginning or end. It's just one long circular process.

So we're going to start here with number one just because that's where they've started but really this cycle could start anywhere. So we're going to start with atrial systole and this is where these two terms come in. So we have systole over on this side of the diagram and we have diastole over on this side of the diagram. Systole is the point in which the point is under maximum pressure. This is the contraction of the heart. This is when the heart is contracting. Diastole is the exact opposite. It's when the heart is under its minimal pressure or going towards its minimal pressure. This is when it's relaxing. So we're going to get another picture of what systole and diastole look like in a moment but the cut-off in terms of the cardiac cycle and its phases is right about here. So systole is these three phases, one, two and three. Diastole is these two phases, four and five.

So let's go through what those look like. We're going to start with atrial systole, again systole is contraction so what we're talking about here is atrial contraction. We already talked about this a little bit earlier but again, what's happening is that the atria are contracting. You can see that they're kind of distorted in this picture and what this is causing is blood to flow from the atria into the ventricles. Now as we talked about, the valves that connect the ventricles with the rest of the circulatory system are shut. So what's happening here is that all of the blood within the atria is being pushed into the ventricles. So we're packing the ventricles full of as much as blood as is possible. We're getting as much blood in there as we can and what this is doing is, A, it's loading the ventricles with blood but, B, it's also putting the ventricles under increased pressure. So as the atria contract, it puts the blood within the ventricles under increased and increased pressure. This is important because what ends up happening is that the valves between the atria and ventricle then shut when all of the blood's been pushed into the ventricles and so we get this, ahh, the, this following phase, phase number two which is called isovolumetric ventricular contraction and what this means is that the ventricles themselves are contracting but what is isovolumetric referring to? It's referring to the fact that the, the valves that are leading out into the vasculature, at this point are still closed. So this is a little different than the picture that I showed earlier and this is a better version of what's actually happening during a heartbeat. These valves that go out to the vasculature, out to the aorta and the pulmonary artery are still closed. So this is an isovolumetric contraction because the volume of the blood is staying the same but the ventricles themselves are contracting and what does that mean? That means that we've already increased the pressure on the blood within the ventricles because the aorta has jam packed it full of blood but now because we're reducing the space that the blood can take up but not allowing the blood to escape, we're putting it under even more pressure and

what's happening during this contraction is a huge spike in the pressure of the blood. And finally, what ends up happening is that the blood becomes so highly pressurised, it becomes, the pressure is so great that the valves between the ventricles and the aorta and the pulmonary artery snap open and when that occurs, the blood then comes rushing out in, in one large burst, lots of blood coming out in one big pressure wave. So we're going to talk about that pressure wave in just a second but the key point here is that blood is being pushed out of the ventricles very rapidly. It's a, a large volume of blood because the aorta and the ventricles have teamed up together to push as much blood as possible out. So the first two steps within this cycle are the generation of a lot of pressure within the ventricle. Ultimately this allows then lots of blood to move out of the ventricle during the actual ejection phase which is the third phase. So now we're pushing all the blood out.

So once this occurs, the valves that lead out to the aorta and the pulmonary artery, this is, we've moved on now to phase four, these snap back shut. There's always a little bit of blood left in the ventricles. It never empties itself completely but if the heart is beating very efficiently, it empties itself mostly. So what happens at this point is that the ventricles themselves actually relax. So again, we're getting isovolumetric relaxation of the ventricles. This again is looking at the exact same type of process as step two, the isovolumetric contraction except this time in reverse. So now we're expanding the volume that the blood is taking up and remember, we've ejected most of it leaving just a little bit. So if we only have a small volume of blood left and we're increasing the volume that it's taking up, that means that it's under much lower pressure. So during this phase of the cardiac cycle, we're seeing a large drop in the pressure that the blood is under.

And then, the final phase is passive filling of the ventricles. So you might be thinking that, okay, the way that the blood gets into the ventricles is by the atria pushing the blood in there as we described in the first phase. That's actually only the way that the last bit of blood gets into the ventricles. If that was the case, if all of the blood in the ventricles came from the atria, then the two wouldn't, there would be no reason for the two not to be the same size since the volume in the ventricles would end up being the same as the volume in the atria themselves. But as we know, the atria are actually much smaller than ventricles. So what we actually need to happen is for the ventricles to be mostly full before we push the blood in through the atria. So we get this passive filling phase of the ventricles. So what happens here is that the valves between the atria and the ventricles opens up and blood passively fills both the atria and the ventricles from the veins. So we get the heart to be refilled with blood and it's now ready for this next cardiac cycle where once again, the atria can contract, put the blood into the ventricles under high pressure. Again, the ventricles then contract after that. Put the blood under more pressure and we're ready for another ejection. And around and around the cycle we go.

