Lippincott Clinical Leaders: Cardiac Output

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Lisa Bonsall: Hello and welcome to the Lippincott Clinical Leaders Podcast. My name is Lisa Bonsall and I am the Senior Clinical Editor for Lippincott NursingCenter. I'm joined today by Doctor Anne Dabrow Woods, Chief Nurse of Wolters Kluwer Health Learning, Research and Practice. Doctor Woods is also adjunct faculty at Drexel University and Neumann University, and also a practicing acute care nurse practitioner for Penn Medicine. Thank you for joining me today.

Anne Dabrow Woods: Happy to be here, Lisa.

Lisa Bonsall: Today we're going to speak about cardiac output and some related terms in cardiology that are often confusing to nurses. So, thank you, Anne. Can we please start with what is cardiac output?

Anne Dabrow Woods: So cardiac output is defined by heart rate to time stroke volume. But what does that really mean? Well cardiac output really talks about how well the heart is actually working. Is the heart being able to meet the metabolic needs of the body. Now sometimes you hear the term “ejection fraction”. And ejection fraction is measured by an echocardiogram and basically what it tells you is how well the heart's functioning. And what it really tells you is how much blood is being ejected out of that left ventricle at one time and how well that heart is working.

You'll also hear about this word “stroke volume”. And stroke volume actually measures the amount of blood that's ejected from the left ventricle every time the heart contracts. And it's normally between 60 and 100 mL per contraction.

So we need to look at the components of cardiac output. And if you look at the true measure of cardiac output, what it basically is is 4 to 8 liters per minute or how much blood the heart is ejecting each minute.

Now we also look at our cardiac output based on how big or how small a person is, and that's called “cardiac index”. And cardiac index is normally 2.5 to 4 liters per minute per meter squared. Now there's a couple other pieces related to cardiac output. We have “preload”. We have “afterload”. We have “heart rate” and we have “contractility”. And all those things have an impact on stroke volume.

Lisa Bonsall: Okay. So let's talk about that a little bit. You mentioned preload and afterload and that those terms can kind of really trip nurses up. So what's the difference between preload and afterload?

Anne Dabrow Woods: Sure. So preload, really when you think about it, it's the amount of venous return to the heart and how much the myocytes in the ventricles have to stretch to be able to fill. So we can actually measure that by putting a catheter into the right atrium. And normal preload pressure is 2 to 6mm of mercury. Now what's going to cause preload to increase? Well if your volume overloaded, your preload is going to go up. If you are dehydrated or hemorrhaging preload actually decreases. Or if a patient is dehydrated.

Now afterload is a little different. Afterload is the pressure the left ventricle has to overcome to eject its contents, and normal pressure for afterload is 800 to 1200 dynes per second per centimeter to the fifth. Now, afterload is also known by another term called “systemic vascular resistance”. So I don't want people to get tripped up on that. Basically, they mean the same thing. Now what causes somebody’s afterload to go up?

Well, remember it's the pressure that left ventricle has to overcome to eject its contents. So what you're actually measuring is how constricted or dilated blood vessels, in this case, the aorta, really is. So it's going to elevate if the aorta and blood vessels are very vasoconstricted. But it's also going to be lower if those vessels are very dilated out.

Lisa Bonsall: So you talked about SVR. Now there's another acronym PVR. What's that, and how is it different?

Anne Dabrow Woods: So PVR is called “pulmonary vascular resistance”. And PVR is the pressure the vessels, the pulmonary veins, coming back from the lungs and into the heart have to overcome to be able to get that blood from the lungs into the left atrium, that is pulmonary vascular resistance. And basically it’s caused by the pressure that's happening within the lungs. So things that are going to increase PVR are things like pulmonary hypertension or pulmonary edema. Someone having a pulmonary embolism is going to have higher PVR or simply cardiovascular disease. The normal pressure in the lungs, the PVR pressure is going to be between 100 to 200 dynes per second to centimeters to the minus five. So that's normally what your normal PVR is going to be.

Lisa Bonsall: Thank you. And now how about heart rate and contractility. What's their role?

Anne Dabrow Woods: So these things kind of go together. If you have to have electrical activity before you have mechanical activity. So you got to have the heart rate. What we see on a monitor that, you know QRS on the monitor. You’ve got to have that first. So somebody’s normal pulse is going to be between 60 to 100 beats-per-minute. That's normal for people. Now we do know that the electrical activity is generated first by our SA node in the right atrium. And then those impulses go throughout the heart. So if the heart rate is too low, what's going to happen is you're not going to have sufficient, contractility. Because, remember, electrical activity before mechanical activity to actually move the blood through the heart, right. And move the blood to the body. The flip side is if your heart rate's too fast, then you don't have enough time for filling of your coronary arteries.

Now here's the thing people forget, our body gets perfused with oxygen-rich blood during systole, when our heart is working. So that top number of our blood pressure. But the heart itself doesn't get perfused during systole. It actually gets perfused during diastole when the heart is not pumping. And if someone

is tachycardic, meaning they have a really high heart rate above 100 beats-per-minute, then they have a shortened diastole time and they get less cardiac filling.

Now normally people are okay with that up to a heart rate about 150 and after about 150, patients start to feel the effect and they will actually, have a lower blood pressure because of it. But they can also develop ischemia because that heart is not getting enough oxygen rich blood.

The next thing is contractility. And that's how well the heart pumps. Remember electrical activity before mechanical activity.

So contractility is really all about how those myocytes or those mechanical cells of the heart actually contract. And we do know we call this “inotropic activity”. And we do know that there's drugs that can have positive inotrophy meaning increasing the strength of the contraction. And then we use drugs that are negative inotropics. So they decrease the strength of our contraction.

So what can increase contractility? Well it's usually a compensatory mechanism. Like if you get hit by a truck and you just hemorrhage a bunch of blood out on the pavement, your heart is going to contract more briskly, because your heart rate's going to go up, right? It's trying to compensate for it. During exercise, our contractility is going to increase because our heart rate goes up.

Drugs like digoxin can also increase our contractility of our heart. What can make it actually decrease are things like having a myocardial infarction because your heart muscle's not working as well. Or if you have a cardiomyopathy or certain drugs like beta blockers and calcium channel blockers actually decrease the strength of the contraction of the heart itself.

Lisa Bonsall: Thank you, Anne. Can you sum this all up for us? What are all the components that affect cardiac output?

Anne Dabrow Woods: Sure. So you have preload, venous return to the heart: how much myocytes in the ventricles have to be able to stretch. So normally 2 to 6. You also have afterload, systemic vascular resistance which is how much pressure the left ventricle has to overcome to eject its contents. So what does that mean? How vasoconstricted or vasodilated the blood vessels are: normally between 800 to 1200 dynes. And then you have heart rate and contractility that go hand-in-hand. And remember you have to have electrical activity before you have mechanical activity. Now, if all these things are working well, then you have adequate cardiac output and your patient will do well. And everything that we do when we talk about, tuning up a patient where we're managing their hemodynamics, we want them to get into the normal parameters.

Lisa Bonsall: Thank you so much, Anne!

Anne Dabrow Woods: You're welcome.

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