Okay. So again I want to remind you of these two words here though, to describe the two main phases. This is systole and diastole so after having gone through this process, what we see is that the contraction of the atria, the contractions of the ventricles and the ejection phase itself are all part of systole but then right after ejection, right after we've pushed that blood out and we're now starting refilling, everything is going into its relaxation phase and so at that point, we're in diastole. So if you were think, all right which phase lasts longer? Systole or diastole. You might guess from this diagram that because there's three steps in systole, one, two, three, and two steps in diastole, four and four, that systole must last longer. But when we look in actual cyclical blood pressure chart, we see that, in fact, that's not the case. So this over here, this diagram is actually showing you what a typical blood pressure chart for a person would look like during heartbeat. This person has a heart rate of 60 beats per minute, 60 seconds in a minute, that means that there's one beat per second. You can see that on the time scale down here where this is one full cycle of a heartbeat. One and two. Here, and that takes one and two seconds. So we have two different pressures. First of all, we have a systemic pressure and pulmonary pressure. The systemic pressure is the pressure of the blood that is moving through your systemic system or all of the different parts of your body that you're supplying with blood. The pulmonary pressure is for your pulmonary system. This is the blood that's going to your lungs and back and you'll notice that the systemic pressure is much, much higher than the pulmonary pressure. We're going to get back to that point in just a moment but when you think about it, this makes a lot of sense because the pulmonary system only needs to pump blood from your heart to your lungs which is pretty rapid and short trip. The systemic system needs to pump blood throughout your entire system but it needs to do all of this at the same time or in the same amount of time. You can't have blood piling up in one system or the other. That means that the blood within the systemic system needs to be moving faster and be under greater pressure.

Now what you can see here is that this phase, this phase and this phase of the two heartbeats are where the blood pressure is rising. This is where the contractions are occurring. You can see that the pressure is going up and so that's systole. But what you can also see is that the systole is actually only about 40 percent of the cardiac cycle. Now this is going to vary a little bit from person to person but in general, it's actually is the shorter portion of the entire cycle. More of the time is actually spent in diastole. This is the relaxation phase and this is where blood is just refilling into the heart. This is where the heart is relaxed, blood is refilling it. This is actually about 60 percent of the cardiac cycle.

So despite what you might think, diastole is the longer of the two cycles or two phases within each cycle. I just want to talk about a couple of more things that we can get from the pressure wave here.

So first of all, the systolic blood pressure and diastolic blood

pressure are considered to be the point at which your blood pressure is the highest and the point at which your blood pressure is the lowest. So if you ever go in to the clinic or to see your doctor, they'll usually take your blood pressure and they'll give you some kind of a pair of numbers. 120 over 70 or 120 over 80, something like that. Well what they're actually giving you is your systolic and diastolic pressure. So for this person that we see in the right panel, their blood pressure would be read to them 120 over 70 because their systolic pressure is 120 and their diastolic pressure is 70. We can also get the pulse pressure and the pulse pressure is the difference between the systolic and diastolic pressure, in this case it's 50mm of mercury. So 120 minus 70 is 50. And we can also, from this get the mean blood pressure. This is the average of blood pressure over the entire cardiac cycle. This one's a little tricky though because what you might think that you need to do is take diastolic pressure and systolic pressure, add them together, divide by two and there you go, that would give you 95. That's not quite right and the reason is because the heart, again spends more time in diastole that it does in systole. In other words, it's spending more time at low pressure than it is at high pressure. So the mean blood pressure that the heart, that the system is experiencing is always going to be a little bit lower than the halfway point between systole and diastole. In this case, it's about 85 to 90.

It's not important that you be able to calculate that figure from looking at one of these. It's just important for you to know that over the course of a cardiac cycle, the mean blood pressure is actually lower than the midpoint between systole and diastole.

And a couple of more things that we can get from this are what the normal blood pressure range for a person are. So our patient here looks like a very normal and healthy adult individual, at least in terms of blood pressure. They have a blood pressure of about 120 over 70 which is in a great range. So you can see here that this white range here is the range that you want your blood pressure to occupy for your systolic and diastolic blood pressure. If your systolic blood pressure starts to get very high, if your blood pressure increasing and it gets over about 130 to 140, somewhere in that range, then that person is said to be hypertensive or going through hypertension which just means high blood pressure and we know that high blood pressure can be a very dangerous thing. So in the short term, blood pressure can be affected by a lot of things, by stress, by diet, by all kinds of things and people can go through temporary spikes in blood pressure without a lot of issues. But if you have long term high blood pressure, long term hypertension, then that contributes to heart disease and stroke, two things that are very, ahh, are big killers both here in New Zealand and around the world. So hypertension is something that we definitely want to avoid.

But on the other end of the spectrum we have another kind of danger zone, this blue area down here. This is hypotension. This is when the blood pressure falls, for most people again this is below around the 60 to 50 range. Usually this is where your diastolic pressure

might fall. What this means is that the pressure within your vessels actually isn't high enough and when that occurs, blood is not being very efficiently moved around your system. It can actually do some pooling and it can actually have trouble getting all the way to the distal portions of your body including your brain and ultimately, if your blood pressure is too low, for example if you suffer some kind of a wound and you're losing blood or even just if you're undergoing some kind of a physiological response, you can go into syncope which is a fancy way of saying, fainting, so, because you're not getting enough blood to your head, to your brain because the blood pressure's too low. You end up fainting because your brain becomes oxygen starved.

Okay. So let's talk quickly about the pulse. What is pulsatile blood flow through the arteries? Well, when you think about the way the heart beats, we build this incredible amount of pressure within the heart and then we push lots of the blood out in one big burst. But if you think about the way that the blood flows through your system, that is not what you would want to have happen. You would not want some big bolus of blood moving around through your system, especially because as you think about it, every time that it moves through into a new part of your vasculature, it actually gets smaller and smaller. Each part of the vasculature branches out. So you actually want a nice smooth, continuous flow of blood. So how do you do that? Because the, for the heart to beat efficiently, you want big bursts of blood to come out of it but for your circulatory system to work properly, you need the blood to flow out.

Well, the way that your system compensates for this is that your arteries actually have some give to them. They're not rigid tubes like glass tubes. They're actually more like a garden hose in that there's some ability to flex and so when the heart has this big pressure wave, when it creates this big change in pressure as it contracts and as it ejects blood, that pressure wave travels through your arteries, out in front of the blood and you can see that in this picture here. So here's the blood being contained within the heart. When the heart opens, this pressure wave moves out in front of the release of the blood and this causes a distension in your larger arteries and this distension is then stored within the arteries as an energetic force and as the blood moves through, it actually then applies that force back to the blood and causes it to even out and continue to move along the path. So as the pressure changes within the heart, as you get this big pressure change from contraction and ejection of the blood, that pressure change is sensed by the arteries. It's absorbed by the arteries and then it's transferred back to the blood as it moves through the system. So when you're feeling your pulse, a lot of people will talk about how if they're feeling their pulse, they're either feeling their heartbeat or they're feeling blood moving through their arteries. Either one of those things is exactly right. What you're actually feeling when you're taking your pulse is that you're feeling through one of your major arteries, that pulse wave, that pulsatile pressure or that pulsatile force moving through your arteries in advance of the blood.

Okay. So last topic and then we're done for the day. I want to talk a little bit about ventricular power. So we talked about how the heart is actually two pumps working in series, the left half and the right half. One half is going to the pulmonary system. One half is going to the systemic system. So we have pulmonary blood flow. We have systemic blood flow. But there's a problem here because there's a power difference between the two and the reason for that is because again, the pulmonary system is much shorter. It's a shorter trip for the blood and so it, not as much power is required for the blood to move through the pulmonary system as through the systemic system and what we recall from this picture back up here is that the systemic system is under much, much more pressure than the pulmonary system. So the systemic system is under more pressure but the flow between the two, and this is the flow right here, is five litres per minute. Essentially, that's all of your blood. So the average person has about five litres of blood so you recycle all of your blood in about one minute and the flow between the two has to be exactly the same. If you're going to put five litres of blood through your systemic per minute, then it needs to go through your pulmonary system per minute because the two link to one another. Again, we can't have blood piling up in any specific spot within the circulatory system. So how can we rectify this difference between the two? If the flow is the same but the pressure is higher in the systemic circuit, then something else must be different and so this is the big question. How can the flow be the same but the pressure be different between the two and the answer goes back to this question that I referred to earlier. It's from the haemodynamics. If flow is the same and pressure is higher in the systemic system, then to get that flow to be the same, the resistance has to be different between the two systems and, in fact that's the case. The resistance through the systemic system is much higher than through the pulmonary system. So it's higher pressure but higher resistance. Pulmonary system is low pressure, low resistance.

Okay, so I just hit you with a lot of information. I just want to go back through all of our main points very quickly. We talked repeatedly about the fact that the heart is actually two pumps that work in series, the left half and the right half but even though they work in series, they're connected with one another and they pump simultaneously. We also talked quite a bit about how the different parts of the heart and the valves work together to move blood working in a unidirectional sort of a way. We talked about the cellular bases of contraction, so look through that again. I want you to really be able to understand what calcium is doing, how actin and myosin are interacting with one another. We talked about the order of contraction, first the atria and the ventricles and we went very thoroughly through the cardiac cycle. Kind of try and keep that in mind and make sure you revise that really well because it's something we're going to be coming back to and something that's very important for the course and finally we talked about the difference between the left pump and the right pump, how those two are working with one another and we looked at that pressure wave and

all of the different little factors that we can get off of that.

If you have any questions, please feel free to come up and see me. Otherwise I'll talk to you on Thursday.